Figure 7.1-9 Subsurface Profile of Cross Section "2-2"

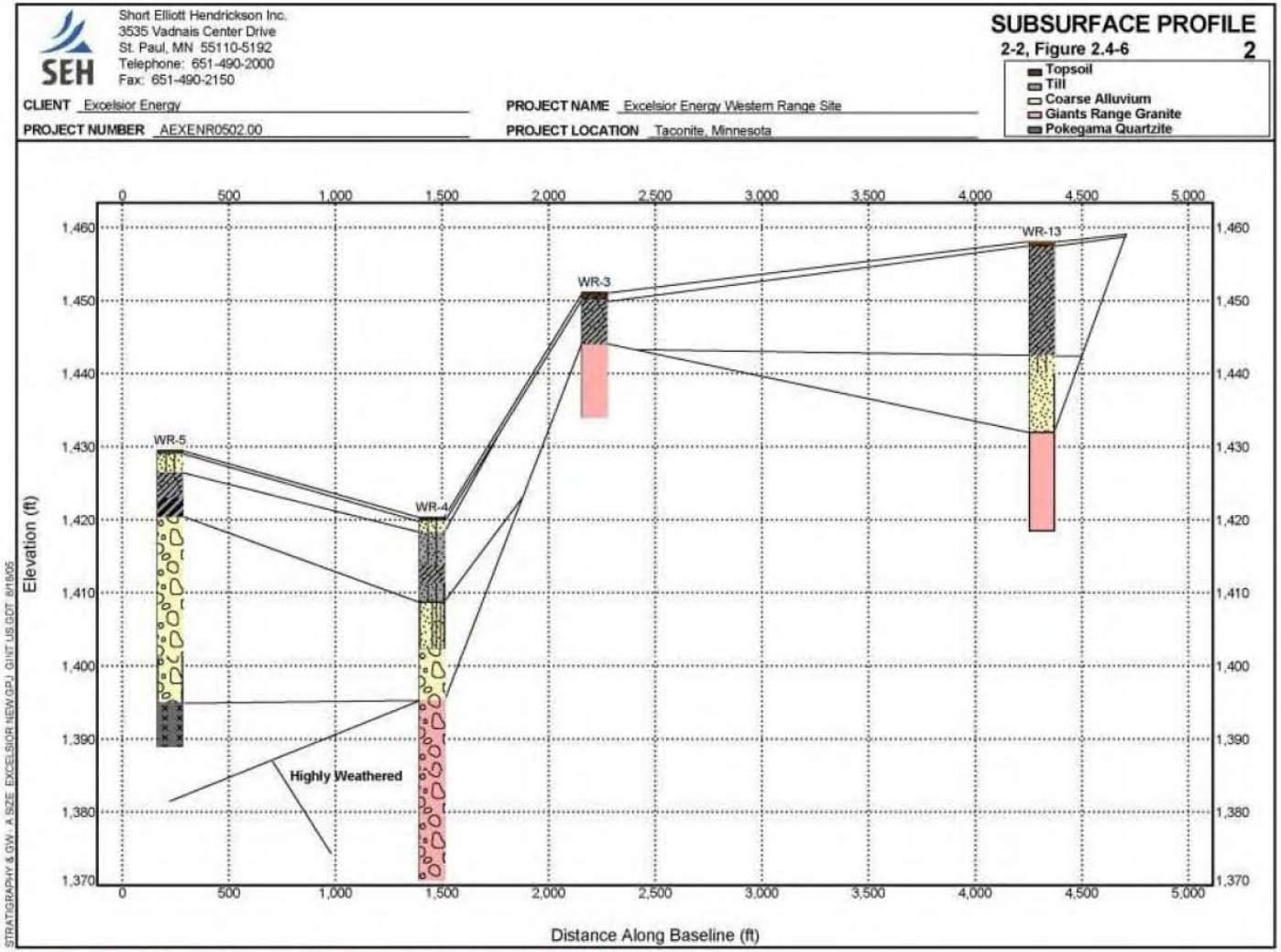


Figure 7.1-10 Subsurface Profile of Cross Section "3-3"

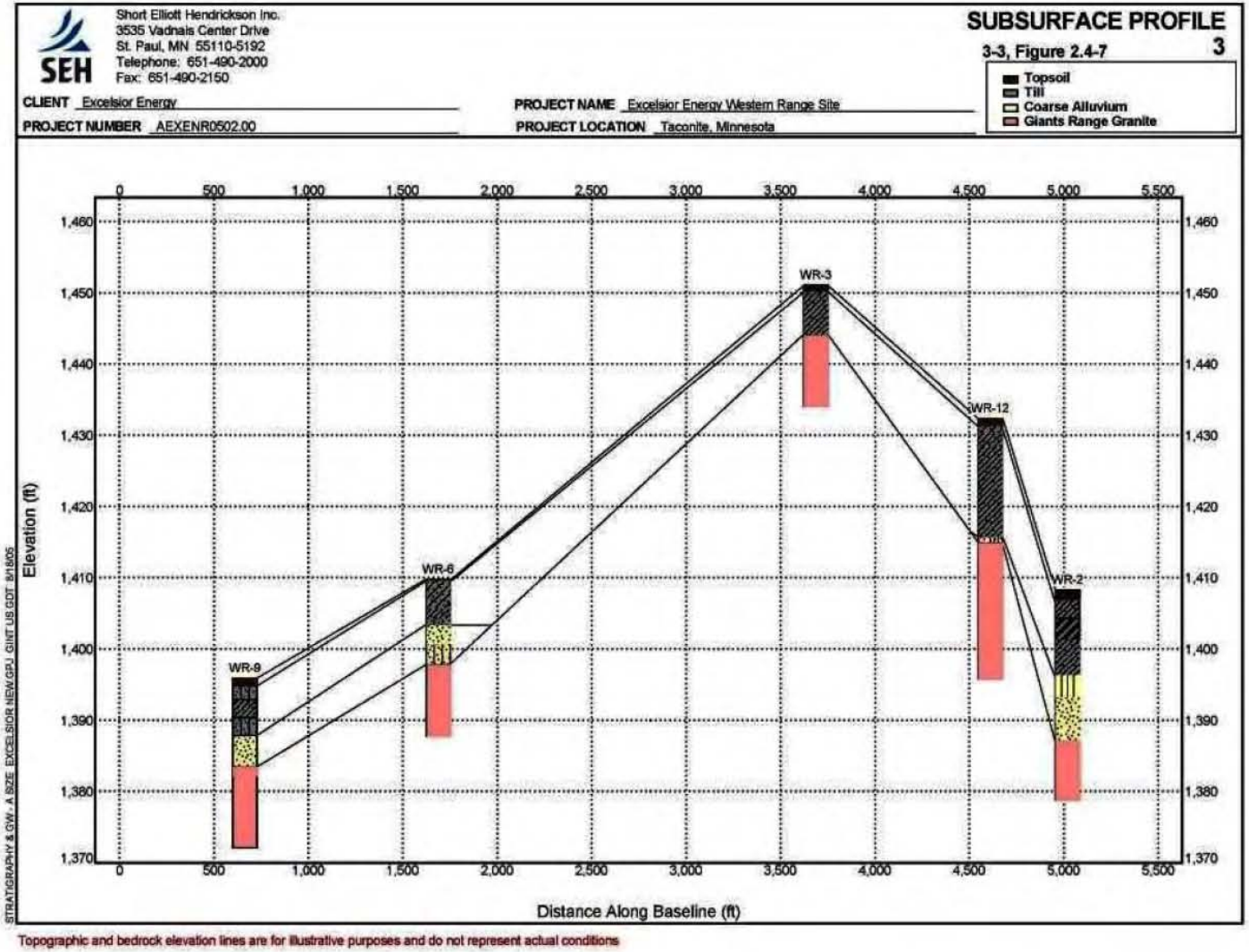
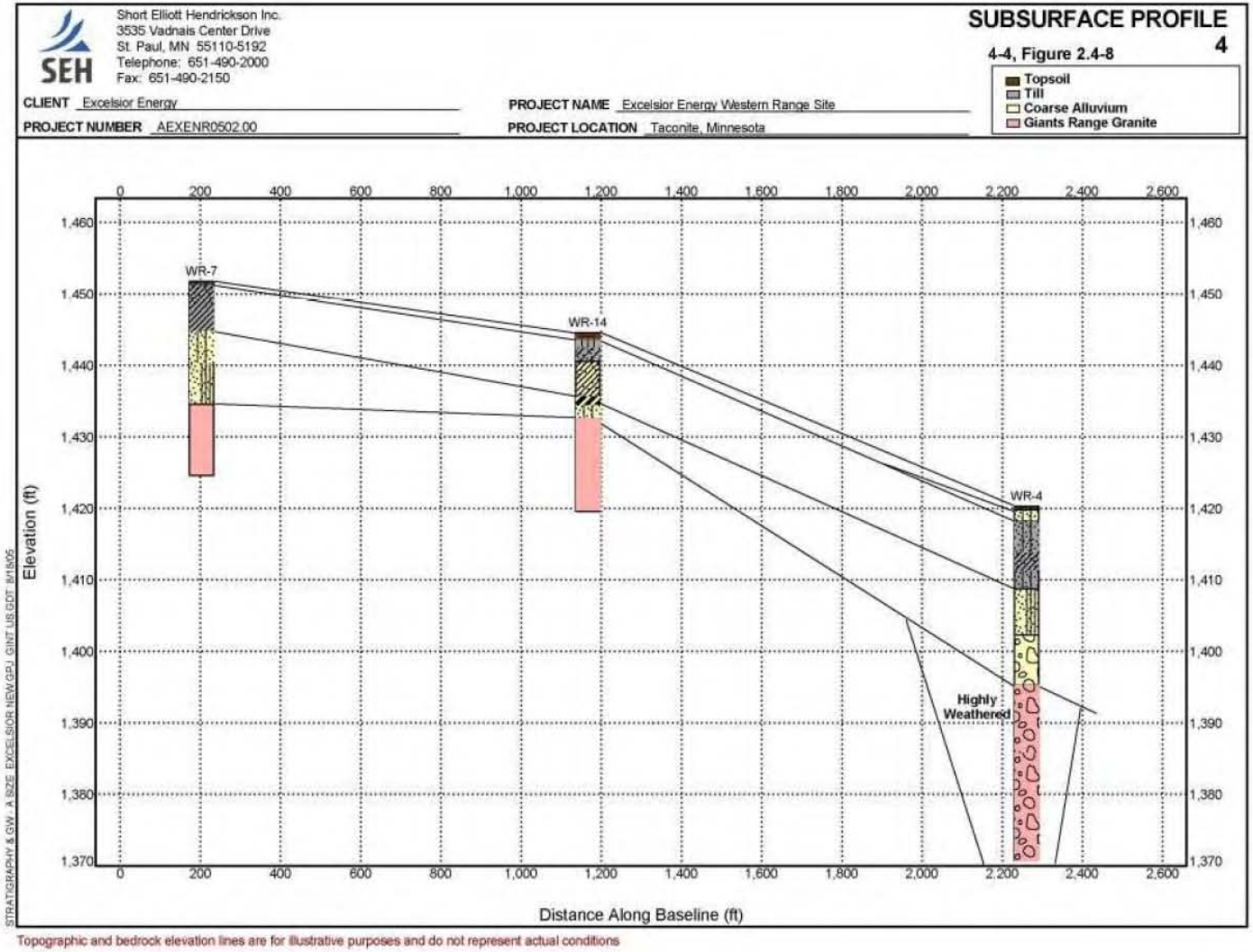


Figure 7.1-11 Subsurface Profile of Cross Section "4-4"



7.1.10 Special Land Uses

7.1.10.1 Recreational Lands

The Receptor Map (Figure 2.5-2) indicates that there are no recreational areas within IGCC Power Station Footprint and Buffer Land, except snowmobile trails that follow existing transmission line ROWs through the Buffer Land (one trail oriented in a north-south alignment along the 28L/45L HVTL tap line to the retired Greenway Substation and the other oriented in an east-west alignment along the 28L HVTL route between the Clay Boswell Station and the Nashwauk Substation). Both trails will likely be required to be diverted and/or closed for construction and operation of the IGCC Power Station.

In addition, the Hill-Annex State Park is about 3.5 miles east of the IGCC Power Station.

Area lakes provide numerous recreational opportunities for area residents. Activities such as swimming, boating, fishing, bird watching and other similar activities are prevalent. Gibs Park is located on Holman Lake about 2.1 miles south-southeast of the IGCC Power Station Footprint. The park includes a fishing pier, swimming beach and picnic area, the fishing pier being a cooperative project between Iron Range Township and the MDNR. Holman Lake will receive cooling tower blowdown from the IGCC Power Station. As cooling tower blowdown will be regulated within prescribed limits and contain predominantly minerals that are present in mine pit waters, no adverse impacts on water or land use will occur.

The forested areas in the Project area apparently also allow for some recreational activities such as hiking, biking, hunting, bird-watching and similar activities. Many of these activities take place on land that is owned by Itasca County but is not specifically designated as a recreation area.

7.1.10.2 Designated Wildlife Areas

There are no designated Federal Wildlife Refuges, Waterfowl Production Areas, or National Preserves within or immediately adjacent to the IGCC Power Station Footprint, Buffer Land, Associated Facilities, or Interconnection Corridors included as part of the West Range Site. No MDNR Wildlife Management Areas (WMAs), Wildlife Refuges, state Scientific and Natural Areas (SNA), designated Game Lakes, or Designated Trout Streams are within or immediately adjacent to such areas.

7.1.10.3 Prime Farmland

Although no active farmland would be disrupted by construction of the IGCC Power Station, its Associated Facilities, or Interconnection Corridors, many of the soils in the West Range Site are officially designated as “prime farmland” or “prime farmland if drained.” As a practical matter, this area and region of the State is not considered to be high quality or desirable farmland. Figure 7.1-12 shows the coverage of soils considered to have potential to be prime farmland in the vicinity of the West Range Site. According to the 1996 Land Cover/Land Use Map, no cultivated farmlands are located within one-mile of the centerline of the Preferred or Alternate HVTL Routes. Several tracts of cultivated farmland are located within one-mile of the centerline of the Proposed Natural Gas Pipeline Route and the Process Water Supply and Process Water

Process Water Blowdown Pipeline corridors. No cultivated farmland is located near the preferred rail line access corridor.

Minnesota Rule 4400.3450, subpart 4. ("Prime Farmland Exclusion") provides that "No large electric power generating plant site may be permitted where the developed portion of the plant site, excluding water storage reservoirs and cooling ponds, includes more than 0.5 acres of prime farmland per megawatt of net generating capacity, or where makeup water storage reservoirs or cooling pond facilities include more than 0.5 acres of prime farmland per megawatt of net generating capacity, unless there is no feasible and prudent alternative." The provision does not apply to areas located within home rule charter or statutory cities, areas located within two miles of home rule charter or statutory cities of the first, second, and third class, or areas designated for orderly annexation under Minn. Stat. § 414.0325.

The only Associated Facilities of the West Range Site that lie outside the City limits of Taconite and Marble (Taconite and Marble abut one another at the eastern-most boundary of Taconite and both are statutory cities) are the LMP pumping station, Segment 1 of the Process Water Supply Pipeline, and the outfall at its point of termination of the Segment 1 pipeline. Figure 7.1-13 shows a map of these features and their proximity to soils meeting the criteria of "prime farmland" or "prime farmland if drained." The proposed HVTL segments do not impact prime farmland.

7.1.11 Regional Zoning

The IGCC Power Station Footprint and Buffer Land are located completely within an area zoned for industrial uses. The current zoning designations for property in the immediate vicinity of the Station Footprint and Buffer Land is shown in Figure 7.1-14. Zoning within the region including the Preferred and Alternate HVTL Routes and Proposed Natural Gas Pipeline Route is shown in Figure 7.1-15.

Figure 7.1-12 Prime Farmland and Other Important Farmlands in the Vicinity of the West Range Site

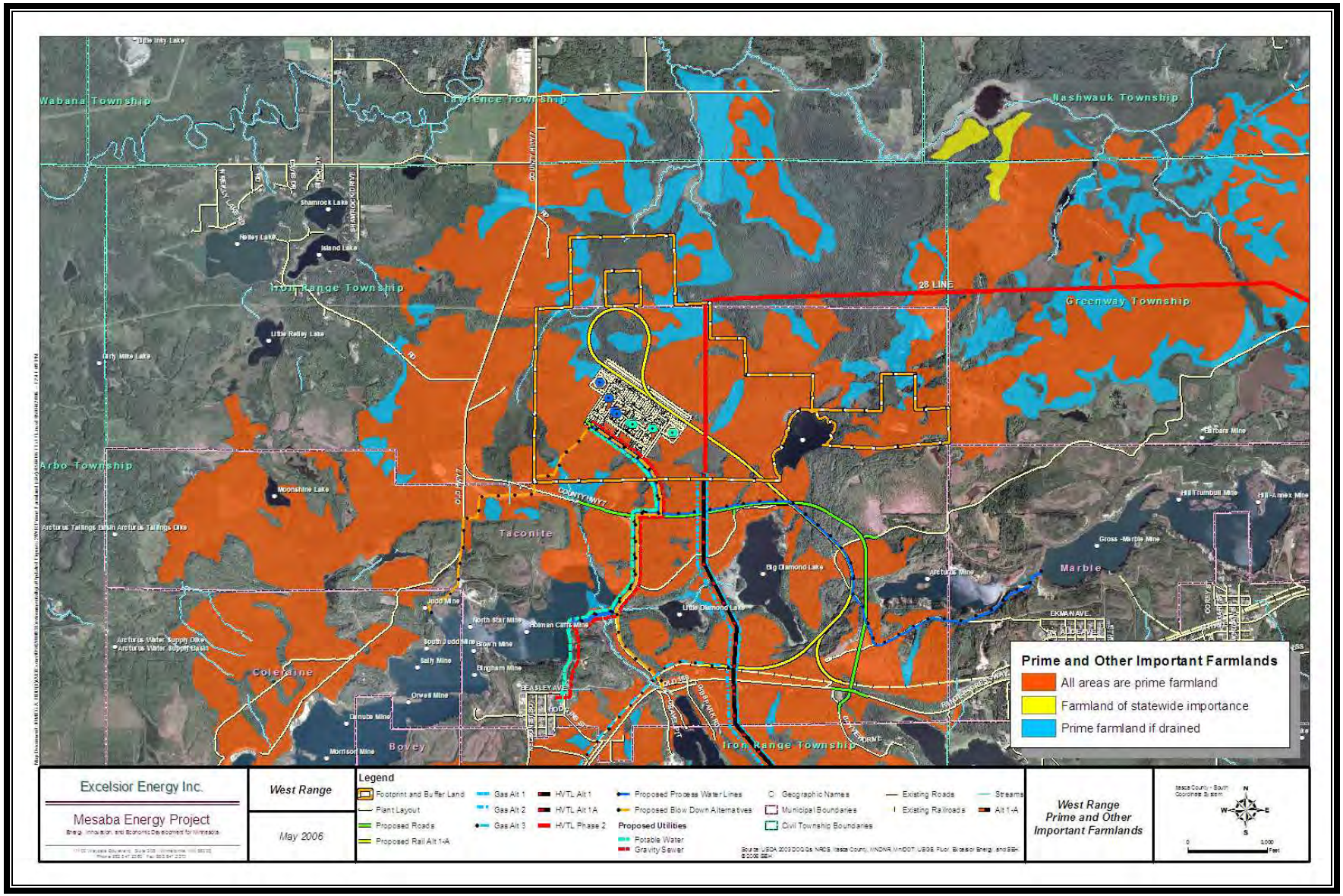


Figure 7.1-13 Prime Farmland and Other Important Farmlands in the Vicinity of Process Water Supply Pipeline Segment 1 the West Range Site

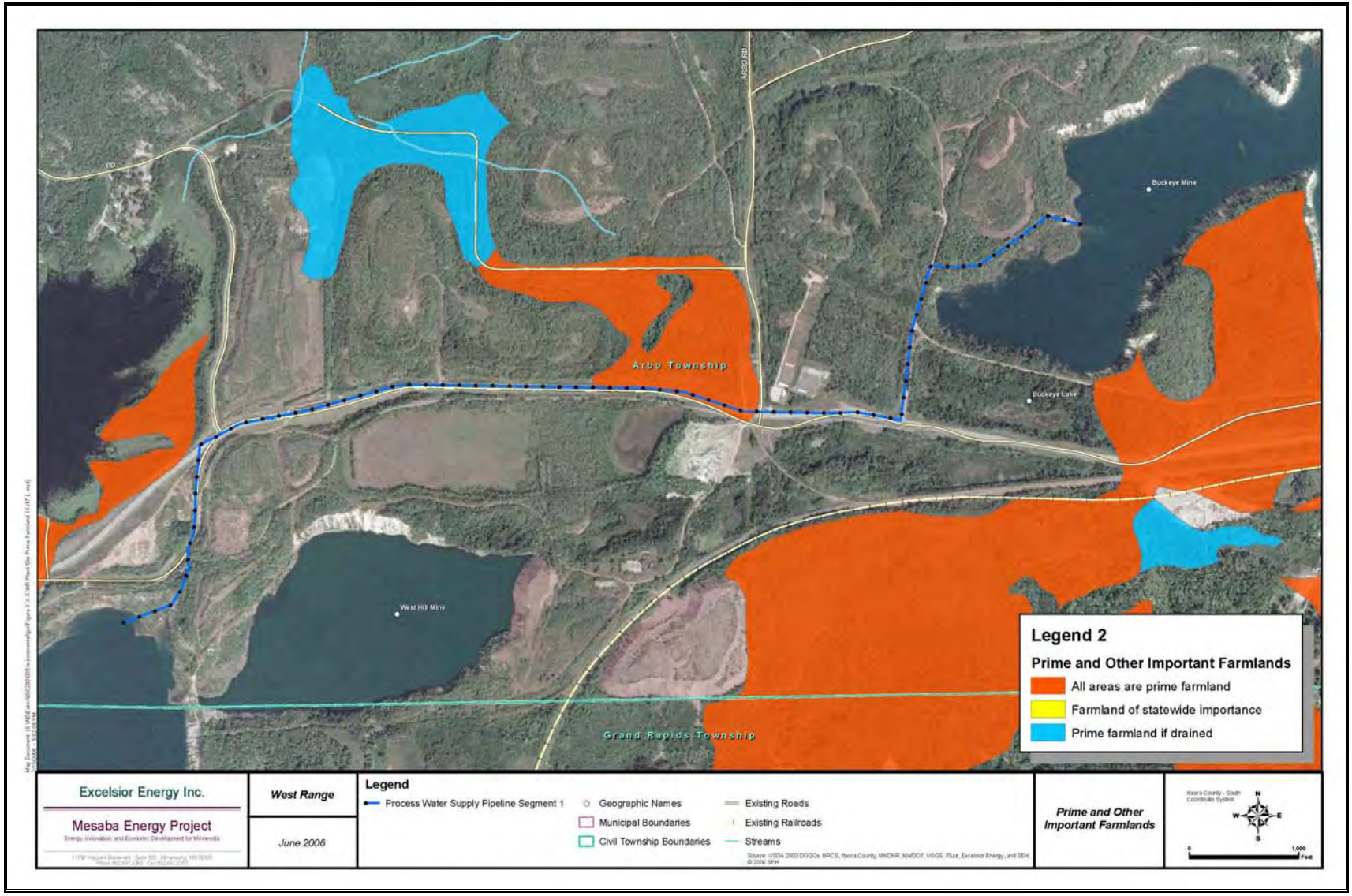


Figure 7.1-14 Zoning in the Immediate Area of the West Range Site

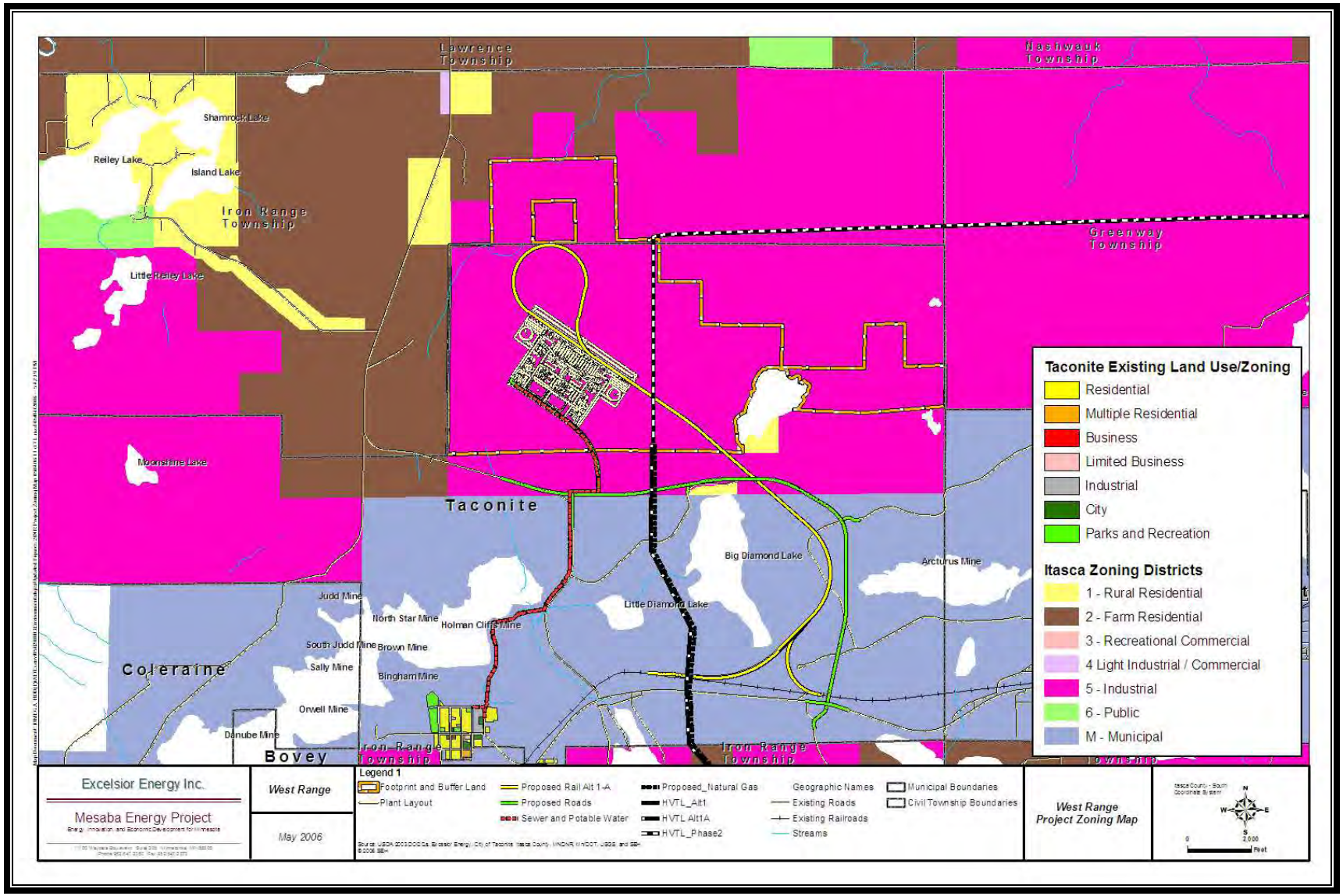
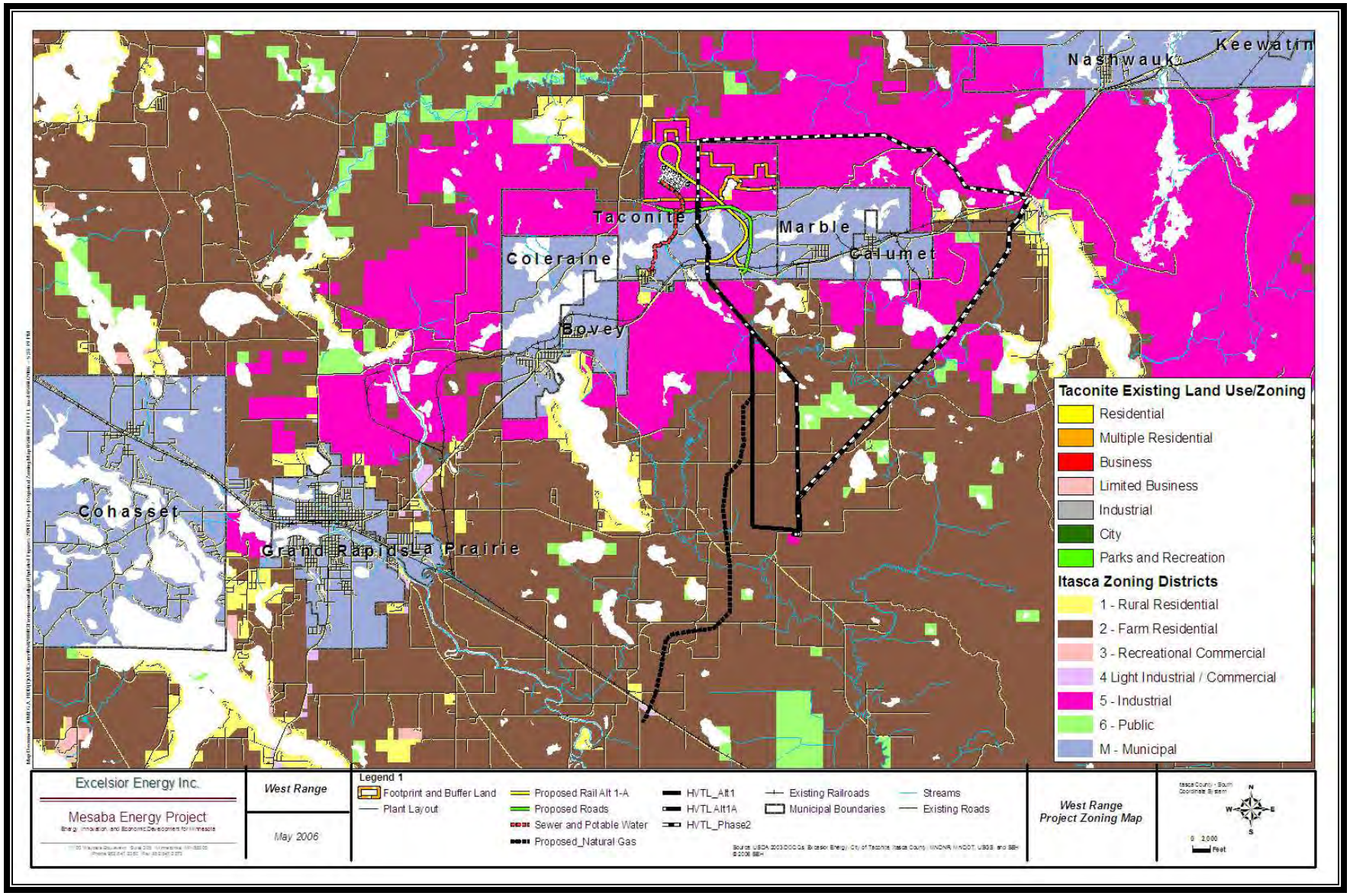


Figure 7.1-15 Zoning in the Region Surrounding the West Range Site, HVTL Routes, and Natural Gas Pipeline Route



The Applicant does not expect any industry to be adversely impacted by the construction and operation of the IGCC Power Station at the West Range Site. Area tourism and recreation areas will not be adversely impacted by the Project. The Hill-Annex State Park will benefit from the IGCC Power Station operations at the West Range Site because the water levels in the Hill-Annex Mine Pit would be better managed, thereby allowing full or expanded use of the Park (See Section 3.6.1.1 for a description of the West Range water management plan).

7.2 NEARBY RESIDENCES AND OTHER SIGNIFICANT RECEPTORS

Significant receptors are locations where people gather in groups or spend extended periods of time. Significant receptors also include locations where children, elderly, or the infirm live or spend time, and include residences, schools, daycare centers, recreation centers, playgrounds, nursing homes and hospitals. Figure 2.5-2 above shows the location of residences and other significant receptors in the vicinity of the West Range Site. Figure 7.1-14 also indicates that the residences on Big Diamond and Dunning Lakes are located immediately adjacent to an area zoned "Industrial." Presumably, owners of such residences purchased such properties with knowledge of such zoning and/or did not object to such designation.

7.2.1 IGCC Power Station Footprint and Buffer Land

7.2.1.1 Distance of Nearby Receptors from IGCC Power Station Emission Points

The closest residence located 0.7 miles west of the IGCC Power Station (see Table 7.2-1 below and Figure 7.2-1). Other residences that are located to the northwest, southwest, southeast, and east-southeast are located about 390 to 850 feet further away than the closest residence.

The residences most likely to be affected by construction and operation of the IGCC Power Station are located to the southwest on the north shore of Big Diamond Lake ("BDL") and the southeast shore of Dunning Lake ("DL"). The residences on the lake shore are a mix of seasonal and year-round dwellings. These properties will not be buffered to the same extent as properties located in all other radial directions. The proposed rail track and the proposed realignment of CR 7 will cut between the two lakes with the rail spur extending in a northwesterly direction and the highway extending directly to the west, just north of the existing haul road now used for access by local residents. Construction of these two transportation elements will likely take place over a two year period interrupting the residents' normal daily activities. Thereafter, increased levels of construction traffic will be ongoing over several years as construction of Mesaba One and Mesaba Two reach peak levels.

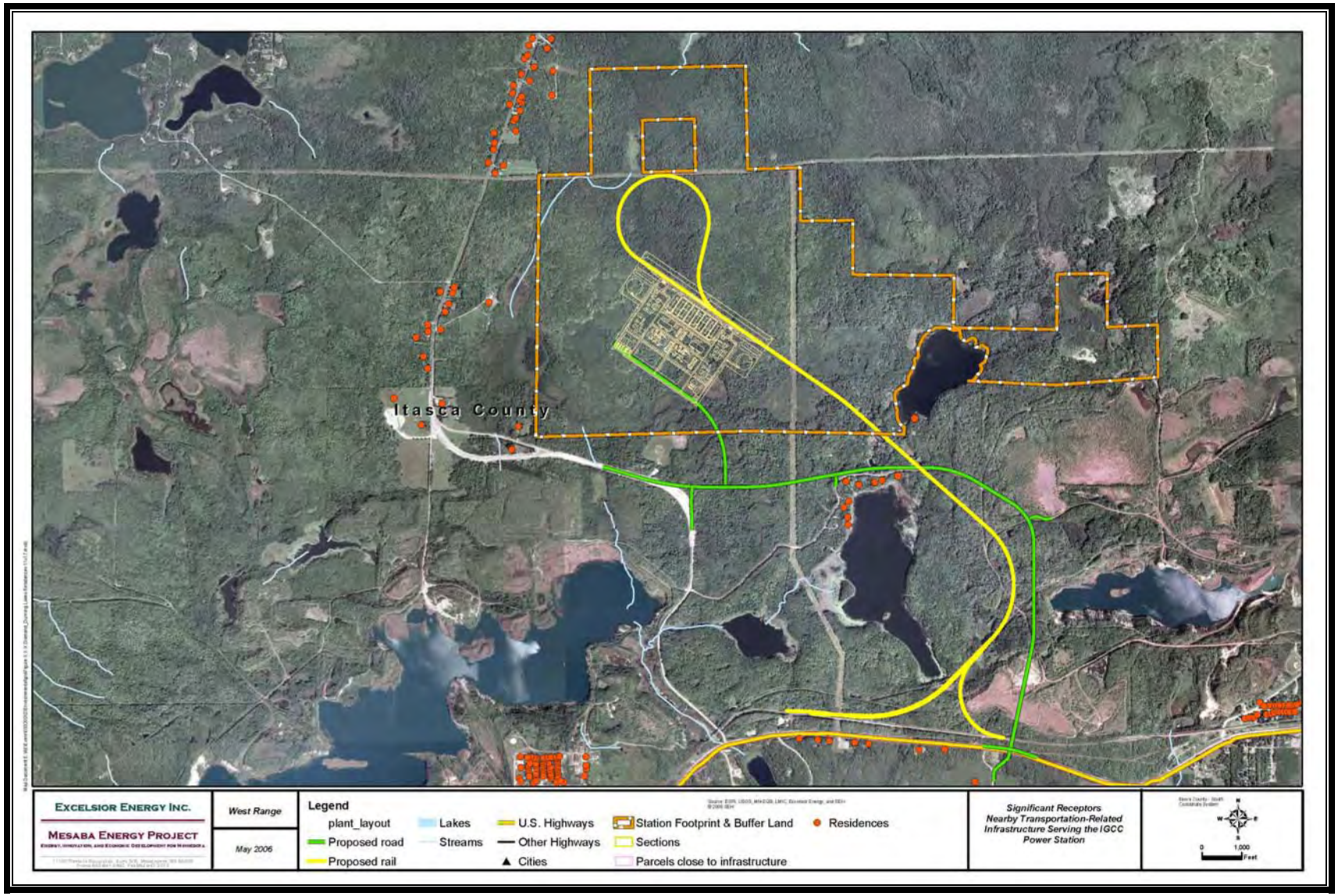
Figure 7.2-1 shows the residential properties on BDL and DL and their proximity to the proposed rail spur and the realignment of CR 7. The closest resident on the shore of BDL is about 500 feet from the track centerline and the single resident on DL is about 800 feet away. The closest resident on Big Diamond Lake to the rail alignment is also the closest to the realigned CR 7, approximately 200 feet distant from its centerline. One other property owner on BDL will be located about that same distance from the centerline of the highway. The properties located in all other radial directions will be far less exposed to construction and operational disturbances. Impacts on all receptors is addressed in the remaining sections of this Chapter 7.

The City of Taconite has both single-family and multi-family residential houses. The majority of these residences are occupied year-round. The closest residents of Taconite are located approximately 1.7 miles south-southeast of the IGCC Power Station Footprint.

Table 7.2-1
Receptors Located Nearby the IGCC Power Station Footprint

Location	Approximate Distance from the nearest edge of IGCC Power Station Footprint
R2 Residence, on Big Diamond Lake	3,850' to the southeast
R3 Residence, 31950 CR 7	3,800' to the west
R4 Residence, 32423 CR 7	4,400' to the west
R5 Residence, on Dunning Lake	4,175' to the east
R6 Lutheran Church	18,000' to the southeast
R7 Catholic Church	10,700' to the NNW

Figure 7.2-1 Residential Receptors Located Nearby the IGCC Power Station



7.2.2 Alternate HVTL Routes

The significant receptors within ½ mile of the Preferred and Alternate HVTL routes were identified from aerial photography and a helicopter overflight of the applicable HVTL routes. Table 7.2-2 summarizes the significant receptor inventory along each route

Table 7.2-2
Residences Along HVTL Routes

HVTL Route	Receptors	Distance from Centerline of Alignment (ft)					
		0-50	50-100	100-300	300-500	500-1,320	1,320-2,640
Preferred Route	66 residences	0	0	1	3	13	49
Plan B Phase II Alternate Route	214 residences	0	0	8	21	69	116
Alternative Route	62 residences	0	0	2	5	14	41

Only residential receptors are located within ½ mile of the centerline of each HVTL alignment. Of those receptors, all appear to be located at a distance from the centerline that exceeds 100 feet. Prior to construction, the Applicant will attempt to shift the HVTL alignment to avoid residences located too close to the centerline of the ROW.

7.2.3 Proposed Natural Gas Pipeline Route

The locations of significant receptors within ½ mile of the centerline of the Proposed Natural Gas Pipeline Route were identified from aerial photography. Table 7.2-3 provides a summary of the significant receptor inventory for this route. An analysis of the Other Considered Natural Gas Pipeline Routes is provided in the ES.

Table 7.2-3
Significant Receptors Located along the
Proposed Natural Gas Pipeline Route

Significant Receptors	Distance from Centerline of Alignment (ft)					
	0-50	50-100	100-300	300-500	500-1,320	1,320-2,640
153 residences	0	0	3	14	61	75
Trout Lake Cemetery	0	0			X	
Trout Lake Church				X		

All significant receptors located within ½ mile of the centerline of the Proposed Natural Gas Pipeline Route are located at a distance greater than 100 feet from its proposed centerline. The Trout Lake Church and Trout Lake Cemetery are about 470 feet and 720 feet away from the centerline of the Pipeline Route alignment and are shown above in Figure 2.5-2.

7.2.4 Process Water Supply Pipelines

The location of significant receptors within ½ mile of each of the Process Water Supply Pipeline Segments were identified by aerial photography. Table 7.2-4 below provides a summary of the significant receptor inventory.

Table 7.2-4
Significant Receptors along the Process Water Supply Pipeline

Significant Receptors	Number	Distance from Centerline of Alignment (ft)					
		0-50	50-100	100-300	300-500	500-1,320	1,320-2,640
Segment 1 (Lind Pit to Canisteo Pit)	15	0	0				15
Segment 2 (Canisteo Pit to West Range Site)	0	0	0	0	0	0	0
Segment 3 (Gross-Marble Pit to Canisteo Pit)	89			1	3	3	82

Only residential receptors are located within ½ mile of any of the process water supply pipeline segments, and only one is less than 100 feet from the centerline of the alignment.

7.2.5 Process Water Blowdown Pipelines

Significant receptors within ½ mile of each of the process water blowdown pipelines were identified from aerial photography. Table 7.2-5 below provides a summary of the significant receptor inventory.

Table 7.2-5
Significant Receptors along the Process Water Blowdown Pipelines

Process Water Blowdown Pipeline	Number	Distance from Centerline of Alignment (ft)					
		0-50	50-100	100-300	300-500	500-1,320	1,320-2,640
Pipeline 1	8	0	0	0	0	0	8
Pipeline 2	6	0	0	1	1	1	3

Only residential receptors are located within ½ mile of any of the process water blowdown pipelines, and all are greater than 100 feet from the centerline of the alignments.

7.2.6 Potable Water and Sewer Pipelines

Significant receptors within ½ mile of each of the Potable Water and Sewer Pipeline alignment were identified from aerial photography. Table 7.2-6 below provides a summary of the significant receptor inventory.

Table 7.2-6
Significant Receptors along the Potable Water and Sewer Pipeline Alignment

Potable Water and Sewer Pipelines	Number	Distance from Centerline of Alignment (ft)					
		0-50	50-100	100-300	300-500	500-1,320	1,320-2,640
Potable Water and Sewer Alignment	114	0	1	3	0	63	46

Only residential receptors are located within ½ mile of the potable water and sewer alignment, and all but one are greater than 100 feet from the centerline of the alignment centerline. The only exception is a home that is located approximately 90 feet from the centerline.

7.2.7 Railroad

Significant receptors within ½ mile of each of the railroad alignment alternatives were identified from aerial photography. Table 7.2-7 below provides a summary of the significant receptor inventory.

Table 7.2-7
Significant Receptors along the Alternative Railroad Alignments

Rail Line Alternative	Number	Distance from Centerline of Alignment (ft)					
		0-50	50-100	100-300	300-500	500-1,320	1,320-2,640
Rail Line Alternative 1A	16	0	0	0	0	10	6
Rail Line Alternative 1B	8	0	0	0	0	8	0

Only residential receptors are located within ½ mile of any of the railroad alternative alignments, and all are greater than 500 feet from the centerline of the alignments.

7.2.8 Roads

Significant receptors within ½ mile of each road alignment were identified from aerial photography. Table 7.2-8 provides a summary of the significant receptor inventory.

Table 7.2-8
Significant Receptors along Access Roads

Roads	Number	Distance from Centerline of Alignment (ft)					
		0-50	50-100	100-300	300-500	500-1,320	1,320-2,640
Access Roads 1 & 2	22	0	0	1	5	4	12

Only residential receptors are located within ½ mile of any of the road alignments, and all but one are located at a distance greater than 300 feet from the centerline of the alignments. The one exception is located approximately 200 ft from the centerline of the alignment.

7.2.9 Displacement

No resident or business will be displaced as a result of the construction or operation of Mesaba One and/or Mesaba Two.

7.3 AESTHETICS

A detailed evaluation of the areas from which the new Power Station and transmission lines will be visible is provided in Section 3 of the Environmental Supplement. This section of the Application provides an overview of the general visual impacts from the plant, HVTL, and pipelines.

The Mesaba Project will affect views in West Range Site area primarily in two ways. First, some of the IGCC Power Station buildings and exhaust stacks will be visible from nearby residential areas and roadways. Second, the new, taller, high-voltage transmission line structures will be visible along the selected transmission line corridors. In addition, along natural gas and water pipeline corridors (and along new roadways or railway) trees will be cleared in some areas and construction will require filling or relocation of wetlands. Permanently cleared right-of-way on these corridors will be visible where the routes follow or cross existing roadways.

7.3.1 IGCC Power Station

Near the West Range IGCC Power Station buildings and stacks will be screened but still be visible from nearby homes, businesses and the nearest public highway, CR 7. The highest buildings within the IGCC Power Station are the rod mill feed bins (150 feet) and generator buildings (90 feet). The tallest stack is on the tank vent boiler (210 feet); however, that stack is only six-feet in diameter and will not be highly visible. The stack on the generator building will be 150 feet tall. An artist's visualization of the Phase I and II Developments is provided above in Figure 3.2-2.

7.3.2 High-Voltage Transmission

The proposed double circuit 345-kV high-voltage transmission line for the West Range Site would be constructed primarily on steel single-pole structures to minimize the width of the right-

of-way needed and to minimize land use conflicts. While single steel poles cost more and are taller than wooden H-frame supports or other alternatives, they do allow for longer spans and require less right-of-way. Longer spans between poles require fewer poles than other structure types. For the proposed double -circuit 345-kV line, the structures would be about 130- to 140-feet tall, with average spans of about 800 feet. Structures on the taller end of this range will be needed on the one-mile segment where the structures share right-of-way with an existing line near the Blackberry Substation. On that segment, the line would carry three circuits, including the existing circuit, on one set of structures. H-frame or other structure types may be necessary near waterfowl areas or interstate crossings. These single-pole structures would be visible to residents along the proposed route between the IGCC Power Station and the Blackberry Substation.

7.3.3 Pipelines and Roadways

Where the pipelines are constructed under a roadway or ATV trail-type surface, the surface condition will be maintained or improved. Since most of the proposed pipeline routes are currently covered with wooded areas, brush, or grassy vegetation, little visual change would result from construction or maintaining the proposed alignments. Where the routes follow the existing highway, clearing of trees and shrubs will be noticeable.

7.4 AIR QUALITY

The projected annual air emissions for Mesaba One and Mesaba Two are provided above in Section 3.4.1. In this section, we summarize the applicable regulatory requirements, emission controls evaluated, and the site-specific impacts of air emissions as modeled at receptors and the Federal Class One areas nearest the West Range Site. The engineering and operational design of the proposed IGCC Power Station, including air emission controls, is provided in Section 3. In addition, the Part 70 Air Permit Application filed with the MPCA provides details regarding all regulatory requirements, emission calculations, ambient air modeling assumptions, and modeling results.

7.4.1 BACT Requirements

Since the Project is subject to Prevention of Significant Deterioration (PSD) review, a Best Available Control Technology (BACT) analysis is required. The BACT analysis is independent of the location of the plant, so the results of the analysis are applicable to both the West and East Range Sites.

The BACT analysis was conducted for the following pollutants that exceed their respective PSD significance threshold: carbon monoxide, nitrogen oxides, sulfur dioxide, particulate matter, particulate matter less than ten microns in diameter (PM_{10}), ozone (volatile organic compounds are the surrogate pollutant for ozone), lead, and sulfuric acid mist.

The analysis was conducted in accordance with USEPA's "top-down" BACT methodology. The "top-down" process involves the identification of all applicable control technologies according to control effectiveness. The owner or operator then evaluates the "top," or most stringent, control alternative. If the most stringent alternative is shown to be technically or economically

infeasible, or if environmental impacts are severe enough to preclude its use, then the next most stringent control technology is similarly evaluated. This process continues until the BACT level under consideration cannot be eliminated by technical or economic considerations, energy impacts, or environmental impacts.

Potential control options were identified by researching the EPA database known as the RACT/BACT/LAER Clearinghouse (RBLC), drawing upon previous environmental permitting for similar units, engineering experience, discussions with equipment vendors, and researching available literature. Available controls were further evaluated based on an analysis of economic, environmental, and energy impacts. The permitted IGCC power stations in the United States that were used in this analysis are as follows:

- SG Solutions, Wabash River Generating Station, West Terre Haute, IN (operating)
- Tampa Electric Company, Polk Power Station, Mulberry, Florida (operating)
- We Energies, Elm Road Generating Station, Wisconsin (permitted)
- Global Energy, Inc.'s Kentucky Pioneer Energy LLC, Trapp, Kentucky (permitted)
- Global Energy, Inc.'s Lima Energy Company, Lima, Ohio (permitted)

A summary of the proposed BACT controls and associated emission rates for each emission unit is shown in Table 7.4-1. This analysis includes the syngas-fired CTGs, tank vent boilers, cooling towers, flare, emergency diesel generators, and diesel fire water pumps.

Table 7.4-1
Proposed BACT for the IGCC Power Station

Pollutant	Control	Emissions Limits
Syngas-Fired Combustion Turbines (emissions shown per CTG)		
NO_x	Diluent Injection	15 ppm NOx @ 15%O_2; 157 lb/hr per CTG on syngas fuel
		25 ppm NOx @ 15%O_2; 198 lb/hr per CTG on natural gas fuel
CO	Good Combustion Practice (GCP)	15 ppm @ actual O_2 (above 50% load) 95 lb/hr per CTG
PM/PM_{10}	GCP, gas cleanup, Gaseous Fuels only	25 lb/hr
SO_2	Gas cleanup/Use of Clean Syngas	76 lb/hr SO_2 per CTG; (approx. 50 ppmv sulfur, as H_2S, in undiluted syngas)
VOC	GCP	9 lb/hr per CTG
Lead	Gas cleanup/Use of Clean Syngas	0.087 tons/yr per CTG
H_2SO_4	Gas cleanup/Use of Clean Syngas	5.3 lb/hr, 23.2 tpy

Pollutant	Control	Emissions Limits
Cooling Towers		
PM_{10}	High Efficiency Drift Eliminators	0.001% drift
Tank Vent Boiler		
NO_x, SO_2, CO, VOC, PM_{10}	GCP, Gas cleanup/Use of Clean Syngas or Natural Gas	
Flare		
NO_x, SO_2, CO, VOC, PM_{10}	Good Flare Design, Flaring only treated Syngas	
Fire Pumps		
NO_x, SO_2, CO, VOC, PM_{10}	GCP, limited hours of operation, and use of low-sulfur diesel	Less than 100 hrs/yr operation; low sulfur diesel
Emergency Diesel Generators		
NO_x, SO_2, CO, VOC, PM_{10}	GCP, limited hours of operation, and use of low-sulfur diesel	Less than100 hrs/yr operation; low sulfur diesel

7.4.2 NAAQS and PSD Increment Impact Analysis

State and federal air quality rules prohibit emissions from a new facility that cause or contribute to an exceedance of State or National Ambient Air Quality Standards (NAAQS). In addition, emissions cannot exceed established Prevention of Significant Deterioration (PSD) increments. To demonstrate compliance with these requirements, the Applicant conducted the required air dispersion modeling analysis for the IGCC Power Station at the West Range Site. The AERMOD air dispersion model, which is MPCA's and USEPA's preferred model, was used for the analysis.

7.4.3 Significant Impact Analysis

Results of AERMOD modeling confirmed that facility emissions from Phases I and II would exceed Significant Impact Levels (SILs). Wherever modeled pollutant concentration increases exceed the SILs, further modeling is required under PSD rules to ensure that the Class II PSD increment for the area is not violated. This further evaluation must include all sources within 50 kilometers of the project's area of impact. The significant area of impact is determined by the farthest distance from the site that exceeded the SILs.

Table 7.4-2 below shows modeled impacts at normal operation, when the flares are operated, and during system startup. Because highest predicted impacts are above the SILs, PSD increment and NAAQS compliance modeling was necessary for SO_2, PM10, and NO_x.

There are no applicable PSD increments for CO, and NAAQS compliance need only be demonstrated for the one-hour ambient standard. The normal operation scenario was addressed in all increment and NAAQS analyses for SO_2, PM10, and NO_x, since normal operations (full load operation) represent the highest emission concentration scenario. A startup scenario was

addressed only for the CO one-hour NAAQS demonstration. No further modeling was conducted for the flaring scenario, since it produces lower concentrations than created under other scenarios.

Table. 7.4-2
Highest Project Impacts (Phase I and II) and PSD SILs West Range IGCC Power Station

Pollutant and Averaging Time	Normal Operation	Flaring	Startup	SIL
	ug/m^3	ug/m^3	ug/m^3	ug/m^3
SO_2				
one-hour	132.0	77.3	N/A	25.0
three-hour	80.7	26.6	N/A	25.0
24-hour	32.3	6.2	N/A	5.0
Annual	1.32	N/A	N/A	1.0
PM10				
24-hour	24.6	N/A	N/A	5.0
Annual	1.86	N/A	N/A	1.0
CO				
one-hour	159.8	421.9	3088	2000
eight-hour	54.3	135.0	462	500
NO_x				
Annual	2.69	N/A	N/A	1.0

7.4.4 PSD Increment

Increment analyses were completed for SO_2, PM10, and NO_x. The modeling included all Mesaba Phase I and II sources at maximum emission rates in normal capacity operation, plus all regional increment consuming (and expanding) emissions listed in inventories provided by the MPCA. Increment consuming emissions were included in the input file as positive numbers, and increment-expanding emissions (decreases since the baseline date) were included as negative numbers, in conformance with good modeling practice. Of note, the major emission reduction plans recently announced by Minnesota Power for its Syl Laskin, Clay Boswell, and Taconite Harbor power generation facilities were not included in the modeling analysis.

The results of the increment analyses are shown in Table 7.4-3, along with a comparison to the allowable Class II PSD increments. The data in Table 7.4-3 demonstrate that the Mesaba Project, in combination with all other regional PSD sources, will be in full compliance with all state and federal increment limits.

Table 7.4-3
Results of Class II PSD Increment Analysis (µg/m^3)
West Range IGCC Power Station

Pollutant/Averaging Time	Highest*Concentration	PSD Increment
SO_2: 3-hour	76.4	512
24-hour	20.8	91
annual	1.56	20
PM10: 24-hour	21.2	30
annual	1.93	17
NO_2: annual	2.70	25

*For short-term periods, the highest second-high concentration from five years of meteorological data is shown. For annual average, the highest concentration for any of the five years is listed.

7.4.5 Class II NAAQS Evaluation

The NAAQS modeling demonstration calculated the maximum impact of Mesaba One and Mesaba Two and all other regional sources and compared the highest total impacts, plus background concentrations, to applicable Minnesota and NAAQS. Maximum emission rates in normal operation were modeled for all Mesaba One and Mesaba Two sources and pollutants, except in the case of CO for which the startup scenario has maximum impacts.

Inclusion of other regional sources utilized a two step procedure pursuant to recommendations of MPCA modeling staff. In the first step, the Mesaba Project was modeled with nearby sources whose emission parameters were provided by the MPCA. The location and time of high and highest second-high concentrations were defined by these model results. These specific high impact events were then remodeled, through use of the AERMOD EVENT option, and included a much larger inventory of regional emission sources. The full regional inventory, referred to as First Approximation Run (FAR) data, was also provided by the MPCA. FAR data files were generated specifically for the Mesaba site location (again, not including the Minnesota Power emission reduction plan) with separate files provided for each pollutant and averaging time.

Application of the FAR data provided an approximation of the combined impacts of all sources for the specific times and receptors that were modeled. If predicted impacts threaten ambient standards, or if there is indication of significant interaction between multiple sources, more refined multiple source modeling could be necessary. In the case of Mesaba One and Two, the models clearly demonstrated that the highest predicted impacts in the analysis are far below applicable standards, and that there are very low impacts of regional sources within the Phase I and II IGCC Power Station's significant area of impact. Therefore, the FAR DATA methodology demonstrated compliance with all NAAQS limits.

Table 7.4-4 summarizes results of the NAAQS model analysis. For SO_2, PM10, and NO_x the table shows maximum impacts of the Mesaba Project alone, the Mesaba Project plus local sources that were explicitly included in the five-year model runs, and the Mesaba Project plus all

regional sources from FAR modeling of the highest impact days. For CO, no inventory of regional emissions is available. However, the data in Table 7.4-4 show CO concentrations from Mesaba One and Mesaba Two alone, and conservative total concentration estimates obtained by adding an urban background concentration to predicted Mesaba One and Mesaba Two impacts. All predicted concentrations are far below allowable levels and the results demonstrate compliance with all Minnesota and Federal ambient air quality standards.

Table 7.4-4
Results of Project Class II NAAQS Modeling West Range

Pollutant Averaging Time	Highest (1) Mesaba Alone	Highest(1) Mesaba & Nearby	Highest(1) All Sources	Back-ground	Total	NAAQS
	ug/m^3	ug/m^3	ug/m^3	ug/m^3	ug/m^3	ug/m^3
SO2						
1-hour	124.5	262.7	267.8	10	277.8	1300
3-hour	76.4	104.2	106.3	10	116.3	915
24-hour	20.8	25.1	37.3	10	47.3	365
annual	1.32	1.95	2.64	2	4.6	60
PM10						
24-hour	11.0	11.7	15.4	20	35.4	150
Annual	1.86	2.16	3.38	10	13.4	50
NOx						
Annual	2.69	3.19	4.88	5	9.9	100
CO						
1-hour	2689.6	N/A	N/A	7000(2)	9690	40,000
8-hour	389.7	N/A	N/A	3000(2)	3390	10,000

(1) Listed Highest Concentrations are highest second-high for one to 24-hour averaging times except for PM10, which is the highest 6th high from five years. Annual average values are the highest for any year.
(2) Background CO concentrations are very conservative estimates from urban monitors in Minneapolis/St. Paul. No background data exist for the Project area.

7.4.6 Risks to Human Health and Ecology

The Applicant conducted an Air Emissions Risk Assessment ("AERA") to determine whether air emissions from the IGCC Power Station may pose an unacceptable health risk to people living or farming nearby, or from eating fish from nearby lakes. The maximum predicted emissions of all potentially toxic chemicals of potential concern ("COPC") are provided in Section 3.4, above. The detailed assumptions, inputs and results of the AERA are available in the air permit application submitted to the MPCA.

The AERA was completed using several methodologies. First, acute (short-term) and sub-chronic (one-month) exposure risks were estimated using MPCA's Risk Assessment Screening Spreadsheet ("RASS") and Equivalent Risk Emission Rate ("ERER") approaches. Second, chronic risks (long-term cancer risk) were estimated using the Industrial Risk Assessment Program ("IRAP") – Health View model. The IRAP model included the evaluation of the risk

associated with inhalation as well as other exposure pathways—such as eating fish indirectly contaminated by facility air emissions. In addition, the Applicant estimated whether mercury emissions from the facility would increase the amount of mercury in fish in nearby Big Diamond Lake as well as the associated health risk due to consumption of mercury-contaminated fish. The incremental health risk of mercury emissions was estimated using both the MPCA Draft Mercury Risk Estimation Method (MPCA 2005a) and a revised version of the MPCA method using results from the AERMOD air dispersion model.

7.4.6.1 Acceptable Health Risk

The MPCA benchmark for determining whether a facility's emissions present either an acute and/or sub-chronic (non-carcinogenic) health risk to nearby residents through inhalation is called a total "hazard index." The total hazard index accounts for the risk due to inhalation of all COPC by a maximally exposed person. The acceptable MPCA total hazard index for acute and sub-chronic exposures is 1.0 or less. For chemicals producing carcinogenic (chronic) effects, the acceptable MPCA regulatory benchmark is a total cancer risk of less than one in 100,000 (10^{-5}) for a maximally exposed person.

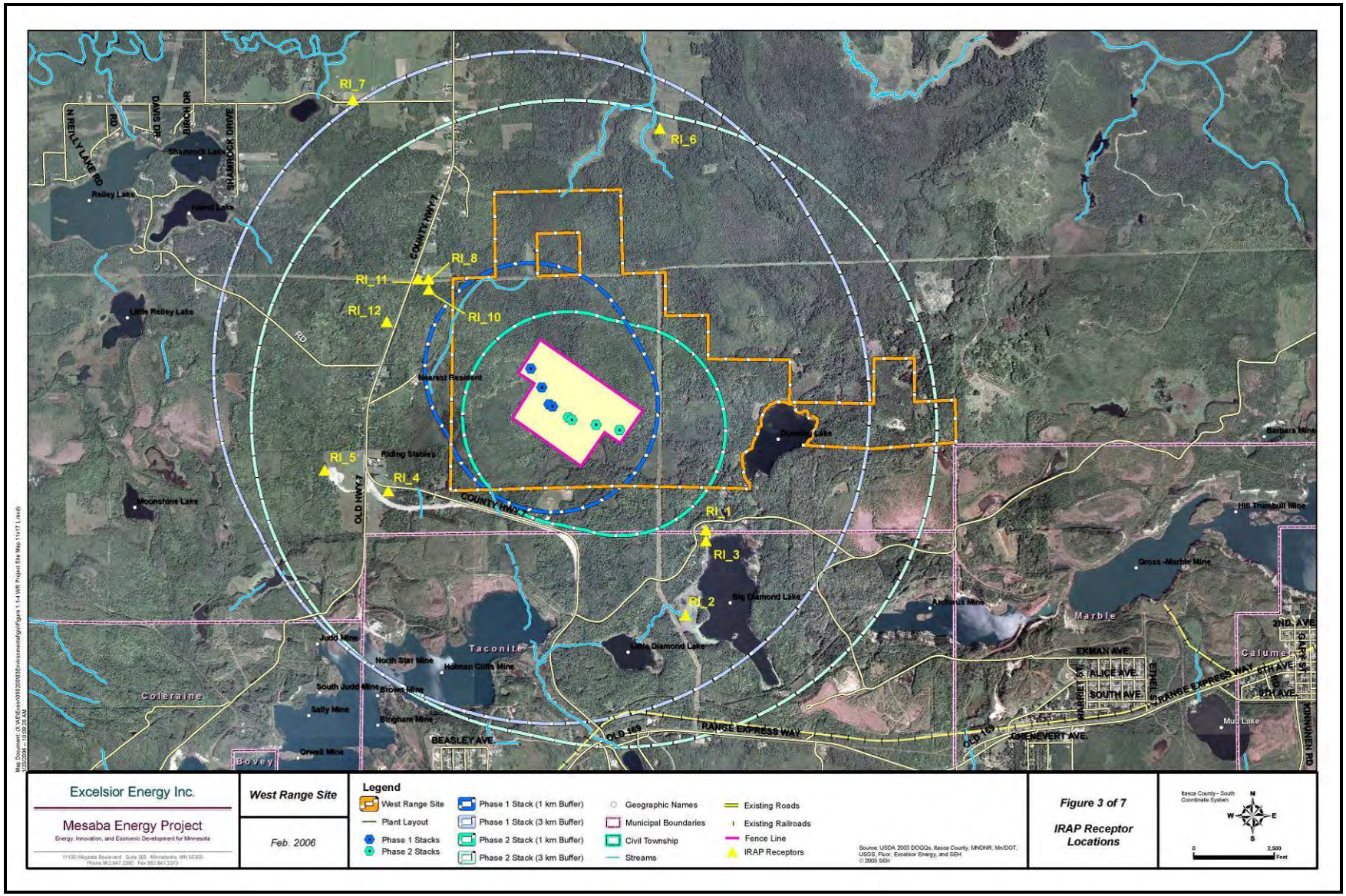
7.4.6.2 Nearby Receptors

Figure 7.4-1 shows the receptors located closest to the IGCC Power Station's point source stack emissions. The closest residence is located more than 1 kilometer away from the closest stack emission point.

7.4.6.3 RASS and ERER Results

The acute and sub-chronic potential "hazard index" was modeled at the receptor locations described above in Section 7.2. Using default, conservative RASS dispersion assumptions, the RASS spreadsheet results exceeded the MPCA hazard index of 1.0. However, the RASS spreadsheet is a "screening" tool with conservative emission dispersion and other assumptions. Therefore, a hazard index of 1.0 using the RASS does not necessarily mean that a project presents a health risk but only that additional detailed modeling must be performed to determine whether such a hazard is in fact likely.

Figure 7.4-1 Health Risk Assessment Residential Receptors



In this case, using the more refined ERER methodology (described in the AERA of the air permit application), the acute and sub-chronic health risks method are 0.52 and 0.13, respectively. Thus when using the ERER method, which is based on actual stack heights, dispersion parameters and receptor locations, both the acute and sub-chronic hazard indices are well within the acceptable MPCA total hazard index of 1.0. The ERER modeled acute and sub-chronic hazard indices are shown in Figure 7.4-2 and Figure 7.4-3, respectively.

7.4.6.4 IRAP Cancer Risk Assessment

The IRAP model was used to estimate the chronic (cancer) risk and non-cancer risks at eleven receptors representing rural residents, hobby and working farmers, and lakeshore residents. Again, using the emission rates provided in Section 3.4, above, the IRAP model predicts cancer risks ranging from 9.1×10^{-7} to $5.0 \times 10,^{-8}$ depending on receptor location. The IRAP predicted total non-cancer hazard index was 0.032 at the highest receptor. The modeled chronic and non-chronic risks indices are shown in Tables 7.4-5, 7.4-6 and 7.4-7.

Thus, the IRAP non-cancer hazard index is about 1/30 the applicable MPCA hazard index risk level for acute and sub-chronic health risks. The IRAP estimated cancer risk is less than 1/10 of the applicable chronic risk level.

Figure 7.4-2 Acute Hazard Indices From ERER Method

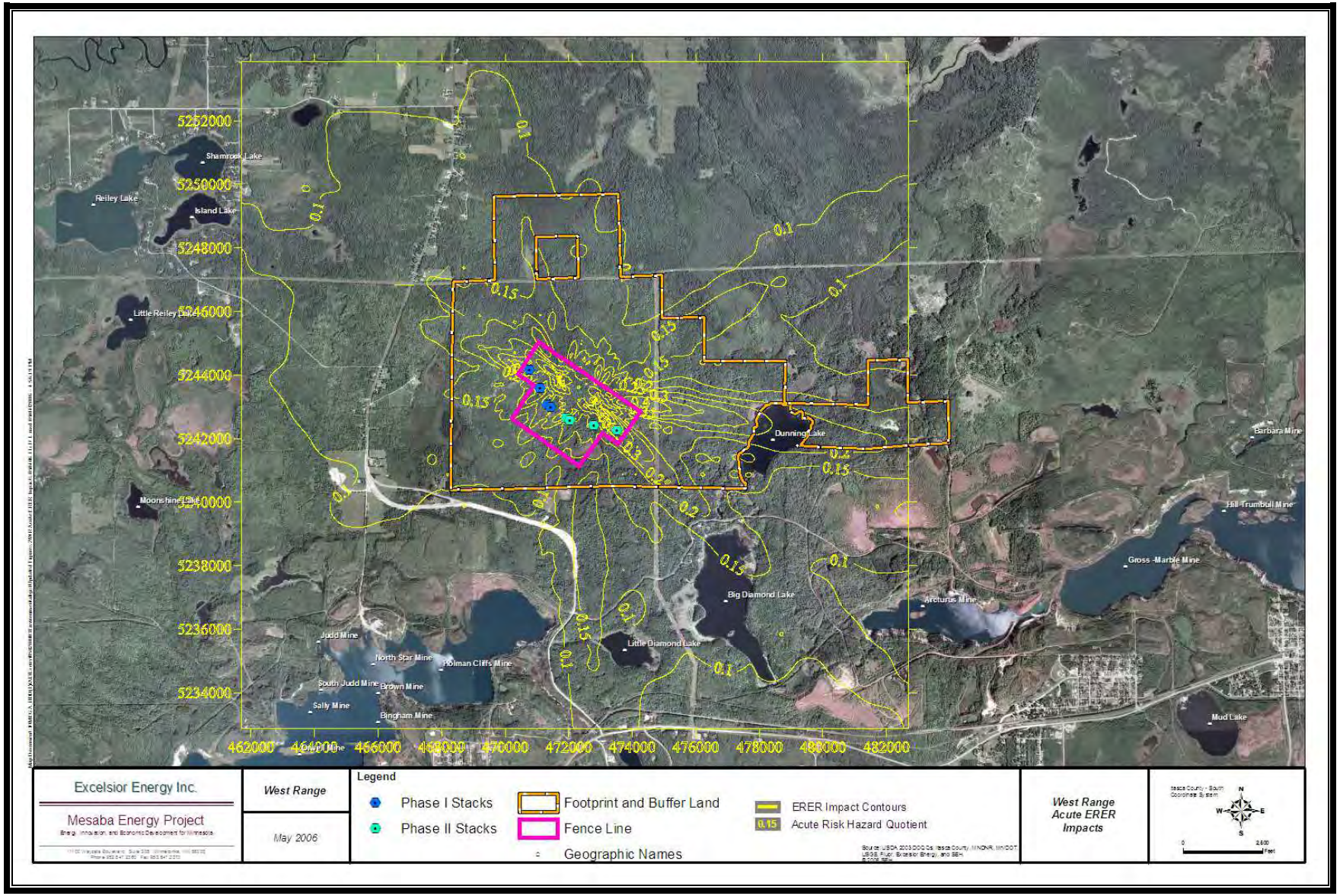


Figure 7.4-3 Subchronic Hazard Indices From ERER Method

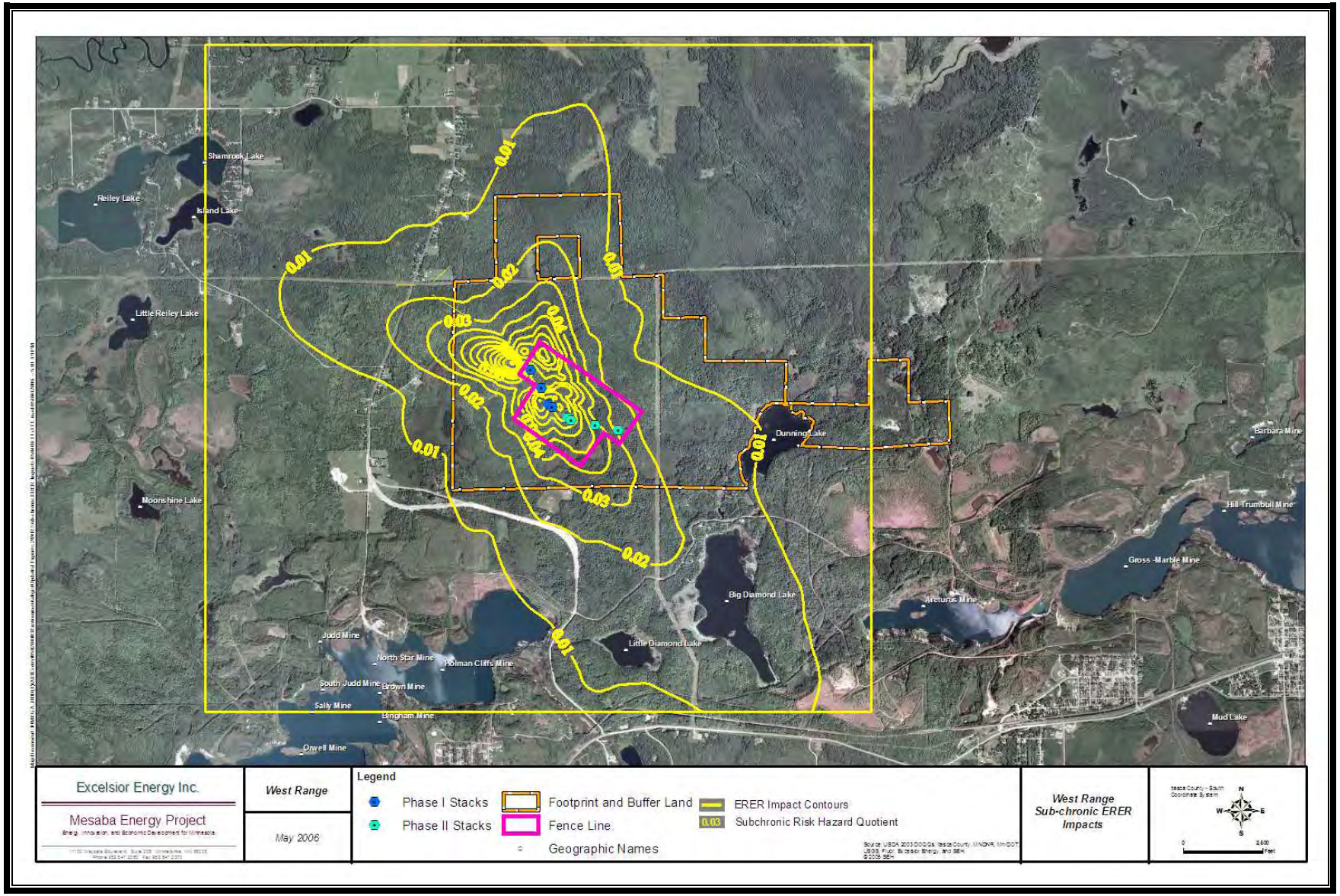


Table 7.4-5
IRAP Risk Summary by Exposure Scenarios

Location	Risk	Exposure Scenario Evaluated						
		Resident		Farmer		Fisher		Risk Acceptance Criteria Ca = 1E05 HQ = 1
		Adult	Child	Adult	Child	Adult	Child	
Rl_1 – Lake Resident	Cancer Risk	6.2E-07	2.5E-07	N/A	N/A	9.1E-07	2.9E-07	Passed
	Hazard Index	0.015	0.032	N/A	N/A	0.015	0.032	Passed
Rl_2 – Lake Resident	Cancer Risk	5.2E-07	2.1E-07	N/A	N/A	8.1E-07	2.4E-07	Passed
	Hazard Index	0.013	0.028	N/A	N/A	0.013	0.028	Passed
Rl_3 – Lake Resident	Cancer Risk	6.2E-07	2.5E-07	N/A	N/A	9.1E-07	2.9E-07	Passed
	Hazard Index	0.015	0.032	N/A	N/A	0.015	0.032	Passed
Rl_4 – Riding Stable	Cancer Risk	1.6E-07	6.5E-08	N/A	N/A	4.6E-07	1.0E-07	Passed
	Hazard Index	0.0036	0.0079	N/A	N/A	0.0037	0.0080	Passed
Rl_5 – Riding Stable	Cancer Risk	1.3E-07	5.0E-08	N/A	N/A	4.2E-07	8.8E-08	Passed
	Hazard Index	0.0028	0.0062	N/A	N/A	0.0029	0.0063	Passed
Rl_6 – NE Hobby Farm	Cancer Risk	2.6E-07	1.1E-07	N/A	N/A	5.6E-07	1.4E-07	Passed
	Hazard Index	0.0064	0.014	N/A	N/A	0.0065	0.014	Passed
Rl_7 – Working Farm	Cancer Risk	1.9E-07	7.4E-08	9.1E-07	2.3E-07	4.8E-07	1.1E-07	Passed
	Hazard Index	0.0047	0.010	0.0050	0.011	0.0048	0.010	Passed
Rl_8 – Rural Resident	Cancer Risk	4.0E-07	1.6E-07	N/A	N/A	6.9E-07	2.0E-07	Passed
	Hazard Index	0.0093	0.021	N/A	N/A	0.0095	0.021	Passed
Rl_10 – Rural Resident	Cancer Risk	4.0E-07	1.6E-07	N/A	N/A	6.9E-07	2.0E-07	Passed
	Hazard Index	0.0093	0.021	N/A	N/A	0.0094	0.021	Passed
Rl_11 – Rural Resident	Cancer Risk	3.7E-07	1.5E-07	N/A	N/A	6.7E-07	1.9E-07	Passed
	Hazard Index	0.0088	0.019	N/A	N/A	0.0089	0.020	Passed
Rl_12 – Rural Resident	Cancer Risk	3.2E-07	1.3E-07	N/A	N/A	6.2E-07	1.7E-07	Passed
	Hazard Index	0.0076	0.017	N/A	N/A	0.0077	0.017	Passed

Table 7.4-6
IRAP Cancer Risk Summary by Exposure Pathways (Page 1 of 3)

Location	Scenario	Pathway									Total Risk	Acceptance Criteria = 1E-5
		Inhalation	Produce	Beef	Poultry	Eggs	Fish	Milk	Pork	Soil		
Rl_1 – Lake Resident	Fisher Adult	2.7E-07	3.3E-07				2.9E-07			1.9E-08	9.1E-07	Passed
	Fisher Child	1.2E-07	9.2E-08				3.8E-08			3.5E-08	2.9E-07	Passed
	Resident Adult	2.7E-07	3.3E-07							1.9E-08	6.2E-07	Passed
	Resident Child	1.2E-07	9.2E-08							3.5E-08	2.5E-07	Passed
Rl_2 – Lake Resident	Fisher Adult	2.3E-07	2.7E-07				2.9E-07			1.5E-08	8.1E-07	Passed
	Fisher Child	1.0E-07	7.5E-08				3.8E-08			2.9E-08	2.4E-07	Passed
	Resident Adult	2.3E-07	2.7E-07							1.5E-08	5.2E-07	Passed
	Resident Child	1.0E-07	7.5E-08							2.9E-08	2.1E-07	Passed
Rl_3 – Lake Resident	Fisher Adult	2.7E-07	3.3E-07				2.9E-07			1.9E-08	9.1E-07	Passed
	Fisher Child	1.2E-07	9.2E-08				3.8E-08			3.5E-08	2.9E-07	Passed
	Resident Adult	2.7E-07	3.3E-07							1.9E-08	6.2E-07	Passed
	Resident Child	1.2E-07	9.2E-08							3.5E-08	2.5E-07	Passed
Rl_4 – Riding Stable	Fisher Adult	6.9E-08	9.0E-08				2.9E-07			5.1E-09	4.6E-07	Passed
	Fisher Child	3.1E-08	2.5E-08				3.8E-08			9.5E-09	1.0E-07	Passed
	Resident Adult	6.9E-08	9.0E-08							5.1E-09	1.6E-07	Passed
	Resident Child	3.1E-08	2.5E-08							9.5E-09	6.5E-08	Passed
Rl_5 – Riding Stable	Fisher Adult	5.3E-08	6.9E-08				2.9E-07			3.9E-09	4.2E-07	Passed
	Fisher Child	2.4E-08	1.9E-08				3.8E-08			7.3E-09	8.8E-08	Passed
	Resident Adult	5.3E-08	6.9E-08							3.9E-09	1.3E-07	Passed

Location	Scenario	Pathway									Total Risk	Acceptance Criteria = 1E-5
		Inhalation	Produce	Beef	Poultry	Eggs	Fish	Milk	Pork	Soil		
	Resident Child	2.4E-08	1.9E-08							7.3E-09	5.0E-08	Passed
Rl_6 – NE Hobby Farm	Fisher Adult	1.2E-07	1.4E-07				2.9E-07			8.0E-09	5.6E-07	Passed
	Fisher Child	5.1E-08	3.9E-08				3.8E-08			1.5E-08	1.4E-07	Passed
	Resident Adult	1.2E-07	1.4E-07							8.0E-09	2.6E-07	Passed
	Resident Child	5.1E-08	3.9E-08							1.5E-08	1.1E-07	Passed
Rl_7 – Working Farm	Farmer Adult	1.1E-07	5.2E-07	6.5E-08	8.7E-09	7.6E-10		2.0E-07	2.1E-09	7.3E-09	9.1E-07	Passed
	Farmer Child	3.7E-08	1.1E-07	4.3E-09	9.1E-10	8.1E-11		6.5E-08	2.4E-10	1.0E-08	2.3E-07	Passed
	Fisher Adult	8.4E-08	9.7E-08				2.9E-07			5.5E-09	4.8E-07	Passed
	Fisher Child	3.7E-08	2.7E-08				3.8E-08			1.0E-08	1.1E-07	Passed
	Resident Adult	8.4E-08	9.7E-08							5.5E-09	1.9E-07	Passed
	Resident Child	3.7E-08	2.7E-08							1.0E-08	7.4E-08	Passed
Rl_8 – Rural Resident	Fisher Adult	1.7E-07	2.1E-07				2.9E-07			1.2E-08	6.9E-07	Passed
	Fisher Child	7.7E-08	5.9E-08				3.8E-08			2.3E-08	2.0E-07	Passed
	Resident Adult	1.7E-07	2.1E-07							1.2E-08	4.0E-07	Passed
	Resident Child	7.7E-08	5.9E-08							2.3E-08	1.6E-07	Passed
Rl_10 – Rural Resident	Fisher Adult	1.7E-07	2.1E-07				2.9E-07			1.2E-08	6.9E-07	Passed
	Fisher Child	7.6E-08	5.9E-08				3.8E-08			2.3E-08	2.0E-07	Passed
	Resident Adult	1.7E-07	2.1E-07							1.2E-08	4.0E-07	Passed
	Resident Child	7.6E-08	5.9E-08							2.3E-08	1.6E-07	Passed
Rl_11 – Rural Resident	Fisher Adult	1.6E-07	2.0E-07				2.9E-07			1.1E-08	6.7E-07	Passed
	Fisher Child	7.2E-08	5.5E-08				3.8E-08			2.1E-08	1.9E-07	Passed
	Resident Adult	1.6E-07	2.0E-07							1.1E-08	3.7E-07	Passed

Location	Scenario	Pathway									Total Risk	Acceptance Criteria = 1E-5
		Inhalation	Produce	Beef	Poultry	Eggs	Fish	Milk	Pork	Soil		
	Resident Child	7.2E-08	5.5E-08							2.1E-08	1.5E-07	Passed
Rl_12 – Rural Resident	Fisher Adult	1.4E-07	1.8E-07				2.9E-07			1.0E-08	6.2E-07	Passed
	Fisher Child	6.0E-08	4.9E-08				3.8E-08			1.9E-08	1.7E-07	Passed
	Resident Adult	1.4E-07	1.8E-07							1.0E-08	3.2E-07	Passed
	Resident Child	6.0E-08	4.9E-08							1.9E-08	1.3E-07	Passed

Note: Blank cells indicate pathway was not evaluated for the scenario.

Table 7.4-7
IRAP Hazard Index Summary by Exposure Pathways (Page 1 of 2)

Location	Scenario	Pathway									HQ Total	Acceptance Criteria = 1
		Inhalation	Produce	Beef	Poultry	Eggs	Fish	Milk	Pork	Soil		
Rl_1 – Lake Resident	Fisher Adult	0.014	0.0003				0.0001			0.000005	0.015	Passed
	Fisher Child	0.032	0.0005				0.0001			0.000042	0.032	Passed
	Resident Adult	0.014	0.0003							0.000005	0.015	Passed
	Resident Child	0.032	0.0005							0.000042	0.032	Passed
Rl_2 – Lake Resident	Fisher Adult	0.012	0.0003				0.0001			0.000004	0.013	Passed
	Fisher Child	0.028	0.0004				0.0001			0.000033	0.028	Passed
	Resident Adult	0.012	0.0003							0.000004	0.013	Passed
	Resident Child	0.028	0.0004							0.000033	0.028	Passed
Rl_3 – Lake Resident	Fisher Adult	0.014	0.0003				0.0001			0.000004	0.015	Passed
	Fisher Child	0.032	0.0005				0.0001			0.000042	0.032	Passed
	Resident Adult	0.014	0.0003							0.000004	0.015	Passed
	Resident Child	0.032	0.0005							0.000042	0.032	Passed
Rl_4 – Riding Stable	Fisher Adult	0.004	0.0001				0.0001			0.000001	0.004	Passed
	Fisher Child	0.008	0.0001				0.0001			0.000011	0.008	Passed
	Resident Adult	0.004	0.0001							0.000001	0.004	Passed
	Resident Child	0.008	0.0001							0.000011	0.008	Passed
Rl_5 – Riding Stable	Fisher Adult	0.003	0.0001				0.0001			0.000001	0.003	Passed
	Fisher Child	0.006	0.0001				0.0001			0.000008	0.006	Passed
	Resident Adult	0.003	0.0001							0.000001	0.003	Passed
	Resident Child	0.006	0.0001							0.000008	0.006	Passed
Rl_6 – NE Hobby Farm	Fisher Adult	0.006	0.0001				0.0001			0.000002	0.006	Passed
	Fisher Child	0.014	0.0002				0.0001			0.000017	0.014	Passed
	Resident Adult	0.006	0.0001							0.000002	0.006	Passed
	Resident Child	0.014	0.0002							0.000017	0.014	Passed

Location	Scenario	Pathway									HQ Total	Acceptance Criteria = 1
		Inhalation	Produce	Beef	Poultry	Eggs	Fish	Milk	Pork	Soil		
Rl_7 – Working Farm	Farmer Adult	0.005	0.0004	0.00001	0.0000	0.0000		0.00001	0.0000	0.000001	0.005	Passed
	Farmer Child	0.010	0.0006	0.00001	0.0000	0.0000		0.00002	0.0000	0.000012	0.011	Passed
	Fisher Adult	0.005	0.0001				0.0001			0.000001	0.005	Passed
	Fisher Child	0.010	0.0002				0.0001			0.000012	0.010	Passed
	Resident Adult	0.005	0.0001							0.000001	0.005	Passed
	Resident Child	0.010	0.0002							0.000012	0.010	Passed
Rl_8 – Rural Resident	Fisher Adult	0.009	0.0002				0.0001			0.000003	0.009	Passed
	Fisher Child	0.020	0.0003				0.0001			0.000027	0.021	Passed
	Resident Adult	0.009	0.0002							0.000003	0.009	Passed
	Resident Child	0.020	0.0003							0.000027	0.021	Passed
Rl_10 – Rural Resident	Fisher Adult	0.009	0.0002				0.0001			0.000003	0.009	Passed
	Fisher Child	0.020	0.0003				0.0001			0.000027	0.021	Passed
	Resident Adult	0.009	0.0002							0.000003	0.009	Passed
	Resident Child	0.020	0.0003							0.000027	0.021	Passed
Rl_11 – Rural Resident	Fisher Adult	0.009	0.0002				0.0001			0.000003	0.009	Passed
	Fisher Child	0.019	0.0003				0.0001			0.000025	0.019	Passed
	Resident Adult	0.009	0.0002							0.000003	0.009	Passed
	Resident Child	0.019	0.0003							0.000025	0.019	Passed
Rl_12 – Rural Resident	Fisher Adult	0.007	0.0002				0.0001			0.000002	0.008	Passed
	Fisher Child	0.017	0.0003				0.0001			0.000021	0.017	Passed
	Resident Adult	0.007	0.0002							0.000002	0.008	Passed
	Resident Child	0.017	0.0003							0.000021	0.017	Passed

7.4.6.5 Mercury Fish Consumption Risk

Based on mercury data for lakes in the region, the 90th percentile northern pike in nearby Big Diamond Lake has a mercury contamination level of 0.56 ppm. As described below, using the MPCA Draft Mercury Risk Estimation Method for the Fish Consumption Pathway (MPCA 2006) it was estimated that emissions from the proposed project would increase the mercury concentration in the 90th percentile pike in Big Diamond Lake by 0.003 ppm.

This predicted mercury increase translates into an incremental increase in the hazard quotient of 0.01 for a subsistence fisher on Big Diamond Lake.

Based on the MPCA assumptions provided below, even if the proposed project is not constructed, the current estimated mercury risk to a subsistence fisher from eating fish from Big Diamond Lake equals a hazard quotient of 1.95 to 2.80, depending on the size of fish consumed (from an average of 21.8 inches to the 90th percentile of 27.8 inches).

In comparison to the existing hazard quotient for subsistence fishers eating fish from Big Diamond Lake, the incremental increase in hazard quotient predicted for inputs of mercury from Mesaba One and Mesaba Two is negligible.

7.4.6.5.1 Mercury Risk Assumptions

Based on the MPCA draft mercury risk assessment method, the Applicant used the following modeling assumptions:

- Background mercury deposition:
  - wet-plus-dry ambient deposition (flux) = 12.5 $\mu g/m^2$-yr – Minnesota default to lake surfaces and 33.6 $\mu g/m^2$-yr to rest of the watershed
  - 10 % watershed deposition transported to water body
  - Lake Finder database lake area for Big Diamond Lake = 122 acres (MNDR Lake Finder)
  - Watershed area for Big Diamond Lake determined using IRAP = 760 acres
- Mercury mass deposited to lake and watershed due to facility emissions
  - Determined by site-specific air dispersion modeling in AERMOD
  - Concentration over lake and watershed = 13 pg/m^3
  - Hg^0 Depositional Velocity = 0.01 cm/sec over the lake and 0.05 cm/sec over the rest of the watershed
  - All mercury emissions are assumed to be elemental mercury (Hg^0)
- Methylmercury estimation in fish fillet
  - Reference species of fish is Northern Pike
  - Database used to determine the current fish tissue concentration = "Allfish 04 NE lakes only" provided electronically as an Excel spreadsheet by MPCA
- Risk assumptions
  - Daily fish consumed = 0.142 kg/day
  - Adult body weight = 70 kg
  - Reference dose for methyl mercury = 1.0×10^{-4} mg/kg-day
- Fish Mercury Assumptions
  - N = Total fish in the modified database = 9,375 Northern Pike

- Minimum length = 6.7 inches
- Maximum length = 45.5 inches
- 90th percentile length fish = 27.8 inches
- Mean mercury concentration of 90th percentile 27.8 inch fish = 0.56 ppm (standard deviation = 0.40)
- Average length fish = 21.8 inches
- Number of fish of 21.8 inches = 105 fish
- Mean mercury concentration of all 21.8 inch fish = 0.39 ppm (standard deviation = 0.26)

7.4.6.5.2 MPCA Mercury Method Results

The MPCA draft local impact mercury assessment produced the following results:

- Mercury Loading Summary:
  - Mercury deposition to the lake from the facility = 0.08 g/yr
  - Background mercury deposition to the lake from rainfall and runoff = 16.51 g/yr
- Incremental increase in mercury in fish tissue from the project – 90th percentile fish size = 0.003 ppm
- Incremental increase in mercury in fish tissue from the project – average fish size = 0.002 ppm
- 90th percentile fish size
  - Ambient Subsistence Fisher Hazard Quotient = 2.80
  - Incremental Subsistence Fisher Hazard Quotient from the project = 0.01
- Average fish size
  - Ambient Subsistence Fisher Hazard Quotient = 1.95
  - Incremental Subsistence Fisher Hazard Quotient from the project = 0.01

7.4.6.5.3 Alternative Mercury Modeling Results

Tests on the Wabash River Coal Gasification Repowering Project showed that 100% of the mercury emitted from the facility was in the volatile, Hg^0, valence state (EPA 2002). Based on these Wabash test results, the limited solubility of elemental mercury in water (Perry and Green, 1984), and the low level emissions of mercury from the IGCC Power Station, the insignificance of the impacts predicted by the MPCA's Draft Mercury Risk Estimation Method for Fish Consumption are realistic. To confirm this, the Applicant also used AERMOD to calculate mercury deposition from the project to Big Diamond Lake and found the deposition rate to be 0.0021 micrograms per square meter per year ($\mu g/m^2$-yr).

The MPCA Hg-2003 evaluation conducted by the Applicant can be found in the application for a Part 70/New Source Review Construction Authorization Permit Mesaba One and Mesaba Two, dated June 2006 and attached as Appendix 5 to this Joint Application.

7.4.7 Class I Impacts and Increment Consumption

An air quality modeling analysis was conducted to estimate impacts of the Project on air quality in the following Class I areas: the Boundary Waters Canoe Area (BWCA), Voyageurs National Park (VNP), and the Rainbow Lakes Wilderness (RLW) located in Wisconsin. The distance from the Station to the closest point in each of these Class I areas is approximately 61 miles (98 km) for the BWCA, 75 miles (121 km) for VNP, and 117 miles (188 km) for RLW. The next closest Class I area, Isle Royale National Park, is more than 300 km from the Station and is located beyond the distance where long-range transport modeling has been shown to provide meaningful impact predictions.

The Class I analyses addressed PSD Class I increments for SO_2, and PM10. The dispersion modeling analysis used standard EPA long-range transport modeling methodologies, and followed the guidance presented in EPA's Guideline on Air Quality Models, the IWAQM Phase II report, and the FLAG Phase I report. The analyses also incorporated the suggestions and guidance received in pre-application meetings with the U.S. Forest Service and the National Park Service.

The CALPUFF model was used to calculate pollutant impacts from the Project for each year of meteorological data, for three-hour, 24-hour, and annual averaging periods. The two-phase Mesaba facility was modeled alone and the results compared with Class I PSD increments and SILs. Table 7.4-8 summarizes the highest CALPUFF model results for each Class I area, and shows the applicable Increment and SIL values. The data indicate that maximum Mesaba impacts are far below allowable increments for all pollutants and Class I areas. Impacts are also below the SIL in most cases, indicating that impacts will be insignificant, with no further analysis necessary. However, for short-term SO_2 concentrations, impacts are indicated to exceed the SIL in the BWCA and VNP. Because of the 3-hour and 24-hour SO_2 projected impacts, it was necessary to conduct a cumulative impact analysis, including other regional SO_2 increment sources, to quantify total PSD increment consumption.

Table 7.4-8
Class I PSD Increment Modeling Results for Mesaba Energy Project West Range

Boundary Waters/Pollutant	Year Evaluated			Class I Inc	Class I SIL	Max
Averaging Period	**1990**	**1992**	**1996**	**(ug/m3)**	**(ug/m3)**	**(ug/m3)**
SO2 3-Hour	1.3804	1.4547	1.5505	25.0	1.00	1.5505
SO2 24-Hour	0.4554	0.3382	0.3589	5.0	0.20	0.4554
SO2 Annual	0.0147	0.0127	0.0095	2.0	0.10	0.0147
NOx Annual	0.0174	0.0152	0.0109	2.5	0.10	0.0174
PM10 24-Hour	0.0866	0.0617	0.0586	8.0	0.30	0.0866
PM10 Annual	0.0041	0.0037	0.0026	4.0	0.20	0.0041

Voyageurs Pollutant/	Year Evaluated			Class I Inc	Class I SIL	Max
Averaging Period	**1990**	**1992**	**1996**	**(ug/m3)**	**(ug/m3)**	**(ug/m3)**
SO2 3-Hour	1.5911	1.0477	1.4836	25.0	1.00	1.5911
SO2 24-Hour	0.2506	0.2943	0.4492	5.0	0.20	0.4492
SO2 Annual	0.0128	0.0110	0.0113	2.0	0.10	0.0128
NOx Annual	0.0151	0.0125	0.0142	2.5	0.10	0.0151
PM10 24-Hour	0.0537	0.0500	0.0745	8.0	0.30	0.0745
PM10 Annual	0.0037	0.0032	0.0031	4.0	0.20	0.0037

Rainbow Lakes Pollutant/	Year Evaluated			Class I Inc	Class I SIL	Max
Averaging Period	**1990**	**1992**	**1996**	**(ug/m3)**	**(ug/m3)**	**(ug/m3)**
SO2 3-Hour	0.7088	0.7567	0.7012	25.0	1.00	0.7567
SO2 24-Hour	0.1806	0.1917	0.1711	5.0	0.20	0.1917
SO2 Annual	0.0075	0.0083	0.0065	2.0	0.10	0.0083
NOx Annual	0.0081	0.0071	0.0068	2.5	0.10	0.0081
PM10 24-Hour	0.0369	0.0462	0.0316	8.0	0.30	0.0462
PM10 Annual	0.0022	0.0028	0.0019	4.0	0.20	0.0028

The short-term cumulative SO_2 increment analysis used the CALPUFF model to calculate the combined impacts of all regional increment consuming and increment expanding sources at each of the Class I areas. In response to a request for SO_2 increment inventories, the MPCA provided emission and stack data for those northern Minnesota sources with a potential for Class I impacts (again, not including Minnesota Power's emission reduction plans).

The results of the cumulative SO_2 increment analysis are shown in Table 7.4-9. The maximum predicted increment consumption in each of the Class I areas was shown to be well within the PSD Class I limits with the conclusion being that the Mesaba Project will not cause or contribute to any violation of Class I PSD increments.

Table 7.4-9
Mesaba Cumulative SO_2 Increment Results (Highest Second Highs)
West Range

Boundary Waters/Pollutant Averaging Period	**1990**	**1992**	**1996**	**Class I Inc (ug/m3)**	**Max (ug/m3)**	**Violation? (Y/N)**
SO2 3-Hour	6.1	7.0	5.8	25.0	7.0	NO
SO2 24-Hour	2.6	2.1	2.2	5.0	2.6	NO
Voyageurs Pollutant/ Averaging Period	1990	1992	1996	Class I Inc (ug/m3)	Max (ug/m3)	Violation? (Y/N)
SO2 3-Hour	5.2	4.6	5.5	25.0	5.5	NO
SO2 24-Hour	1.6	1.5	1.6	5.0	1.6	NO
Rainbow Lakes Pollutant/ Averaging Period	1990	1992	1996	Class I Inc (ug/m3)	Max (ug/m3)	Violation? (Y/N)
SO2 3-Hour	4.5	4.3	4.7	25.0	4.7	NO
SO2 24-Hour	1.4	1.2	1.3	5.0	1.4	NO

7.4.8 Visibility

A visibility impact analysis was carried out for BWCA and VNP (visibility analysis is not required for Rainbow Lakes). Detailed modeling inputs, assumptions, and results are contained in the Applicant's MPCA air permit application. The recommended methodology for assessing visibility impacts according to the FLAG guidance involves the use of CALPOST to process the data on concentrations of pollutants from the CALPUFF modeling of 24-hour emissions. In CALPOST a daily value of light extinction is defined by the concentrations of each pollutant that can affect visibility, taking into account the efficiency of each particle type in scattering light, and the relative humidity which influences the size of hygroscopic pollutants (sulfates and nitrates). The 24-hour average light extinction caused by emissions from the modeled source(s) is then compared to the background light extinction, a value based upon "natural" or pristine unpolluted conditions for each Class I area.

The Federal Land Managers (FLMs) have established threshold changes in light extinction (as a percentage of natural background) that are believed to represent potential adverse impacts on visibility. These thresholds are 5% (a potentially detectable change) and 10% (a level that may represent an unacceptable degradation).

Table 7.4-10 presents results of the initial CALPUFF visibility analysis following the FLAG methodology, and using "Method 2" of CALPOST for calculation of visibility impacts. In Method 2, relative humidity data from the nearest surface weather station is used to calculate both source and background light extinction. Other methods, discussed below, use average relative humidity values, consider natural visibility impairment, and take into account average light extinction over a line-of-sight rather than extinction at a single receptor location.

Table 7.4-10
Mesaba CALPUFF Visibility Results

Speciated PM	**1990**	**1990**	**1990**	**1992**	**1992**	**1992**	**1996**	**1996**	**1996**
12/5/2005	**Method 2**	**Method 2**	**Method 2**	**Method 2**	**Method 2**	**Method 2**	**Method 2**	**Method 2**	**Method 2**
	> 5 %	> 10 %	Max %	> 5 %	> 10 %	Max %	> 5 %	> 10 %	Max %
Boundary Waters Wilderness	39	10	16.43	36	15	24.11	17	6	14.98
Voyageurs National Park	16	1	11.82	25	4	18.97	18	4	22.47
Speciated PM	**1990**	**1990**	**1990**	**1992**	**1992**	**1992**	**1996**	**1996**	**1996**
12/5/2005	**Method 6**	**Method 6**	**Method 6**	**Method 6**	**Method 6**	**Method 6**	**Method 6**	**Method 6**	**Method 6**
	> 5 %	> 10 %	Max %	> 5 %	> 10 %	Max %	> 5 %	> 10 %	Max %
Boundary Waters Wilderness	24	1	12.12	19	2	11.54	9	0	8.13
Voyageurs National Park	13	0	8.43	14	1	10.22	8	1	12.49
Speciated PM	**1990**	**1990**	**1990**	**1992**	**1992**	**1992**	**1996**	**1996**	**1996**
12/5/2005	**Method 7***	**Method 7**	**Method 7**	**Method 7**	**Method 7**	**Method 7**	**Method 7**	**Method 7**	**Method 7**
	> 5 %	> 10 %	Max %	> 5 %	> 10 %	Max %	> 5 %	> 10 %	Max %
Boundary Waters Wilderness	11	1	10.43	7	1	19.22	2	0	7.63
Voyageurs National Park	3	0	7.93	2	0	6.13	3	0	8.13

* - Hibbing MN used as primary weather station for Boundary Waters Wilderness, International Falls used for Voyageurs NP.

The data in Table 7.4-10 indicate that calculated visibility impacts greater than 5% or 10% could occur at some times within the BWCA and VNP on a small number of days each year. Because these data suggest a potential for detectable visibility degradation due to Mesaba emissions, additional analyses were carried out to better quantify and evaluate the real possibility of visibility impacts. These analyses are described in following sections.

The CALPOST post-processing software contains several alternative algorithms for calculating the change in light extinction due to the modeled source. Method 6 substitutes monthly average relative humidity values (specific to each Class I area) for the hourly relative humidity data at nearby weather stations. This substitution mitigates, to some extent, the high extinction values calculated when very high humidity values are reported throughout the day at the nearest observation site. It is intended to account for the fact that the observed humidity may be unrepresentative of the Class I area, and that very high relative humidities are frequently associated with natural impairment by fog, clouds, and precipitation. The Method 6 calculation is recommended by the U.S. EPA for state regional haze BART analyses.

Method 7 is another modification of the standard Method 2 and attempts to account for natural visibility reduction due to fog or precipitation. In Method 7, the actual measured visibility at the nearest weather station is used as background on those hours when fog or precipitation are reported. Method 7 represents another attempt to account for natural visibility reduction in assessing the impact of man-made pollution which may affect visibility.

A criticism of Method 7 is that it tends to minimize the effect of source-induced haze on days when natural impairment may only exist for a small part of the day. It is possible that the impact of the source could still be significant during other hours of the day. This is a valid point, but the FLAG procedures specify visibility calculation on a 24-hour daily basis, on the grounds that model predictions for any single hour are subject to significant error. If the 24-hour averaging is appropriate for the basic visibility calculation, it is reasonable to also utilize some 24-hour averaging in calculating the effect of natural visibility impairing events. As an example, the occurrence of fog or precipitation on specific hours at a nearby weather station indicates the likelihood of these events at other times and/or locations within the Class I area.

Table 7.4-10 shows the results of Method 6 and Method 7 visibility calculations for the Mesaba Project, with comparison to the Method 2 data. Both alternative analyses indicate lower frequency and magnitude of impacts relative to Method 2. For Method 7, there are only two days of predicted impacts in the three year data period which exceed the 10% change in light extinction at the BWCA, and no predicted impacts at VNP.

In EPA's BART guidance for regional haze, an average of seven days per year or more exceeding a 5% increase indicates a significant impact. Using this criterion, the Method 7 results show no significant visibility impact of Mesaba One and Mesaba Two at either BWCA or Voyageurs National Park.

7.4.9 Vehicle Traffic Emissions

Emissions will be generated from vehicles operated during construction and operation of the IGCC Power Station and its Associated Facilities. These emissions will be by-products of combustion from vehicle engines and fugitive dust generated from traffic on the roadways near and on the IGCC Power Station Footprint and Buffer Land.

On-site personnel during peak construction activities is expected to reach about 1,500 persons. Assuming a 20 percent reduction in vehicle trips from carpooling, peak vehicle trips during this time are estimated to be about 1,200 trips per day of personal vehicles, and 20 to 30 delivery vehicles per day. During operation of Phases I and II, employees, on-site contractors, and visitors are expected to total between 125 and 175 persons. Estimated vehicular emissions from the East and West Range Sites are comparable.

When compared with emissions from the facility, vehicular emissions are small. Table 7.4-11 shows estimated peak daily emission rates from personal vehicles during construction. The estimated emission rate of carbon monoxide, the pollutant emitted at the greatest rate, is 22 pounds per day.

Table 7.4-11. Daily Emission Rates from Vehicle Traffic

Pollutant	Emission Factor[1] gram/mile	Number of Vehicle Trips/day	Distance Per Trip mile/trip	Emission Rate lb/day
NOx	0.3	1,200	1	0.8
CO	4.2	1,200	1	11
NMOC[2]	0.18	1,200	1	0.48
PM	0.06	1,200	1	0.2

Notes:
[1] Emission Factors taken from EPA Green Vehicle Guide using EPA's assumed average engine performance (www.epa.gov/greenvehicles/select.htm).
[2] NMOC = non methane organic compounds, which is equivalent to volatile organic compounds.

Roadways and parking lots where emissions from mobile sources will occur are referred to as indirect sources. The State of Minnesota does not have an indirect source permitting program. According to Minnesota Department of Transportation Highway Project Development Process Handbook (HPDP Handbook, Part II, Section D "Air Quality"), a detailed air quality analysis is required if anticipated traffic volumes exceed traffic volumes of the top ten intersections in Minnesota (see HPDP Handbook, Appendix 5). The smallest traffic volume of the top ten intersections is 35,800 annual average daily traffic (AADT). As previously stated, peak traffic counts associated with the construction and operation of Mesaba One and Mesaba Two will be a small fraction of the AADT threshold, and further detailed air quality analysis is not appropriate or necessary.

7.4.10 Soil and Vegetation

The PSD regulation requires analysis of air quality impacts on sensitive vegetation and soil types. Evaluation of impacts on sensitive vegetation and soils was performed at the West Range Site by comparing predicted project impacts to screening levels presented in the 1980 EPA document entitled: *A Screening Procedure for the Impacts of Air Pollution Sources on Plants, Soils and Animals* (EPA, December 1980, EPA 450/2-81-078). These screening levels represent the minimum concentrations in either plant tissue or soils at which adverse growth effects or tissue injury was reported in the literature. The procedures specify that predicted impact concentrations used for comparison account for facility impacts and ambient background concentrations. Most of the designated vegetation screening levels are equivalent to or exceed PSD standards. The 3-hour and 1-hour SO_2 sensitive vegetation screening levels are more stringent than comparable NAAQS and State Ambient Air Quality Standards. The Applicant's air quality modeling indicates that project impacts will be below NAAQS and PSD thresholds.

A comparison of the SO_2 sensitive vegetation screening levels with SO_2 impacts from the proposed project is presented in Table 7.4-12. The comparison includes ambient background concentrations in the impact levels. As shown in the table, maximum impacts for the 1-hour and 3-hour averaging periods are less than 20% of allowable concentrations.

Since dispersion modeling results at the East and West Range Sites are similar, impacts on vegetation at each site will be similar.

Table 7.4-12
Vegetative Sensitivity Screening for SO_2 Concentrations

Averaging Time	Background Concentration ($\mu g/m^3$)	Maximum Contribution from Project ($\mu g/m^3$)	Maximum Total Concentration ($\mu g/m^3$)	Sensitive Vegetation Screening Level ($\mu g/m^3$)
1-Hour	10	124.5	134.5	917
3-Hour	10	76.4	86.4	786

7.5 GEOLOGY AND SOILS

This subsection summarizes the geology in the vicinity of the West Range Site, with a more detailed description provided in Section 2 of the ES. Mesaba One and Mesaba Two will not have a significant impact on geology or soils outside the IGCC Power Station Footprint, Buffer Land, and Interconnection Corridors. The impacts on farmland are described in Section 7.1.10.3.

The West Range Site is located within the Superior Upland Section of the Laurentian Upland of the Canadian Shield physiographic province (Leonards, 1962). The physical landscape of the region is typified by forests, lakes and bogs in glacial till over somewhat shallow bedrock. The landscape has been greatly affected by the glaciers that covered the land, the last of which left the area about 12,000 years ago. Physical relief is generally limited to a thousand feet or less

around the West Range Site. The maximum elevation range in the region is from elevation 600 feet msl at Lake Superior to elevation 2,301 feet msl at Eagle Mountain, the highest point in Minnesota.

The West Range Site is located in a regional area that generally consists of low glacial moraines and till plains. Physically, the local landscape is dotted with 300-to-400-foot deep mine pits, large mine-pit overburden spoil piles, and tailing basins, all of which are associated with former iron ore mining activity. The extent of the mining disturbance in the West Range Site area is shown in Figure 7.1-3.

The West Range Site straddles the border of Giants Range and the Chisholm-Embarrass physiographic area of Minnesota, as defined by Wright (Sims and Morey, 1972). The elevation atop the Giant's Range Batholith is about elevation 1,430 feet msl to 1,470 feet msl at the site. The Chisholm/Embarrass Till area exists north of the Giant's Range. The Chisholm/Embarrass Till Area is an area of low glacial moraines and outwash plains that extends north and south of the Giants Range in the vicinity of the project site. The till is typically on the order of 25 feet thick or less at the West Range Site, but extends to greater depths in the general area north and south of the site. Bedrock outcrops also exist in the area. Much of the till has been stripped and removed along the Iron Range as part of past mining operations. The elevation of the till plains to the north and south of the site are at about elevation 1,330 feet msl.

7.6 WATER RESOURCES AND WATER QUALITY

7.6.1 Surface Waters in the Vicinity of the West Range Site

The West Range Site lies within the northernmost region of the Upper Mississippi River Basin (UMRB) Watershed. The major surface waters in the vicinity of the Site are listed in Table 7.6-1.

Table 7.6-1
Surface Waterbodies

Surface Water	Watershed
Big Diamond Lake	Swan River
CMP	Swan River
Dunning Lake	Swan River
Greenway Mine Pit	Prairie River
Hill-Annex Mine Pit	Swan River
Holman Lake (Hill Lake)	Swan River
Lind Mine Pit	Prairie River
Little Diamond Lake	Swan River
Lower Panasa Lake	Swan River
Mississippi River	Mississippi River
Oxhide Creek	Swan River

Surface Water	Watershed
Oxhide Lake	Swan River
Prairie River	Mississippi River
Snowball Creek	Swan River
Sucker Brook	Prairie River
Swan River	Mississippi River
Trout Creek	Swan River
Trout Lake	Swan River
Twin Lakes	Swan River
Upper Panasa Lake	Swan River
West Hill Mine Pit	Prairie River

The Prairie River Watershed includes the northern portions of the project site. The CMP (CMP) Watershed is isolated from the other watersheds as the CMP does not have an outlet. The remaining surface waterbodies listed in Table 7.6-1 are all within the Swan River Watershed. The Prairie River and the Swan River both drain to the Mississippi River.

The Site consists of small surface depressions, wetlands, and several intermittent unnamed streams. The IGCC Power Station Footprint would be constructed over an existing drainage divide. The northern portion of the Station Footprint lies in the Sucker Brook watershed that drains into the Prairie River. The southern portion of the Station Footprint lies in a subwatershed that drains into the CMP.

Potential impacts to these waterbodies from stormwater discharges from the West Range Site during construction and operation will be minimized by appropriate stormwater management practices.

7.6.2 Historical Information

This Section describes the historical background of the resources the Applicant will use for its water supply. Water supply lines will originate in the CMP, HAMP Complex, LMP and the Prairie River. Process water discharge lines will terminate in the CMP and Holman Lake. The West Range Site process water infrastructure is discussed in Section 3.6.2.2. The Applicant's water management plan is discussed below in Section 7.6.3.

1.1.1.3 Stage/Storage Relationships

7.6.2.1.1 CMP Complex

The Canisteo Mine was one of 18 different properties operated over time by six different companies that made up a 4.5 mile long natural ore mining complex. In 1907, the Holman-Cliffs, Diamond, and Canisteo properties were the first to begin shipping ore. By 1985, mining across the entire length of the ore complex had ceased after having shipped more than 194,500,000 long tons of ore (MDNR, 2001).

During active mining it was necessary to pump water from the individual pits making up the mining complex to permit mining of the iron ore body. Once the pits were abandoned, dewatering operations ceased and they began to fill with water. Waters that had received pumped input, but no natural drainage, were cut off from this water supply, but runoff and ground water began to fill the formerly mined pits. Water in such pits rose dramatically in the first several years following abandonment but was not monitored.

Bathymetric data has been collected by the MDNR and was used to develop a stage-storage relationship for the pit. The MDNR has also collected stage (water elevation) data since 1989, but stage data was not collected on a daily basis until 1995. Because less detailed stage data exists for the period from 1989 to 1995, it was necessary to calculate long-term average inflows. Based on the available stage data and the stage-storage relationship for the pit, an average recharge of 3,160 gpm for the CMP was calculated over this period. Bathymetric mapping of the pit has been developed by the Applicant, based on electronic sampling of the mined surface through the water column.

Daily stage data from 1995 to present is available from the MDNR, although some data gaps exist. From 1995 to present, recharge rates range from 810 gpm to 4,190 gpm, with an average of 2,580 gpm. The water surface elevation in the mine pit on November 1, 2005 was 1,309 feet, with a surface area of 1,393 acres and a water volume of 149,500 acre-feet. Although groundwater movements are difficult to quantify, it appears that the amount of seepage out of the mine pit increases significantly when the water surface elevation is above the bedrock elevation (approximately 1300 feet msl).

When the data from years in which the stage was above 1300 feet msl (after year 2000) are not included, recharge rates ranged from 1,820 gpm to 4,190 gpm, with an average of 2,980 gpm.

The CMP does not currently discharge directly to surface waters. Water input to the pit results exclusively from surface water runoff and groundwater inflow. Water only leaves the pit through groundwater seepage and evaporation.

The west end of the mining complex, the Buckeye Mine Pit, filled with water and began being used for some recreational fishing after a boat launch was installed and the MDNR began to stock lake trout. As water continued to rise in each of the pits across the abandoned mining complex, the pits became interconnected, with the Buckeye Mine Pit becoming connected to the other pits in the early 1990's. The connected series of pits is locally referred to as the CMP and the pit now receives occasional recreational use by virtue of the Buckeye Mine Pit boat launch. For safety, security, and operational reasons, the Applicant proposes that this boat launch eventually be removed and access to the CMP be restricted.

Some stocking of lake trout in the west end of the conjoined pits (formerly the Buckeye Mine Pit) has occurred since 1999, and as a result lake trout have begun to populate waters in the eastern end of the CMP. Illegal stocking and/or unintended transport of other species may have also occurred.

According to the MDNR's sampling of the CMP, rainbow smelt are present in the pit. It is unknown if the effects of rainbow smelt on the fishery in this area are negative or positive.

The trophic state of the CMP is considered to be oligotrophic. As water that enters into the mine pit is mostly groundwater and there is relatively little surficial inflow to the pit, the quantity of nutrients and biota is resultingly low. This low amount of nutrients and biota results in a deficiency in the food chain within the pit with a resulting poor fishery and slow fish growth.

Some black crappie, bluegill, horneyhead chub, largemouth bass, pumpkinseed sunfish, painted turtle, rainbow trout, rock bass, snapping turtle, walleye, white sucker, and yellow perch have been found in the Canisteo and Buckeye Mine Pits by the MDNR. Bass appear to be in the pit, but they also grow slowly.

7.6.2.1.2 HAMP Complex

The HAMP Complex consists of four main mine pits: Arcturus, Gross-Marble, Hill-Trumbull, and Hill-Annex. Mining operations kept the pits completely dewatered until 1979. Following 1979, some dewatering took place and some of the pits began to fill with water. By 1981, all mining operations had ceased (Barr, 1987). Hill-Annex was established as a state park in 1988 by the Minnesota Legislature and is controlled by the MDNR Division of Parks and Recreation.

The Arcturus, Gross-Marble, and Hill-Trumbull/Hill-Annex were separated by large masses of rock during mining operations. Following the cessation of mining, water levels in the pits began to rise, and the Gross-Marble became connected to the Hill-Trumbull/Hill-Annex when the water surface elevation reached approximately 1,215 feet msl. The water surface in the Arcturus is higher than that of the other pits, and has not become connected by water to the other pits. Water currently overflows out of the Arcturus into the Gross-Marble. On November 1, 2005, the stage measured by the Applicant in the Gross-Marble, Hill-Trumbull/Hill-Annex Pit was 1,247 feet msl. At that same time, the stage measured in the Arcturus Pit was 1,269 feet msl.

Pumping records have been maintained by the MDNR since 1973, and the MDNR Hill-Annex staff continues to report dewatering volumes on a monthly basis.

Bathymetric data was collected by the Applicant in the fall of 2005, and a stage-storage relationship was developed for the Arcturus, Gross-Marble, and Hill-Trumbull/Hill-Annex pits from such data.

Stage data were collected by the MDNR from 1993 through 2002 for Hill-Annex, but because such data were not collected on a regular basis, it could not be used for a detailed yearly estimate of pit recharge. The stage in Gross-Marble, Hill-Trumbull/Hill-Annex was measured at 1,247 feet msl, and Arcturus was measured at 1,269 feet msl on November 1, 2005. The Applicant continues to measure stage at all of the pits within the HAMP Complex.

Actual recharge rates when the pits were dewatered from 1973-1979 were calculated based upon pumping records. Recharge rates during this period range from 3,230 gpm to 4,030 gpm.

Based on the stage-storage relationship, pumping records, and stage measurements, some long-term average recharge rates were calculated. Assuming that the pits were completely dewatered on January 1, 1979 and the Arcturus was completely full by 1999, an average recharge rate of 2,150 gpm was calculated using the stage-storage relationship, the stage measured on December 9, 1999, and historical pumping records.

A second long-term average recharge rate was calculated by adding the difference in volume in the pits between December 9, 1999 and November 1, 2005, and adding the volume of water pumped over this time period. The average recharge rate between the end of 1999 to 2005 was determined to be 1,590 gpm.

Uncertainties in the long-term average recharge rates calculated arise because of potentially missing data and pumping records, as well as the potential of highly variable groundwater head conditions. Recharge rates were calculated by the MDNR and independently by the Applicant's consultants. The HAMP Complex is not managed as a fishery and the MDNR has never stocked the pit. Sampling in 1990 failed to identify any game species in the mine pit, but some small species such as brook sticklebacks and common shiner were captured in minnow traps.

7.6.2.1.3 Lind Mine Pit

Very little historical water surface elevation and outflow data are available on the LMP. The pit has filled with water and has an outlet pipe that discharges to the Prairie River.

Bathymetric mapping of the pit has been developed by the Applicant based on electronic sampling of the mined surface through the water column.

Common shiner and black crappie were sampled by the MDNR in this pit. Black crappie appear to be naturally reproducing and the black crappie sampled appear to be near average with respect to growth rate. West Hill Mine Pit filled with water following the cessation of mining and currently discharges to the LMP through two 8 inch diameter HDPE pipes.

On November 2, 2005, the Applicant's consultants measured the pipe size, flow depth, and flow velocity at the LMP pipe outlet and determined the outflow from the pump at 1,800 gpm (4 cfs). A majority of the outflow appears to come from the West Hill Mine Pit. During the November 2, 2005 field trip, field personnel measured the pipe size, flow depth, and flow velocity at the West Hill Mine Pit pipe outlet and determined the outflow to be approximately 1,570 gpm (3.5 cfs). Follow-up flow measurements confirmed this flow rate.

7.6.2.1.4 Prairie River

The Applicant will also appropriate water from the Prairie River to supply and supplement the water resource needs of Mesaba One and Two. Applicant's consultants obtained river gauge data and conducted statistical analyses of appropriate data to assess the potential supply characteristics of the Prairie River. Average monthly flow rates are shown in Figure 7.6-2. MP reported an average flow of 27 cfs at the gauge station on August 29, 2005.

Gauge data have been collected intermittently at a USGS gauging station for a period of 16 years. The USGS gauge is located near the CR 7 crossing of the river, north of Taconite, Minnesota.

Gauge data have also been collected intermittently at the Prairie Lake Hydropower Dam, which is several miles downstream of the USGS gauge station. Flow data were collected from 1925 to 1957 on a monthly average basis while under the control and ownership of Blandin Paper Company. MP assumed control and ownership of the facility and collected flow data from 1997

to 2005 on a daily basis. Since the river flows are buffered by the lake and managed at the dam, the variability in the daily flow rates is not as extreme as at the USGS Prairie River gauge site.

Section 316(b) of the Clean Water Act ("CWA") contains criteria regarding Cooling Water Intake Structures (CWIS) that would apply to Prairie River appropriations. The rule specific to CWIS on fresh water rivers states that the maximum amount of water that can be taken is "5% of the mean annual flow or 25% of the 7Q10, whichever is the lesser."

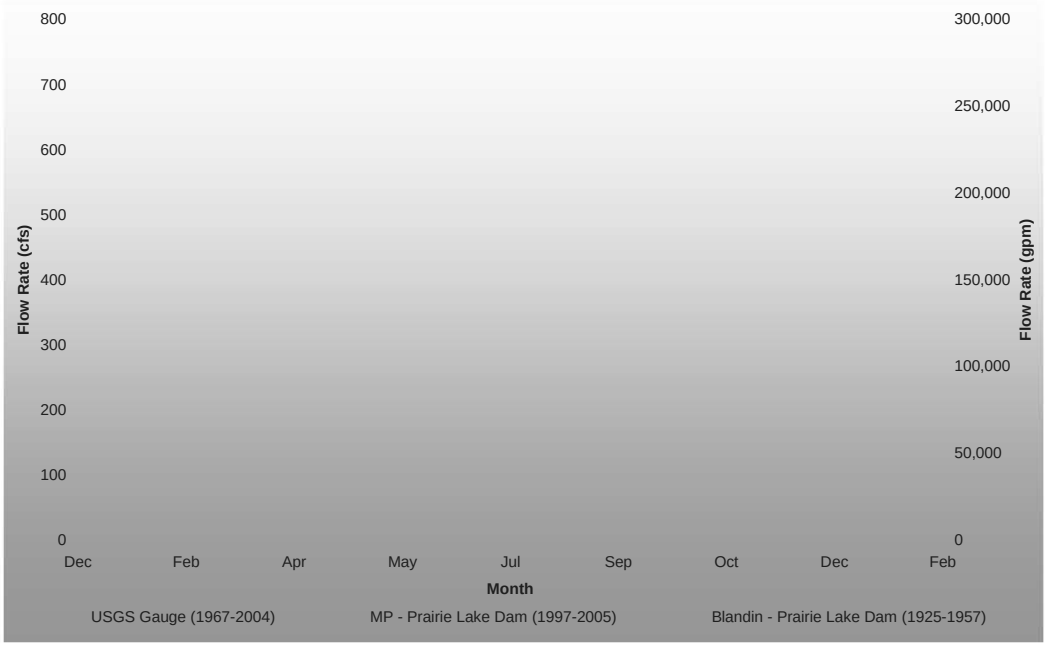
The 7Q10 is the seven day low flow average with a 10-year recurrence interval. The Weibull distribution[17] is the preferred statistical method used to determine the 7Q10, and requires that the top 80 percent of flow measurements be dropped as they are not considered to be true "low flows." The basis for the calculations used in determining the 7Q10 flow rate for the Prairie River is presented in Appendix F of the West Range IGCC Power Station Water Appropriation Permit Application filed with the MPCA and attached as Appendix 9. In general, river flows are plotted (on a log scale) against reoccurrence interval (on a normal scale) and an exponential regression is used to best fit a regression line to the data points. The point on the graph at which the best fit line intersects the 10-year recurrence interval is defined as the 7Q10.

To be conservative, only the data collected by Minnesota Power at the Prairie Lake Dam from 1998 to 2004 were utilized in the determination of the mean annual flow and the 7Q10 determination. Since full year records for 1997 and 2005 were not available, such data were eliminated from consideration. The Blandin data from 1925-1957 were recorded on a monthly average basis and could not be used to determine the 7Q10. Data from the USGS gauge were also not used because the point of taking is several miles downstream of the USGS gauging station and Prairie Lake.

The mean annual flow in the Prairie River is 319 cfs, and 5% of that flow is equal to 16 cfs. The 7Q10 in the Prairie River was determined to be 22 cfs, and 25% of that flow is equal to 5.5 cfs. Based upon Applicant's calculations and the data available for such calculations, with 25% of the 7Q10 being the smaller amount, the maximum rate at which water can be appropriated from the Prairie River at one time pursuant to Section 316(b) is 2,468 gpm (5.5 cfs). The Applicant will thus limit its Prairie River appropriations to this amount unless subsequent observations and determinations permit greater usage.

[17] Weibull, Wallodi, "A Statistical Distribution Function of Wide Applicability," Journal of Applied Mechanics, Vol. 18, pp. 293-297, 1951.

Figure 7.6-1 Average Monthly Flow Rates for Prairie River



7.6.3 West Range Water Management Plan: Modeled Water Level Impacts

The operation of water infrastructure for Mesaba One and Two and its impacts on water levels in the CMP have been modeled. The description of the model utilized is found in Section 4.3 of the Water Appropriation Permit Application filed with the MDNR. Modeling results indicate that water levels in the CMP could fluctuate up to two feet during a year with average rainfall. Under drought conditions, water levels in the CMP could fluctuate up to six feet. Figure 7.6-2 shows the average and drought-case scenario in the CMP during one years' operation with Mesaba One and Two in service. Figure 7.6-3 shows the impact of operating Mesaba One and Two on water levels in the CMP when drought conditions extend to five years. In this instance, CMP water levels are predicted to be lowered by up to 30 feet.

Based on the model runs conducted and other engineering considerations, the Applicant proposes to operate the CMP within a range of 1,260-1,290 feet msl, with a contingency plan range of 1,250-1,260 feet msl. The contingency plan range is proposed to accommodate the five-year drought scenario. The Applicant will operate within the 1,290-1,300 feet msl range during extremely wet periods. In the event water levels in the CMP cannot be controlled (that is, continue to rise) through water withdrawals required for operating Mesaba One and/or Mesaba One and Two, a cross-tie into the Holman Lake discharge pipe will allow excess CMP waters to be pumped to Holman Lake on an as needed basis. The cross-tie will contain sufficient protection to ensure that unwanted species are not inadvertently directed into Holman Lake. The Applicant and/or the MDNR through an approved mechanism derived during the permitting process will also have the capability to operate the existing pump in the Hill-Annex Mine Pit to manage water levels in that complex during overly wet periods.

Figure 7.6-2 Modeled Annual Variation in CMP Water Levels

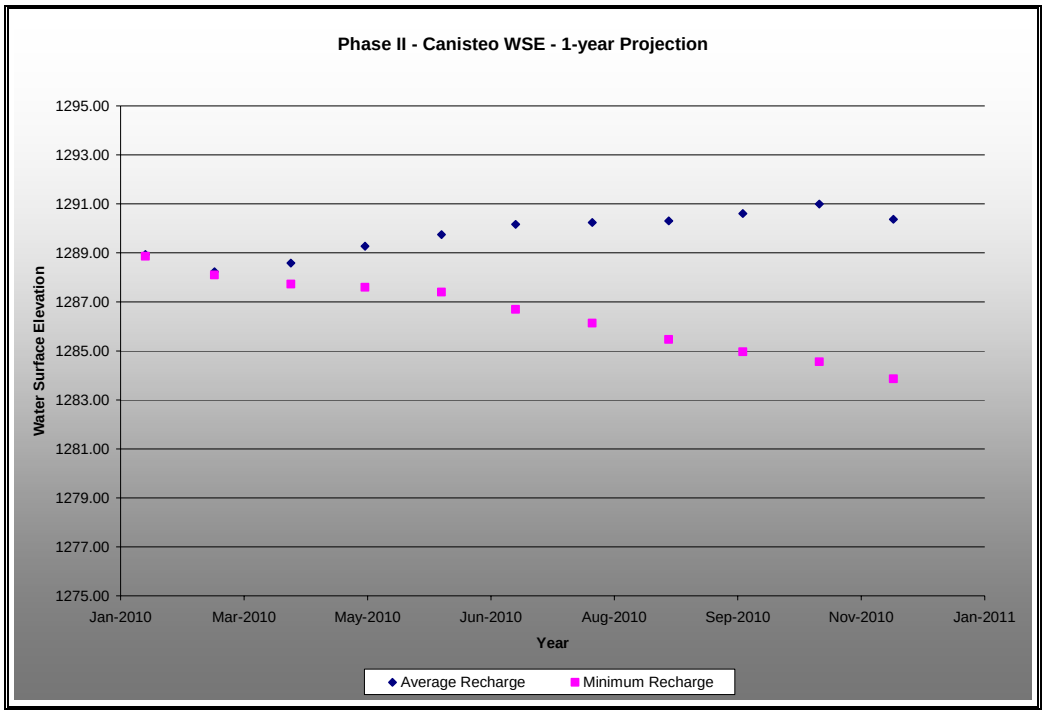
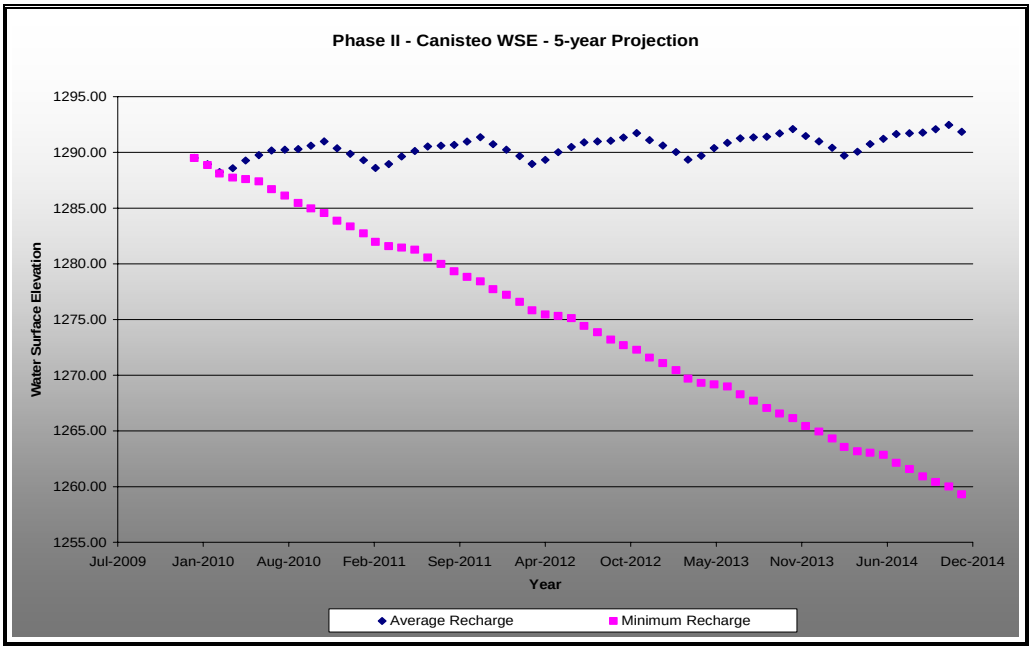


Figure 7.6-3 Modeled 5-Year Variation in CMP Water Levels



7.6.3.1 Water levels and Water Balance During Operation of Phase I and Phase II

The CMP contains some land bridges that are below a water surface elevation of approximately 1,260 feet msl. The intended operation of the CMP will be to maintain water levels above 1,260 feet msl, unless such other levels are otherwise necessary or required.

The pit water surface elevation will be 1,290 ± 2 feet msl during a typical year. Water from the other pits will augment water levels in the CMP, and should help to prevent significant water level changes.

The GMMP will typically be operated in the range of 1,220-1,230 feet msl. Significantly higher flows are believed to be available if the water level in the HAMP is reduced below the now-submerged land bridge located between the GMMP and the HAMP. Discussions will be required between the Applicant and the MDNR to determine whether operation at greatly reduced water levels in the HAMP is advisable and, if so, under what conditions such operation would be desirable.

The LMP will be operated in the range of 1,190-1,250 feet msl during a typical year. The operating ranges in the GMMP and LMP will allow for storage of water during non-pumping periods. Pumping is unlikely to occur during the winter unless there is equipment failure or system maintenance needs.

Within the context of the permitting process, the Applicant will create a monitoring plan to record levels within the mine pits from which water supplies for the IGCC Power Station will be derived, levels within the receiving waters to which cooling tower blowdown will be discharged, and the pumping rates at which waters are transferred.

7.6.4 Water Permits

7.6.4.1 Existing Permits

The Minnesota DNR currently holds a Minnesota DNR Water Appropriations Permit (Permit #042088) and a MPCA NPDES/SDS Permit (Permit #MN00 30198) for the withdrawal and discharge of water for the existing Hill-Annex State Park dewatering operation. The on-going data collection and cooperative study of the mine pit by the Applicant and the MDNR will be covered under the existing permits.

7.6.4.2 New Permits

7.6.4.2.1 Minnesota DNR Water Appropriation Permit

A MDNR Water Appropriation Permit for Non-Irrigation (FORM #A-02623-06) is required for appropriations from the CMP, Hill-Annex Mine Pit, Lind Pit and the Prairie River. A separate permit application will be submitted for each water source with a request that one permit be issued for appropriation from all such sources.

An annual Water Use Report is required by the MDNR for all Water Appropriations Permits.

7.6.4.2.2 Minnesota DNR Public Waters Work Permits

A MDNR Public Waters Work Permit (FORM #NA-026620-03B) is required for temporary and permanent impacts to Public Waters.

The CMP and the Hill-Annex Mine Pit are Waters of the State, but are not classified by the MDNR as public waters. Since they are not public waters, a MDNR Public Waters Work Permit is not required for work that is done below the Ordinary High Water Level (OHWL) within these waterbodies.

A MDNR Public Waters Work Permit is required for work that takes place in identified public waters (see Table 2.5-1), and stream crossings must be reviewed and approved for any proposed hydraulic changes to the stream.

The following proposed activities will require coverage under a MDNR Public Waters Work Permit:

- Gas line crossing of the Swan River (2 locations)
- HVTL crossing of the Swan River (2 locations)
- HVTL crossing of the Lower Panasa Lake Outlet
- HVTL crossing of Snowball Creek
- HVTL crossing of Oxhide Creek
- HVTL crossing of Oxhide Lake
- HVTL crossing of Big Diamond Lake Outlet
- Process water orifice at the Prairie River

More detailed discussions of these water crossings are provided in the ES.

7.6.4.3 NPDES Construction Storm Water Permitting

This permit is required for storm water discharges associated with construction activity.

The NPDES Construction Permit requires a Storm Water Pollution Prevention Plan (SWPPP) to address erosion and sediment control during and after construction

Best management practices (BMPs) will be followed in accordance with the NPDES Permit and MPCA BMP Manual, 2000. Temporary and permanent erosion control features include timely re-vegetation of disturbed areas, silt fence, inlet protection, ditch checks, and sedimentation ponds.

The SWPPP will address erosion prevention measures, sediment control measures, permanent storm water management, dewatering, environmental inspection and maintenance, and final stabilization.

The project will create more than one acre of new impervious surfaces, and therefore a permanent storm water management system is required under the NPDES permit. The permanent

storm water management system must provide water quality treatment for ½ inch of runoff from the new impervious surfaces before discharge to surface waters. This treatment may be obtained by construction of wet sedimentation basins, infiltration/filtration, regional ponds, or a combination of practices. Design criteria for wet sedimentation basins can be found in the MPCA NPDES General Permit for Construction Activities.

7.6.4.4 Process Water Discharge Permit

Process water discharges of cooling tower blowdown are discussed in detail in the NPDES Permit Application and in Section 3.4.2 of this Application. Potential impacts resulting from the proposed four process water outfalls are summarized in this section. Figure 3.4-12 shows the location of each of the four outfalls receiving process water discharges. The outfalls associated with discharges of stormwater are also shown on this figure.

7.6.4.4.1 Surface Water Quality Standards

The key water quality constituents associated with Outfall 001 and 002 discharges will be mercury, total dissolved solids (TDS), and hardness. Mercury will be addressed by operating the IGCC Power Station such that the concentration of mercury in its effluent discharges will not exceed the water quality standard of 6.9 ng/L. TDS and hardness discharge concentrations will be acceptable with the use of an approved mixing zone.

7.6.4.4.2 Impaired Waters

Holman Lake, Panasa Lake, the CMP and the HAMP Complex are not impaired waters. However, the water from those waterbodies, either now or in the future, will ultimately discharge into the Swan River, which is impaired for mercury. The Clean Water Act requires states to publish every two years an updated list of streams and lakes that are not meeting their designated uses because of excess pollutants. The list, known as the 303(d) list, is based on violations of water quality standards and is organized by river basin. The most recent draft of Minnesota's list of impaired waters was placed on the MPCA's web site on December 19, 2005 (MPCA, 2005b). The draft list indicates that the entire length of the Swan River from Swan Lake to the Mississippi River is impaired for dissolved oxygen ("DO") and mercury. See http://www.pca.state.mn.us/water/tmdl/index.html.

The Swan River flows into a reach of the Mississippi River between Swan River and Sandy River, which reach is also impaired for mercury. Other reaches further downstream on the Mississippi are impaired for:

- Mercury
- Fecal Coliform
- PCBs
- Low DO (excess nutrients, primarily phosphorus)
- Turbidity

Project waste streams consist of cooling tower blowdown, HRSG blowdown, boiler feedwater demineralizers and other minor sources. All other contact process water will be managed in the

ZLD System. The Applicant is proposing to eliminate discharges of nutrients and fecal coliform to the Swan River (a water impaired for DO and a tributary to a water impaired for PCBs) by discharging its domestic wastewater to the Coleraine-Bovey-Taconite municipal wastewater treatment facility.

Concerns over the environmental effects of PCBs led to a North American ban in 1977 on their manufacture, importation and most non-electrical uses, and also to restrictions on their use in existing electrical and mechanical equipment (Health Canada, 1998[18]). Effluents from Mesaba One and Mesaba Two will not contain PCBs.

Phosphorus concentrations in recent samples collected from proposed source waters (CMP, HAMP Complex and the LMP) have been shown to be below 0.1 mg/L. While there is currently no water quality standard for phosphorus, the MPCA has established a discharge standard of 1.0 mg/L that is applied at end-of-pipe discharges. However, even though such a discharge may meet the discharge standard of 1 mg/L, because it is upstream of an impaired body of water, no additional contribution of phosphorus is permitted.

As noted in Section 3.4.2.2.2, the Applicant has taken steps to eliminate the Station's use of phosphorus-containing chemicals that might otherwise cause the discharge of blowdown to cause or contribute to a violation of water quality standards in waters impaired for DO. As well, the Applicant will avoid discharges of turbidity through use of a contained conveyance to Holman Lake and a well designed discharge structure. Both of these design features will avoid the possibility of scouring sediment from wetlands known to have been receiving waters for direct discharges of domestic wastewaters and/or natural swales.

The proposed operation of the IGCC Power Station will result in no increase in the mass of mercury or phosphorus over that currently permitted from the HAMP Complex under NPDES Discharge Permit MN0030198. The MDNR also holds a water use permit, No. 510144 for appropriating water from the Hill-Annex Mine Pit. General permit information is summarized in Table 7.6-2. The MDNR has been pumping water out of the Hill-Annex Mine Pit since 1989 to control water levels in the pit and has discharged the water into Panasa Lake and ultimately to the Swan River.[19] Prior to 1989, the HAMP Complex was pumped to allow mining activities.

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[18] Health Canada 1998. The Health and Environment Handbook for Health Professionals. http://www.hcsc.gc.ca/ehp/ehd/catalogue/bch_pubs/98ehd211/98ehd211.htm

[19] Discharges of Canisteo Mine Pit water to the Swan River watershed have also occurred during past mining operations. NPDES permits for those discharges are available in MPCA files but detailed records of actual pumping activities are limited.

Table 7.6-2
Summary of Hill-Annex Mine Pit NPDES and Appropriations Permits

Permit Number	Date Issued	Expiration Date	Permit Holder	Average Discharge Rate (MGD/ gpm)	Maximum Discharge Rate (MGD/ gpm)	Annual Average Discharge Volume (acre-feet)	Receiving Water Body
NPDES Permit							
0030198	June 3, 2003	May 31, 2008	MDNR	4.5/3,125	9.0/6,250	--	Panasa Lake
Appropriations Permit							
510144	Not available	NA	MDNR	10.08/7,000	--.	10,485	--

Based on the permitted average discharge rate from the existing NPDES permit, and an assumed mine pit water concentration based on the analytical results from the HAMP Complex, the mass of a constituent permitted to be discharged to the Swan River watershed under the existing HAMP Complex pumping permit was estimated. The estimated mass of mercury and phosphorus permitted annually is shown in Table 7.6-3.

Table 7.6-3
Estimated Annual Mass Permitted to the Swan River Watershed From the Hill-Annex Mine Pit

Constituent	Estimated Concentration	Permitted Average Annual Discharge Rate	Permitted Annual Mass Discharge
Mercury	0.9 ng/L	3,125 gpm	5.6 g
Phosphorus	0.1 mg/L		621 kg

The Applicant will operate Mesaba One and Mesaba One and Two such that the actual mass of mercury and phosphorus discharged to the Swan River will be equal to or less than that currently allowed under the existing NPDES permit. The mass discharged will be the sum of each constituent associated with:

1. Water discharged into Holman Lake at Outfall 002 from the IGCC Power Station or the CMP (The mercury and phosphorus contained in the minor volume water streams that ultimately flow to the ZLD system are expected to be small and need not be considered in water discharge mass balance calculations. Similarly, mercury volatization in the cooling towers and elsewhere in the process is expected to be negligible and need not be considered in this calculation.).

2. Water pumped to Panasa Lake from the Hill-Annex Complex Mine Pits for water level control permitted under existing NPDES Permit MN0030198.

Mass balance calculations, based on expected source water quality, expected IGCC Power Station operation, and assumed HAMP Complex water level management pumping rates show that mercury and phosphorus discharged from Outfall 002 and the existing Panasa Lake outfall will be maintained at annual quantities less than that allowed under the current permit. The mass balance model and the results of its use associated with Mesaba One and Two are reported in Appendix D of the Applicant's NPDES Permit Application for the West Range IGCC Power Station attached as Appendix 6.

7.6.4.4.3 Cooling Water Intake Structures (Clean Water Act § 316(b))

See Section 3.6.2.1.1 for a discussion of rules applicable to Mesaba One and Mesaba Two.

7.6.5 Domestic Wastewater Discharge

The proposed approach for treating domestic wastewaters is to connect to the Coleraine-Bovey-Taconite wastewater collection and treatment system because it avoids discharging nutrients to Holman Lake, a tributary to the Swan River (which in turn is a tributary to the Mississippi River), a receiving water that is impaired for DO.

7.6.5.1 Construction Period

About 1,500 construction personnel are expected to be on site during the periods of peak construction activity associated with Mesaba One and Mesaba Two. Assuming each worker generates an average of 20 gallons of wastewater per day, the estimated peak wastewater flows from the West Range Site during construction would be about 30,000 gallons per day (gpd). Construction activities associated with Mesaba One are projected to take approximately 44 months.

Construction of Mesaba Two would overlap activities associated with construction of Mesaba One. Mesaba Two is expected to be complete about 24 months after Mesaba One begins commercial operation. Peak construction periods associated with each phase are expected to be staggered in such a way that peak construction activities for Mesaba One would not overlap with those for Mesaba Two.

7.6.5.2 Operation

At the conclusion of construction, the number of permanent personnel at the Phase I IGCC Power Station would number about 110 people. The volume of wastewater generated by each person during operation versus that during construction is expected to be higher due to the use of showers and locker room facilities. During normal plant operation, each person will generate about 30 gallons of wastewater per day and the amount of domestic wastewater generated per day would total about 3,300 gpd. This total does not reflect the likelihood that the number of people on site at any one time will be greater than the operations staff due to the presence of equipment contractors, outage crews, and visitors.

The operational work force for Mesaba Two will number about 75 individuals (that is, somewhat fewer than that required for Mesaba One due to the overlap with Mesaba Two of managerial responsibilities, supervisory and administrative personnel responsibilities, and some

maintenance). The overall operational work force for Mesaba One and Two is expected to total about 185 people. Using the 30 gpd wastewater generation rate identified above, the expected amount of wastewater from Mesaba One and Two would total 5,600 gpd.

To accommodate the additional flows as a result of the added number of people on site during tours, special maintenance/construction activities, and outages, the capacity of the system has been conservatively set at 7,500 gpd. This capacity provides the capability to accommodate about 140 additional individuals on-site during operation of Mesaba One and about 80 individuals during operation of Mesaba One and Two. This flow is based on Mesaba One and Two having restrooms, locker rooms, showers, and break room facilities.

7.6.5.3 Adequacy of Taconite-Bovey-Coleraine WWTF

The cities of Taconite, Bovey and Coleraine have a joint wastewater commission that manages the wastewater treatment facility (WWTF) located in Coleraine, approximately 4 miles southwest of the West Range Site. The WWTF receives wastewater from the three cities and discharges treated effluent to the Swan River. The WWTF has a design capacity of 499,000 gpd and received an average flow of 334,000 gpd during the period from January 1 through May 31, 2005. During the wettest 30-day period, the WWTF received an average of 444,000 gpd with a peak day of 969,000 gpd.

An issue relating to Taconite's collection system is the amount of inflow and infiltration (I/I) entering the system during periods of rainfall or high groundwater. At such times, excess flow can exceed the capacity of the main wastewater pump station in Taconite, creating a need to bypass untreated wastewater into a natural pond system. Larger pumps could be installed in the pump station to remedy this problem or the City's collection system could be rehabilitated to prevent extraneous water from entering the sewers.

7.6.6 Water Crossings

Lakes and streams in the vicinity of the West Range Site are described in Section 7.6.1 above. Utility crossings over, under, or through waterbodies listed as protected waters or wetlands on the MDNR Protected Waters Inventory ("PWI") will require Licenses for Utility Crossings of Public Lands and Waters under Minn. Stat. § 84.415 and Minnesota Rules Chapter 6135. The MDNR Division of Land and Minerals is the administrative agency issuing such licenses, which may be renewed at the end of the licensing period. Protected waters or wetlands that will be crossed by HVTLs, gas pipelines, and process water blowdown pipelines and which require licenses for such crossings, are identified in the following subsections.

Water crossings associated with the development of Mesaba One and Mesaba Two are limited to the corridors associated with the HVTLs, gas pipeline, and one process water blowdown pipeline. There are no water crossings associated with siting, placement, or construction on the IGCC Power Station Footprint or on Buffer Land, the railroad alternatives, sewer and water lines, and roads. The following subsections describe the water crossings within the HVTLs, gas pipelines, water supply, and process water discharge lines.

7.6.6.1 Preferred HVTL Route

7.6.6.1.1 Preferred Double Circuit Corridor

There are a total of two river or stream crossings associated with this route. These crossings are over the Swan River (perennial) and a perennial stream between Big and Little Diamond Lakes. The perennial stream between Big and Little Diamond Lakes was the only water crossing along this alternative that was field surveyed during the 2005 field season due to access limitations. The Swan River is identified as a protected water by the MDNR Protected Waters Inventory (PWI) and requires a license to cross this waterbody for this HVTL.

Wetland habitat associated with the water crossings is based on NWI classification and mapping. In areas where 2005 field surveys were conducted, the classification given is based on observations made during the field surveys. The wetland habitat for the Swan River crossing is mapped by NWI as Type 6 (PSS/EM5C) scrub-shrub habitat. The perennial stream between Big and Little Diamond Lake was mapped during the 2005 field surveys and included Type 3 (PEMC) shallow marsh habitat. Total length of water crossings for this alternative is estimated at 123 linear feet. The location and wetland types associated with the water crossings for the preferred HVTL are summarized in the following Table 7.6-4.

Table 7.6-4
Water Crossings for the Preferred West Range HVTL

Stream Crossing Location	MDNR PWI?	Milepost (mile + linear feet)	Length of Waterbody Crossing	Adjacent Wetland Types	
				Cowardin	Circular 39
Perennial stream between Big & Little Diamond Lakes (Basin E1)*	Yes	0+3980	3 linear feet	PEMC	Type 3
Swan River (perennial)	No	3+1630	120 linear feet	PSS/EM5C	Type 6
Total: 123 linear feet					

* = This information has been field verified.

7.6.6.2 Alternate HVTL Route

As described Sections 2 and 4, an alternative route for the Preferred 345kV Double Circuit HVTL Route serving both Phases I and II was proposed for consideration. For this route, there are a total of six river or stream crossings. Five of these crossings are over the Swan River (perennial) and one crossing is over a perennial stream between Big and Little Diamond Lakes. The perennial stream between Big and Little Diamond Lakes was the only water crossing in this alternative that was field surveyed during the 2005 field season due to access limitations. The Swan River is identified as a protected water by the MDNR Protected Waters Inventory (PWI) and would require a license to cross this waterbody.

The water crossings and wetland impacts for this alternative is based on NWI classification and mapping. In areas where 2005 field surveys were conducted, the classification given is based on observations made during the field surveys. The wetland habitat for one of the Swan River crossings is mapped by NWI as Type 6 (PSS/EM5C) scrub-shrub habitat. The other Swan River crossings have no wetland habitats mapped by the NWI, and no classification is given for the streambed in these areas. The perennial stream between Big and Little Diamond Lake was mapped during the 2005 field surveys and included Type 3 (PEMC) shallow marsh habitat. Total length of water crossings for this alternative is estimated at 533 linear feet. The location and wetland types associated with the water crossings for HVTL Alternative are summarized in the following table. Table 7.6-5 details locations of water crossings associated with the HVTL alternatives.

Table 7.6-5
Water Crossings for West Range Preferred Alternate HVTL

Stream Crossing Location	MDNR PWI?	Milepost (mile + linear feet)	Length of Waterbody Crossing	Adjacent Wetland Types	
				Cowardin	Circular 39
Perennial stream between Big & Little Diamond Lakes (Basin E1)*	No	0+3980	3 linear feet	PEMC	Type 3
Swan River (perennial)	Yes	3+1700	60 linear feet	PSS/EM5C	Type 6
Swan River (perennial)	Yes	3+2960	60 linear feet	No classification given by NWI	No classification given by NWI
Swan River (perennial)	Yes	3+3575	50 linear feet	No classification given by NWI	No classification given by NWI
Swan River (perennial)	Yes	3+4400	270 linear feet	No classification given by NWI	No classification given by NWI
Swan River (perennial)	Yes	4+360	90 linear feet	No classification given by NWI	No classification given by NWI
Total: 533 linear feet					

* = This information has been field verified.

7.6.6.2.1 Plan B Phase II Alternative 345kV Route

This route alternative utilizes 18 miles of existing MP ROW. There are a total of five water crossings associated with this more easterly 18-mile Phase II alternative. Two crossings are under the Swan River (perennial) and one of its perennial tributaries. The other three crossings are associated with Snowball and Oxhide Creeks (both perennial), and Oxhide Lake. The Swan River and its tributary, Snowball Creek, and Oxhide Lake are identified as protected waters by the MDNR Protected Water Inventory (PWI), and would require a license to cross these waterbodies for the HVTL at this location. Lakes and wetlands designated as MDNR Protected

Waters or Wetlands receive a unique identification number, but streams and rivers do not. In this case, the PWI identification number for Oxhide Lake is 106P.

Wetland habitat associated with the water crossings for this HVTL is based on NWI classification and mapping. The wetland habitat for the Swan River crossing is mapped by NWI as Type 3 (PEM5C) shallow marsh habitat. The tributary of the Swan River tributary, which is the outlet of Lower Panasa Lake, and Oxhide Creek have no wetland habitats mapped by the NWI, and no classification is given for the streambed in this area. Snowball Creek is mapped by NWI as Type 2 (PEM5B) wet meadow habitat. Oxhide Lake is identified as Type 5 (L1UBH) deep water habitat and Type 6 (PSS1/EM5C) scrub-shrub habitat. Total length of water crossings for this alternative is estimated at 283 linear feet. The location and wetland types associated with the water crossings for this alternative Plan B Phase II route are summarized in the following table.

Table 7.6-6
Water Crossings for West Range Alternative 345kV HVTL (East Corridor)

Stream Crossing Location	MDNR PWI?	Milepost (mile + linear feet)	Length of Waterbody Crossing	Adjacent Wetland Types	
				Cowardin	Circular 39
Swan River (perennial)	Yes	14+0	190 linear feet	PEM5C	Type 3
Tributary of Swan River, outlet of Lower Panasa Lake (perennial)	Yes	12+4640	3 linear feet	No classification given by NWI	No classification given by NWI
Snowball Creek (perennial)	Yes	11	10 linear feet	PEM5B	Type 2
Oxhide Lake	Yes (PWI 106P)	8+2220	70 linear feet	L1UBH, PSS1/EM5C	Type 5, Type 6
Oxhide Creek (perennial)	Yes	9+2880	10 linear feet	No classification given by NWI	No classification given by NWI
Total: 283 linear feet					

7.6.6.3 Proposed Natural Gas Pipeline Route

There are a total of four river or stream crossings associated with Natural Gas Pipeline Alternative 1. Two of these crossings are under the Swan River (perennial). The other crossings are under a tributary of the Swan River (perennial) and a perennial stream between Big and Little Diamond Lakes. The perennial stream between Big and Little Diamond Lakes was the only water crossing in this alternative that was field surveyed during the 2005 field season due to access limitations. The Swan River is the only waterbody identified as a protected water by the

MDNR Protected Waters Inventory (PWI), and therefore would require a License to cross this waterbody for Gas Pipeline 1.

Wetland habitat associated with the water crossings for Natural Gas Pipeline Alternative 1 is based on NWI classification and mapping. In areas where 2005 field surveys were conducted, the classification given is based on observations made during the field surveys. The wetland habitat for the two Swan River crossings is mapped by NWI as Type 1 (PFO1A) seasonally flooded and Type 6 (PSS/EM5C) scrub-shrub habitats. The wetland habitat at the tributary to the Swan River is mapped by NWI as Type 2 (PEM5Bd) wet meadow habitat. The perennial stream between Big and Little Diamond Lake was mapped during the 2005 field surveys and included Type 3 (PEMC) shallow marsh habitat. Total length of water crossings for this alternative is estimated at 133 linear feet. The location and wetland types associated with the water crossings for Natural Gas Pipeline Alternative 1 are summarized in Table 7.6-7.

Table 7.6-7
Water Crossings for West Range Gas Pipeline

Stream Crossing Location	MDNR PWI?	Milepost (mile + linear feet)	Length of Waterbody Crossing	Adjacent Wetland Types	
				Cowardin	Circular 39
Swan River (perennial)	Yes	4+2170	60 linear feet	PFO1A	Type 1
Tributary of Swan River (perennial)	No	5+1460	10 linear feet	PEM5Bd	Type 2
Swan River (perennial)	Yes	9+4560	60 linear feet	PSS/EM5C	Type 6
Perennial stream between Big and Little Diamond Lakes (Basin E1)*	No	12+2000	3 linear feet	PEMC	Type 3
Total: 51 linear feet					

* = This information has been field verified.

7.6.6.4 Process Water Supply Pipeline

7.6.6.4.1 Segment 1 – Lind Pit to Canisteo Pit

There are no water crossings associated with Process Water Supply Pipeline Segment l.

7.6.6.4.2 Segment 2 – Canisteo Pit to IGCC Power Station

There are no water crossings associated with Process Water Supply Pipeline Segment 2.

7.6.6.4.3 Segment 3 – Gross-Marble Pit to Canisteo Pit

There are no water crossings associated with Process Water Supply Pipeline 3 alternative.

7.6.6.5 Process Water Blowdown Pipelines

7.6.6.5.1 Process Water Blowdown Pipeline 1 (West Range Site to Holman Lake)

There are two stream crossings associated with the Process Water Blowdown Pipeline 1. Both crossings are under perennial streams, one which drains from Little Diamond Lake, and the other draining to Holman Lake. Neither stream was field surveyed during the 2005 field season due to access limitations. The National Wetlands Inventory is the basis for evaluating wetlands associated with the stream crossings.

The perennial stream draining from Little Diamond Lake has no wetland habitats mapped by the NWI, and no classification is given for the streambed. The perennial stream draining to Holman Lake is mapped by NWI as Type 3 (PEM/UBF) shallow marsh and Type 6 (PSS1/EM5C) scrub-shrub habitat. Both crossings are approximately three feet in length. The location and wetland types associated with the stream crossings for Blowdown Pipeline 1 are summarized in the following table. Table 7.6-8 details locations of water crossings associated with the water process line.

Table 7.6-8
Water Crossings for West Range Process Water Blowdown Pipeline 1

Stream Crossing Location	MDNR PWI?	Milepost	Length of Waterbody Crossing	Adjacent Wetland Types	
				Cowardin	Circular 39
Perennial stream from Little Diamond Lake	Yes	1+3990	3 linear feet	No classification given by NWI	No classification given by NWI
Perennial stream, drains to Holman Lake	Yes	2+2280	3 linear feet	PEM/UBF, PSS1/EM5C	Type 3, Type 6
Total: 48 linear feet					

* = This information has been field verified.

7.6.6.6 Process Water Blowdown Pipeline 2 (West Range Site to Canisteo Pit)

There are no water crossings associated with the Process Water Blowdown Pipeline 2 (West Range Site to Canisteo Pit).

7.7 WETLANDS

A detailed summary of wetlands in the project site and associated utility corridors is provided in the ES (Sections 2 and 3). This section summarizes wetland impacts for the site and each

alternate utility corridor associated with the West Range Site. Table 7.7-1 below summarizes the expected acres of wetlands impacted.

7.7.1 Overview of Impacts and Contact with Agencies

The following subsections describe effects on wetlands due to construction and operation activities, particularly where impacts may be minimized or avoided due to construction practices, or where temporary impacts may be restored. Under Minnesota law, and through a memorandum of understanding between the Minnesota Board of Water and Soil Resources (BWSR) and the U.S. Army Corps of Engineers (USACE) – St. Paul District, wetland impacts are generally evaluated on a per acre basis, without regard to wetland type being affected when the Minnesota Wetland Conservation Act (WCA) *de minimis* thresholds have been exceeded (Minn. Stat. § 103G.2241, subd. 9). An exception to this rule is for wetlands that may have particular ecological uniqueness or protection status (for example, calcareous fens) or are otherwise legally protected under other state and/or federal law (for example, wetlands in state Scientific and Natural Areas, state-designated trout waters, Outstanding Resource Value Waters, etc.). Higher replacement ratios are sometimes utilized when regulatory agencies determine that impacted wetlands have a higher value relative to other wetland types (e.g., impacts to tamarack bogs may be regulated at higher levels than impacts to a disturbed, urbanized wetland).

Table 7.7-1
Summary of Total Temporary and Permanent ROW Wetland Impacts for West Range Site and Associated Utility and Transportation Corridors

Project Alternative	Total Wetland Impacts (Acres)	
	Temporary ROW	Permanent ROW
IGCC Facility	n/a [1]	30.95
HVTL Preferred	n/a [1]	0.01 [2]
HVTL Alternative	n/a [1]	0.01 [2]
HVTL Plan B Alternative	n/a [1]	0.03 [2]
Gas Pipeline 1	24.69	17.47
Process Water Blowdown Pipeline 1 (IGCC Facility to Holman Lake)	5.86	4.07
Process Water Blowdown Pipeline 2 (IGCC Facility to Canisteo Pit)	20.38	13.60
Process Water Supply Pipeline Segment 1 (Lind Pit to Canisteo Pit)	0	0
Process Water Supply Pipeline Segment 2 (Canisteo Pit to West Range Site)	5.48	3.73

Project Alternative	Total Wetland Impacts (Acres)	
	Temporary ROW	Permanent ROW
Process Water Supply Pipeline Segment 3 (Gross-Marble Pit to Canisteo Pit)	6.17	3.79
Railroad Alternative 1A and Center Loop	26.45 [3]	77.08
Railroad Alternative 1B and Center Loop	18.11 [3]	64.23
Potable Water and Sewer Pipelines	4.48	1.79
Access Road 1	9.72	5.67
Subtotal[4]	**103.2**	**158.2**
Total Permanent Impacts (including Railroad construction limit impacts)	**n/a**	**172.4 acres**

[1] Temporary construction areas for the IGCC or temporary ROW for the HVTL corridors are not defined for the project area, therefore temporary wetland impacts are not anticipated for these project alternatives.
[2] Permanent impacts in the permanent ROW for HVTL is limited to placement of new power poles.
[3] Impacts in Railroad temporary ROW are permanent impacts due to grading in the construction limits, which should be included with total permanent wetland impacts for mitigation purposes.
[4]Total wetland impacts assuming West Range Proposed Natural Gas Pipeline Route, Railroad Alternative 1A and Center Loop. The Applicant will seek to avoid wetland impacts within the railroad center loop, however, including it here represents a worst case analysis.

Special or protected wetlands are not known to occur within the IGCC Power Station Footprint, Buffer Land, Associated Facilities or their Interconnection Corridors. However, to some extent, areas of tamarack and spruce bogs are located within each of these areas on the Site. No wetland type is anticipated to require higher mitigation requirements over any other type (e.g., Type 7 forested wetlands will not require higher mitigation requirements than Type 6 scrub-shrub or Type 3 emergent wetlands). However, these mitigation requirements will be negotiated during the wetland permitting phase of the project. Summaries of wetland impacts by wetland types are provided in detail in Section 3.1 of the ES for the West Range IGCC Power Station Footprint, Buffer Land, Associated Facilities and their Interconnection Corridors.

7.7.2 IGCC Power Station Footprint and Buffer Land

Minimization of wetland impacts has been addressed by adjusting the IGCC Power Station Footprint to straddle the two large wetland complexes (A1 and A4) located within the property on which the Applicant has obtained an option. However, the mitigation of wetland impacts will of necessity result in more permanent impacts to forest land and disturbances related to blasting. Some movement of the Station Footprint is to be expected as the FEED moves forward into a final design evaluation and negotiations regarding wetland impacts are undertaken between the Applicant and the agencies having jurisdiction over the particular wetlands affected.

Siting the Station Footprint was largely driven by establishing an acceptable grade for and length of the railroad trackage (that is, grades that will accommodate unit coal train deliveries, allow for automatic unloading via a rotary dumper system, and a length that will avoid interferences with other railroad traffic occurring on the main line) and avoid extensive cut/fill activity.

Permanent wetland impacts are estimated at 30.95 acres for the IGCC Power Station Footprint with wetland impacts for Mesaba One and Mesaba Two estimated at 17.33 acres and 13.62 acres, respectively. These wetlands are primarily Type 3/7 or Type 7 basins, many of which appear to be isolated and/or ephemeral type basins. Figure 7.7-1 shows the occurrence of wetlands on the IGCC Power Station Footprint.

The greatest amount of impacts from the IGCC Power Station Footprint and Buffer Land would be to Wetlands A1 (Type 3/6/8) and A4 (Type 7), which are larger wetland complexes that extend beyond the Station Footprint and Buffer Land. Figure 7.7-2 shows the occurrence of wetlands on the IGCC Power Station Footprint.

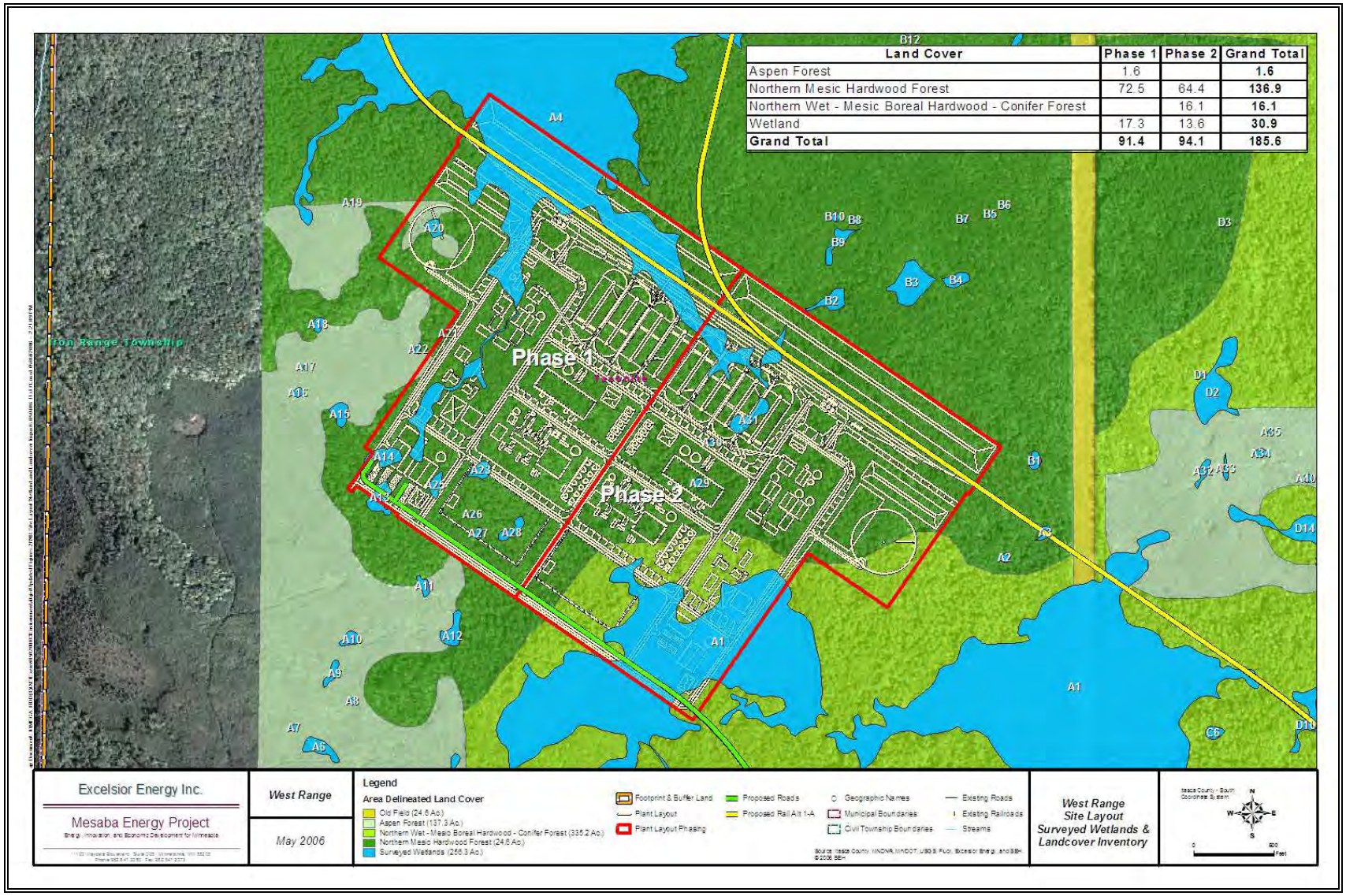
Type 7 wetlands are the most abundant wetland type present within the Station Footprint and Buffer Land and have the most impacts for both phases of the IGCC Facility, most of which being affected by the rail loop. Phase I will cause the majority of wetland impacts for the facility, most of which are Type 7 wetlands. Phase II involves less wetland impact, but includes impacts to Type 3 and Type 3/6/8 wetlands, including bog habitats, which are not impacted by Phase I.

7.7.3 Preferred HVTL Route

For purposes of comparing permanent wetland impacts, the Applicant estimated only the area actually needed for the required structure foundations and not the entire area in the right-of-way. This methodology is deemed to be a reasonable and proper approach for such analysis. Using this criteria, permanent impacts in the Preferred HVTL Route will be limited to about 0.01 acres of wetlands where new HVTL towers will be placed within wetland habitat. To the extent practicable, wetlands will be avoided when installing new towers and construction activities will be undertaken during winter months when wetlands are frozen and more resistant to equipment-related impacts.

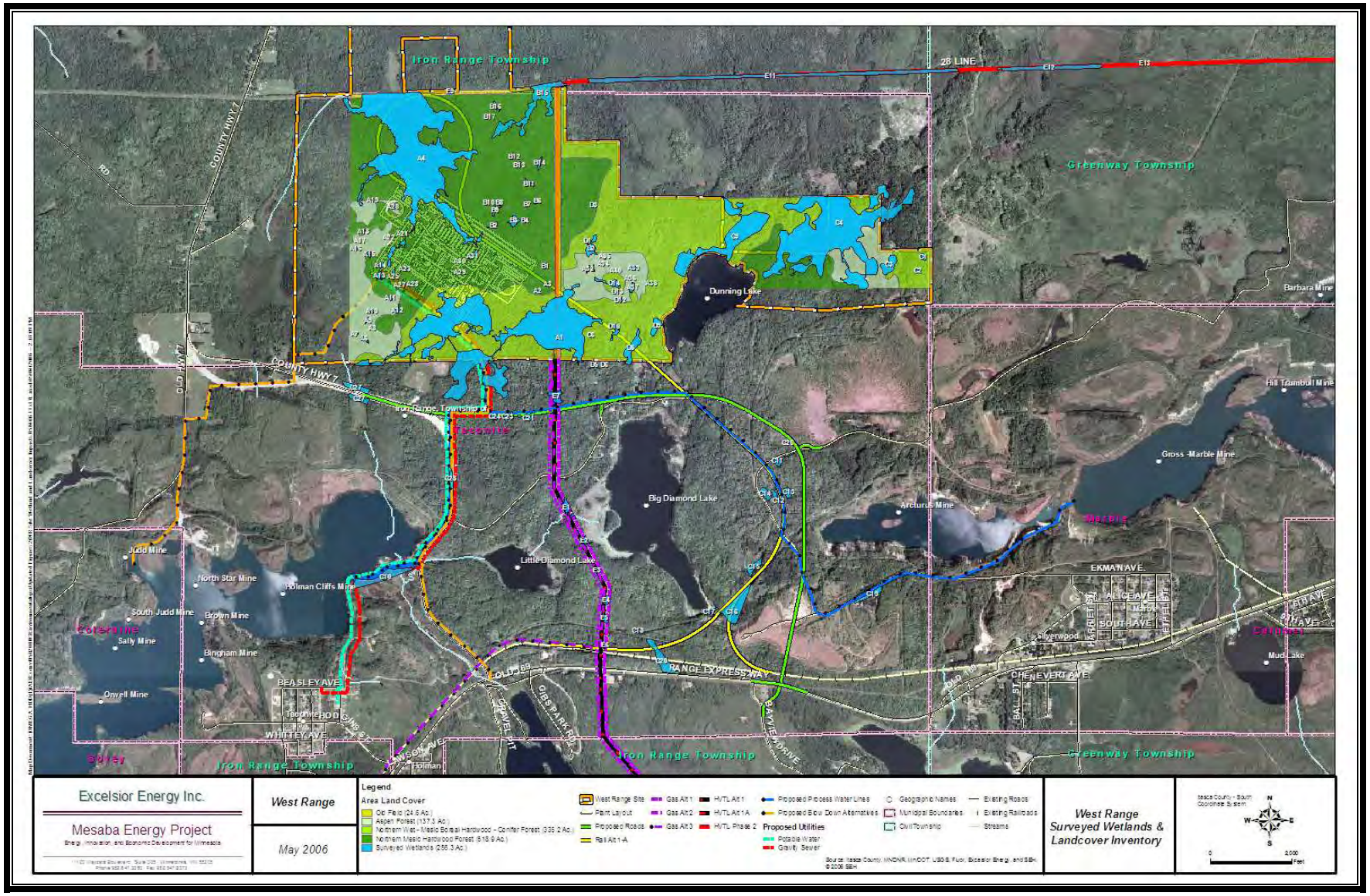
Tree and shrub clearing in wetlands will be initiated along new areas of ROW of the Preferred HVTL Route. A total of 30.21 acres of trees and shrubs is estimated to be cleared in Type 6, 7, and 8 wetlands. No vegetation clearing is anticipated in Type 1-5 wetlands (i.e., herbaceous dominated vegetation in seasonal basins, wet meadow, shallow marsh, or open water wetlands). Direct wetland impacts are not anticipated as no stump grubbing, excavation, or fill is planned for the areas to be cleared of woody vegetation. Ultimately, some wetland areas may be converted to different types (e.g., Type 6 scrub-shrub habitat may convert to Type 2/3 wet meadow/shallow marsh); however, direct loss of wetland is not anticipated. To comply with the federal Migratory Bird Treaty Act, tree clearing activities will be completed during the winter months to avoid the bird nesting period. Work during the winter months when the wetlands are frozen will further reduce equipment-related impacts. Future maintenance of the ROW will likely include the clearing of trees and shrubs that re-establish in wetlands and will be completed during the winter months to avoid equipment-related impacts and disturbances to nesting birds.

Figure 7.7-1 IGCC Power Station Footprint Impacts on Vegetation and Wetlands



Land Cover	Phase 1	Phase 2	Grand Total
Aspen Forest	1.6		1.6
Northern Mesic Hardwood Forest	72.5	64.4	136.9
Northern Wet - Mesic Boreal Hardwood - Conifer Forest		16.1	16.1
Wetland	17.3	13.6	30.9
Grand Total	91.4	94.1	185.6

Figure 7.7-2 Wetland and Land Use Impacts on the IGCC Power Station Footprint and Buffer Land



7.7.4 Alternate HVTL Route

Tree and shrub clearing in wetlands will be initiated along new ROW. A total of 24.53 acres is estimated to be cleared in Type 6, 7, and 8 wetlands. No vegetation clearing is anticipated in Type 1-5 wetlands (i.e., herbaceous dominated vegetation in seasonal basins, wet meadow, shallow marsh, or open water wetlands). Direct wetland impacts are not anticipated as no stump grubbing, excavation, or fill is planned for the areas to be cleared of woody vegetation. Ultimately, some wetland areas may be converted to different types (e.g., Type 6 scrub-shrub habitat may convert to Type 2/3 wet meadow/shallow marsh);but direct loss of wetlands is not anticipated. Tree clearing will be completed during the winter months to avoid equipment-related impacts and, in compliance with the federal Migratory Bird Treaty Act, avoiding the bird nesting period. Future maintenance of the Alternate HVTL Route ROW will likely include clearing of trees and shrubs that re-establish in wetlands and will be completed during the winter months to avoid equipment-related impacts and disturbances to nesting birds.

Although wetlands will be impacted during construction, only 0.01 acres of wetlands will be permanently impacted by placement of new HVTL towers. To the extent practicable, wetlands will be avoided when installing the new towers and construction activities will be undertaken during the winter months to minimize direct wetland impacts.

7.7.5 Plan B Phase II Alternate HVTL Route Route

An estimated 0.03 acres of wetland will be permanently impacted by placement of new HVTL towers on the Plan B Phase II Alternate Route. To the extent practicable, wetlands will be avoided when towers are installed and construction activities will be planned during the winter months when wetlands are frozen.

No tree and shrub clearing in wetlands is anticipated as this alternative is proposed along an existing utility corridor maintained by MP. Future maintenance of the ROW will include clearing of trees and shrubs that re-establish in wetlands and will be completed during the winter months to avoid equipment-related wetland impacts and disturbances to nesting birds.

7.7.6 West Range Proposed Natural Gas Pipeline Route

A total of 24.69 acres of wetland habitat is located in the proposed temporary ROW. For permanent ROW, these wetland impacts will be reduced to 17.47 acres. Temporary impacts will result from construction activities and will be mitigated by restoring the area after construction is completed. Temporary wetland impacts will include tree and shrub clearing for construction staging areas paralleling the pipeline corridor.

To minimize wetland impacts at water crossings, the natural gas pipeline will be directionally drilled under the water body starting at approximately 100 feet from the edge of each bank. In this instance, wetland impacts associated with water crossings will include 1.34 acres in the temporary ROW and 0.94 acres in the permanent ROW. The remainder of the gas pipeline will be placed within the West Range Proposed Natural Gas Pipeline Route using open trench installation techniques. Soils and vegetation that become compacted as a result of construction will be restored by loosening such soils and reseeding the area with grasses and broad-leafed herbaceous plants native to the region.

7.7.7 Process Water Supply Pipeline

7.7.7.1 Segment 1 - Lind Pit to Canisteo Pit

According to the National Wetlands Inventory, no wetlands are present on the route Process Water Supply Pipeline Segment 1. However, field investigations will be performed prior to construction activities to determine if any wetlands are within the ROW and to determine if any impacts will occur.

7.7.7.2 Segment 2 – Canisteo Pit to West Range Site

Wetland impacts were minimized by routing the process water lines along existing and proposed roadways, railroads, and utility rights-of-way. Wetland impacts within the proposed Process Water Supply Pipeline Segment 2 corridor include a total of 5.48 acres in the 150-foot temporary ROW, but these impacts would be reduced to 3.73 acres for the 100-foot permanent ROW. The largest impact for the Process Water Supply Pipeline Segment 2 is within Wetland A1, the wetland complex near the southern boundary of the West Range Site. There are no water crossings associated with the Process Water Supply Pipeline Segment 2. Temporary wetland impacts may include tree and shrub clearing for construction staging areas paralleling the water process line corridor. Where soils and vegetation may become disturbed in the construction areas, such areas will be restored by loosening the soils from compaction and reseeding with grasses and forbs native to the region.

7.7.7.3 Segment 3 – Gross-Marble Pit to Canisteo Pit

Wetland impacts were minimized by routing the process water lines along existing and proposed roadways, railroads, and utility rights-of-way. Process Water Supply Pipeline Segment 3 will include a total of 6.17 acres of wetland impacts in the 150-foot temporary ROW, with these impacts reduced to 3.79 acres for the 100-foot permanent ROW. Type 6 scrub-shrub wetland would sustain the greatest impacts due to this alternative. There are no water crossings associated with the Process Water Supply Pipeline Segment 3. Temporary wetland impacts may include tree and shrub clearing for construction staging areas paralleling the water process line corridor. Where soils and vegetation may become disturbed in the construction areas, such areas will be restored by loosening the soils from compaction and reseeding with grasses and forbs native to the region.

7.7.8 Process Water Blowdown Pipeline Alternative 1 (Facility to Holman Lake)

Wetland impacts were minimized by routing the process water lines along existing and proposed roadways, railroads, and utility rights-of-way. The blowdown process water line includes a total of 5.86 acres of wetland impacts in the temporary ROW, and is reduced to 4.07 acres for the permanent ROW. The process water blowdown line will be placed in wetlands and below waterbodies through open-cut trenching.

There are two water crossings associated with the process water blowdown line. Wetland impacts include the total length of the crossing through waterbodies and adjacent wetlands. The total length of water crossings is 6 linear feet over water, and a total of 50 linear feet in the adjacent wetlands. Impacts to wetlands due to the water crossings are based on a 150-foot

temporary ROW and 100-foot permanent ROW. Wetland habitats associated with the water crossings that will be affected include 7,500 ft^2 (0.17 acres) in the temporary ROW and 5,000 ft^2 (0.11 acres) in the permanent ROW.

Temporary wetland impacts will include tree and shrub clearing for construction staging areas paralleling the water process line corridor. Where soils and vegetation become disturbed in the construction areas, such areas will be restored by loosening the soils from compaction and reseeding with grasses and forbs native to the region.

7.7.9 Blowdown Process Water Alternative 2 (Facility to Canisteo Pit)

Wetland impacts were minimized by routing the process water lines along existing and proposed roadways, railroads, and utility rights-of-way. The blowdown process water line includes a total of 20.38 acres of wetland impacts in the temporary ROW, but will be reduced to 13.60 acres for the permanent ROW. The process water blowdown pipeline will be placed in wetlands and below waterbodies through open-cut trenching. There are no water crossings associated with Alternative 2 process water blowdown pipeline.

Temporary wetland impacts will include tree and shrub clearing for construction staging areas paralleling the water process line corridor. Where soils and vegetation become disturbed in the construction areas, such areas will be restored by loosening the soils from compaction and reseeding with grasses and forbs native to the region.

7.7.10 West Range Potable Water and Sewer Pipelines

Wetland impacts were minimized by routing the sewer and water lines adjacent to the process water lines, which were placed along existing and proposed roadways, railroads, and utility rights-of-way. Wetland impacts within the proposed sewer and water corridor include a total of 4.48 acres in the 100-foot temporary ROW and 1.79 acres for the 40-foot permanent ROW. The largest impact for the sewer and water pipeline are within Wetland A1, the large wetland complex near the southern boundary of the West Range Site. There are no water crossings associated with the water and sewer lines.

7.7.11 West Range Rail Line Alternative 1A

Siting the location of the railroad alternatives first considered avoidance of both Dunning and Big Diamond Lakes. Preliminary alignments for the railroad included some designs that would have required filling as much as ¼ of Big Diamond Lake and were removed from further consideration. At the southeast corner of Big Diamond Lake, Alternative 1A was shifted away from Big Diamond Lake to reduce direct impacts on the lakebed itself and its surrounding aquatic habitat.

Because the railroads must be designed for the straightest possible alignment, some unavoidable wetland impacts will occur. The railroad alternatives are the only utility or transportation corridors that have precisely established construction limits, which may be considered as temporary ROW. For the West Range Railroad Alternative 1A, the construction limits (temporary ROW) vary in width from 80 – 450 feet. The permanent ROW for the railroad will be an established 100-foot ROW, and includes the ROW width needed for the center loop.

Permanent wetland impacts within the railroad alternatives will occur within the construction limits (temporary ROW), permanent ROW, and the center loop. There are no temporary wetland impacts anticipated for the railroad alternatives due to the necessary grading required for the railroad bed. Permanent wetland impacts within the construction limits (temporary ROW) include 26.45 acres. Permanent wetland impacts within the permanent ROW (the railroad bed itself) total 12.23 acres. The center loop of the rail spur for Alternative 1A has an estimated 64.85 acres of permanent impacts. The impacts estimated for the center loop may be reduced upon completion of final design when the layout of storage areas within the center loop is determined. There are no water crossings associated with Railroad Alternative 1A.

7.7.12 West Range Rail Line Alternative 1B

For the West Range Railroad Alternative 1B, the construction limits (temporary ROW) vary in width from 60 – 760 feet. The permanent ROW for the railroad would be an established 100-foot ROW, and includes the ROW width needed for the center loop.

Permanent wetland impacts within the construction limits (temporary ROW) total 18.11 acres. Permanent wetland impacts within the permanent ROW (the railroad bed itself) totals 12.00 acres. The center loop of the rail spur for Alternative 1A has an estimated 64.23 acres of permanent impacts. The impacts estimated for the center loop may be reduced upon completion of final design when the layout of storage areas within the center loop is determined. There are no water crossings associated with Railroad Alternative 1B.

7.7.13 West Range Roads

Appropriate road corridors were identified by assessing grading requirements, existing topography, accessible properties, and the presence of wetlands in providing necessary access to the West Range Site. Although there will be impacts to wetlands due to the placement of the corridors, these impacts were balanced by the overall site grading requirements. In some instances it became more feasible to impact a small area of wetland than attempt to grade hillsides or steep slopes.

Roads that will serve the facility will impact a total of 9.72 acres of wetlands in the 200-foot temporary ROW. These impacts will be reduced to 5.67 acres for the 120-foot permanent ROW. The largest wetland impacts for roads are within Wetland A1, the large wetland complex near the southern boundary of the West Range Site. There are no water crossings associated with the access roads.

7.8 ECOLOGICAL RESOURCES: PLANTS, ANIMALS, AND ENDANGERED SPECIES

This section describes the ecological conditions and biological communities that are present on the West Range Site, including an analysis of flora and fauna and occurrences of habitat for state and federally rare, special concern, threatened, or endangered species.

The following state and federal agencies are anticipated to provide comments on the environmental review documents, and require permits or approvals on natural resource related

subjects. Wetland agency requirements and its involvement are discussed in detail in Section 3.6 of this report.

Anticipated Involvement of Federal and State Agencies

Federal Agency	Potential Project Roles
U.S. Fish and Wildlife Service	Endangered Species Act, Migratory Bird Treaty Act, Fish and Wildlife Coordination Act, Environmental Document Review and Comments
U.S. Army Corps of Engineers	Section 404 Federal Wetland Permit, Federal Clean Water Act, Environmental Document Review and Comments
U.S. Environmental Protection Agency	Environmental Document Review and Comments
State Agency	**Potential Project Roles**
Minnesota Department of Natural Resources	Minnesota Endangered Species Statute, Public Waters Work Permit, License for Utility Crossings of Public Lands and Waters, Water Appropriation Permit, Environmental Document Review and Comments
Minnesota Pollution Control Agency	Section 401 Water Quality Permit, Federal Clean Water Act, Environmental Document Review and Comments
Minnesota Board of Water and Soil Resources	Minnesota Wetland Conservation Act, Environmental Document Review and Comments

Timber harvesting is the primary upland use that has impacted the West Range Station Footprint and Buffer Land, and that has influenced the composition and dynamics of the forest cover. Both clear cutting and selective harvesting of timber have occurred on different tracts of land within the upland Buffer Land, resulting in a patchwork like pattern of cleared recently cut (10-20 years) areas and stands of forest cover of varying ages and compositions in the uplands.

Biological communities and habitats for and occurrences of flora and fauna were assessed in conjunction with the field reconnaissance for wetland habitat. Review of MDNR Natural Heritage Information System (NHIS) data for known records of protected habitats or species within or near the project area provided information for potential target habitats to assess. Habitats were first identified through off-site methods including review of aerial and satellite imagery. This was followed by a field reconnaissance by field biologists during June 6-10, 2005. The terrestrial (upland) habitats described below are based on observations collected during the June 2005 field reconnaissance. Supplemental information describing terrestrial habitats was provided by wetland scientists during a June-August, 2005 wetlands survey.

7.8.1 Terrestrial Flora

The list of vegetative communities found in the vicinity of the West Range Site is derived from the *Field Guide to the Native Plant Communities of Minnesota: The Laurentian Mixed Forest Province* (MDNR 2003), a vegetation classification system for north central and northeastern Minnesota. The following discussion describes terrestrial habitats present on the IGCC Station

Footprint and Buffer Land. State and federally protected flora species in the vicinity of the West Range Site are addressed in Section 7.8.3 below. The general land use impacts on trees and other plants and aquatic areas from the power station and associated utility corridors are described in Section 7.1 above. Detailed estimates of the amount of trees and brush required to be cleared within each area or right-of-way are presented in Section 3.9 of the ES.

Within the Station Footprint there was no old growth or mature conifer forest observed during field reconnaissance. White pines were observed infrequently and red pine (*Pinus resinosa*) was not observed. All of these upland terrestrial communities onsite have been impacted by timber activities at some point in time. Some areas appear to have been logged for several consecutive decades. The western half of the Station Footprint and Buffer Land also had evidence of logging activities in the past 10-20 years, with dense quaking aspen regrowth.

Table 7.8-1 below provides a summary of impacts by habitat type for the Station Footprint and Buffer Land. A summary of total land use impacts for the associated facilities, transmission lines, and pipelines is provided above in Section 7.1.

Table 7.8-1
Summary of Biological Communities within Station Footprint and Buffer Land

Vegetation Cover Type	Acres within Buffer	Phase I Impacts (acres)	Phase II Impacts (acres)	Total Impacts Acres
Aspen Forest	137.30	1.64	0	1.64
Northern Wet-Mesic Boreal Hardwood-Conifer Forest	335.2	0	16.1	16.1
Northern Mesic Hardwood Forest	518.9	72.47	64.42	136.9
Old Field	24.64	0	0	0
Wetland	177	17.3	13.6	30.95
Total Acres	**1193**	**88**	**111**	**179**

Given that the Station Footprint and Buffer Land is located within a timber production area subject to frequent clear cutting, comprised entirely of secondary growth, and within the forest setting of northern Minnesota, trees are not rare and no significant impacts to trees are anticipated. No tree mitigation will occur nor will any mitigation for impacts to vegetative communities, all of which are abundant throughout the region.

7.8.2 Fauna

Fauna (animals) in the vicinity of the West Range Site include species that are typical to northern Minnesota (State and federally protected fauna are addressed in Section 7.8.3.). The most abundant habitat found on the Station Footprint and Buffer Land is northern mesic hardwood forest (red oak-sugar maple-basswood-(bluebead lily) forest). Bird diversity is highest within this community compared to other habitats, and includes nesting and foraging habitats for songbirds and raptors.

The list of potential mammals that utilize land within the boundary of the Buffer Land includes predators, bears, and large ungulates such as moose and deer. Many deer were observed within this forest type, and evidence of deer browse was commonly observed. Suitable forage for moose occurs in the wetlands and uplands areas within the boundaries of the Buffer Land, and a moose skeleton was observed within the Station Footprint and Buffer Land. Beaver also utilize this site for forage and beaver activity was prevalent, especially within the eastern half of the Station Footprint and Buffer Land. During the June 2005 field reconnaissance, field biologists saw a lone timber wolf (*Canis lupus* – federally threatened) preying on a deer fawn.

The northern wet-mesic boreal hardwood-conifer forest is patchy and discontinuous within the Station Footprint and Buffer Land. The fauna found are common to second growth forests and the varying upland habitats found in northern Minnesota.

Wetland habitats for fauna are relatively diverse and common throughout the vicinity of the West Range Site. The Type 8 bog habitat is the most unique and is potential habitat for rare species of fauna, primarily birds and small mammals. All other types of wetlands (Types 3-7) within the Station Footprint and Buffer Land, but not connected to lakes, are the most important for amphibians. The wetlands that provide optimum amphibian breeding habitats are ones lacking fish (predators) populations. Adult Anurans (frogs) were observed during the field reconnaissance and included American toad (*Bufo americanus*), grey treefrog (*Hyla versicolor*), Northern leopard frog (*Rana pipiens*), and wood frog (*Rana sylvatica*). Potential habitats were also observed for the spring peeper (*Psuedacris crucifer*), Western chorus frog (*Psuedacris triserata*), green frog (*Rana clamitans*), and mink frog (*Rana septentrionalis*), all species common to the area. The mink frog is common to lakes and lake fringe wetlands. Wetlands also provide potential habitat for the Eastern newt (*Notopthalmus viredescens*) and the blue-spotted salamander.

No breeding concentrations of migratory birds are present within the Station Footprint and Buffer Land. These include nesting swallow colonies, waterbird colonies, heron and egret nests, or other colonial nesting species. The entire area contains breeding bird habitats as evidenced by the songbirds engaging in territorial behaviors and calls during the June and July 2005 field surveys. These were thought to be from nesting birds. Raptor nesting was assumed to occur throughout the site as well. Two adult unidentifiable Accipiters and a barred owl (*Strix varia*) were observed. Of the three potential Accipiters found in this area, the Northern goshawk (*Accipiter gentiles*) is the only Accipiter considered rare and is a designated sensitive species in Minnesota by the U.S. Forest Service. Goshawk habitat was relatively absent from the site as this animal prefers old growth and undisturbed conifers. Ruffed grouse (*Bonasa umbellus*) were commonly observed in the second growth aspen forest, further indicating the widespread occurrence of timber harvesting activities within the Station Footprint and Buffer Land.

Habitat quality varies and the overall habitat quality within the Station Footprint and Buffer Land. Wetlands are the highest quality habitats within the Station Footprint and Buffer Land. Habitat fragmentation and loss of habitat connectivity is most prevalent southwest of the Station Footprint and Buffer Land due to the impacts of past mining. The existing roads and transmission lines in and around the area have also resulted in permanent conversions and represent a habitat fragmenting vector for some species. Land uses and habitats are similar in lands surrounding and extending outward from the West Range Site.

7.8.3 Rare and Unique Natural Resources

The West Range Site has potential habitat for and is within the distributional range of three federally-listed species: the bald eagle (*Haliaeetus leucocephalus* – recently delisted), Canada Lynx (*Lynx canadensis*) and grey wolf (*Canis lupus*). As previously noted, a wolf was observed within the Station Footprint and Buffer Land during the 2005 field reconnaissance and wolf habitat and prey items widely occur throughout the vicinity of the West Range Site. Verified and unverified sightings (MDNR Online Data, 2005) of Canada lynx are found within Itasca County and potential habitats for this predator exist within and adjacent to the Station Footprint and Buffer Land.

There are no federally protected plant species identified by the USFWS within the Station Footprint and Buffer Land or any of the proposed utility or transportation corridors and, therefore no adverse effects are anticipated on any federally protected plant species.

Section 7 Formal Consultation will need to occur for the Canada lynx and grey wolf. Currently, population studies are being conducted on these species in conjunction with the Formal Consultation that has been initiated by the other projects in the vicinity of the West Range Site and are under current consultation. Based on this Formal Consultation, the determination of the significance of effects on the Canada lynx and grey wolf will be made by the USFWS.

According to MDNR data (MDNR Online Data, 2005), there have been both "verified without evidence of breeding" and "unverified" sightings of Canada lynx within Itasca County through 2005. Potential Canada lynx habitat and prey items were observed in the vicinity of the West Range Site during the 2005 habitat assessments and field reconnaissance for wetlands. The request for Formal Consultation under Section 7 of the Endangered Species Act will determine the need for additional studies and coordination for this species. The MDNR Natural Heritage and Information System (NHIS) database shows no bald eagle nesting areas within the Station Footprint and Buffer Land, nor within a 0.5-mile radius of the project boundary.

Additional field survey during winter 2005-2006 by U.S. Fish and Wildlife Service to determine the potential effects on habitat for the Canada lynx and grey wolf is ongoing. The significance of effects to these species will be determined through the Section 7 consultation process. Similarly, effects on bald eagles should also be determined by the U.S. Fish and Wildlife Service through the Section 7 consultation process.

7.8.3.1 Minnesota Endangered Species Act

The MDNR Natural Heritage Information System (NHIS) database contains documented occurrences of non-status (tracked), special concern, threatened, and endangered species; sensitive ecological and natural resources; and results of the Minnesota County Biological Survey (MCBS). State-listed threatened or endangered species are protected under the Minnesota Endangered Species Statute (Minn. Stat. § 84.0895). The MDNR was contacted to request a review of the NHIS for occurrences within the Station Footprint and Buffer Land and its associated utility and transportation corridors, which includes the Nashwauk, Taconite, and Bovey areas. At the request of the MDNR, the specific locations of these occurrences are not provided in this report to protect the integrity of these rare or protected species. A summary of

the known species occurrences relative to the West Range Site project alternatives are provided in the following paragraphs.

7.8.3.2 West Range IGCC Power Station Footprint and Buffer Land

No MDNR NHIS species occurrences within the Station Footprint and Buffer Land are documented within the Station Footprint and Buffer Land. There are 17 occurrences of state-listed rare or protected species identified by the MDNR NHIS within the Nashwauk, Taconite, and Bovey areas. Of these occurrences, only three species are within a one-mile radius of the Station Footprint and Buffer Land. These three species are located southeast of the project and are comprised of *Botrychium spp* observed in mine spoil and tailings areas, as summarized in Table 7.8-2 below.

Most of the 17 NHIS occurrences in the vicinity of the West Range Site are associated with mine spoil, tailings ponds, or otherwise disturbed soils near mine sites. None of these mine areas or associated disturbed lands are within the Station Footprint and Buffer Land. Potential habitats for species identified in the NHIS database search were investigated during the 2005 habitat assessments and field reconnaissance for wetlands.

Of greatest concern are those records for the state-listed endangered orchid species, *Platanthera flava var. herbiola* (tubercled-rein orchid), that have colonized in disturbed mine spoil areas. Typical habitat for this species is within fringe wetland habitats such as wet meadow habitats dominated by native graminoids and sedges. Similar habitat is present within the Buffer Land. Consideration must be given to the potential presence of the species in designing the final layout of the IGCC power station.

Two plant species records from the NHIS database that are found in areas other than disturbed mine areas are *Myriophyllum tenellum* (leafless water milfoil – non-status) and *Torreyochloa pallida* (Torrey's manna grass – special concern). *Myriophyllum tenellum*, a non-status species, is associated with aquatic environments along shorelines. Dunning Lake is likely the only area within the Buffer Land that may provide potential habitat for this species. However, Dunning Lake and its associated aquatic habitats will be avoided for construction of the IGCC Power Station and associated facilities.

Torreyochloa pallida, a species of special concern, is associated with shallow marsh habitats in mixed hardwood forests. This type of habitat is abundant throughout the West Range Site, although this species was not observed during the field reconnaissance for habitat or during the wetland surveys. Shallow marsh habitat that could contain *Torreyochloa pallida* will be impacted by the construction of the IGCC Power Station and its associated facilities. However, species of special concern are not protected by the Minnesota Endangered Species Statute (Minn. Stat. § 84.0895). Nonetheless, the Applicant will coordinate with the MDNR to determine whether there could be any significant effects on this species.

During the field reconnaissance in June 2005, a plant species that closely resembled *Botrychium minganense*, a state-listed species of special concern, was observed in the mixed-hardwood conifer forest. Only one individual was observed, and no voucher specimens were collected. This area of forest may require a more thorough review for potential occurrences of state-listed *Botrychium spp.* to determine if these resources could be affected. Again, even though species of

special concern are not protected by the Minnesota Endangered Species Statute (Minn. Stat. § 84.0895), the Applicant will coordinate with the MDNR to determine whether there could be any significant effects on this species.

For the records of state-listed species within the vicinity of the West Range Site that are listed as species of special concern or non-status, impacts to these species or their habitats are not regulated by State law. However, this does not preclude coordination with the MDNR to determine significance of potential impacts. For these reasons coordination with MDNR will be completed as soon as possible to determine the potential effects on all State-listed species or their habitats within the vicinity of the West Range Site, particularly for State-listed endangered *Platanthera flava var. herbiola.*

Table 7.8-2
MDNR NHIS Plant Species Occurrences in Vicinity of West Range Site

NHIS Occurrence Number	Scientific Name	Common Name	Protection Status	Associated Habitat Near Project Area
#28507, #28508	*Botrychium campestre*	Prairie moonwort	SPC	High iron content and gravel soils
#24083, #24098, #24107, #28518, #28536, #28639	*Botrychium simplex*	Least moonwort	SPC	Mine tailings basin, disturbed utility ROW
#28509, #30927	*Botrychium matricariifolium*	Matricary grapefern	Non-status	Grassy opening, near mine area
#24653, #28537	*Liparis lilifolia*	Lilia-leaved twayblade	Non-status	Tailings basin
#27799	*Myriophyllum tenellum*	Leafless water milfoil	Non-status	Lake shoreline
#24655, #28510	*Platanthera flava var. herbiola*	Tubercled rein-orchid	END	Tailings basin
#30846	*Spiranthes casei*	Case's ladies'-tresses	Non-status	Tailings basin
#28514	*Torreyochloa pallida*	Torrey's manna grass	SPC	Shallow marsh in mixed hardwood forest

[1] END – Endangered
SPC – Species of Special Concern
No status – No state protection status, but species may be monitored due to other concerns

7.8.4 West Range Preferred HVTL Route

Seven (7) known occurrences of state-listed species are documented within one mile of the West Range Preferred HVTL Route, as detailed in Table 7.8-3. Of greatest concern are those records for the state-listed endangered orchid species, *Platanthera flava var. herbiola* (tubercled-rein orchid), which is known to occur in fringe wetland habitats such as wet meadow habitats dominated by native graminoids and sedges. The known records for this species near the West Range Preferred HVTL Route are within mine spoil areas, and it is not fully understood how this species has recruited into these highly disturbed areas. There are no mine spoil areas that are within the West Range Preferred HVTL Route.

Because of the rarity of *Platanthera flava var. herbiola* in the state, the probability is low for encountering this species in wet meadow habitat within the West Range Preferred HVTL Route.

The remaining records of state-listed species within one mile of the West Range Preferred HVTL Route are listed as species of special concern or non-status species. These species were all observed within mine spoil areas which are not found within any area of the West Range Preferred HVTL Route. Although impacts to these species or their habitats are not regulated, coordination with MDNR will be completed to determine the potential effects on these species or their habitats within or near this HVTL route, particularly for state-listed endangered *Platanthera flava var. herbiola*.

Table 7.8-3
MDNR NHIS Occurrences within One Mile of HVTL Preferred Route

NHIS Occurrence Number	Common Name	Scientific name	State Protection Status[1]	Potential Habitats
#30922	Matricary grapefern	*Botrychium matricariifolium*	No status	Site records also within mine spoil areas.
#28637	Species of moonwort	*Botrychium michiganense*	No status	Site records also within mine spoil areas.
#28638	Least moonwort	*Botrychium simplex*	SPC	Site records also within mine spoil areas.
#28172, #29121, #24088	Tubercled-rein orchid	*Platanthera flava var. herbiola*	END	Occurs in fringe wetland habitats. Site records also within mine spoil areas.
#29124	Case's ladies'-tresses	*Spiranthes casei*	No Status	Occurs in fringe wetland habitats. Site records also within mine spoil areas.

[1] END – Endangered
SPC – Species of Special Concern
No status – No state protection status, but species may be monitored due to other concerns

7.8.5 West Range Alternate HVTL Route

Seven (7) known occurrences of state-listed species are documented within one mile of the West Range Alternate HVTL Route, as detailed in Table 7.8-4. Of greatest concern are those records for the state-listed endangered orchid species, *Platanthera flava var. herbiola* (tubercled-rein orchid), which is known to occur in fringe wetland habitats such as wet meadow habitats dominated by native graminoids and sedges. The known records for this species near the West Range Alternate HVTL Route are within mine spoil areas, and there are no mine spoil areas that are within the West Range Alternate HVTL Route.

As with the West Range Preffered HVTL Route, because of the rarity of *Platanthera flava var. herbiola* in the state, the probability is low for encountering this species in wet meadow habitat.

The remaining records of state-listed species within one mile of the West Range Alternate HVTL Route are listed as species of special concern or non-status species. These species were all observed within mine spoil areas, which are not found within any area of the West Range Alternate HVTL Route. Although impacts to these species or their habitats are not regulated, coordination with MDNR will be completed to determine the potential effects on these species or their habitats within or near the West Range Alternate HVTL Route, particularly for state-listed endangered *Platanthera flava var. herbiola*.

Table 7.8-4
MDNR NHIS Occurrences within One Mile of the West Range Alternate HVTL Route

NHIS Occurrence Number	Common Name	Scientific name	State Protection Status[1]	Potential Habitats
#30922	Matricary grapefern	*Botrychium matricariifolium*	No status	Site records also within mine spoil areas.
#28637	Species of moonwort	*Botrychium michiganense*	No status	Site records also within mine spoil areas.
#28638	Least moonwort	*Botrychium simplex*	SPC	Site records also within mine spoil areas.
#28172, #29121, #24088	Tubercled-rein orchid	*Platanthera flava var. herbiola*	END	Occurs in fringe wetland habitats. Site records also within mine spoil areas.
#29124	Case's ladies'-tresses	*Spiranthes casei*	No Status	Occurs in fringe wetland habitats. Site records also within mine spoil areas.

[1] END – Endangered
SPC – Species of Special Concern
No status – No state protection status, but species may be monitored due to other concerns

7.8.6 Plan B Phase II Alternate HVTL Route Route

The Plan B Phase II Alternate HVTL Route is an existing HVTL corridor that would be utilized as an alternate Phase II route if the Proponent's Plan A Preferred 345kV option is not approved. Twelve (12) known occurrences of state-listed species are documented within one mile of the Plan B Phase II Alternate HVTL Route, as detailed in Table 7.8-5. Of greatest concern are those records for the state-listed endangered orchid species, *Platanthera flava var. herbiola* (tubercled-rein orchid), that have colonized in disturbed in mine spoil areas. Typical habitat for this species is within in fringe wetland habitats such as wet meadow habitats dominated by native graminoids and sedges. However, the known record for this species near the Plan B Phase II Alternate HVTL Route is within a mine spoil area and there are no mine spoil areas within the Plan B Phase II Alternate HVTL Route.

The remaining records of state-listed species within one mile of this corridor are listed as species of special concern or non-status. Although impacts to these species or their habitats are not regulated, coordination with MDNR will be completed to determine the potential effects on these species or their habitats within the corridor, particularly for state-listed endangered *Platanthera flava var. herbiola*.

Table 7.8-5
MDNR NHIS Occurrences within One Mile of Plan B Alternative Route

NHIS Occurrence Number	Common Name	Scientific name	State Protection Status[1]	Potential Habitats
#26408	Northern goshawk	*Accipiter gentiles*	No Status	Mature conifer forest.
#28507	Prairie moonwort	*Botrychium campestre*	SPC	Site record is within mine spoil area.
#28509	Matricary grapefern	*Botrychium matricariifolium*	SPC	Site record is within mine spoil area.
#27896	Species of moonwort	*Botrychium michiganense*	No Status	Site record is within mine spoil area.
#28534, #23754, #27894	Pale moonwort	*Botrychium pallidum*	SPC	Site records are within mine spoil areas.
#28533, #27895	Least moonwort	*Botrychium simplex*	SPC	Site records are within mine spoil areas.
#28537	Lilia-leaved twayblade	*Liparis lilifolia*	SPC	Occurs in fringe wetland habitats. Site record is within mine spoil area.
#24655	Tubercled-rein orchid	*Platanthera flava var. herbiola*	END	Occurs in fringe wetland habitats. Site record is within mine spoil area.
#19111	Lapland buttercup	*Ranunculus laponnicus*	SPC	Species is found in wetland habitats.

[1] END – Endangered
SPC – Species of Special Concern
No status – No state protection status, but species may be monitored due to other concerns

7.8.7 West Range Natural Gas Pipeline Alternative

Nine (9) known occurrences of state-listed species are documented within one mile of the Natural Gas Pipeline Alternative 1, as detailed in Table 7.8-6. Of greatest concern are those records for the state-listed endangered orchid species, *Platanthera flava var. herbiola* (tubercled-rein orchid), that have colonized in disturbed in mine spoil areas. Typical habitat for this species is within in fringe wetland habitats such as wet meadow habitats dominated by native graminoids and sedges. However, the known records for this species near the Natural Gas Pipeline

Alternative 1 are within mine spoil areas, and there are no mine spoil areas within the alignment for the Natural Gas Pipeline Alternative.

Because of the rarity of *Platanthera flava var. herbiola* in the state, the probability is low for encountering this species in wet meadow habitat within the Natural Gas Pipeline Alternative.

The remaining records of state-listed species within one mile of Natural Gas Pipeline Alternative 1 are listed as species of special concern or non-status. Although impacts to these species or their habitats are not regulated, coordination with MDNR will be completed to determine the potential effects on these species or their habitats within Natural Gas Pipeline Alternative 1, particularly for state-listed endangered *Platanthera flava var. herbiola*.

Table 7.8-6
MDNR NHIS Occurrences within One Mile of Gas Pipeline Route

NHIS Occurrence Number	Common Name	Scientific name	State Protection Status[1]	Potential Habitats
#12178	American bittern	*Botaurus lentiginosus*	No status	Wet meadow, shallow and deep marsh, and fringe lakeshore dominated by herbaceous vegetation.
#30922	Matricary grapefern	*Botrychium matricariifolium*	No status	Site record is within mine spoil area.
#28637	Species of moonwort	*Botrychium michiganense*	No status	Site record is within mine spoil area.
#28638	Least moonwort	*Botrychium simplex*	SPC	Site record is within mine spoil area.
#27799	Leafless water milfoil	*Myriophyllum tenellum*	No Status	Lake shoreline.
#28172, #29121, #24088	Tubercled-rein orchid	*Platanthera flava var. herbiola*	END	Occurs in fringe wetland habitats. Site records are within mine spoil areas.
#29124	Case's spiranthes	*Spiranthes casei*	No Status	Occurs in fringe wetland habitats. Site record is within mine spoil area.

[1] END – Endangered
SPC – Species of Special Concern
No status – No state protection status, but species may be monitored due to other concerns

7.8.8 West Range Process Water Supply Pipeline

7.8.8.1 Segment 1 (Lind Pit to Canisteo Pit)

Four (4) known occurrences of state-listed species are documented within one mile of Process Water Supply Pipeline Segment 1 (Lind Pit to Canisteo Pit), as detailed in Table 7.8-7. These

four records are for the state-listed *Botrychium spp.*, which were documented through field survey completed by Critical Connections Ecological Services, Inc. in 2005. It is assumed these records have been reported to the MDNR and are now part of the NHIS database.

All four *Botrychium spp.* were observed in mine spoil areas. One species, *Botrychium pallidum* (pale moonwort) is listed endangered in the state. The remaining *Botrychium spp* are listed as species of special concern or non-status species. All four of these species may be within the temporary or permanent ROWs for Process Water Supply Pipeline Segment 1 and could be impacted due to construction activities.

State-listed endangered or threatened species that will be impacted by the project will require securing a "takings permit" from the MDNR. This would involve detailed descriptions of the type of habitat and number of species/populations affected, minimization and avoidance measures, and compensatory mitigation for the takings. Compensatory mitigation would be negotiated with the MDNR if *Botrychium pallidum* is directly or indirectly affected by the project.

Although impacts to species of special concern or non-status species or their habitats are not regulated, coordination with MDNR will be completed as soon as possible to determine the potential effects on these species or their habitats within or near Process Water Supply Pipeline Segment 1, particularly for state-listed endangered *Botrychium pallidum*.

Table 7.8-7
MDNR NHIS Occurrences within One Mile of
West Range Process Water Supply Pipeline Segment 1

NHIS Occurrence Number	Common Name	Scientific name	State Protection Status[1]	Potential Habitats
n/a [2]	Prairie moonwort	*Botrychium campestre*	SPC	Observed in mine tailings near Lind Pit and West Hill Pit.
n/a [2]	Matricary grapefern	*Botrychium matricariifolium*	No status	Observed in mine tailings near Lind Pit and West Hill Pit.
n/a [2]	Pale moonwort	*Botrychium pallidum*	END	Observed in mine tailings near Lind Pit and West Hill Pit.
n/a [2]	Least moonwort	*Botrychium simplex*	SPC	Observed in mine tailings near Lind Pit and West Hill Pit.

[1] END – Endangered
SPC – Species of Special Concern
No status – No state protection status, but species may be monitored due to other concerns

7.8.8.2 West Range Process Water Supply Pipeline Segment 2 (Canisteo Pit to West Range Site)

There are no known occurrences of state-listed protected or otherwise rare species within one mile of Process Water Supply Pipeline Segment 2 (Canisteo Pit West Range Site), and impacts to these resources or their habitats are not anticipated for this alternative.

7.8.8.3 West Range Process Water Supply Pipeline Segment 3 (Gross-Marble Pit to Canisteo Pit)

One (1) known occurrence of a state-listed species is documented within one mile of Process Water Supply Pipeline Segment 3 (Gross-Marble Pit to Canisteo Pit), as detailed in Table 7.8-8. This record is for the state-listed threatened *Botrychium rugulosum* (St. Lawrence grapefern) which was observed within a mine tailings basin among aspen. Although this record is not directly within the proposed alignment for Process Water Supply Pipeline Segment 3, there are mine spoil areas within the proposed alignment that may contain undocumented occurrences of this species.

State-listed endangered or threatened species that will be impacted by the project will require securing a "takings permit" from the MDNR. This would involve detailed descriptions of the type of habitat and number of species/populations affected, minimization and avoidance measures, and compensatory mitigation for the takings. Compensatory mitigation would be negotiated with the MDNR if state-listed threatened *Botrychium rugulosum* is directly or indirectly affected by the project.

Table 7.8-8
MDNR NHIS Occurrences within One Mile of
West Range Process Water Supply Pipeline Segment 3

West Range Process Water Supply Pipeline – Segment 3 (Gross-Marble Pit to Canisteo Pit)				
#30926	St. Lawrence grapefern	*Botrychium rugulosum*	THR	Site record within mine tailings basin among aspen.

[1] THR – Threatened

7.8.9 Process Water Blowdown Pipeline 1 (IGCC Facility to Holman Lake)

There are no known occurrences of state-listed protected or otherwise rare species within one mile of Process Water Blowdown Pipeline 1 (IGCC Facility to Holman Lake), and impacts to these resources or their habitats are not anticipated for this alternative.

7.8.10 Process Water Blowdown Pipeline 2 (IGCC Facility to Canisteo Pit)

There are no known occurrences of state-listed protected or otherwise rare species within one mile of Process Water Blowdown Pipeline 2 (IGCC Facility to Canisteo Pit), and impacts to these resources or their habitats are not anticipated for this alternative.

7.8.11 West Range Potable Water and Sewer Pipelines

There are no known occurrences of state-listed protected or otherwise rare species within one mile of the alignment for the Potable Water Sewer Pipelines Alternative 1, and impacts to these resources or their habitats are not anticipated for this alternative.

7.8.12 West Range Rail Line Alternative 1A and Center Loop

There are no known occurrences of state-listed protected or otherwise rare species within one mile of Rail Line Alternative 1A and Center Loop, and impacts to these resources or their habitats are not anticipated for this alternative.

7.8.13 West Range Rail Line Alternative 1B and Center Loop

There are no known occurrences of state-listed protected or otherwise rare species within one mile of Rail Line Alternative 1B and Center Loop, and impacts to these resources or their habitats are not anticipated for this alternative.

7.8.14 West Range Roads

There are no known occurrences of state-listed protected or otherwise rare species within one mile of the road alignments, and impacts to these resources or their habitats are not anticipated for this alternative.

7.9 NOISE

The Applicant estimated the amount of noise that Mesaba One and Mesaba Two would create at nearby residences and other receptors during construction and operation. Noise will be generated by the IGCC Power Station as well as by associated rail and roadway traffic. Overall, noise mitigation methods are available such that noise from the IGCC Power station will be not be audible at nearby residences (less than 3dBA increase) and will not exceed MPCA noise standards at any surrounding receptors. While the State noise standards are not applicable to Itasca County's proposed realignment of CR 7, traffic noise levels are projected to exceed MPCA standards at some nearby receptors during both construction and CR7 operation. Mitigation of the roadway traffic noise is economically infeasible.

7.9.1 Noise Standards

The Minnesota Pollution Control Agency (MPCA) has set noise standards for both residential and industrial zones. These standards, measured in dBA, are stipulated in the form of L_{10} and L_{50}. L_{10} means that the measured sound pressure level ("SPL") (in dBA) must not exceed a certain threshold more than 10% of the time (for a one hour survey), and L_{50}, being a level that must not

be exceeded more than 50% of the time (for a one hour survey). These thresholds are listed as SPL (dBA) maximums by the MPCA, as shown in Table 7.9-1. The Noise Area Classification (NAC) has 4 classes. NAC-1 includes household units, including farmhouses, as well as religious activities. NAC – 2 applies to more commercial development, such as retail, businesses, government services, and parks. NAC-3 and 4 (not considered here) are less stringent, and are composed primarily of industrial uses. Applicable limits for NAC-1 areas where there is no overnight lodging are daytime values only.

Table 7.9-1 Minn. R. 7030.0050 Noise Area Classification (NAC)

	NAC - 1		NAC - 2	
	L_{50}	L_{10}	L_{50}	L_{10}
Daytime	60dBA	65dBA	65dBA	70dBA
Nighttime (10:00 p.m.-7:00 a.m.)	50dBA	55dBA	65dBA	70dBA

7.9.2 Site Setting and Receptors

The West Range Site and its HVTL and natural gas pipeline routes are rural, generally forested areas, and sparsely populated with residences. A total of 11 noise receptor stations were positioned nearby the IGCC Power Station Footprint and Buffer Land. Four receptors were positioned on the boundary of the Buffer Land and seven others at more distant locations. These receptor locations are summarized in Table 7.9-2 below.

Table 7.9-2 West Range Site Receptor Locations

Location	Approximate Distance from the nearest edge of West Range Site	Used for Analyses Type(s)
1. County Landfill, south of proposed Plant	1,625' to the south	Ambient Monitoring; Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
2. Residence, North Big Diamond Lake	3,850' to the southeast	Ambient Monitoring; Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
3. Residence, along CR 7	3,800' to the west	Ambient Monitoring; Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
4. 32423 CR 7	4,400' to the west	Ambient Monitoring; Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
5. Dunning Lake site	4,175' to the east	Ambient Monitoring; Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
6. Lutheran Church	18,000' to the southeast	Plant Operations Modeling
7. Catholic Church	10,700' to the NNW	Plant Operations Modeling
AAC-6. Near Beasley Ave., City of Taconite	8,800' to the SSW	Construction Impacts; Rail Operations Impacts
AAC-7. North side of Twin Lakes; near City of Marble	14,800' to the southeast	Construction Impacts; Rail Operations Impacts
AAC-8. Between O'Reilly Lake & Island Lake (off Reilly Beach Rd.)	11,260' to the northwest	Construction Impacts; Rail Operations Impacts

7.9.3 Existing Noise Levels

Noise monitoring was completed at five locations throughout the West Range Site. All monitoring was completed using a Type II, ANSI approved noise level meter with calibration performed before and after each monitoring cycle.

A windscreen was used to counter any wind effects and no monitoring was performed during times when winds greater than 15 mph were measured, or when precipitation was occurring. Monitoring at each location was performed for no less than one hour and during both times specified as "night" and "day" by MPCA classification.

Table 7.9-3 and Table 7.9-4 summarize ambient noise conditions for the time frames measured in and around the West Range Site, and for daytime and nighttime conditions, respectively.

Table 7.9-3 Monitored levels at all Receptors, Daytime

Daytime Receptors	L_{10}	L_{50}	L_{10} dB over State Compliance	L_{50} dB over State Compliance
Receptor 1, 1700' West, Day (9:15AM-10: 15AM)	53dBA	52dBA	0dB	0dB
Receptor 2, 3900' SE, Day (3:15PM-4: 15PM)	54dBA	53dBA	0dB	0dB
Receptor 3, 3900' West, Day (1:03PM-2: 04PM)	59dBA	55dBA	0dB	0dB
Receptor 4, 4400' West, Day (2:30PM-3:30PM)	59dBA	52dBA	0dB	0dB
Receptor 4, 4100' SE, Day (4:00PM-5:00PM)	51dBA	50dBA	0dB	0dB

Table 7.9-4 Monitored Levels at all Receptors, Nighttime

Nighttime Receptors	L_{10}	L_{50}	L_{10} dB over State Compliance	L_{50} dB over State Compliance
Receptor 1, 1700' West, Night (10:04PM-11:04PM)	51dBA	49dBA	0dB	0dB
Receptor 2, 3900' SE, Night (11:15PM-12: 16AM)	50dBA	49dBA	0dB	0dB
Receptor 3, 3900' West, Night (10:23PM-11:23PM)	59dBA	55dBA	4dB	0dB
Receptor 4, 4400' West, Night (11:45PM-12:45PM)	56dBA	53dBA	1dB	3dB
Receptor 5, 4100' SE, Night (from Receptor 2)	50dBA	49dBA	0dB	0dB

All locations, with the exception of Receptors 3 and 4, experience ambient noise conditions at or below state and federal standards during the most critical nighttime hours (10PM-7AM). Receptors 3 and 4 experience ambient noise levels above state standards during nighttime conditions, likely due to their proximity to CR 7.

Noise levels at all locations are typical for townships and locales of this size, and below those of typical urban environments in close proximity to major transportation corridors. Detailed descriptions of the monitoring locations and conditions are provided in Section 2.11 of the ES.

7.9.4 Construction Noise Impacts

The construction process for the IGCC Power Station and Associated Facilities would be expected to generate noise during the following phases:

- Site Preparation
- Excavation
- Foundation Placement
- Plant and Building Construction
- Exterior Finish and Cleanup

Equipment utilized during the construction process would differ from phase to phase. In general, heavy equipment (bulldozers, scrapers, dump trucks, and concrete mixers) will be used during excavation and concrete pouring activities. Most other phases involve the delivery and erection of the building and equipment components. The Applicant assumed that there will be no driven piles during the construction process, although the necessity for such construction activity has not been finally determined.

Noise associated with the construction of the Project will be attenuated in a variety of ways. The most significant is the divergence of the sound waves with distance (attenuation by divergence). In general, this mechanism results in a 6 dBA decrease in the sound level with every doubling of distance from the source. For example, the 84 dBA average sound level (at 50 feet) associated with clearing and grading will be attenuated to 78 dBA at 100 feet, 72 dBA at 200 feet, and to 66 dBA at 400 feet. Noise attenuation from dampening due to ground effects was not included in the construction noise analysis to allow for some conservatism.

During final construction, a method used for testing and cleaning steam piping called "steam blows" creates substantial noise. A steam blow results when high-pressure steam is allowed to escape into the atmosphere when cleaning the steam piping. A series of short steam blows, lasting two or three minutes each, will be performed several times daily over a period of two or three weeks. Steam blows can produce noise as loud as 130 dBA at a distance of 100 feet. Under such circumstances, the resultant sound level at the nearby receptors would range from 86 to 103 dBA. To minimize these short-term temporary noise impacts, the piping would be equipped with silencers that would reduce noise levels by 20 dBA to 30 dBA at each receptor location. Detailed information regarding methods to mitigate noise can be found in Section 2.11 of the ES.

Although nighttime construction is not currently anticipated, if construction activities took place during the nighttime hours (after 10:00 p.m.), noise would be expected to exceed the MPCA residential nighttime noise limit of 50 dBA (L_{50}) at receptor locations R2 and R3, and could cause a significant impact.

Although construction noise would be below daytime state standards, because of its transitory nature and common fluctuations in the background noise level, construction activity will occasionally be discernable at the nearest receptors. Given ideal atmospheric conditions for noise propagation, construction noise could be discernable at the receptors located furthest from the IGCC Power Station Footprint and Buffer Land.

Rail line construction will encroach within 500 feet of Receptors R2 and R5. Construction noise would be expected to range from 57 to 69 dBA during the short period that the linear construction operation is nearest to the homes represented by each of these receptors. Blasting of rock will be required for some of the cuts needed to establish an acceptable grade for the rail track and will affect local residents. Because of the temporary nature of the linear construction activities, rail construction noise will result in short-term temporary noise impacts. However, these impacts would be diminished once the construction operation moves away from Receptors R2 and R5.

7.9.5 Operating Noise Impacts

7.9.5.1 Methodology

A proprietary computerized noise prediction program was used to simulate and model the operation of IGCC Power Station equipment. The modeling program uses industry-accepted propagation algorithms based on ANSI and ISO standards (ISO 9613, ISO 1913 (Part 1), ANSI 126, or ISO 3891). The calculations account for classical sound wave divergence (spherical spreading loss with adjustments for source directivity from point sources) plus attenuation factors due to air absorption, ground effects, and barriers/shielding.

Calculations were performed using octave band sound power levels (abbreviated PWL or Lw) as inputs from each noise source. The computer outputs are in terms of octave band and overall A-weighted noise levels (sound pressure levels, abbreviated SPL or Lp) at discrete receptor positions or at grid map nodes (in preparation for computing a contour map). The output listing is ranked by relative noise contribution from each noise source.

The IGCC Power Station was assumed to operate 24 hours per day at its design capacity, meaning that its noise output would be constant, regardless of time-of-day (and, thus, the statistical sound levels would all be the same – that is, L100=L90=L50=L10). Major buildings, as well as the stepped terracing, were included as barriers to account for propagation losses due to shielding between a given noise source and a receptor location. However, for conservatism, low-lying buildings (such as power distribution centers and water treatment buildings) and the coal piles were not included in the provision for shielding benefits.

A total of 11 receptor locations – four along the boundary of the Buffer Land and seven at locations outside of the Buffer Land – were used for the predictive analyses to assess future noise conditions due to the proposed equipment. The modeled off-site receptor locations were the same as the ambient measurement locations used by the Applicant and reported in Section 2.11 of the ES. See Section 2.11 of the ES for further details.

7.9.5.2 Results

The noise model was run for the base case plant configuration for both Phase I only and for the combined Phase I and Phase II operations. The dominant noise sources for the base case configuration included HRSG and ASU stack exits, large buildings with major process equipment inside (including the GTG and STG buildings, the ASU buildings, Rod Mill buildings, and Slurry Feed buildings) Acid and Tail Gas burners, Power Block and ASU cooling towers, and several large water-handling pumps.

Because noise standards were exceeded under the base case assumptions, the Applicant evaluated ways to further reduce noise from plant operations. Specifically, the Applicant's noise consultant, ACC, evaluated a mix of low-noise designs for some equipment items, using available noise control technologies (such as stack silencers), and applying external treatments such as enclosures or noise control panels on selected building walls. The ACC-suggested noise mitigation for the IGCC Power Station is summarized in Table 7.9-5. With the proposed mitigation, the facility is predicted to meet state standards (both L_{50} and L_{10}) at all receptors that currently meet these standards (L_{10} noise levels at Receptors 3 and 4 are already above the

MPCA nighttime limits due to roadway traffic on CR 7, and the noise from the IGCC Power Station would not increase noise at these sites.). With the proposed mitigation, noise levels would not increase at any nearby residence by more than one decibel, which is an imperceptible increase.

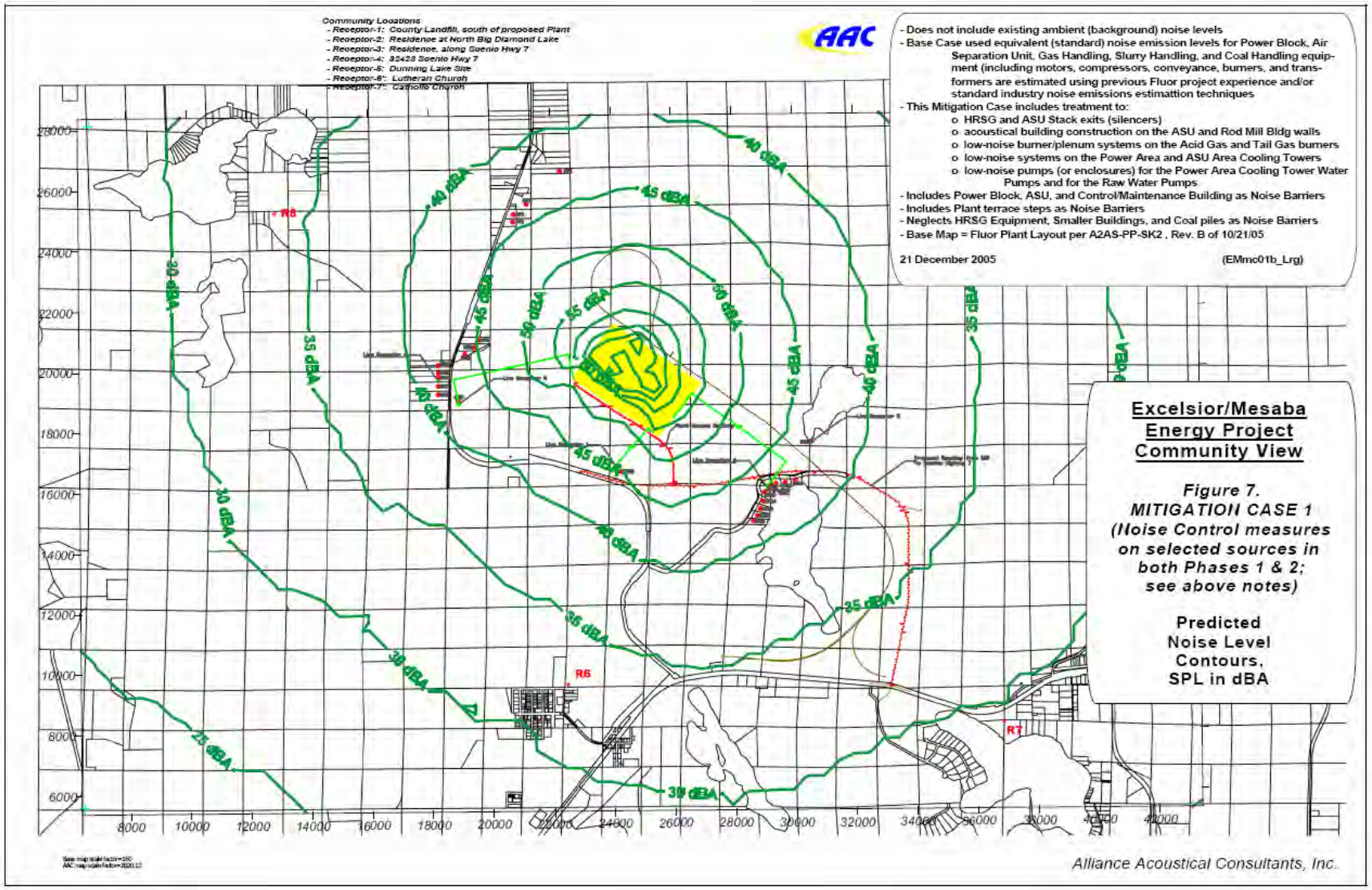
The predicted noise contours around the IGCC Power Station, with the suggested noise mitigation, are shown in Figure 7.9-6 below. While the ACC-recommended noise mitigation is listed below, the actual noise mitigation to be used for the IGCC Power Station to meet the applicable noise limits will be determined during final design. Details of the noise mitigation measures and the levels resulting from the modeling can be found in Section 2.11 of the ES.

Table 7.9-5
Summary of Noise Mitigation Project Design Features

Noise Source (Original Noise Emissions Rating)	Conceptual Noise Mitigation Feature(s)
Power Block Cooling Tower (60 dBA at 400' from tower edge)	Reduced 6 dB to 54 dBA at 400' from tower edge. Tower vendors can use a combination of slower-speed fans with special blade design, low-noise drive systems, splash control features, and/or tower baffling materials.
Gas Turbine, Steam Turbine, & HRSG 2-on-1 Power Island (70 dBA at 400' from island envelope)	(a) Include acoustical panel specifications for GTG and STG buildings walls in the detailed design such that interior space noise levels are adequately absorbed and encased within these building shells. (b) Specify GTG components that are outside buildings to be less than 90 dBA at 3 feet from the equipment surface envelope, as an aggregate.
HRSG Stack Exit (alone) (60 dBA at 400')	Reduced 10 dB to 50 dBA at 400' from stack base. Power Island vendor should use a stack silencer (either before or after the up-turn bend) to reduce HRSG stack noise.
Power Block Cooling Tower Pumps (94 dBA at 1')	Reduced 6 dB to ≤88 dBA at 1'. Can be accomplished via noise limit specification to equipment vendor (for a quiet design). As an alternative, install an acoustical enclosure around the pump and drive mechanics.
ASU System (varies)	(a) Include acoustical panel specifications for ASU building walls in the detailed design such that interior space noise levels are adequately absorbed and encased within the building shell. (b) Specify ASU components that are outside buildings to be less than 90 dBA at 3 feet from the equipment surface envelope, as an aggregate.
ASU Stack Exit (alone) (50 dBA at 400')	Reduced 10 dB to 40 dBA at 400' from stack base. ASU System vendor should use a stack silencer to reduce stack noise.

Noise Source (Original Noise Emissions Rating)	Conceptual Noise Mitigation Feature(s)
Rail Dumping Building (73 dBA at 50')	Assumes acoustical panel specifications for building walls in the detailed design such that interior space noise levels are adequately absorbed and encased within the building shell to meet the assumed emissions levels.
Slurry Feed and Slurry Prep Building (60 dBA at 50')	Same as immediately above.
Slag Handling Building (65 dBA at 50')	Same as immediately above.
Rod Mill Building (75 dBA at 50')	Reduced 10 dB to 65 dBA at 50' from any building facade. Specify acoustical panel specifications for Rod Mill building walls in the detailed design such that interior space noise levels are adequately absorbed and encased within the building shell to meet the reduced emissions levels.
SynGas and TailGas Burners (96 dBA at 3')	Reduced 10 dB to 86 dBA at 3' from the burner box. Specify low-noise burners to equipment vendors or use noise control enclosures/ plenums around burner systems.
Raw Water Pump Sets (91 dBA at 3')	Reduced 10 dB to 81 dBA at 3' from the pump set envelope. Noise limit specification to equipment vendor to supply either quiet-design pump sets or to utilize equipment enclosure.
All other Mechanical Equipment not specified above (various)	Noise limit specification to equipment vendor; no more than 85 dBA at 3’.
All building HVAC units and fans (various)	Noise limit specification to equipment vendor; no more than 85 dBA at 3’.

Figure 7.9-1 Noise Level Mitigation Modeling Results



7.9.6 Vehicular Traffic Noise Impacts

Traffic noise analysis was performed according to Federal Highway Administration (FHWA), Mn/DOT, and MPCA guidelines with regard to noise in and around proposed neighborhoods affected by proposed road improvements. The methods used are in accordance with State and Federal mandated noise simulation methods and on-site measurement. Specifically, the MINNOISE model was used as a basis for identifying potential noise impacts along the corridor, in conjunction with on-site measurement of traffic noise during peak hours.

On-site ambient measurement sites were used as a basis for modeled results and included in the modeled receptor sites. The measurement sites include areas of existing residential housing and common use areas regarded by Federal standards as Federal Activity Category B. For reference, FHWA Noise Abatement Criteria is listed in Table 7.9-6.

Table 7.9-6
FHWA Noise Abatement Criteria

Table 5: Noise Abatement Criteria (NAC)
Hourly A-Weighted Sound Level in Decibels (dBA)*

Activity Category	$L_{eq}(h)$	$L_{10}(h)$	Description of Activity Category
A	57 (Exterior)	60 (Exterior)	Lands on which serenity and quiet are of extraordinary significance and serve an important public need and where the preservation of those qualities is essential if the area is to continue to serve its intended purpose.
B	67 (Exterior)	70 (Exterior)	Picnic areas, recreation areas, playgrounds, active sports areas, parks, residences, motels, hotels, schools, churches, libraries, and hospitals.
C	72 (Exterior)	75 (Exterior)	Developed lands, properties, or activities not included in Categories A or B above.
D	--	--	Undeveloped lands.
E	52 (Interior)	55 (Interior)	Residences, motels, hotels, public meeting rooms, schools, churches, libraries, hospitals, and auditoriums.

* Either $L_{eq}(h)$ or $L_{10}(h)$ (but not both) may be used on a project.

In accordance with FHWA requirements, Mn/DOT has adopted a statewide noise policy that clarifies the FHWA terminologies of noise impacts. “Mn/DOT Noise Policy for Type I and Type II Federal-aid Projects as per 23 C.F.R. 772” includes the following descriptions:

- Noise Level Approaching the NAC; Mn/DOT defines a level as “approaching” the criterion level when it is 1dB, or less, below the criterion level. For example, 69 dBA is considered “approaching” the FHWA NAC category B level of 70 dBA.
- Substantial Increase in Noise; Mn/DOT defines a substantial increase in noise as those future predicted noise levels that exceed the FHWA NAC category B level of 70 by 5dB or greater, or 75dBA.
- Substantial Noise Reduction; Mn/DOT identifies feasibility requirements for the use of abatement procedures such as noise walls and their associated costs. These requirements require that every reasonable effort be made to obtain a substantial noise reduction. Mn/DOT defines a substantial noise reduction as 5dBA or more from a noise impact.

The augmented FHWA noise prediction software MINNOISE was used to predict noise levels at 20 receptor sites along the study corridor. Ten of the receptors were placed in and around Big Diamond and Dunning Lakes to represent residences in close proximity to the proposed roadway. The remaining receptors were placed at other residential locations near the proposed roadway. All modeled results were judged using the L10 metric, as both federal and state guidelines specify only one metric be used when determining impacts, with the L10 standard being commonly utilized in both federal and state guidelines.

Due to the proximity of the proposed access roadway running between Big Diamond Lake and Dunning Lake, the following noise issues were identified by the model:

- The nighttime state L_{10} standard was exceeded at ten receptor sites during construction. This construction period noise impact would be temporary; that is, limited to the construction period.
- The "Daytime" L10 standard was exceeded during construction at one receptor, MR19, because it is 275 feet from the proposed roadway. This construction period noise impact would also be temporary. Nighttime state L10 standards were exceeded at five receptors during plant operation.

The modeled operating noise levels at the five receptors are predicted to be "substantially" above current noise levels in the project design year. "Substantial" is defined as a 5dB or greater increase in noise. Therefore, a mitigation analysis was performed to determine whether noise walls would be cost effective in accordance with Mn/DOT cost reasonableness criteria. "Cost reasonableness" defines a ratio of cost per square foot to effectiveness of attenuation. This is determined by dividing the total cost of a wall (Mn/DOT currently estimates this as $15 per square foot) by the total decibel reduction for residences that are predicted to have a >5dBA reduction in noise. As described in detail in the ES, a 2,200-foot wall, placed between receptors MR15-MR19, which are homes located to the north of Big Diamond Lake, would cost about $660,000—or a price per total decibel reduction of $20,625.

Since the proposed access roadway would be constructed and owned by Itasca County (See Section 3.5.1.1.1), it would be designed to meet Minnesota State Aid standards for construction of roadways eligible for state funding. Under these standards, the criteria for a noise wall must meet MnDOT minimum of $3,250 per total decibel reduction. The hypothetical 20-foot noise wall would not meet the MnDOT minimum criteria and is therefore not considered feasible.

7.9.7 Railroad Noise and Vibration Impacts

Freight train noise levels would range from 38 to 58 dBA at the Receptor locations during a train pass-by. Typical daytime background noise levels were measured to be in the low 50's dBA (L_{50}). Some instances of train pass-bys would be noticeable at receptors with quieter background noise levels, but the noise would not be expected to contribute appreciably to the ambient background on an hourly or 24-hour basis. Further, the maximum noise levels generated by freight train operations would be clearly below the ATPA guideline of 70 dBA at each residential receptor location and would not be considered significant.

Vibrations generated by the Project's future rail operations were estimated by AAC for the surrounding receptors using FRA and FTA methodologies. Adjustments were also made to the vibration calculations to conservatively account for stiff rail car suspension systems, welded rail, train speed, and efficient soil propagation conditions. Vibrations at the nearest receptors were not predicted to be greater than FRA guidelines.

Horn soundings would be expected to be clearly audible to the nearest residential receptors. Because train horns are a requirement of the FRA, the noise impact would be considered an unavoidable adverse noise impact.

Noise generated by rail yard operations was also estimated by AAC. The noise from yard activities, involving loading and unloading of freight trains, would be greatly attenuated due to the distance between the nearby receptors and the yard. Rail yard noise is estimated to be between 8 to 23 dBA at the nearby residences. When compared to the FRA and ATPA noise guidelines, noise generated by yard operations would not be expected to be significant.

In summary, significant impacts from freight rail operations are expected to be limited to the audibility of train horns, which noise is unavoidable due to FRA regulations.

7.10 TRANSPORTATION AND TRAFFIC

7.10.1 Roads

The proposed 3.2-mile access roadway (Access Road 1) that will serve the IGCC Power Station will be constructed and owned by Itasca County. The new two-lane roadway starts at a new access point on State Highway 169, approximately 7,000 feet east of CR 7. The new road will cross underneath the adjacent rail line and proceed north, then curve west between Big Diamond and Dunning Lakes before terminating as it connects with CR 7, just southwest of the plant site. The section of existing CR 7 between the new connection and U.S. 169 will remain in place, and the only modifications required will be to orient the north end of CR 7 to meet the new connection perpendicularly. About 800' to 1000' of the existing curve between the east/west section and the north/south connection of CR 7 will then be abandoned and reclaimed.

The plant would be served by a 4,900-foot paved driveway (Access Road 2), about 32 feet wide, connecting the plant site with Access Road 1. This proposed access roadway is shown above in Figure 2.1-3.

Access to the HVTL, natural gas pipeline, and other utility corridors will come from various existing roadways at points that they are crossed by the proposed utilities. As design and construction progress, there could be a need for temporary access roads to be constructed to facilitate utility construction.

One benefit of the proposed Access Road 1 is that it would give local residents north of the IGCC Power Station a new route alternative when traveling east on State Highway 169. It will also reduce traffic volumes on the southerly portion of County CR 7, an area that has experienced problems with slope stability. In addition, the new roadway will have a better

intersection with State Highway 169 than County CR 7, including longer sight distance and flatter grades.

Access roads will have wetland impacts and also pass near several residences in the area between Big Diamond and Dunning Lakes. Noise, wetland, and other impacts on existing land use/land cover as a result of roadway construction are discussed in more detail in Section 7.9, Section 7.7, and Section 7.1, respectively.

7.10.2 Traffic Impacts

The predicted 2028 traffic forecast indicates that if Mesaba One and Mesaba Two are built at the West Range Site, traffic on State Highway 169 and C.H. 7 (north of Site) will only be slightly higher than that forecast under the 1.5% annual growth No-Build scenario. The proposed access roadway between State Highway 169 and the plant site will see its highest volumes (about 3,100 vehicles per day) during peak construction (2008), then will drop off to an estimated 1,550 vehicles per day. The section of existing County Highway 7 that lies between the IGCC Power Station and State Highway 169 will see its volume drop by more than 50% as traffic diverts to the proposed access roadway. The forecast traffic volumes are shown below. These traffic forecasts assume that Itasca County constructs Access Road 1 before plant construction begins. If not, a longer driveway off of C.H. 7 would serve the plant until the proposed access road is built. Under either scenario, the existing or proposed roadways are adequate to handle predicted traffic.

Table 7.10.2
Forecast Traffic Volumes West Range Site

Year	T.H. 169		C.H. 7		New C.H. 7	
	West of CSAH 7	East of CH 7	North of New C.H. 7	South of New C.H. 7	West of Entrance	East of Entrance
2000	5,800	5,500	1,100	1,100	0	0
2002	6,500	5,800	N/A	N/A	0	0
2004	7,200	5,700	N/A	N/A	0	0
2008 Build	8,900	7,100	1,500	470	1250	3100
2028 Build	10,500	8,400	1,700	460	1250	3100

7.10.3 Railroad

The proposed railroad will be a single track using a 100 foot wide permanent right-of-way. As provided in Table 3.5-2, railroad tracks, especially tracks designed for unit coal trains, have limited parameters for curvature and grades. These criteria make it difficult to avoid impacts to the environment. Grading impacts, based on cut and fill sections, will vary from 60 feet to 760 feet wide. Except for the track bed, disturbed areas will be revegetated.

Current and expected train traffic on the BNSF and CN rail line that would serve the IGCC Power Station at the West Range Site is discussed in Section 3.5.2.2. About six trains per day currently travel on the BNSF line through Grand Rapids at speeds up to 25 miles per hour. Nine

grade crossings (a location where a public highway, road, street, or private roadway, including associated sidewalks and pathways, crosses one or more railroad tracks at grade) are located within the city limits of Grand Rapids and La Prairie. The track from Gunn to the West Range Site (about 12.5 miles in length) also operates at speeds of 25 miles per hour and has traditionally carried 4 to 10 trains per day. This track segment has another six public grade crossings.

Traveling at 25 miles per hour, a unit coal train would take about four minutes to clear a grade crossing. With both Phase I and Phase II operating at full load conditions using PRB coal, the IGCC Power Station is expected to require five unit coal train deliveries about every four days, or approximately nine full and empty unit train deliveries per week.

7.11 SOCIAL AND CULTURAL IMPACTS

7.11.1 Public Services

This section describes existing local government units located near the West Range Site that may be affected by the proposed project. It is divided into the following three subsections:

- Fire and Emergency Medical
- Police
- Utilities

7.11.1.1 Fire and Emergency Medical

Emergency services for the West Range Site would be primarily provided by the City of Taconite. Taconite has an Emergency Medical Technician (EMT) staff of seven volunteers that handle emergency medical services for the area. Taconite currently has a total of 14 volunteer fire department personnel. Ambulance service may be provided from Nashwauk or Grand Rapids, depending on exact location of the 911 caller. The nearest hospitals are the Itasca Memorial Center in Grand Rapids (13 miles) Hibbing (30 miles), and Bigfork (29 miles.) The City of Taconite also has a mutual aid agreement with nearby Cohasset and Grand Rapids. Itasca County provides additional emergency response as needed.

According to the City of Taconite, if the facility is built at the West Range Site, its volunteer fire department would likely have to be expanded from the current 14 up to perhaps a staff of 20, similar to the number of fire and emergency personnel available in Grand Rapids and Cohasset (The Cohasset fire and emergency response team of 21 has served the Minnesota Power Clay Boswell plant successfully for over 25 years, with a response requirement of three or four visits a year.). Also, as at the existing coal-fired power plant at Boswell, the City of Taconite would expect the Mesaba IGCC facility to train its own first responders and first aid specialists to respond until local emergency personnel arrive. In a large emergency, Itasca County, Grand Rapids, and Cohasset fire and EMT personnel would respond pursuant to existing mutual aid agreements with the City of Taconite.

7.11.1.2 Police

Itasca County provides police protection to the City of Taconite and the surrounding area. The Itasca County Sheriff's Office has 64 employees working as Jailers, Dispatchers, Clerical and Road Deputies. Itasca County employees have specialized training to work in the following areas: D.A.R.E. (Drug Abuse Resistance Education), pre-employment background investigation, Boat and Water Safety, Snowmobile Safety, Drug Task Force, Emergency Response Team, Dive Team, and special enforcement projects.

7.11.1.3 Utilities

The utilities in the rural areas are all on-site utilities including wells and septic systems. The City of Taconite uses wells to appropriate water and has a wastewater collection system that conveys wastewater to the joint Coleraine-Bovey-Taconite WWTF located in Coleraine. Potable water service and sanitary sewer collection system will be extended from the City of Taconite's existing utility systems to the IGCC facility site. The utility corridor will be approximately 12,400 feet in length and will affect about 17 acres.

There are also several private utilities that provide services to the area. QWest provides local telephone service. Natural gas and electricity are also provided by various utility companies and municipalities.

7.11.2 Archaeological and Historical Considerations

Section 106 of the National Historic Preservation Act requires consideration of impacts on historic, archaeological and cultural properties determined eligible for listing on the National Register of Historic Places (NRHP). The area of potential effects (APE) for archaeological resources is defined as all areas of potential effects from aspects of direct, physical impacts through the construction of the facility site itself, as well as its associated transportation systems (road and railroads), HVTL (high voltage transmission lines), gas pipelines and other associated utilities. The potential area of impact due to transmission line construction includes not only the area within the right-of-way but also nearby areas used during project construction. Specifically, the recommended APE for the architectural history resources extends to 0.25 miles from the centerline of proposed HVTL routes along existing or new proposed rights of way.

7.11.2.1 Archaeological Resource Model and Survey

During June and July 2005, an initial screening-level cultural resources assessment of the West Range Site and portions of associated corridors was conducted. The project study area for the West Range Site is approximately 1,344 acres (544 hectares), and 4,970 acres (2,011 hectares) for associated corridors. Thus, a total of 6,314 acres (2,555 hectares) were evaluated.

This evaluation consisted of three major steps: a review of SHPO file, the development of a GIS based sensitivity model, and a limited field survey to verify model predictions. Background research was first conducted using the SHPO site files for information on previously identified archaeological sites and cultural resource surveys within one mile (1.6 kilometer [km]) of the project area. The archaeological sensitivity model was developed to establish areas of archaeological sensitivity within the West Range Site and associated corridors. A 10-mile radius

around the West Range Site was used to determine the type of cultural affiliations and locations of archaeological sites that could be encountered within the project area. Previously recorded sites located within the study area were grouped according to the model.

The model established a general set of criteria that were based upon previous work throughout Minnesota and conversations with the Minnesota State Historic Preservation Office. The criteria included undisturbed portions of the following areas:

- Within 500 feet (ft.) (150 meters [m]) of an existing or former water source (lake, pond, river, stream).
- Elevated, comparatively well-drained areas within, or immediately adjacent to, a marsh or wetland of 10 acres (4.0 hectares) or greater in extent.
- Topographically prominent areas that command a wide view of the surrounding Landscape.
- Areas adjacent to a known or suspected portage or transportation route.
- Located within 300 feet (100 m) of a previously reported site.
- Located within 300 feet (100 m) of a former or existing historic structure or feature (such as a building foundation or cellar depression).

Areas of sensitivity were ranked in terms of the frequency in which previously recorded sites occurred. Areas were then categorized in terms of high, moderate and low potential for the location of archaeological sites.

Based on this sensitivity model, the Applicant conducted a limited archaeological survey covering 31 acres of within the Station Footprint and Buffer Land (6 acres identified as high-potential; 25 acres as moderate). No archaeological resources were encountered in either the high or moderate potential areas identified. The complete report of this investigation is included in an Appendix to the ES.

The Minnesota SHPO and appropriate federal agencies and tribes will be consulted to address the proposed strategy area prior to any additional testing. Reports outlining the results of the investigation will be forwarded to the SHPO and other appropriate agencies for review and comment. Construction will not commence until appropriate consultation, identification, and treatment of historic, archaeological and cultural resources has occurred.

7.11.2.2 Architectural Resources

A Phase I architectural history survey was conducted within the project boundaries to identify previous studies conducted in or near the project area, and to identify expected resource types. As described below, for the HVTL, rail and road corridors, the recommended APE for the architectural history resources extends to 0.25 miles from the centerline. For underground pipelines, the APE for is limited to the width of the corridor itself.

Several properties currently listed or eligible to be on the National Register of Historic Places (NRHP) are located in the vicinity of the area of potential effect (APE), but no such properties are located within the APE. In addition, eleven architectural history properties within the recommended APE have been previously recorded in SHPO records. Two properties, the Great Northern Railway Nashwauk-Gunn Line, and the Duluth, Missabe & Northern Railway Alborn Branch have been determined eligible for listing on the NRHP. Two previously recorded properties no longer exist. As to the gas pipeline corridors, since the area of potential impact of the pipeline is limited to the corridor itself, no historic buildings will be affected as no buildings are located within the corridor of the proposed pipeline route.

To date, the level of potential impact to NRHP registered or eligible archaeological sites, resources, or architectural resources has not been determined. Scoping studies to be conducted as part of the PPSA are expected to help identify areas of special concern that may warrant further investigation. At that time studies will be commissioned to address any identified concerns. Reports outlining these investigations will be provided to SHPO and other appropriate agencies for review and comment. Construction will not commence until appropriate consultation, identification, and treatment of historic, archaeological and cultural resources has occurred.

7.11.2.3 Programmatic Agreement

All federally-recognized tribes with historic or current affiliation to Minnesota and the project area have been invited to participate in the consultation process, and become a signatory to a Programmatic Agreement. Initial consultation letters were sent in September 2005 from the Department of Energy to all federally recognized tribes that have expressed a cultural and historical interest in Minnesota. As of the date of this Application, one tribe has indicated an interest in participating in the review of the Project.

7.11.3 Population Trends and Demographics

This section summarizes demographic data for the area surrounding the West Range IGCC Power Station near the City of Taconite. Regional population trends, demographics, and racial justice issues are addressed in Section 6.1.

7.11.3.1 Taconite Population Trends

The population trends for the City of Taconite are shown below in Table 7.11-1. The data show that the population of Taconite has remained essentially constant since 1980.

Table 7.11.1
Population Trends Since 1980 for Taconite (West Range)

	1980	1990	2000	2004
Taconite	331	310	315	323

The regional area gets a large influx of temporary residents and visitors at lake cabins, resorts and campgrounds in the summer. These temporary residents are not counted in these population statistics, but they do impact the capacity of local government services to meet local needs.

7.11.3.2 Predicted Impacts

A new industrial facility of the magnitude of Mesaba One and Mesaba Two is expected to result in a positive impact on the area population. The most recent example of a baseload power plant's impact on rural Minnesota population trends comes from the Sherburne County Generating Plant's (Sherco) in the City of Becker, Minnesota. Prior to construction and operation of Sherco, Becker was a small rural community with few businesses. Now, after more than twenty years of operation of Sherco, the City has changed dramatically. Many businesses operate within the community and the City has become a magnet for commuters, increasing its population nearly four-fold since 1980. A similar, positive impact may occur from the construction and operation of Mesaba One and Two.

7.11.3.3 Demographics and Environmental Justice

The population of Taconite is over 95% white, with about 2.5% American Indian. To help determine whether the project could disproportionately impact minority or low-income residents, demographic data was evaluated for local site areas. A review of the demographics at the census block level also did not show any concentrations of minority or low income populations along the alternative transmission line route. The Applicant is not aware of any minority populations that are disproportionately affected in the area should the project be built at this site.

The racial demographics of the census tracts and block groups in the vicinity of the West Range Site and associated facilities (transmission lines, water lines, pipelines, etc.) were examined and compared with the demographics of Itasca County. The minority population in the census tract throughout the West Range Site, including the associated facility areas, ranges between 2.0% to 3.6%. The overall minority population for Itasca County is 4.1%. Therefore, the demographics of the block groups surrounding the site, the City of Taconite, and the surrounding area consist of minority population percentages that are slightly less than those found at the county or state levels. The percentage of population that earns above the poverty level is approximately the same in these block groups as in the rest of Itasca County and the Arrowhead Region. Based on this data, no significant numbers of minorities or low-income people are represented in the vicinity of the proposed project location, and it is unlikely that the project will create a disproportionate impact on minorities or those below the poverty line.

As described in Section 7.4 above and in the AERA Report attached as an appendix to the West Range IGCC Power Station Application for a Part 70 New Source Review Construction Authorization Permit (attached as Appendix 5), the Project's mercury emissions will not contribute appreciably to mercury concentrations in fish in nearby lakes. Therefore the IGCC Power Station will not disproportionately affect Native Americans or others in the area who may rely upon locally caught fish as a regular part of their diet.

8. EAST RANGE (ALTERNATE) SITE ENVIRONMENTAL IMPACTS

This Section describes the environmental impacts expected at the East Range Site. Section 2.1 of this Application describes the general location of the East Range Site. Section 2.6.1 describes the IGCC Power Station Footprint and Buffer Land as configured on the East Range Site. The Associated Facility corridors located outside of the Station Footprint are shown in Figure 2.1-4.

The East Range Preferred and Alternate HVTL Routes required to link Mesaba One and Mesaba Two to the Forbes Substation are described in Section 2.6.3 and depicted in Figures 2.6-4 through 2.6-17. As described in more detail in Section 2, two 345-kV HTVLs are proposed for the East Range Site to provide the necessary generator outlet interconnection between the IGCC Power Station and the Forbes Substation POI. Both 345-kV HVTLs would use existing 115-kV transmission ROW, and place both the new and old HVTLs on new steel pole structures. The ROWs within which the two existing 115-kV HVTLs are located are referred to as the 39L/37L Route and the 38L Route.

The route for the East Range Proposed Natural Gas Pipeline is described in Section 2.6.4 and depicted in Figure 2.6-18 (the East Range Natural Gas Pipeline Milepost Map) and in Figures 2.6-19 through 2.6-24. The Applicant is proposing this supply line as it is the only reasonable option for obtaining natural gas capacity at the East Range Site. The only alternative to using NNG for providing transportation of natural gas at the East Range Site would be to tap the GLG pipeline near Carlton, Minnesota. This tapping point would be 65 miles (in straight line distance) from the East Range Site and would require an entirely new route between Carlton and Hoyt Lakes. Given the option of working with NNG to deliver gas directly to the footprint of the East Range Site using an existing route, a new route alternative has little merit. As described in Section 1.8.2.8, NNG would permit this route under its blanket authorization from the FERC.

A detailed description of the environmental setting for the East Range Site is provided in Section 2 of the ES, and an assessment of the impacts associated with the construction and operation of the IGCC Power Station at the East Range Site is provided in Section 3 of the ES.

8.1 LAND USE

This section summarizes the direct land use impacts on the East Range Site, including activities related to construction and operation of the IGCC Power Station, its Associated Facilities, the Additional Lands, the Preferred and Alternate HVTL Routes, and the East Range Proposed Natural Gas Pipeline Route. The Applicant is providing a discussion of the impacts of constructing and operating the Proposed Natural Gas Pipeline even though it is not requesting a permit from the MPUC. A detailed land use/land cover map showing the IGCC Power Station Footprint and Buffer Land is provided in Figure 8.1-1 and a regional-scale land use/land cover map showing the Proposed and Alternate HVTL Routes and the East Range Proposed Natural Gas Pipeline Route is provided in Figure 8.1-2. Tables 8.1-1 and 8.1-2 summarize the predicted permanent and temporary land use impacts described in the subsections below for the IGCC Power Station, its Associated Facilities, the Additional Lands, Preferred and Alternate HVTL routes, and the Proposed Natural Gas Pipeline.

Section 2.8 of the ES provides a detailed description of existing local and regional land use and the information sources used in compiling such information. Section 3.7 of the ES provides a more detailed analysis of the permanent and temporary impacts that will result from development of the East Range Site. The following discussions summarize that information.

8.1.1 Historical Overview

The East Range Site has been disturbed through years of mining activity. Past and present mining activity in the area (the former LTV Mining Company location) is shown in Figure 8.1-3, which identifies the locations and extent of mine pits, waste-rock dumps and tailing basins in the vicinity of the East Range Site. The large area highlighted in yellow and located immediately to the west of the IGCC Power Station Footprint and Buffer Land is a 80-100 foot mound of waste rock approximately 300 acres in size that has been planted with natural grasses.

8.1.2 IGCC Power Station Power Station Footprint and Buffer Land

A description of the IGCC Power Station Footprint and Buffer Land is provided in Section 2.6.1.

No existing structures are located within the 810-acre parcel. The land use/land cover is primarily coniferous forest, mixed wood forest and regeneration/young forests. Surveyed wetlands also are present on the Station Footprint and Buffer Land.

Within the past year, much of this area has been clear-cut for timber production. The remaining forest cover is relatively young given that these lands have been harvested within the past 25 years. There is no old growth forest cover within or near the Station Footprint and Buffer Land. The upland forest composition and character demonstrates that the area has served as a timber source and has been impacted by timber production for several decades.

Permanent land use impacts from construction of Mesaba One and Two in the area of the Station Footprint are shown in Table 8.1-1. Such impacts are associated with clearing and grading the area to accommodate construction and to provide an acceptable grade allowing unit train access to the Power Station. Extensive cuts and filling will be required to create the proper grade for the railroad. Figure 3.2-5 in Section 3.2.3 shows the preliminary grading plan for the East Range IGCC Power Station. Figure 8.1-4 shows the configuration of the IGCC Power Station Footprint and the location of wetlands.

Approximately 142 acres of forested land and 16 acres of surveyed wetlands will be cleared to build the facility.

Figure 8.1-1 Land Use and Land Cover in the Vicinity of the East Range Site

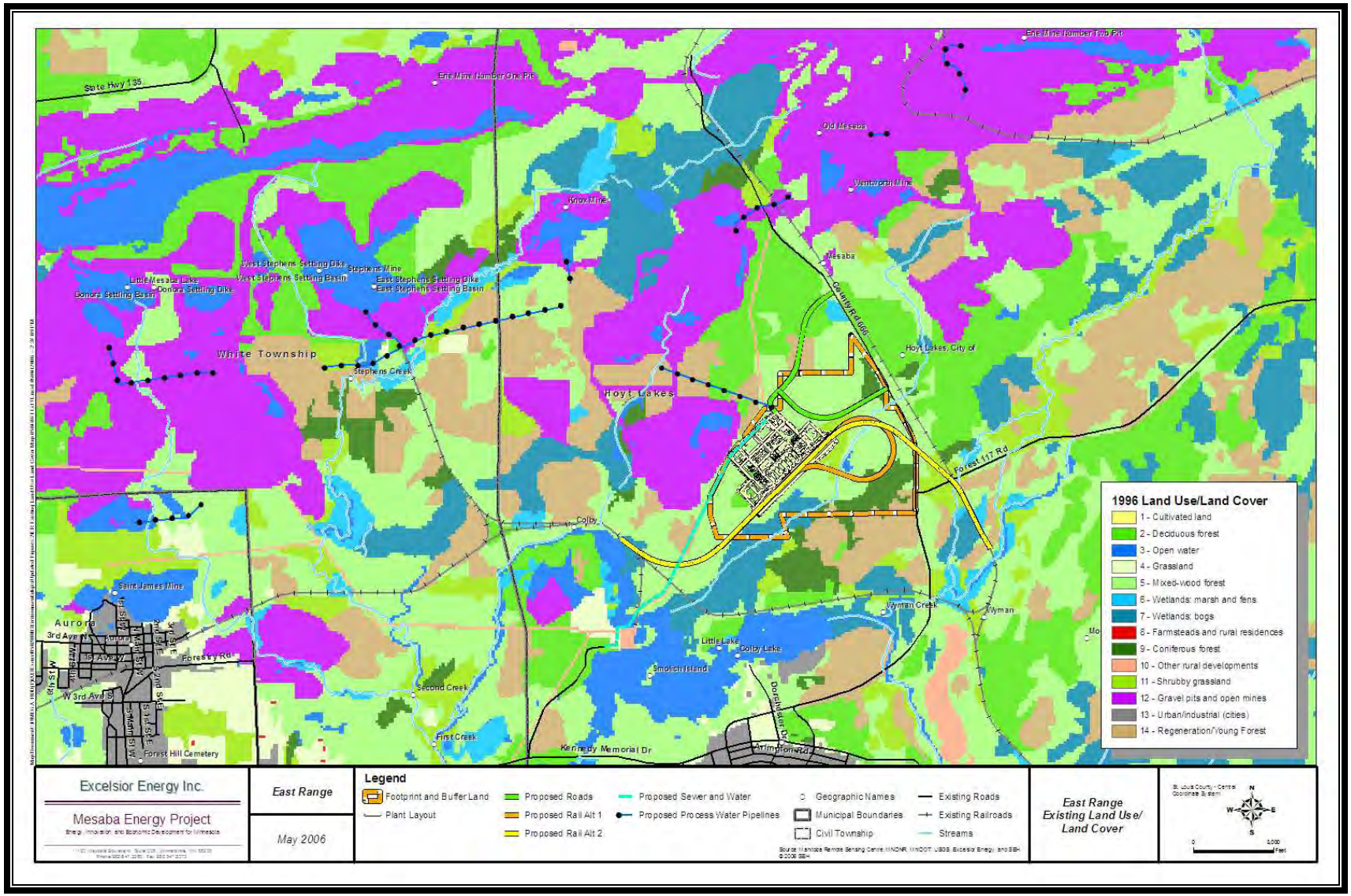


Figure 8.1-2 Land Use/Land Cover Map Showing the Preferred and Alternate HVTL Routes and East Range Proposed Natural Gas Pipeline Route

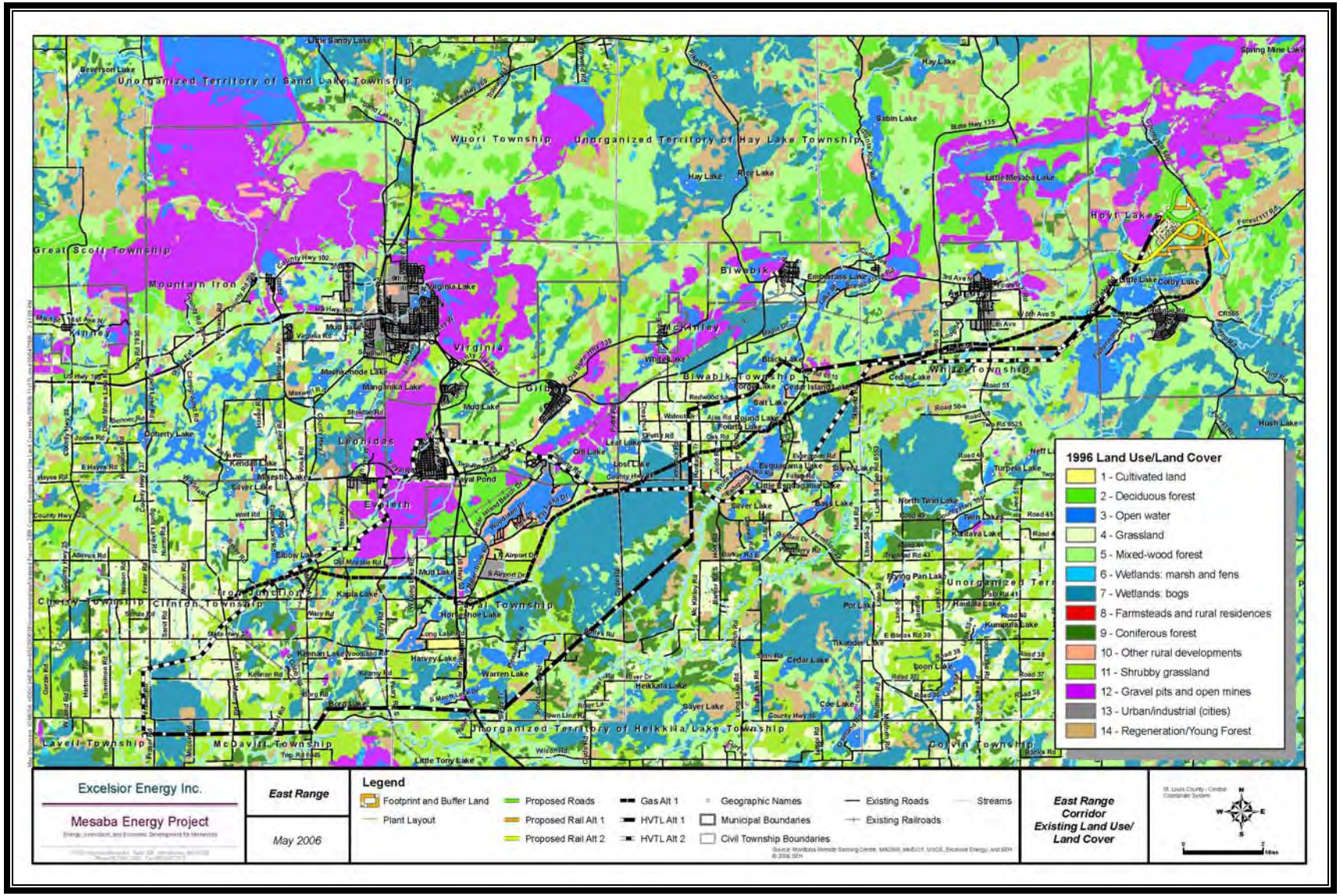


Table 8.1-1 East Range Site Land Use Permanent Impacts (Acres)

	East IGCC	Proposed HVTL Route	Alternative HVTL Route	Gas Pipeline	Process Water Lines (8 Segments)	Potable Water Lines	Rail Alt 1	Rail Alt 1 C. Loop	Rail Alt 2	Roads
Coniferous forest	0.0	2.5	4.0	5.6	0	0.2	0	0	0	1.5
Deciduous forest	22.4	2.4	0	3.5	3.4	0	0	0	0	4.8
Cultivated Farmland	0	0.2	0.0		0	0	0	0	0	0
Farmsteads / residences	0	0.5	0.2	0.7	0	0	0	0	0	0
Grassland	0	6.9	10.4	32.0	0	0.7	0	0	0	0
Gravel Pits and open mines	0	33.5	27.7	0.2	21.4	0	0	0	0	0
Mixed wood forest	119.7	44.5	50.3	84.5	21.8	4.4	22.1	30.4	21.8	12.8
NWI Wetlands	0	30.2	32.6	0	0	0	0	0	0	0
Open water	0.0	0.1	0	1.2	6.5	0.8	0.2	0.2	0.2	0
Other rural developments1	1.2	3.0	1.6	3.3	0.3	3.3	0.2	0	0.2	0
Regeneration/ Young Forests	0	15.5	12.1	38.3	11.2	0.8	0	0	0.8	3.8
Shrubby grassland	8.2	20.8	23.0	54.8	5.7	0	5.8	25.2	4.8	1.9
Wetlands - bogs	0.0	0	0	19.2	0	0	1.3		1.3	0
Surveyed Wetlands	15.6	0	0	0	0	0	10.7	47.9	13.4	3.2
Wetlands-Fens	0.0	0	0	0.9	0.3	0	0	0	0	0
Roads-improved trails	0	5.3	2.7	0.4	0	0	0	0	0	0
Total	**167.0**	**165.6**	**164.6**	**244.7**	**70.5**	**10.2**	**40.4**	**103.7**	**42.9**	**28.0**

Note (1) HVTL Impacts include entire area required for new right-of-way

Table 8.1-2 East Range Site Land Use Temporary Impacts (Acres)

	East IGCC	Proposed HVTL Route	Alternative HVTL Route	Gas Pipeline	Process Water Lines (8 Segments)	Potable Water Lines	Rail Alt 1	Rail Alt 1 C. Loop	Rail Alt 2	Roads
Coniferous forest	19.5	2.5	4.0	7.6	0	0	0	0	0	2.4
Deciduous forest	32	2.4	0	5.0	1.76	0.6	0	0	0	8.0
Cultivated Farmland	0	0.2	0	0	0	0	0	0	0	0
Farmsteads /residences	0	0.5	0.2	1.0	0	0	0	0	0	0
Grassland	0	6.9	10.4	46.1	0	1.8	0	0	0	0
Gravel Pits / mines	0	33.9	4.7	.24	31.3	0	0	0	0	0
Mixed wood forest	360	44.5	50.3	120.7	31.5	11.1	37.2	30.4	35.2	21.8
NWI-Wetlands	0	76.7	100.4	0	0	0	0	0	0	0
Open water	0.2	0.5	0	1.7	9.5	1.8	0.1	0.2	0.1	0
Other rural developments	6.1	242.8	247.3	4.8	0	8.2	.52	0	0.5	0
Regeneration/ Young Forests	9.2	15.5	12.1	53.8	8.6	1.9	0	0	0.7	6.2
Shrubby grassland	51.6	20.8	23.0	78.9	8.6	0	20.7	25.2	17.8	3.1
Surveyed Wetlands	329	0	0	0	0	0	17.2	47.9	18.4	5.5
Wetlands - bogs	0.2	0	0	28.2	0	0	1.1	0	1.1	0
Wetlands - marsh and fens	0	0	0	1.2	.44	0	0	0	0	0
Urban/Industrial	0	0	4.5	.53	0	0	0	0	0	0
Total	**807**	**456.8**	**454.6**	**349**	**92.1**	**25.5**	**76.8**	**104**	**73.8**	**47**

Figure 8.1-3 Historical Mining Disturbances in the Vicinity of the East Range Site

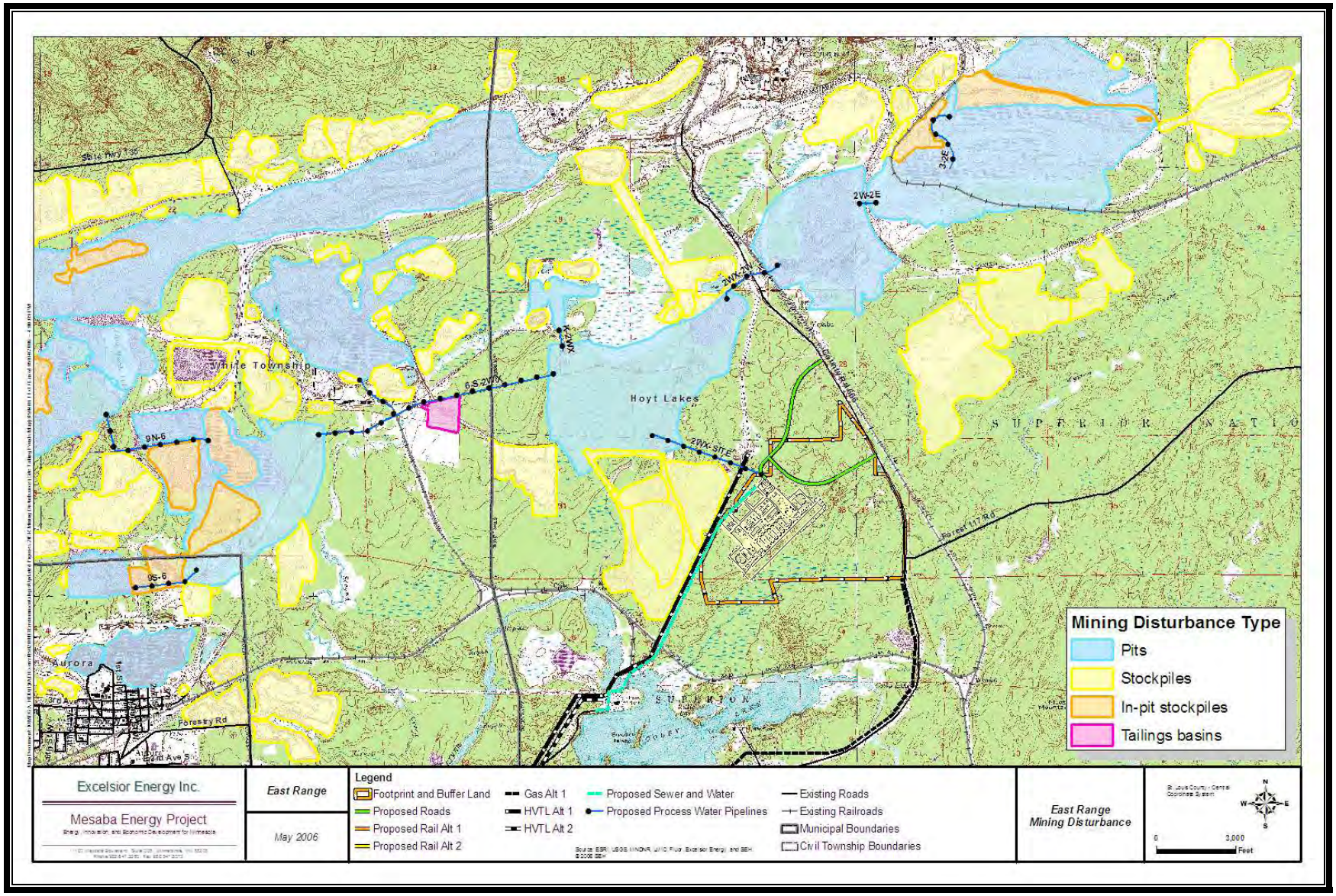
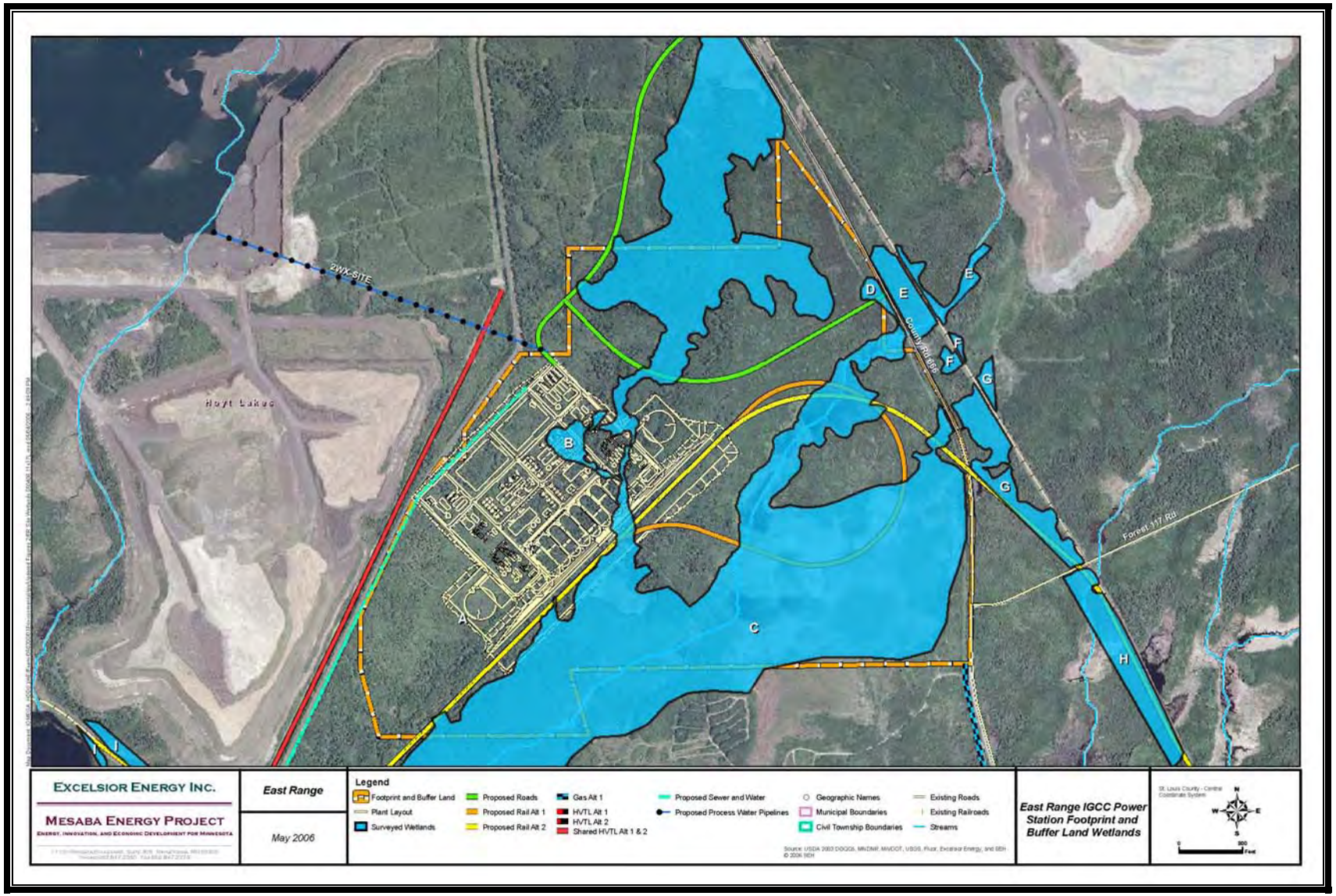


Figure 8.1-4 Distribution of Forested Areas and Wetlands Across the IGCC Power Station Footprint and Buffer Land



8.1.3 HVTL Routes

8.1.3.1 Preferred HVTL Route Configuration

The routes proposed for the two 345-kV HVTL needed for development at the East Range Site are described in Section 2.6.3 and shown with milepost markings in Figures 2.6-4 through 2.6-17. A milepost map superimposed on a USGS map is provided for the two routes in Figure 2.2-5. The design of the HVTL structures that will be used in each route and the ROW required to accommodate such structures are provided in Section 4. Total permanent and temporary impacts are shown in Tables 8.1-1 and 8.1-2 above. The two routes generally follow existing MP 115-kV HVTLs in the 39L/37L and 38L Routes.

8.1.3.1.1 39L/37L Route

This corridor of the Preferred 345-kV HVTL Route extends about 35.5 miles from the IGCC Power Station Footprint to the Forbes Substation and requires about 4 miles of new 150-foot wide right-of-way. The remaining 31 miles of the Preferred HVTL Route would share the existing ROW with the existing 39L/37L 115kV HVTLs. Along the 39L/37L Route, an additional 30 feet of new ROW adjacent to and contiguous with the existing 39L/37L Route is required. The details regarding specific right-of-way requirements for each section of the new line are provided in Section 4. After construction of the new 345kV HVTL, the existing 115-kV HVTL conductors would be moved to the new structures, and the old structures would be permanently removed. Some of the right-of-way in the existing 39L/37L Route may be allowed to revert to its original land use, although that has not been accounted for in the land use impact estimates.

The 39L/37L Route passes through forest land, shrub swamp wetlands, wooded swamps and seasonally flooded basins or flats. Table 8.1-3 below identifies the land uses/land cover that will be permanently impacted by taking the additional 30 feet of ROW along the 39L/37L Route.

Table 8.1-3
Permanent Impacts Associated with Expansion of the 39L/37L Route

Land Use/Land Cover Category	Permanent Impacts			Potential to Re-establish Vegetation (acres)
	30' ROW Along Entire Route (acres)	New ROW Linking 39L to 37L (acres)	Total Impact (acres)	
Coniferous forest	2.5	--	2.5	
Deciduous forest	2.4	--	2.4	
Farmlands and rural residences	0.5	--	0.5	
Grassland	6.9	--	6.9	
Gravel pits and open mines	6.1	23.6	29.7	0.1
Mixed wood forest	34.5	--	34.5	
Open water	0.1	--	0.1	0.2
Other rural developments	2.9	--	2.9	103.7

Land Use/Land Cover Category	Permanent Impacts			Potential to Re-establish Vegetation (acres)
	30' ROW Along Entire Route (acres)	New ROW Linking 39L to 37L (acres)	Total Impact (acres)	
Regeneration/Young Forests	13.3	--	13.3	
Shrubby grassland	17.3	--	17.3	
NWI-Wetlands	24.7	0.7	25.4	26.4
Roads-improved trails-rail lines	3.8	1.3	5.1	3.9
Cultivated land	0.2	--	0.2	

Some impacts identified in Table 8.1-3, although labeled as permanent, may be allowed to re-establish after the construction phase of the project such that the total impacts identified would be lessened. For example, approximately 6.9 acres of grassland will need to be cleared for the HVTL line but much of this would re-establish after the construction phase of the project.

8.1.3.1.2 38L Route

The 38L Route of the preferred configuration would be constructed almost entirely as a new double circuit HVTL with the existing 38L 115-kV HVTL, which runs between the proposed IGCC facility and the Forbes Substation. Under the preferred HVTL route configuration, the new single-pole double circuit line would be constructed within the existing right-of-way of the 38L HVTL.

8.1.3.1.3 Common New ROW

A short length of new ROW (150-feet wide) would be required for the initial 2 mile connection between the IGCC Power Station Footprint and the Syl Laskin Energy Center's Substation. Table 8.1-4 below identifies the land uses/land cover that would be permanently impacted by this new ROW. The impacts associated with this short length of ROW will be identical to the impact that would occur as part of the Alternate HVTL Route analyzed below in Section 8.1.3.2.

Table 8.1-4
Permanent Impacts Derived from Constructing Two Miles of New ROW Between the East Range IGCC Power Station and Syl Laskin Energy Center Substation

Land Use/Land Cover Category	New 2 Mile ROW (acres)
Coniferous forest	--
Deciduous forest	--
Farmlands and rural residences	--
Grassland	--
Gravel pits and open mines	3.8
Mixed wood forest	10

Land Use/Land Cover Category	New 2 Mile ROW (acres)
Open water	--
Other rural developments	0.1
Regeneration/Young Forests	2.2
Shrubby grassland	3.6
NWI-Wetlands	4.8
Roads-improved trails-rail lines	0.2

8.1.3.2 Alternate HVTL Route Configuration

Under the alternative route configuration, the additional 30 feet of ROW required for the new double circuit towers (a 345kV circuit and a 115kV circuit) would be taken along the 38L Route instead of the 39L/37L Route. Then, on the existing 39L/37L Route, the new double circuit HVTL would be placed entirely within the existing ROW (except for the two miles of new ROW between the IGCC Power Station and the Forbes Substation).

The areas impacted by this configuration include shrub swamp wetlands, wooded swamps, and seasonally flooded basins or flats. With the extra 30-feet of ROW taken along 38L, about 54 acres of forest land and about 30 acres of grassland would require clearing. Although labeled as permanent impact, some vegetation will be allowed to re-establish after the construction phase of the project.

A summary of the permanent impacts associated with taking the additional 30 feet of ROW along the existing 38L Route instead of the 39L/37L Route are provided in Table 8.1-5 below.

Table 8.1-5
Permanent Impacts from Taking 30 Feet of New ROW from the 38L Route

Land Use/Land Cover Category	Permanent Impacts	
	30' ROW Along Entire Route (acres)	Potential to Re-establish Vegetation (acres)
Coniferous forest	4.0	
Deciduous forest		
Farmlands and rural residences	0.2	
Grassland	10.4	0.4
Gravel pits and open mines	0.3	
Mixed wood forest	40.2	
Open water		
Other rural developments	1.5	98.5
Regeneration/Young Forests	9.9	

Land Use/Land Cover Category	Permanent Impacts	
	30' ROW Along Entire Route (acres)	Potential to Re-establish Vegetation (acres)
Shrubby grassland	19.4	
NWI-Wetlands	27.1	32.1
Roads-improved trails-rail lines	1.2	1.8

8.1.3.3 Justification of Preferred HVTL Route

Table 8.1-6 provides a summary and comparison of the permanent impacts that will occur along the existing 39L/37L Route and the land uses/land cover that will be permanently impacted by utilizing an additional 30 feet of ROW and the permanent impacts that would occur if an additional land was instead taken along the existing 38L Route.

Table 8.1-6
Permanent Impacts Comparison Between Taking 30 Feet of New ROW from the 39L/37L Route or from the 38L Route

Land Use/Land Cover Category	39L/37L (acres)	38L (acres)	Difference (39L/37L-38L)
Coniferous forest	2.5	4.0	-1.6
Deciduous forest	2.4	--	2.4
Farmlands and rural residences	0.5	0.2	0.3
Grassland	6.9	10.4	-3.5
Gravel pits and open mines	6.1	0.3	5.8
Mixed wood forest	34.5	40.2	-5.7
Open water	0.1	--	0.1
Other rural developments	2.9	1.5	1.4
Regeneration/Young Forests	13.3	9.9	3.4
Shrubby grassland	17.3	19.4	-1.9
NWI-Wetlands	24.7	27.1	-2.4
Roads-improved trails-rail lines	3.8	1.2	2.6
Cultivated land	0.2	--	0.2

The comparison shows that the preferred route configuration would impact less forest and wetlands than the alternative route configuration.

Again, some of the impacts labeled as permanent in Table 8.1-6 would be mitigated by allowing for the re-establishment of vegetative growth following the construction phase of the HVTL. For example, approximately 12 acres of grassland that will need to be cleared for the HVTL line will re-establish itself after construction.

8.1.4 East Range Proposed Natural Gas Pipeline Route

The East Range Proposed Natural Gas Pipeline Route will require a 100-foot ROW for construction activities and a 70-foot permanent ROW for maintenance. See Figure 8.1-2 for a detailed map showing existing land use/land cover and Tables 8.1-1 and 8.1-2 for the acreage and type of land impacted within the permanent and temporary ROWs.

NNG currently provides natural gas transportation to Cliffs Erie with a 10 inch diameter pipeline. The branch line taps NNG's 20-inch diameter pipeline that, in turn, taps the 36 inch line in Carlton, Minnesota owned by GLG. The tap of NNG's 20 inch pipeline occurs near the junction of St. Louis County Roads 454 and 315, about one mile due west of Iron Junction, Minnesota. From that point the 10 inch diameter branch line travels about 29 miles to where it abuts the eastern boundary of the Buffer Land.

Permanent impacts have already occurred given the construction and operation of the Cliffs Erie branch line route. The current ROW that NNG maintains on this route is 70 feet. NNG would follow the pre-construction process outlined in Section 1.8.2.8. As noted in Section 1.8.2.8, NNG would undertake this activity under the rules of the FERC. For purposes of conservatively establishing an upper limit on the impacts that could occur along this route, the impacts identified in Table 8.1-7 are described as if the pipeline did not currently exist and, thus, Table 8.1-7 provides the upper limit and marks each value with the "less than" symbol "<".

Table 8.1-7
East Range Proposed Natural Gas Pipeline Route:
Impacts to Land Use/Land Cover

Land Use/Land Cover	Temporary Impact (Acres)	Permanent Impact (Acres)
Coniferous forest	7.56	<5.63
Deciduous forest	5.00	<3.50
Farmsteads and rural residences	0.98	<0.74
Grassland	46.12	<31.97
Gravel pits and open mines	0.24	<0.24
Mixed wood forest	120.71	<84.54
Open water	1.69	<1.22
Other rural developments	4.80	<3.25
Regeneration/Young Forests	53.83	<38.25
Shrubby grassland	78.89	<54.79
Urban/industrial	0.53	<0.44
Wetlands - bogs	28.16	<19.24

Land Use/Land Cover	Temporary Impact (Acres)	Permanent Impact (Acres)
Wetlands - marsh and fens	1.19	<0.91
Total	349.70	<244.72

8.1.5 Process Water Supply Pipelines

The Process Water Supply Pipelines consist of nine separate segments connecting the East Range Water Resources (such Resources having been identified in Table 3.6-5 and shown in Figure 3.6-2), pumping stations (discussed in Section 3.6.2.3.3, and Table 3.6-10), and the IGCC Power Station. These pipeline segments are identified in Table 3.6-9 and are shown in Figure 2.1-5. The overall permanent and temporary land use impacts are provided in Tables 8.1-1 and 8.1-2.

8.1.5.1 Segment 1: 2WX-IGCC

Process Water Supply Pipeline Segment 1 would require a 100-foot permanent ROW and a 150-foot temporary ROW. Approximately 15 acres of forest lands would be cleared to build this process water line corridor, resulting in about 10 acres of permanent impact. The remaining 5 acres will be allowed to revert back to original condition.

8.1.5.2 Segment 2: 2W–2WX

Process Water Supply Pipeline Segment 2 would require a 100-foot permanent ROW and a 150-foot temporary ROW. Approximately 9 acres of gravel pits and open mines would be impacted to build this process water line corridor, resulting in about 6 acres of permanent impact. The remaining 3 acres would revert to original condition or use.

8.1.5.3 Segment 3: 2E–2W

Process Water Supply Pipeline Segment 3 would require a 100-foot permanent ROW and a 150-foot temporary ROW. Approximately 2.90 acres of gravel pits and open mines would be impacted to build this process water line corridor, resulting in about 1.85 acres of permanent impact. Approximately 1 acre would revert to original condition or use.

8.1.5.4 Segment 4: 3–2E

Process Water Supply Pipeline Segment 4 would require a 100-foot permanent ROW and a 150-foot temporary ROW. Approximately 10 acres of gravel pits and open mines would be impacted to build this process water line corridor, resulting in about 7 acres of permanent impact. Approximately 3 acres would revert to original condition or use.

8.1.5.5 Segment 5: K–2WX

Process Water Supply Pipeline Segment 5 would require a 100-foot permanent ROW and a 150-foot temporary ROW. Approximately 1.5 acres of forest lands would be cleared to build

this process water line corridor, resulting in about 1 acre of permanent impact. Approximately 1/2 acre would revert to original condition.

8.1.5.6 Segment 6: 6-S–2WX

Process Water Supply Pipeline Segment 6 would require a 100-foot permanent ROW and a 150-foot temporary ROW. Approximately 19 acres of forest lands would be cleared to build this process water line corridor, resulting in about 13 acres of permanent impact. Six acres would revert to original condition.

8.1.5.7 Segment 7: 9S–6

Process Water Supply Pipeline Segment 7 would require a 100-foot permanent ROW and a 150-foot temporary ROW. Approximately 5 acres of forest lands would be cleared to build this process water line corridor, resulting in about 4 acres of permanent impact. Approximately 1 acre would revert to original condition. Approximately 4 acres of open water would be impacted to build this corridor, resulting in about 3 acres of permanent impact.

8.1.5.8 Segment 8: 9N-6

Process Water Supply Pipeline Segment 8 would require a 100-foot permanent ROW and a 150-foot temporary ROW. Approximately 16 acres of forest lands would be cleared to build this process water line corridor, resulting in about 10 acres of permanent impact. Approximately 6 acres would revert to original condition.

8.1.5.9 Segment 9: Colby Lake–2WX

Process Water Supply Pipeline Segment 9 would use the existing Colby Lake pipeline extending from Cliffs Erie's Colby Lake pumphouse to the IGCC Power Station Footprint. At the Station Footprint, Process Water Supply Pipeline Segment 9 would be placed in the same excavation as Process Water Supply Pipeline Segment 1. Therefore, Segment 9 would not cause additional permanent impacts.

8.1.6 Process Water Blowdown Pipelines

Because discharges of cooling water blowdown would not be released at the East Range Site, no Process Water Blowdown Pipelines would be required to be constructed.

8.1.7 Potable Water and Sewer Pipelines

The East Range Process Water and Sewer Pipelines shown in Figure 2.1-5 would require a 40-foot permanent ROW and a 100-foot temporary ROW. Approximately 14 acres of forest land would be cleared to build the Potable Water and Sewer Pipelines corridor, resulting in about 5 acres of permanent forest impact. Nine acres would revert to original condition. Approximately 8 acres of other rural developments, including a private roadway, would be impacted, resulting in about 3 acres of permanent impact. The Potable Water and Sewer Pipelines would be buried alongside the roadway.

8.1.8 Rail Lines

Railroad Alternative 1 contains a loop track that would reduce a number of off-site impacts compared to Rail Line Alternative 2.

8.1.8.1 Rail Line Alternative 1

8.1.8.1.1 Approach to Rail Loop

Rail Line Alternative 1 would require a 100-foot permanent ROW and a 75 to 490-foot temporary ROW. Approximately 37 acres of forest land must be cleared to build the rail line Alternative 1 corridor, resulting in about 22 acres of permanent impact. Fifteen acres would revert to original condition. The total length of the Rail Line Alternative 1 corridor is 3.39 miles, and includes the trackage for the rail loop.

8.1.8.1.2 Rail Loop

The track for the rail loop would require a 100-foot permanent ROW, 50 feet of which would extend from the centerline of the track into the center of the loop (the other 50 feet of permanent ROW would extend from the centerline of the track outward). The area within the loop would not include the inner 50 feet of the trackage ROW. The upper limit of permanent impacts occurring within the rail loop assumes that none of the present land use/land cover can be maintained, in which case the permanent impacts would equal the entire area within the loop. The lower limit on permanent impacts assumes that 75 percent of the original land use/land cover designation can be maintained. Thus, the permanent impacts associated with construction would include 8 to 30 acres of mixed wood forests, 12 to 48 acres of wetlands, and 6 to 25 acres of shrubby grasslands. Impacts to land cover will be determined upon completion of final design and location of storage facilities within the rail loop.

8.1.8.1.3 Permanent Impacts

Total permanent land use/ land cover impacts for the Rail Line Alternative 1 are provided in Table 8.1-1.

8.1.8.2 Rail Line Alternative 2

Approximately 35 acres or 48 percent of this rail line corridor is mixed wood forest land, with surveyed wetlands making up about 18 acres or 25 percent of the corridor. Shrubby grassland comprises about 18 acres or 25 percent of the corridor. The total length of this corridor is 3.49 miles.

Rail Line Alternative 2 would require a 100-foot permanent ROW and a 75- to 490-foot temporary ROW. Approximately 36 acres of forest land would be cleared to build the Rail Line Alternative 2 corridor resulting in about 23 acres of permanent impact. Thirteen acres would revert to original condition.

8.1.9 Roads (Access Road 1)

The land use/land cover along the proposed East Range Access Road 1 corridor is primarily forest land. Approximately 38 acres or 82 percent of the access road corridor is forest land of some type. Shrubby grassland makes up 3.12 acres or 7 percent of the corridor and surveyed wetlands make up the remaining 5.52 acres of the corridor. The length of the road corridor is 1.91 miles.

Access Road 1 will require a 120-foot permanent ROW and a 200-foot temporary ROW. Approximately 44 acres of forest would be cleared to build the East Range road corridors resulting in about 26 acres of permanent impact.

Approximately 38 acres of forest land would be cleared to construct the East Range road corridors resulting in about 23 acres of permanent impact. Fifteen acres would revert to original condition. The permanent impacts on all land use/land cover types is summarized in Table 8.1.1.

8.1.10 Special Land Uses

8.1.10.1 Recreational Lands

The St. Louis County Plat Book and the St. Louis County Land Department provided information regarding publicly owned lands. According to the 1996 Land Use/Land Cover map there are no recreational areas within the East Range Site. The general area in the region of the site contains lakes that provide recreational opportunities for area residents. Activities such as swimming, boating, fishing, and bird watching are prevalent. Forested areas also allow for recreational activities such as hiking, biking, hunting, bird-watching and related activities. Many of these activities take place on land that is County owned but not specifically designated as a recreational area.

8.1.10.2 Designated Wildlife Areas

No designated federal Wildlife Refuges, Waterfowl Production Areas, or National Preserves are within or immediately adjacent to the East Range Site or its associated utility or transportation corridors. No MDNR Wildlife Management Areas (WMAs), State Natural Areas (SNAs), designated Game Lakes, or Designated Trout Streams are within or immediately adjacent to the East Range Site. Violations of the Fish and Wildlife Coordination Act will not occur as a result of the construction or operation of the project at the East Range Site.

8.1.10.3 Prime Farmland

In accordance with Minn. R. 4400.3450, subp. 4 (“Prime Farmland Exclusion”), “No large electric power generating plant site may be permitted where the developed portion of the plant site, excluding water storage reservoirs and cooling ponds, includes more than 0.5 acres of prime farmland per megawatt of net generating capacity, or where makeup water storage reservoir or cooling pond facilities include more than 0.5 acres of prime farmland per megawatt of net generating capacity, unless there is no feasible and prudent alternative.” "Prime farmland" means those soils that meet the specifications of Code of Federal Regulations 1980, title 7, section 657.5(a).

Subpart 4 also states that the provisions regarding the prime farmland exclusion do not apply to areas located within home rule charter or statutory cities; areas located within two miles of home rule charter or statutory cities of the first, second, and third class; or areas designated for orderly annexation under Minn. Stat. § 414.0325.

The IGCC Power Station Footprint and Buffer Land and many of the Station's Associated Facilities and Additional Lands are located entirely within the City limits of Hoyt Lakes, a statutory city. The Process Water Supply Pipeline Segment 7 is located within the City of Aurora, also a statutory city. The only Associated Facilities of the East Range Site that lie outside the City limits of Hoyt Lakes and Aurora are Segment 6 and Segment 8 of the Process Water Supply Pipeline. Therefore, the prime farmland exclusion does not apply to either the IGCC Power Station Footprint, Buffer Land, any of the Associated Facilities or Additional Lands except for the two identified Process Water Supply Pipeline Segments. The land area impacted by these two segments, assuming they were 100 percent prime farmland, would total 35 acres, a de minimis figure that would not be prohibited under the Statutes.

Additionally, farming is not widely practiced in the area. According to the 1996 Land Use/Land Cover map there is no widespread indication of cultivated farmland in the vicinity of the East Range Site. The Land Use/Land Cover map shows one tract of cultivated farmland in Fayal Township, but it is more than 20 miles west southwest of Hoyt Lakes (see Figure 8.1-1 for the location of this cultivated land).

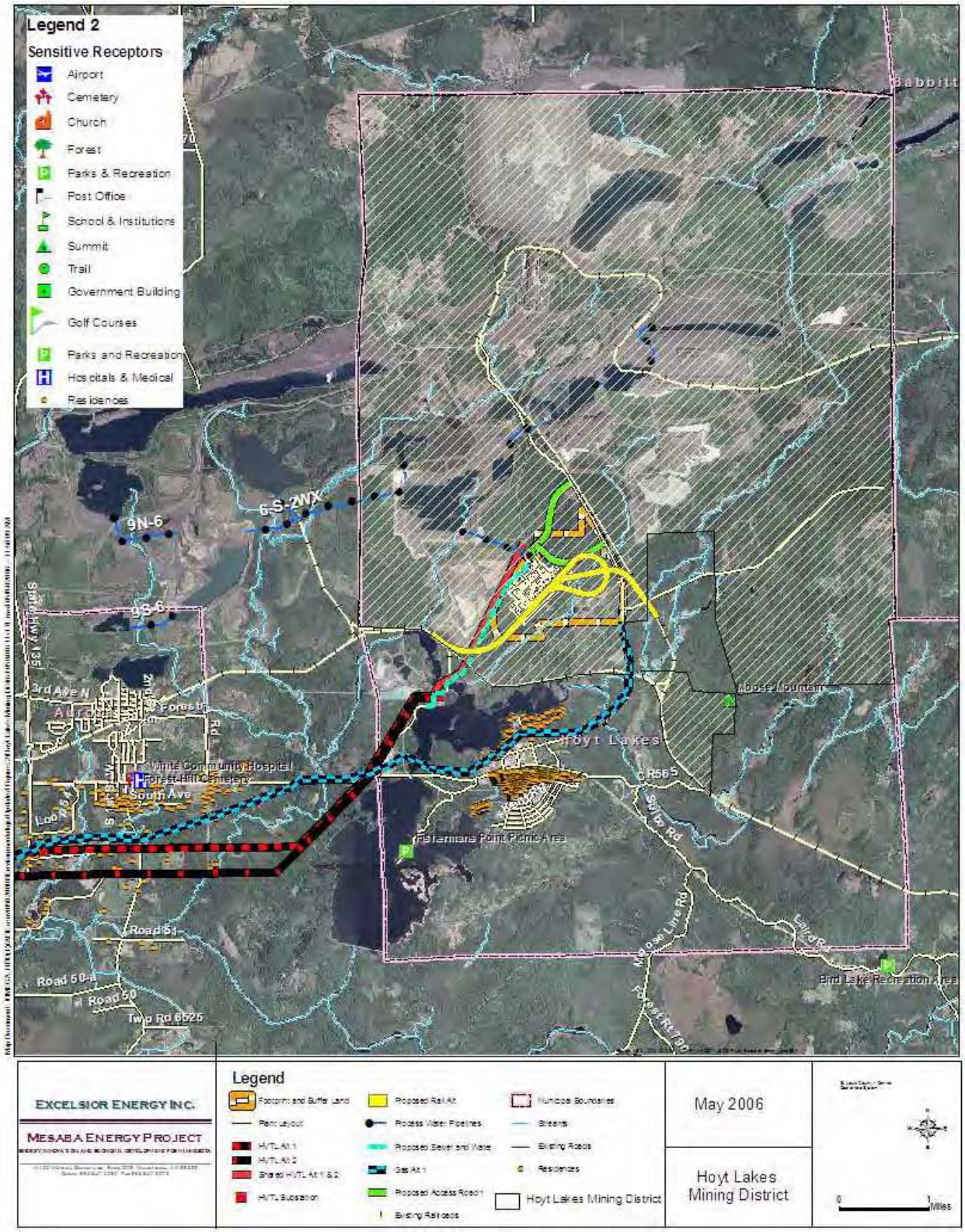
Only preliminary soils data exist for parts of St. Louis County, Minnesota because the county soil survey is currently in progress. Available data were analyzed qualitatively. Farmland designated as being of statewide importance does occur within the vicinity of the East Range Site and its Associated Facilities.

The U.S. Department of Agriculture tracks conversions of prime or statewide important soils to other uses through its Natural Resources Conservation Service (NRCS). Impacts or direct conversions of prime or statewide important farmland require completion of a Farmland Conversion Impact Rating (Form AD-1006) to be completed by the NRCS in St. Louis County in the Environmental Impact Statement review process. The NRCS in St. Louis County will calculate specific impacts on farmlands of statewide importance, which will be used to complete the Farmland Conversion Impact Rating.

8.1.11 Regional Zoning

Figure 2.6-1 shows the zoning map for the City of Hoyt Lakes. Figure 8.1-4 shows that the IGCC Power Station Footprint, Buffer Land, most of the Associated Facilities, and most of the Additional Lands lie within the "mining district" zoning designation (see Section 2.5.1 for a description of the mining district designation). The only Associated Facilities lying outside the City Limits include Segments 6, 7, and 8 of the Process Water Supply Pipeline. Segment 7 of the Process Water Supply Pipeline lies within the City limits of Aurora. The other two segments lie within White Township. In general, the Hoyt Lakes zoning designation is consistent with the industrial nature of power production and the expectations of other cities and townships located in the immediate vicinity of the East Range Site.

Figure 8.1-5 Zoning Designation for the City of Hoyt Lakes: North Half of City



8.2 NEARBY RESIDENCES AND OTHER RECEPTORS

Figure 8.1-5 shows the location of residences and other receptors around the IGCC Power Station Footprint, Buffer Land, Associated Facilities, Additional Lands, Preferred and Alternate HVTL Routes, and the East Range Proposed Natural Gas Pipeline Route.

There are no residences located within the East Range Site. The nearest residences are located some two-miles from the project site in the City of Hoyt Lakes. Existing HVTL corridors to be utilized by the Project are located near various farmsteads/rural residential land uses and structures (see Table 8.2-2). The HVTL corridor is located approximately two miles from the cities of Aurora, Gilbert, Virginia, and Eveleth. There are many residential areas in each of these cities.

The Natural Gas Pipeline is also within two-miles of Hoyt Lakes, Aurora, Gilbert, Eveleth, and Virginia and the proposed rail lines would be within two miles of residential areas in Hoyt Lakes.

8.2.1 IGCC Power Station Footprint and Buffer Land

8.2.1.1 Distance of Nearby Receptors from IGCC Power Station Emission Points

No significant receptors, such as schools, daycares, recreation centers, playgrounds, nursing homes or hospitals are located within 0.62 miles of the proposed facility stacks. The closest residence to any of the elevated stationary source stack emission points is located about 1.6 miles south of the IGCC Power Station Footprint (see Figure 8.1-5 and Table 8.2-1). Table 8.2-1 below identifies the locations of several of the receptors included in the noise monitoring or subsequent impact analysis, and excludes other industrial monitoring locations (i.e., the Reclaimed Landfill).

Table 8.2-1
Significant Receptors Near the IGCC Power Station Footprint and Buffer Land

Location	Approximate Distance From The Nearest Edge Of Project Site
R2, Boat Landing and Park	9,200' to the southwest
R3, Residence (Colby Ridge)	8,300’ from the plant site
R4, Residence, 321 Kent Street	11,500’ from the plant site
R5, Faith Lutheran Church, Hoyt Lakes	10,000’ from the plant site
R6, Queen of Peace Catholic church, Hoyt Lakes	10,200’ from the plant site
R7, Trinity Methodist Church, Hoyt Lakes	10,300’ from the plant site

The residences most likely to be affected by construction and operation of the IGCC Power Station are located on the south shore of the eastern arm of Colby Lake, 1.6 miles due south of the IGCC Power Station Footprint. The impacts on this residential area are most likely to be heavy equipment construction noise and trains. Most of these residences will be somewhat

insulated from construction traffic to and from the Station Footprint and Buffer Land. The City of Hoyt Lakes is two miles south of the IGCC Power Station.

8.2.2 HVTL Alternatives

The significant receptors along the HVTL alternatives were identified from aerial photography. The location of significant receptors within ½ mile of the HVTL alternative alignments was determined, both such alignments utilizing existing HVTL corridors. Table 8.2-2below provides a summary of the significant receptor inventory.

Table 8.2-2
Significant Receptors along the HVTL Alternatives

HVTL Route	Significant Receptors	Distance from Centerline of Alignment (ft)					
		0-50	50-100	100-300	300-500	500-1,320	1,320-2,640
38L Route	271 residences	0	1	10	11	91	155
	Camp Olcott	0	0	0	0	1,157 ft	0
	Eveleth Scout Camp	0	0	0	0	0	1,601 ft
	Forbes Cemetery	0	0	0	0	0	2,396 ft
39L/37L Route	962 residences	0	2	14	33	317	593
	Fayal School	0	0	0	0	0	2,636 ft
	Lincoln School	0	0	0	0	0	2,083 ft
	Mamrelund Church	0	0	0	0	1,301 ft	0

Residential receptors are located within ½-mile of both of the HVTL alternative corridors. Most of the residences are greater than 100 feet away from the alignments, with only one within 100 feet of Alternate 1. Camp Olcott, Eveleth Scout Camp, and Forbes Cemetery are within ½-mile of Alternate 1. Table 8.2-2 above identifies the actual separation of the residences within 100 feet of the alignment centerline and the actual separation of Camp Olcott, Eveleth Scout Camp, and Forbes Cemetery.

8.2.3 Natural Gas Pipeline

Significant receptors along the gas pipeline were identified from aerial photography, and the location of such receptors within ½ mile of the gas pipeline alignment was determined. Table 8.2-3 below provides a summary of the significant receptor inventory.

Table 8.2-3
Significant Receptors along the Gas Pipeline Alternatives

Significant Receptors	Distance from Centerline of Alignment (ft)					
	0-50	50-100	100-300	300-500	500-1,320	1,320-2,640
856 residences	2	5	39	41	199	570
Eveleth-Virginia Airport						1,852 ft
4H Camp						1,956 ft

Residential receptors are located within ½ mile of the gas pipeline alignment. All but seven of the 849 residences within ½ mile are greater than 100 feet away from the gas pipeline centerline. Two residences are within 50 feet of the alignment centerline and five are within 100 feet. The Eveleth-Virginia Municipal Airport and 4H Camp are more than 100 feet away from the gas pipeline alignment. Table 8.3-4 below identifies the actual separation of the residences within 100 feet of the alignment centerline and the actual separation of the Eveleth-Virginia Municipal Airport and the 4H Camp.

Table 8.2-4
Significant Receptors within 100 feet of Gas Pipeline and Public Gathering Places

Approximate Distance to Centerline of Gas Pipeline			
Significant Receptor	Significant Receptors within 50 feet of Centerline	Significant Receptors 50 to 100 feet of Centerline	Significant Receptors 100 to 2,640 ft of Centerline
Residence	38 feet		
Residence	39 feet		
Residence		58 feet	
Residence		72 feet	
Residence		73 feet	
Residence		84 feet	
Residence		92 feet	
Eveleth-Virginia Municipal Airport			1,852 feet
4H Camp			1,956 feet

8.3 AESTHETICS

The Mesaba Project will affect views at the East Range Site primarily in two ways. First, the IGCC Power Station buildings and exhaust stacks will be screened, but will still be slightly visible from nearby residential areas and roadways. Second, the new high-voltage transmission

line structures will be visible along the selected transmission line corridors because even though the new transmission would use existing transmission right-of-way, the new structures will be taller than the existing structures. In addition, along natural gas and water pipeline corridors (and along new roadways or railway) trees will have to be cleared in some areas and construction will require filling or relocation of wetlands. Permanently cleared right-of-way on these corridors will be visible where the routes follow or cross existing roadways.

Aesthetic impacts are discussed in Section 3.1 of the Environmental Supplement.

8.3.1 IGCC Power Station

Near the East Range IGCC Power Station, buildings and stacks will be screened but may still be visible from nearby homes, businesses and nearby roadways. The highest buildings within the IGCC Power Station are the rod mill feed bins (150 feet) and generator buildings (90 feet). The tallest stack is on the tank vent boiler (210 feet); however, that stack is only six-feet in diameter and will not be highly visible. The stack on the generator building will be 150 feet tall. An artist's visualization of the Phase I and II Developments is shown in Figure 3.2-2.

8.3.2 High-Voltage Transmission

The proposed 345-kV high-voltage transmission lines for the East Range Site would be constructed primarily within existing 115-kV ROW on new taller structures (double circuit). For most of the route, the Applicant is proposing to use steel single-pole structures to minimize the width of the right-of-way needed and land use conflicts. While single steel poles cost more and are taller than wooden H-frame supports or other alternatives, they do have longer spans and require a narrower right-of-way. Longer spans between poles allow for construction of fewer poles when compared to the existing 115-kV transmission lines or other structure types. For the proposed 345-kV line, the structures would be about 140-feet tall, with average spans of about 750 feet. Shorter structures (110-feet) would be sufficient on those segments constructed along new right-of-way as a single circuit 345-kV line. In either case, the new single-pole structures would be more visible to residents along the proposed route between the IGCC Power Station and the Forbes Substation. As described in Section 4.1, H-frame or other structure types may be necessary near waterfowl areas or interstate crossings.

8.3.3 Pipelines and Roadways

Where the pipelines are constructed under a roadway or ATV trail-type surface, surface conditions will be maintained or improved. Since most of the proposed pipeline routes are currently covered with wooded areas, brush, or grassy vegetation, little visual change would result from construction or maintenance of the proposed alignments. Where the routes follow the existing highway, clearing of trees and shrubs will be noticeable.

8.4 AIR QUALITY

The projected maximum annual air emissions for Mesaba One and Mesaba Two are provided in Section 3.4.1. Air emissions would be the same at the East Range IGCC Power Station as for the West Range Station, except for PM_{10}. Because of the lower water quality used for cooling at the

East Range Station, PM_{10} emissions from cooling towers will be greater, with cooling tower PM_{10} emissions from the East Range cooling towers being approximately 254 tons per year, compared to 38 tons per year at the West Range cooling towers.

Applicable regulatory emission controls and rules are summarized in Section 7.4 for the West Range Station. The same regulations apply to the East Range Station. This section summarizes the site specific impacts of air emissions as modeled at receptors and the nearby Federal Class One areas. The preliminary detailed design of the IGCC Power Station, including air emission controls, is provided in the West Range IGCC Power Station application for a Part 70/New Source Review Construction Authorization Permit. That application provides details regarding all regulatory requirements, emission calculations, ambient air modeling assumptions, and modeling results.

8.4.1 East Range Site Dispersion Modeling

For the East Range IGCC Power Station modeling, the Applicant used the same Mesaba One and Mesaba Two facility emissions profile as the West Range Station (except as described above for PM_{10}), and applied the same boundary and rectangular grid receptors. Receptor elevations for all areas surrounding the site were determined from USGS DEM data so as to accurately represent topography at the site.

The modeling used the same models, methodology and specifications as the West Range permitting analyses. However, the East Range analysis had the primary objective of identifying significant differences relative to the West Range Site. Accordingly, the East Range Site modeling was less comprehensive than that completed for the West Range permit application but is sufficient for the matters to be determined in this Joint Permit Application. The analysis considered near-field impacts for Class II areas within 50 km of the site, and impacts on Class I areas using the CALPUFF long-range transport model.

8.4.1.1 Near Field Results

Maximum air quality impacts within 50 km of the East Range IGCC Power Station were determined by application of the AERMOD model. Preprocessed meteorological data applicable to the site location were obtained from the MPCA. For comparison to West Range results, model runs were made for SO_2 and PM10 emissions from the East Range Site, and assumed normal (worst case) operation of the two-phase facility.

Table 8.4-1 shows a comparison of East Range results to those for the West Range Site. The highest predicted concentrations for the two sites are comparable. Predicted SO_2 impacts are slightly lower for the East Range Site. Differences due to slightly different meteorological data and terrain were not significant. PM_{10} concentrations are slightly higher at the East Range Site as a result of higher cooling tower emissions. Because of similar predicted impacts at the two sites, additional modeling for other pollutants and operating scenarios at the East Range Site was deemed to be unnecessary.

Table 8.4-1.
Comparison of East Range Site Near-Field Model Predictions to West Range Site Impacts as Shown in Permit Application

Pollutant/Scenario	East Range Site		West Range Site	
	Highest ($\mu g/m^3$)	High Second-High ($\mu g/m^3$)	Highest ($\mu g/m^3$)	High Second-High ($\mu g/m^3$)
SO_2 – Normal Operation				
One-hour average	126.9	103.1	130.2	122.4
Three-hour average	64.8	64.7	77.6	73.4
24-hour average	27.5	20.3	31.2	21.1
PM10 – Normal Operation				
24-hour average	30.5	26.1	27.9	23.4

However, additional modeling was performed at the East Range Site to include other local or regional pollution sources. Because predicted maximum impacts were far below applicable ambient air and increment limits, and because such significant impacts were limited to very small areas in close proximity to the site, it is highly unlikely that interaction with any nearby or regional sources would contribute to compliance issues. As expected, regional multi-source modeling for the East Range Site showed negligible combined impacts of Mesaba and other sources.

Based upon the similarity between predicted impacts for the East and West Range Sites, the Applicant concluded that there is no significant Class II area permitting issues associated with the East Range Site.

8.4.1.2 Class I Results

A complete CALPUFF model analysis was conducted for the East Range Station, analogous to that performed for the West Range air permit application, as the site lies in close proximity to the Boundary Waters Canoe Area. The modeling did indicate visibility impacts in the Boundary Waters, but it is believed such impacts can be mitigated to allow for issuance of air permits.

Table 8.4-2 shows PSD increment consumption results for both the East and West Range Stations at the three Class I areas evaluated. As at the West Range, SO_2 impacts are above the Significant Impact Level (SIL), but are far below allowable increment limits. A cumulative SO_2 increment analyses was carried out for the West Range Site and it demonstrated no threat to Class I PSD increments. The same regional increment-consuming sources are relevant to the East Range Site. Based upon the small fraction of allowable increment that would be consumed by an East Range Mesaba facility, it can be concluded that there would be no Class I increment violation attributable to the Project.

Table 8.4-2
Mesaba Project (Phases I and II) Increment Impacts
East Range Site/ West Range Site

Class I Area and Pollutant	Max Mesaba		Class I SIL ($\mu g/m^3$)	Class I Increment ($\mu g/m^3$)
	E. Range ($\mu g/m^3$)	W. Range ($\mu g/m^3$)		
Boundary Waters CA				
SO_2 3-hr	6.08	1.55	1.0	25.0
24-hr	2.26	0.46	0.2	5.0
annual	0.06	0.01	0.1	2.0
NOx annual	0.09	0.02	0.1	2.5
PM10 24-hr	0.36	0.09	0.3	8.0
annual	0.01	0.004	0.2	4.0
Voyageurs NP				
SO_2 3-hr	2.12	1.59	1.0	25.0
24-hr	0.68	0.45	0.2	5.0
annual	0.01	0.01	0.1	2.0
NOx annual	0.02	0.01	0.1	2.5
PM10 24-hr	0.11	0.07	0.3	8.0
annual	0.004	0.004	0.2	4.0
Rainbow Lake WA				
SO_2 3-hr	1.19	0.76	1.0	25.0
24-hr	0.39	0.19	0.2	5.0
annual	0.01	0.01	0.1	2.0
NOx annual	0.01	0.01	0.1	2.5
PM10 24-hr	0.08	0.05	0.3	8.0
annual	0.003	0.003	0.2	4.0

8.4.2 Air Emission Risk Assessment (AERA)

The results of the AERA analysis conducted at the West Range Site were used to assess potential health risks associated with the East Range Site, as emission rates of hazardous air pollutants from the plant are the same at both plant sites. The northern-most portion of the City of Hoyt Lakes and a development on the south shore of Colby Lake are located within the 3-kilometer AERA buffer area. No farms, schools, nursing homes, assisted living facilities, or licensed daycares are located with 3 kilometers of the facility.

The results of the RASS evaluation are independent of the plant location as the methodology employed does not require the use of site-specific dispersion modeling. With the RASS, "look up tables" based on stack height and distance to fenceline are used to determine dispersion characteristics, rather than detailed dispersion modeling. Therefore, the results of the RASS conducted at the East Range Site are the same as at the West Range Site.

With the ERER and IRAP evaluations, site specific AERMOD dispersion modeling was used to calculate risk. AERMOD was used to evaluate NAAQS compliance for two pollutants, SO_2 and PM_{10}, at the East Range Site. The results of this evaluation showed that dispersion characteristics are very similar between the East and West Range Sites. This was expected given that the two sites share many similarities: identical stack parameters, similar topography, and the same meteorology.

Dispersion at the two sites is very similar. Calculated pollutant concentrations are slightly higher at the West Range Site than the East Range Site.

ERER and IRAP evaluations at the East Range Site are similar to the West Range results. Acute and subchronic health risks at the West Range Site using the ERER Method were 0.52 and 0.13, respectively, and both are within the acceptable MPCA total hazard index of 1.0. Chronic risks estimated using the IRAP Model are 9.1×10^{-7} to 5.0×10^{-8}. Non-cancer hazard indices range between 0.032 and 0.0028. Both ranges are within acceptable MPCA risk levels.

Since dispersion is similar between the two sites, background and project mercury deposition to lakes located near the plant will be similar as well. Colby Lake is similar in size and approximate distance from the East Range Site as Big Diamond Lake is from the West Range Site. With the West Range AERA, the MPCA mercury in fish tissue database included fish from lakes throughout Northeastern Minnesota. The data is therefore representative of fish in both Big Diamond and Colby Lakes. Because of these factors, the results of the risk evaluation due to fish consumption completed for Big Diamond Lake are likely similar to an evaluation of risk at Colby Lake. For Big Diamond Lake, the predicted increase in hazard quotient from consumption of fish attributed to the project ranges between 0.45 and 0.65 (depending on the size of fish consumed). The predicted cancer risks from all contaminants of potential concern (COPC) range from 2.9×10^{-7} to $3.8 \times 10,^{-8}$ and are both well within the acceptable MPCA risk levels.

8.4.3 Cooling Tower Emissions

The issue of fogging, icing, and visibility impacts due to steam from the cooling towers is addressed in Section 7 for the West Range Site. Meteorology at the two sites is similar, so the

potential impacts due to the cooling water steam would be the same. At either site, the only predicted problem is the visual impact of the steam plume in cold, moist weather, and occasional fallout of snow crystals.

8.4.4 East Range Visibility

Visibility modeling assumptions and related issues common to both sites are described above in Section 7.4. Visibility modeling results for the East Range Site are shown in Table 8.4-3. The results for Voyageurs National Park are comparable to those derived for the West Range Site. Predicted visibility impacts in the Boundary Waters, however, are substantially higher than the West Range Site results.

Table 8.4-3.
Mesaba Class I Area Visibility Impacts (Phases I and II)
CALPUFF Method 2 Model Results

Class I Area and Meteorological Data Year	Days ≥ 5%	Days ≥ 10%	Maximum Δb_{ext}
Boundary Waters Canoe Area			
1990	131	68	33.4
1992	137	69	58.3
1996	92	44	34.7
Voyageurs National Park			
1990	26	6	18.5
1992	28	8	18.4
1996	15	4	13.8

Table 8.4-3 shows that the models indicate the Project would reduce visibility in the Boundary Waters by more than 10% from 40 to 60 days per year. However, there are several reasons why the predicted Boundary Waters impacts are likely over-estimated and do not preclude permitting, including:

- Some portions of the BWCA are closer than 50 km to the East Range Site. More refined visibility analyses using other methods would be appropriate for such areas.
- As shown in the West Range permit application, detailed analysis of the visibility impact days is likely to demonstrate natural visibility impairment on many of those days, with Project-related impacts not relevant on such days.
- Emission reductions at other regional sources will be required under Minnesota's developing Regional Haze/BART regulations. Emission reduction plans have also been announced by Minnesota Power.
- The Applicant could also install additional air emission controls or purchase emission reductions at other nearby sources.

Additional visibility analysis and negotiated mitigation measures would likely be necessary for the East Range Station to satisfy all potential concerns about visibility impacts. However, recent permit actions in Minnesota together with developing regional haze strategies indicate that such concerns can be addressed and visibility protection achieved. Mesaba contributions to potential visibility impacts, though not insignificant under conservative FLM methodologies, are small relative to existing regional source contributions and existing air quality. In addition, as low-emission IGCC displaces existing conventional baseload coal plants currently operating in the area, regional air emissions will decline and thereby reduce visibility problems in Minnesota Class I areas.

8.4.5 Vehicle Traffic Emissions

Emissions will be generated from vehicles operated on the site during construction and operation of the plant. These emissions will be by-products of combustion from the exhaust from the vehicle engines and fugitive dust generated from traffic on the roadways near and on the plant site. Such traffic during construction or operation of the facility will not create a local air quality problem at the site.

8.4.6 Soil and Vegetation

The PSD regulation requires analysis of air quality impacts on sensitive vegetation and soil types. Evaluation of impacts on sensitive vegetation and soils was performed at the West Range Site by comparing predicted project impacts to screening levels presented in the 1980 EPA document titled: *A Screening Procedure for the Impacts of Air Pollution Sources on Plants, Soils and Animals* (EPA, December 1980, EPA 450/2-81-078). As at the West Range Site, predicted air concentrations at the East Range Site are well below applicable EPA Screening levels (see Table 7.1-1 of the Air Permit Application).

8.5 GEOLOGY AND SOILS

This subsection provides a brief summary of the geology in the East Range Site area.

8.5.1 Bedrock

A generalized geologic cross section through the Iron Range area is shown on Figure 8.4-2. The location of the site is shown on the right side of that figure, illustrating its location southeast of the Giant's Range Batholith. Bedrock underlying most of the East Range Site is the Virginia Formation, consisting of interbedded argillite, argillaceous siltstone, and fine-grained feldspathic greywacke. This formation lies southeast of Giants Range (discussed in the West Range Site), with the Biwabik Iron Formation between the Giants Range and the Virginia Formation. The east edge of the project site is underlain by the Duluth Complex bedrock, which consists of troctolite and locally grades of gabbro, with numerous inclusions of hornfels and anorthositic rocks.

8.5.2 Surface Geology

The surface geology of the site consists of glacial till. The site is covered predominately with end moraine deposits from Culver Moraine Association of the Des Moines Lobe of the Wisconsinan glaciation. The soil is reddish brown and clayey with sporadic clasts of shale. This glacial till is commonly non-calcareous (MGS, 1982). The end moraine deposits form rolling and hilly topography including numerous lakes and potholes. Sand and gravel aquifers occur beneath till and in ice contact features on the flanks of end moraines (USGS, 1968).

At the extreme east side of the East Range Site the surface geology consists of ground moraine of the Vermillion Moraine Association of the Rainy Lobe. This glacial till is extremely stony and sandy and contains only trace amounts of clay. In this eastern area, the till is thin and patchy over a hilly terrain of scoured bedrock (MGS, 1982).

Buried bedrock valleys in the region create variability in thickness of quaternary deposits. At the project site, such quaternary deposits are typically less than 25 feet thick (Wright, 1972) whereas southwest of Aurora quaternary deposits are as much as 453 feet thick (MGS Publication M-158).

8.6 WATER RESOURCES AND WATER QUALITY

This section addresses impacts on water resources and water quality in the region of the East Range Site as a result of operating the IGCC Power Station

8.6.1 East Range Surface Waters

A detailed description of the location and status of the surface waters in and near the East Range Site is provided in ES at Section 3.4. In general, the East Range Site lies within the northwest region of the Lake Superior Watershed. The major receiving waters of surface runoff originating from the site are listed in Table 8.6-1 and are shown above in Figure 3.6-2.

Table 8.6-1 Surface Waterbodies

Surface Water	Watershed	FEMA[1] Designated Floodplain	Public Water[2]	Special Water[3]	Impaired Water[4]	Target TMDL Study[5]	Impairment
St. Louis River	Lake Superior	X	X		X	2011	Mercury FCA[6]
Partridge River	St. Louis River	X	X				Mercury FCA[6]
Mine Pit 6							
2WX Pit	Partridge River						
2E Pit	Partridge River						
3 Pit	Partridge River						
Wyman Creek	Partridge River		X	X			
5S Pit	Wyman Creek						

Table 8.6-1 Surface Waterbodies

Surface Water	Watershed	FEMA[1] Designated Floodplain	Public Water[2]	Special Water[3]	Impaired Water[4]	Target TMDL Study[5]	Impairment
Colby Lake	Partridge River		X		X	2011	Mercury FCA[6]
Whitewater Reservoir	Partridge River	X	X		X	2011	Mercury FCA[6]
First Creek	Partridge River	X	X				
St. James Mine	First Creek			X			
9S Pit	First Creek						
Donora Mine / 9N	First Creek						
1W / 1 Pit	First Creek						
Little Mesaba Lake	First Creek						
Second Creek	First Creek	X	X				
Stephens Creek	Second Creek						
Stephens Mine	Second Creek						
Knox Mine	Second Creek						
2W Pit	Second Creek						

1 Federal Emergency Management Agency
2 Minnesota DNR Designated Public Water
3 MPCA Designated Special Water
4 MPCA Designated Impaired Water, 2006 EPA Draft 303(d) list of impaired waters
5 Total Maximum Daily Load
6 Fish Consumption Advisory

The Site itself consists of small surface depressions, wetlands, and small streams. Runoff leaving these on-site features generally enters larger streams and wetland systems prior to entering the surface waters listed in Table 8.6-1.

The major drainage areas throughout the project area have been delineated and are shown in Figure 3.6-2. All of the waterbodies listed in Table 8.6-1 are within the St. Louis River watershed, which ultimately drains into Lake Superior.

Most surface water runoff from the East Range Site eventually flows into Colby Lake or the Partridge River. Mining activities within this drainage area have significantly altered the hydrology in this region. Changes to the hydrology within the watershed include removal of trees and soil, creation of mine pits and other depressions, and changes in topography. The drainage area boundaries shown on Figure 3.6-2 were delineated from the USGS maps of the area, but do not represent the altered hydrology that has taken place due to mining and other activities.

The land use and land cover within the watersheds are primarily made up of northern hardwoods, wetlands, surface waterbodies, mine pits, and other mining features. Impervious surfaces, including highways and urban development, make up a small percentage of the total land use.

The soils in the watershed area consist mostly of Hydrologic Soil Group C and D. Hydrologic Soil Group C and D soils typically have relatively low rates of infiltration and high rates of runoff (Minnesota Hydrology Guide, 1992). See Section 8.5, Soils and Geology, for more information.

8.6.2 East Range Water Resource Impacts

Mine pits within the project area are in various stages, from pre-mining activities to the cessation of mining. In locations where mining activities have ceased, the mine pits are filling with water. The MDNR completed a report entitled the "East Range Hydrology Project" in 2004. The MDNR collected data and completed a hydrologic model (using WATBUD, a water balance model developed by the MDNR) for pits 2E, 2W, 2WX, and 6, in order to predict when the pits would overflow and what the average and peak overflow rates would be. In addition, hydrologic changes to Colby Lake, Whitewater Reservoir, and St. James Pit were evaluated as part of the project. Pits 5N, 5S, 9N, and 9S have reached their static water levels and were not studied as part of the East Range Hydrology Project.

Minimal impacts are expected to occur in the region as a result of using water resources on the East Range Site to support power generation at the IGCC Power Station. None of the mine pits support local recreational use. The MDNR has not stocked fish in these water resources. The development of the East Range Site and use of the indicated water resources will have impacts on several of the waterbodies. The following outlines and describes such impacts.

8.6.3 New Discharge

The Mesaba Energy Project will use the zero-discharge system described in Section 3.6.1.2. There will be no process wastewater discharge at the East Range Site.

8.6.4 Regulatory Requirements

8.6.4.1 New Water Related Permits

8.6.4.1.1 Minnesota DNR Water Appropriation Permit

A Minnesota DNR Water Appropriation Permit for Non-Irrigation (FORM #A-02623-06) is required for water appropriations from the East Range Site supply sources. A separate permit application would be completed for each water source, with applications and supporting data submitted in one filing. The MDNR would issue a single permit to the Applicant that would cover all of the water sources. An annual Water Use Report is required by the Minnesota DNR for all Water Appropriation Permits.

8.6.4.1.2 Minnesota DNR Public Waters Work Permit

A Minnesota DNR Public Waters Work Permit (FORM #NA-026620-03B) would be required for temporary and permanent impacts to Public Waters. A Minnesota DNR Public Waters Work Permit will be required for work that takes place in any of the identified public waters listed in Table 8.6-1. For stream crossings, the Minnesota DNR must review and approve any proposed hydraulic changes to the stream.

The following proposed activities in or near public waters would require coverage under a Minnesota DNR Public Waters Work Permit:

8.6.4.1.2A East Range HVTL

Embarrass River (2 crossings)
Cedar Island Lake
Norcund River
Colby Lake
Whitewater Lake
Partridge River (2 crossings)
St. Louis River (3 crossings)
Two River (2 crossings)

8.6.4.1.2B East Range Gas Pipeline

Two River
Unnamed Creek
Elbow Lake
Maryt Lake
Lost Lake
Forth Lake
Esquagama Lake
Unnamed Tributary to St. Louis River
Colby Lake
Whitewater Lake
Partridge River
First Creek

8.6.4.1.2C East Range Rail Line Alternative 1

Unnamed Creek

8.6.4.1.2D East Range Rail Line Alternative 2

Unnamed Creek
Colby Lake

More detailed discussions of these utility corridors are provided in the ES.

8.6.4.1.3 MPCA NPDES/SDS Permit

MPCA NPDES Permits will be required for storm water discharges associated with industrial activity and construction activities. No discharges of cooling tower blowdown will occur, therefore, no NPDES permit for this discharge will be required. Sanitary discharges will routed to the Hoyt Lakes POTW and will require a permit from the local authority. Such discharges do not require an NPDES pre-treatment permit.

8.6.4.1.4 Industrial Storm Water Permitting

Discharge of storm water associated with industrial activities from the Project area to waters of the U.S. and State will be permitted as part of the NPDES/SDS permit described in Section 7.6.4.3 for a listing of Project storm water requirements.

8.6.5 Water Crossings

There are several small streams and one lake that would be crossed for the various utility alternatives associated with the East Range Site. These water crossings are associated with the corridors required for the HVTL, gas pipelines, water process line, processed sewer and water pipelines, and the proposed rail alignment. There are no water crossings associated with siting, placement, or construction of the IGCC Facility or roads to access the facility. The following discussion and tables provide information on the habitats and associated fisheries within these waterbodies. Section 2.7 of the ES provides details about the wetland habitat associated with the water crossings.

The water crossings identified under the various alternative alignments generally affect only two categories: small perennial streams and Colby Lake. These two basic classifications have somewhat unique fisheries components, and will be discussed in general terms. None of the waterbodies proposed to be crossed are designated as trout streams or would be considered cold water streams, although it is possible that trout are occasionally present in some of the undesignated area waterways.

The small streams that would be crossed are typically less than three feet across, tend to be very shallow, have low discharge, are often associated with vegetated wetlands, and tend to act as conveyance systems between the multiple wetlands and waterbodies located in the project vicinity. These small waterways are also highly prone to hydrologic alteration due to the abundance of beaver and beaver dams. The fisheries habitat in these small streams is limited due to lack of space and cover, and occasional lack of water during dry periods. Beaver dams can block fish passage and also create small ponds suitable for some species. Smaller streams can be important for allowing fish to move between more permanent suitable habitat, but are generally not primary fisheries resources. If fish species are present in these small stream systems, they tend to be dominated by small non-game species such as Cyprinids (minnows, dace, creek chub) and Percids (darters).

Colby Lake is a significant aquatic resource, and would be crossed for the HVTL alternatives and the processed water and sewer pipelines. These crossings would occur at one of the narrowest portions of the lake so as to tie into MP's Syl Laskin Station. Colby Lake is a 539 acre lake that has inlets from the Partridge River, Wyman Creek, and Whitewater Lake. MP's coal fired electrical generating plant on Colby Lake utilizes a fish screen in front of its cooling water intake structure. The power plant discharge enters a long narrow arm of the lake that leads to the Partridge River. A fish survey completed in 2000 identified Colby Lake as a below average lake for fish abundance compared to other lakes in the region. Fish populations in 2000 were dominated by bluegill sunfish (*Lepomis* spp.), followed by northern pike (*Esox lucius*), yellow perch (*Perca flavescens*), and white sucker (*Catostomus commersoni*). Other species

were present in low numbers, but included walleye (*Sander vitreus*), black crappie (*Pomoxis nigromaculatus*), yellow bullhead (*Ameiurus natalis*), and rock bass (*Ambloplites rupestris*).

Construction methods at the water crossings will vary depending on the particular facility in question. HVTL lines are suspended and would avoid any direct impacts to the water resources. Gas, water, and sewer pipelines, would be buried, and have the potential to directly impact aquatic resources. Construction methods have been evaluated to minimize impacts with the primary solution being to directionally drill beneath the aquatic resource. If directional drilling cannot be utilized, an open cut will be used. This method can be timed to coincide with low water levels, and can be done using coffer dams, bypass flumes, diversionary channels, or other short-term methods for undertaking work in a dry channel. Guidance published by the U.S. Fish and Wildlife Service, U.S. Army Corps of Engineers, Federal Energy Regulatory Commission, and Minnesota Department of Natural Resources would be consulted and evaluated once final alignments have been determined. These sources will allow a minimally invasive construction method to be used depending on the type of crossing required.

8.6.5.1 East Range Preferred HVTL Routes

8.6.5.1.1 39L/37L Route

There are 21 crossings of streams and other bodies of water associated with East Range HVTL 39L/37L Route. Colby Lake (249P) and an unnamed pond (430W) are protected by the MDNR Protected Waters Inventory (PWI). There are also nine rivers and streams that are protected by the MDNR PWI. As the HVTL line is suspended, there would be no direct impacts associated with these crossings. However, the protected waters will require a License for Utility Crossings of Public Lands and Waters to be granted by the MDNR Division of Lands and Minerals. Table 8.6-2 below summarizes the stream crossings associated with the preferred HVTL routes.

Table 8.6-2
Water Crossings for East Range HVTL Preferred Route: 39L/37L Route

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Crossing (linear feet)
Colby Lake	1+4670	Yes 249 P	540
Partridge River	5+1190	Yes	110
Perennial Tributary to St. Louis River	6+3680	No	3
Perennial Tributary to St. Louis River	6+4590	Yes	3
Perennial Tributary to St. Louis River	8+1215	No	3
Perennial Tributary to St. Louis River	8+2420	No	3
Unnamed Pond	9+0480	Yes, 430 W	180
Perennial Stream between North and South Cedar Island Lake	11+1780	Yes	60

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Crossing (linear feet)
Perennial Stream South of Forge Lake	13+1850	No	95
Perennial Tributary to Esquagama Lake	15+0670	Yes	3
Perennial Ditch to Esquagama Lake	15+3590	No	3
Perennial Tributary to Embarrass River	16+3900	No	60
Intermittent Stream to Embarrass River	16+4900	No	3
Ely Creek	22+0090	Yes	3
Perennial Stream south of Half Moon Lake	23+4750	No	3
Intermittent Stream north of Long Lake Creek	26+4020	No	3
Long Lake Creek	27+0360	Yes	3
Perennial Stream north of St. Louis River	29+3250	Yes	3
Elbow Creek	30+1230	Yes	15
Perennial Stream north of Elbow Creek	30+4100	No	3
Two River (in 3 places due to meander)	31+2840	Yes	95
Total			**1194 linear feet**

8.6.6 38L Route

There are 20 crossings of streams and other bodies of water associated with the East Range 38L Route. Colby Lake (249P) and Deep Lake (666P) are protected by the MDNR Protected Waters Inventory (PWI). There are also seven rivers and streams that are protected by the MDNR PWI. Crossings of such protected waters would require a License for Utility Crossings of Public Lands and Waters to be granted by the MDNR Division of Lands and Minerals. Table 8.6-3 below summarizes the stream crossings associated with this alternative.

Table 8.6-3
Water Crossings for East Range the38L Route

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Waterbody Crossing
Colby Lake	1+4760	Yes 249 P	540
Partridge River	5+3020	Yes	250
Perennial Tributary to St. Louis River	7+1110	Yes	80
Perennial Tributary to St. Louis River	8+2300	Yes	3
Perennial Tributary to St. Louis River	8+2980	No	3
Perennial Drainage Ditch to wetland	12+1410	No	6
Embarrass River	15+1140	No	3
Embarrass River	15+1490	Yes	70

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Waterbody Crossing
Deep Lake	19+2260	Yes 666 P	690
Perennial Stream west of Deep Lake (2 crossings in meander)	19+4840	No	6
Perennial Stream west of Deep Lake	20+1540	No	3
Unnamed Intermittent Stream	22+4080	Yes	3
Perennial Ditch to Mine Dump	25+0960	No	3
Perennial Stream to Mine Dump	25+1960	No	3
Elbow Creek	28+5130	Yes	15
Perennial Ditch to East Two River	30+2190	No	3
Perennial Stream to East Two River	31+1910	No	3
East Two River	32+0810	Yes	70
Unnamed Perennial Stream	33+0340	No	3
Perennial Ditch to Two River	34+4960	No	3
Total			**1760 linear feet**

8.6.6.1 Gas Pipeline Route

The gas pipeline for the East Route would be constructed by an interstate pipeline company and would not require a state pipeline routing permit. Details on the route are provided in the ES. There are 19 crossings of streams and other bodies of water associated with East Range HVTL Alternative 1. Colby Lake (249P) is protected by the MDNR Protected Waters Inventory (PWI). There are also 12 rivers and streams that are protected by the MDNR PWI. The crossings will require either directional drilling or an open cut trench to install the gas line. The crossing of protected waters will require a License for Utility Crossings of Public Lands and Waters to be granted by the MDNR Division of Lands and Minerals.

8.6.6.2 Process Water Supply Pipelines

8.6.6.2.1 Pipeline 6-S-2WX

There are two crossings of streams and other bodies of water associated with East Range Process Water Supply Pipeline from Area 6 and Stephens Mine to Area 2WX. Stephens Creek and Second Creek are both perennial streams protected by the MDNR Protected Waters Inventory (PWI). The crossings will require an open cut trench to install the process water line. The crossing of protected waters will require a License for Utility Crossings of Public Lands and Waters to be granted by the MDNR Division of Lands and Minerals. Table 8.6-4 below summarizes the stream crossings associated with this alternative.

Table 8.6.4
Water Crossings for East Range Pipeline 6-S-2WX

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Waterbody Crossing
Stephens Creek	n/a	Yes	3
Second Creek	n/a	Yes	30
Total			**33 linear feet**

8.6.6.2.2 Pipeline 9S-6

There is one crossing of water associated with East Range Process Water Supply Pipeline from Area 9 South to Area 6. First Creek is a perennial stream protected by the MDNR Protected Waters Inventory (PWI). The crossing would utilize an open cut trench to install the process water line, and would require a License for Utility Crossings of Public Lands and Waters to be granted by the MDNR Division of Lands and Minerals. Table 8.6-5 below summarizes the stream crossings associated with this alternative.

Table 8.6-5
Water Crossings for East Range Pipeline 9S-6

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Waterbody Crossing
First Creek	n/a	Yes	3
Total			**3 linear feet**

8.6.6.2.3 Pipeline 9N-6

There is one crossing of water associated with East Range Process Water Supply Pipeline from Area 9 North to Area 6. First Creek is a perennial stream protected by the MDNR Protected Waters Inventory (PWI). The crossing would utilize an open cut trench to install the process water line, and would require a License for Utility Crossings of Public Lands and Waters to be granted by the MDNR Division of Lands and Minerals. Table 8.6-6 below summarizes the stream crossings associated with this alternative.

Table 8.6-6
Water Crossings for East Range Pipeline 9N-6

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Waterbody Crossing
First Creek	n/a	Yes	3
Total			**3 linear feet**

8.6.6.2.4 Potable Water and Sewer Pipelines

There is one crossing of water associated with East Range Potable Water and Sewer Pipelines. Colby Lake is protected by the MDNR Protected Waters Inventory (PWI). The pipelines will either be installed via open cut trench method or by laying the pipes across the bottom of Colby Lake. This crossing of protected waters will require a License for Utility Crossings of Public Lands and Waters to be granted by the MDNR Division of Lands and Minerals. Table 8.6-7 below summarizes the stream crossings associated with this alternative.

Table 8.6-7
Water Crossings for East Range Potable Water and Sewer Pipeline

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Waterbody Crossing
Colby Lake	1+3720	Yes 249 P	460
Total			**460 linear feet**

8.6.6.3 Rail Lines

8.6.6.3.1 Rail Line Alternative 1

Railroad Alternative 1 will require two crossings of a small tributary to Colby Lake. Culverts under the newly constructed railbeds would permit the flow of water after construction. Impacts to the stream from the center loop would be reduced or negated upon final design when placement of storage areas within the center loop is determined. Both of the tributaries are protected by the MDNR Protected Waters Inventory (PWI) and would require a License for Utility Crossings of Public Lands and Waters to be granted by the MDNR Division of Lands and Minerals. Table 8.6-8 below summarizes the stream crossings associated with this alternative.

Table 8.6-8
Water Crossings for East Range Rail Line Alternative 1

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Waterbody Crossing
Tributary to Colby Lake (North Crossing)	n/a	Yes	3
Tributary to Colby Lake (South Crossing)	n/a	Yes	3
Total			**6 linear feet**

8.6.6.3.2 Rail Line Alternative 2

Railroad Alternative 2 would require crossings of 2 small streams that feed into Colby Lake. Culverts under the newly constructed railbeds would permit the flow of water after construction. Both the tributary to Colby Lake and Wyman Creek are protected by the MDNR Protected Waters Inventory (PWI), and would require a License for Utility Crossings of Public Lands and Waters to be granted by the MDNR Division of Lands and Minerals. Table 8.6-9 below summarizes the stream crossings associated with this alternative.

Table 8.6-9
Water Crossings for East Range Rail Line Alternative 2

Stream Crossing Location	Milepost (mile + linear feet)	MDNR PWI?	Length of Waterbody Crossing
Tributary to Colby Lake	n/a	Yes	3
Wyman Creek	n/a	Yes	3
		Total	**6 linear feet**

8.7 WETLANDS

A detailed summary of impacts on wetlands in the project site and associated utility corridors is provided in the ES (Sections 2 and 3). This subsection summarizes those wetland impacts at the East Range Site and its associated facilities and corridors. Table 8.7-1 below summarizes the expected acres of wetlands impacted.

8.7.1 Overview of Impacts and Contacts with Agencies

Wetland permitting and agency consultation will begin in earnest after the MPUC has issued final Site Certificates and Permits. In general, wetland permitting can be initiated after 80 percent or more of the final design has been completed. A Combined Wetland Permit Application and Replacement Plan will be prepared and submitted to the following agencies:

- U.S. Army Corps of Engineers (USACE) – Section 404 Clean Water Act wetland dredge and fill activities permit.
- Minnesota Pollution Control Agency (MPCA) – Section 401 Clean Water Act water quality certification.
- Itasca County Soil and Water Conservation District (SWCD) – Minnesota Wetland Conservation Act (WCA) approval (West Range Site and Associated Corridors).
- Minnesota Board of Water and Soil Resources (BWSR) – Replacement Plan approval under the rules of the WCA.
- Minnesota Department of Natural Resources (MDNR) – Public Waters work permit.

- St. Louis County, Minnesota – Minnesota Wetland Conservation Act (WCA) approval (East Range Site and associated corridors not within the city limits of Hoyt Lakes, Minnesota).
- City of Hoyt Lakes, Minnesota - Minnesota Wetland Conservation Act (WCA) approval (Associated corridors for East Range Site within the city limits of Hoyt Lakes, Minnesota).

Table 8.7-1
Summary of Total Temporary and Permanent ROW Wetland Impacts for East Range Site and Associated Utility and Transportation Corridors

Project Alternative	Total Wetland Impacts (Acres)	
	Temporary ROW	Permanent ROW
IGCC Facility	n/a [1]	15.61
HVTL 39L/37L Route	n/a [1]	0.05 [2]
HVTL 38L Route	n/a [1]	0.04 [2]
Gas Pipeline	67.29	46.81
Process Water Supply Pipeline (Area 2WX to Site)	1.45	0.87
Process Water Supply Pipeline (Area 2WX to Area 2W)	0	0
Process Water Supply Pipeline (Area 2W to Area 2E)	0	0
Process Water Supply Pipeline (Area 3 to Area 2E)	0.41	0.23
Process Water Supply Pipeline (Knox Mine to Area 2WX)	0	0
Process Water Supply Pipeline (Area 6 and Stephens Mine to Area 2WX)	0.45	0.26
Process Water Supply Pipeline (Area 9 South to Area 6)	0.54	0.29
Process Water Supply Pipeline [Area 9 North (Donora Mine) to Area 6]	0	0
Railroad Alternative 1 and Center Loop	17.21 [3]	58.59
Railroad Alternative 2 (no center loop)	18.35 [3]	13.37
Potable Water and Sewer Pipelines	1.12	0.45
Roads	5.53	3.23
Total[4]	**94.0**	**126.4**
Total Permanent Impacts (including Railroad construction limit impacts)	**n/a**	**133.0 acres**

[1] Temporary construction areas for the IGCC or temporary ROW for the HVTL corridors are not defined for the project area, therefore temporary wetland impacts are not anticipated for these project alternatives.

[2] Permanent impacts in the permanent ROW for HVTL is limited to placement of new power poles.
[3] Impacts in Railroad temporary ROW are permanent impacts due to grading in the construction limits, which should be included with total permanent wetland impacts for mitigation purposes.
[4]Total impacts assuming Railroad Alternative 1 and center loop. The Applicant will seek to minimize impacts to wetlands within the rail loop, but including it here provides a conservative assessment.

8.7.2 IGCC Power Station Footprint and Buffer Land

The IGCC plant facility preliminary layout was designed to minimize wetland impacts by initiating the early stages of wetland impact sequencing (avoidance and minimization, followed by mitigation as necessary). Wetland impacts for the East Range IGCC Facility are estimated at 15.61 acres for the facility layout, with Phase I wetland impacts estimated at 11.91 acres and Phase II wetland impacts estimated at 3.70 acres. Type 7 wetlands are the most abundant wetland type present within the project limits and would receive the greatest impact for both phases of the IGCC Facility. Phase I would cause the majority of wetland impacts on Type 7 wetlands. Phase II construction would involve less wetland impact, involving a small Type 2 wetland not previously impacted by Phase I.

8.7.3 Preferred HVTL Route

The permanent impacts to wetlands due to HVTL foundations and construction are the same for both the preferred and alternative route configurations. The slight difference in structure locations does not affect total wetland impacts.

8.7.3.1 39L/37L Route

About 0.05 acres of wetlands would be permanently impacted by placement of new utility poles. To the extent practicable, wetlands would be avoided for installation of the HVTL, with construction activities planned during winter months to minimize direct impacts to wetlands. Permanent wetland impacts would be limited to those areas where utility poles are placed within a wetland habitat.

Tree and shrub clearing in wetlands would usually be initiated along new areas of ROW. Trees and shrubs would be cleared in Types 6, 7, and 8 wetlands. No vegetation clearing would be anticipated in Type 1-5 wetlands (i.e., herbaceous dominated vegetation in seasonal basins, wet meadow, shallow marsh, or open water wetlands). Wetlands are not anticipated to be cleared of trees in shrubs for HVTL Alternative 1 as it is located entirely within existing ROW which is currently maintained free of trees and shrubs. Future maintenance of the ROW would likely include clearing of trees and shrubs that re-establish in wetlands, and would be completed during winter months to avoid direct wetland impacts by equipment or disturbance to nesting birds.

There are 21 crossings of streams or waterbodies associated with HVTL Alternative 1 that would require the crossing of 1194 linear feet of water. Placement of the power poles supporting the HVTL will be designed to avoid direct impacts to these streams, rivers, or other bodies of water. The average expanse between poles would be approximately 650 feet, but in sensitive or otherwise important areas that should be avoided, the expanse between power poles may be altered if necessary to protect habitat.

8.7.3.2 38L Route

For HVTL Alternative 2, an estimate of 0.04 acres of wetlands would be permanently impacted by placement of new utility poles. To the extent practicable, wetlands will be avoided and construction activities would be planned for winter months to further minimize direct impacts to wetlands. Permanent wetland impacts will be limited to those areas where overhead utility poles would be placed within wetland habitat.

The majority of HVTL Alternative 2 would be located within the existing 38L 100-foot ROW. Approximately 2.5 miles of the proposed corridor is new and would require tree and shrub clearing in wetlands. A total of 0.58 acres of trees and shrubs is estimated to be cleared in Type 6 wetlands. No vegetation clearing is anticipated in Type 1-5 wetlands (i.e., herbaceous dominated vegetation in seasonal basins, wet meadow, shallow marsh, or open water wetlands). Direct wetland impacts to these wetlands is not anticipated as no stump grubbing, excavation, or fill is planned for the areas to be cleared of woody vegetation. Ultimately, some of these wetland areas may be converted to different types (e.g., Type 6 scrub-shrub habitat may convert to Type 2/3 wet meadow/shallow marsh) but direct loss of wetland is not anticipated. In addition, tree clearing activities would be completed during the winter months to avoid direct impacts to wetlands from equipment and to avoid the bird nesting season in compliance with the federal Migratory Bird Treaty Act. Future maintenance of the ROW would likely include clearing of trees and shrubs that re-establish in wetlands, but would be completed during winter months to avoid direct wetland impacts and potential nesting birds.

There are 20 crossings of streams or waterbodies associated the 38L Route that would require the crossing of 1760 linear feet of water. Placement of the power poles supporting the HVTL will be designed to avoid direct impacts to streams, rivers, or other bodies of water. The average expanse between poles would be approximately 530 feet, but would be altered in sensitive or otherwise important areas that should be avoided.

8.7.4 East Range Process Water Line

8.7.4.1 Area 2WX to Site

Wetland impacts were minimized to the maximum extent possible by routing the process water lines along existing and proposed roadways, railroads, and utility rights-of-way. Wetlands impacts within the proposed Process Water Line (Area 2WX to the Site) corridor include a total of 1.45 acres in the 150-foot temporary ROW. These impacts would be reduced to 0.87 acres for the 100-foot permanent ROW. There are no stream crossings associated with the Process Water Line – Area 2WX to Site. Temporary wetland impacts may include tree and shrub clearing for the construction staging areas paralleling the water process line corridor. Where soils and vegetation may become disturbed in the construction areas, they would be restored by loosening the soils from compaction and reseeding with grasses and forbs native to the region.

8.7.4.2 2WX to Area 2W

No wetland impacts have been identified for Water Process Line – Area 2WX to Area 2W, and no effects from construction or operation are anticipated for this alignment.

8.7.4.3 2W to Area 2E

No wetland impacts have been identified for Water Process Line – Area 2W to Area 2E, and no effects from construction or operation are anticipated for this alignment.

8.7.4.4 Area 3 to Area 2E

Wetland impacts were minimized by routing the process water lines along existing and proposed roadways, railroads, and utility rights-of-way. Wetland impacts within the proposed Process Water Line – Area 3 to Area 2E corridor include a total of 0.41 acres in the 150-foot temporary ROW. These impacts would be reduced to 0.23 acres for the 100-foot permanent ROW. There are no stream crossings associated with the Process Water Line – Area 3 to Area 2E. Temporary wetland impacts may include tree and shrub clearing for construction staging areas paralleling the water process line corridor. Where soils and vegetation may become disturbed in the construction areas, such areas will be restored by loosening the soils from compaction and reseeding with grasses and forbs native to the region.

8.7.4.5 Knox Mine to Area 2WX

No wetland impacts have been identified for Water Process Line – Knox Mine to Area 2WX, and no effects from construction or operation are anticipated for this alignment.

8.7.4.6 Area 6 and Stephens Mine to Area 2WX

Wetland impacts were minimized by routing the process water lines along existing and proposed roadways, railroads, and utility rights-of-way. Wetland impacts within the proposed Process Water Line – Area 6 and Stephens Mine to Area 2WX corridor include a total of 0.45 acres in the 150-foot temporary ROW. These impacts would be reduced to 0.26 acres for the 100-foot permanent ROW.

There are two stream crossings associated with the Process Water Line – Area 6 and Stephens Mine to Area 2WX corridor. Wetland impacts include the total length of the crossing through streams and adjacent wetlands. There is no wetland mapped on the NWI adjacent to the crossing at Second Creek, and impacts to adjacent wetlands would be avoided for this crossing. The total length of stream crossings is 33 linear feet over water, and a total of 270 linear feet in the adjacent wetlands. Impacts to wetlands due to the stream crossings are based on a 150-foot temporary ROW and a 100-foot permanent ROW. Wetland habitats associated with the stream crossings that will be affected include 40,500 ft^2 (0.93 acres) in the temporary ROW and 27,000 ft^2 (0.62 acres) in the permanent ROW. Temporary wetland impacts may include tree and shrub clearing for construction staging areas paralleling the water process line corridor. Where soils and vegetation may become disturbed in the construction areas, such areas will be restored by loosening the soils from compaction and reseeding with grasses and forbs native to the region.

8.7.4.7 Area 9 South to Area 6

Wetland impacts were minimized by routing the process water lines along existing and proposed roadways, railroads, and utility rights-of-way. Wetland impacts within the proposed Process Water Line – Area 9 South to Area 6 corridor include a total of 0.54 acres in the 150-foot

temporary ROW. These impacts would be reduced to 0.29 acres for the 100-foot permanent ROW.

There is one stream crossing associated with this alternative. There is no wetland mapped on the NWI adjacent to this crossing, and impacts to adjacent wetlands would be avoided. The total length of the stream crossing is 3 linear feet over water.

8.7.4.8 Area 9 North (Donora Mine) to Area 6

No wetland impacts have been identified for Water Process Line – Area 9 North (Donora Mine) to Area 6 corridor but the UGSG topographic map for the area identifies one stream that flows from Donora Mine to the Partridge River. Because of the past mining activity in the area, it is not clear from aerial photographs whether this stream currently exists or the measures taken to divert its path. No field investigation has been conducted in this area to date. As such, this crossing is addressed below assuming the stream exists.

There is no wetland mapped on the NWI adjacent to this crossing, therefore impacts to adjacent wetlands due to stream crossings will be avoided. The total length of the stream crossing is 3 linear feet over water.

8.7.4.9 East Range Potable Water and Sewer

Wetland impacts were minimized by routing the sewer and water lines along existing and proposed roadways and utility rights-of-way. Wetland impacts within the proposed sewer and water corridor include a total of 1.12 acres in the 100-foot temporary ROW. These impacts would be reduced to 0.45 acres for the 40-foot permanent ROW. The only impact from the Potable Water and Sewer Pipelines are associated with the crossing of the northern section of Colby Lake. The total length of such crossing is 460 linear feet over water, and there are no wetlands mapped by the NWI adjacent to Colby Lake where the crossing occurs.

8.7.4.10 East Range Railroad

8.7.4.10.1 Railroad Alternative 1

Because the railroads must be designed for the straightest possible alignment so as to minimize curves, unavoidable wetland impacts will occur. Also, the railroad alternatives are the only utility or transportation corridors that have strictly established construction limits, which may be considered as temporary ROW. For the East Range Railroad Alternative 1, the construction limits (temporary ROW) vary in width from 75 – 490 feet. The permanent ROW for the railroad would be an established 100-foot ROW, which would include the ROW width needed for the center loop.

Permanent wetland impacts within the railroad alternatives will occur within the construction limits (temporary ROW), permanent ROW, and the center loop. There are no temporary wetland impacts anticipated for the railroad alternatives due to the necessary grading required for the railroad bed. Permanent wetland impacts within the construction limits (temporary ROW) include 17.21 acres. Permanent wetland impacts within the permanent ROW (the railroad bed itself) include 10.68 acres. The center loop of the rail spur for Alternative 1 has an estimated

47.91 acres of permanent impacts. The impacts estimated for the center loop may be reduced upon completion of final design when the layout of storage areas within the center loop is finally determined.

Railroad Alternative 1 will require crossing approximately 6 linear feet of streams and bodies of water. Permanent impacts to wetlands adjacent to streams being crossed are 14.98 acres within the established construction limits. This includes 7.95 acres that would be maintained in the corridor's permanent ROW. Permanent impacts from construction in the streambed for the center loop would be minimized by use of culverts under the railroad bed.

8.7.4.10.2 Railroad Alternative 2

For the East Range Railroad Alternative 2, construction limits (temporary ROW) vary in width from 60 – 500 feet. The permanent ROW for the railroad would be an established 100-foot ROW. There is no center loop associated with East Range Railroad Alternative 2.

Permanent wetland impacts will occur within the construction limits (temporary ROW) and the permanent ROW. There are no temporary wetland impacts anticipated due the necessary grading required for the railroad bed. Permanent wetland impacts within the construction limits (temporary ROW) include 18.35 acres. Permanent wetland impacts within the permanent ROW include 13.37 acres.

Railroad Alternative 2 would require crossing approximately 6 linear feet of streams and bodies of water. Permanent impacts to wetlands adjacent to streams being crossed are 6.30 acres within the established construction limits. This includes 2.59 acres that would be maintained in the corridor's permanent ROW.

8.7.4.11 East Range Roads

Road corridor impacts were identified and assessed by considering grading requirements, existing topography, accessible properties, and the presence of wetlands. Although there would be impacts to wetlands due to the placement of the road corridors, such impacts were balanced by the overall site grading requirements. In some instances it was more feasible to impact a small area of wetlands than grading hillsides or steep slopes.

Roads that will serve the facility will impact a total of 5.53 acres of wetlands in the 200-foot temporary ROW. These impacts would be reduced to 3.23 acres for the 120-foot permanent ROW. The largest wetland impacts for roads are within Wetland C, the large wetland complex found throughout the East Range Site. There are no crossings of water associated with the road corridors.

8.7.5 Wetland Mitigation

Wetland impacts mitigation strategy is the same for either site. Wetland mitigation planning is generally described in Section 7.7.1 in relation to the West Range Site. Site specific mitigation plans would be developed for the East Range Site during detailed project design.

8.8 ECOLOGICAL RESOURCES: PLANTS, ANIMALS, AND ENDANGERED SPECIES

This section describes the ecological conditions and biological communities that are present in the vicinity of the East Range Site, including flora, fauna and the potential occurrences of and habitat for state and federally rare, special concern, threatened, or endangered species.

The following state and federal agencies are anticipated to provide comments on the environmental review documents, and issue permits or approvals for natural resource related subjects.

Table 8.8-1
Anticipated Involvement of Federal and State Agencies

Federal Agency	Potential Project Roles
U.S. Fish and Wildlife Service	Endangered Species Act, Migratory Bird Treaty Act, Fish and Wildlife Coordination Act, Environmental Document Review and Comments
U.S. Army Corps of Engineers	Section 404 Federal Wetland Permit, Federal Clean Water Act, Environmental Document Review and Comments
U.S. Environmental Protection Agency	Environmental Document Review and Comments
State Agency	**Potential Project Roles**
Minnesota Department of Natural Resources	Minnesota Endangered Species Statute, Public Waters Work Permit, License for Utility Crossings of Public Lands and Waters, Water Appropriation Permit, Environmental Document Review and Comments
Minnesota Pollution Control Agency	Section 401 Water Quality Permit, Federal Clean Water Act, Environmental Document Review and Comments
Minnesota Board of Water and Soil Resources	Minnesota Wetland Conservation Act, Environmental Document Review and Comments

The East Range IGCC Power Station Footprint and Buffer Land was reviewed for vegetative cover types during the 2004 and 2005 field surveys. Where portions of existing HVTL and road corridors were accessible, assessments to these areas were limited to wetland surveys and did not include assessments of terrestrial habitat cover types. For this reason, all impacts to terrestrial communities within the East Range utility and transportation corridors were based on land coverages provided by the LandSat-Based Land Use-Land Cover (Raster) data provided by the Manitoba Remote Sensing Centre. Impacts to terrestrial communities within the HVTL corridors were based upon the LandSat-Based Land Use Land Cover (Raster) data, coupled with a review and interpretation of 2003 Farm Service Agency (FSA) aerial photographs.

8.8.1 Terrestrial Flora

The list of vegetative communities found in the vicinity of the East Range Site is derived from the *Field Guide to the Native Plant Communities of Minnesota: The Laurentian Mixed Forest Province* (MDNR 2003), a vegetation classification system for north central and northeastern Minnesota. The following discussion describes terrestrial habitats present on the Station Footprint and Buffer Land. State and federally protected flora species in the vicinity of the East Range Site are addressed in Section 8.8.3 below. The general land use impacts on trees and other plants and aquatic areas due to the power station and associated utility corridors are described in Section 8.1, above. Detailed estimates of the amount of trees and brush required to be cleared within each area or right-of-way are provided in the ES at Section 3.7.2.

The linear maintained rights-of-way that transect the Station Footprint and Buffer Land are dominated with herbaceous vegetation and occasional shrubs. Wetlands within these linear features are described in Section 2.7. Uplands in the right-of-way are dominated with old field vegetation comprised of common Timothy (*Phleum pratense*), Canada blue-joint (*Calamagrostis canadensis*), goldenrods (*Solidago sp.*), smooth brome (*Bromus inermis*), reed canary grass (*Phalaris arundinacea*), big-leaf aster (*Aster macrophylla*), bracken fern *Ptenidium aquilinum*), wild sarsaparilla (*Aralia nudicaulis*), and other pioneer vegetation typical of disturbed areas. Old field areas that are disturbed or maintained do not receive specific classification in the MDNR system for the Laurentian Mixed Forest Province. These old field areas are limited to the maintained utility rights-of-way.

There was no old growth or mature conifer forest observed during the field reconnaissance. White pines were observed infrequently and red pine (*Pinus resinosa*) was not observed within the Station Footprint and Buffer Land. All of these terrestrial communities onsite have been impacted by timber management activities at some point in time. Some areas appear to have been logged for several consecutive decades. The west half of the site had areas of recent (2005) clear cutting of aspen stands. The eas/south half of the site also had evidence of logging activities in the past 10-20 years, with dense quaking aspen regrowth. Beaver activity was also observed where the majority of the tree cover had been recently removed. Beaver activity was highest in the eastern half of the site.

8.8.2 Animals

Fauna (animals) within the East Range Site include species that are typical to northern Minnesota. State and federally protected fauna are addressed in Section 8.8.3. The following discussion describes the wildlife habitats as related to the wetland and terrestrial vegetative communities described above, and faunal assemblages that are expected to occur within each community. Fauna that were observed during the field investigations are also addressed.

The Station Footprint and Buffer Land upland habitats have been widely impacted by recent clearcutting. All of the uplands are classified as northern mesic mixed forest, aspen birch forest *(balsam fir subtype*) (MDNR Code FDn43B1). Most of the un-harvested stands of this habitat are located in the eastern third of the site, and clearcuts dominant elsewhere have modified and qualitatively reduced wildlife habitat in those areas. Avifauna (birds) diversity is highest within the un-harvested stands, which provides nesting and foraging habitats for songbirds and raptors.

Similarly, suitable habitats for reptiles, amphibians and mammals has been diminished by clearcutting in the project area.

During the 2004 and 2005 habitat assessments and wetland surveys, no raptor nests were observed. An adult merlin (*Falco columbaris*) was observed in flight exhibiting territorial behaviors. A great horned owl (*Bubo virginianus*) was observed as well. Habitat for the red-shouldered hawk (*Buteo jamaicensis*) and northern goshawk (*Accipiter gentiles*) was lacking within the Station Footprint and Buffer Land, also attributable to timber activities and recent clearcutting. Probable habitats and improved habitat quality for these two rare species increase east and south of the Station Footprint and Buffer Land, especially when entering the U.S. Forest Service property. No breeding concentrations of migratory birds were observed within the Station Footprint and Buffer Land. These would include nesting swallow colonies, waterbird colonies, heron and egret nests, or other colonial nesting species.

The list of potential mammals that potentially utilize the Station Footprint and Buffer Land is comprehensive and includes predators, bears, and large ungulates such as moose and deer. A calf moose was observed during the wetland assessments in 2004 and sign of moose was widespread throughout the Station Footprint and Buffer Land. Grey timberwolf (*Canis lupus* – federally threatened) tracks and scat were also observed occasionally throughout the site. Grey wolf implications are addressed in the threatened and endangered species sections of this report in more detail. Deer were observed often, and a family of otters was observed on the eastern side of the Station Footprint and Buffer Land. Beaver activity was widespread. Many of the wetlands within the Station Footprint and Buffer Land contained beaver lodges, dams, and active evidence of foraging. Several beavers were observed as well. Habitat for fisher (*Martes pennanti*) and pine martin (*M. americana*) was confined to the forested wetlands where clearcutting has not occurred. Snowshoe hare habitat is also mostly confined to the forested wetlands for the same reason. This species is the primary prey item for the federally threatened Canada lynx (*Lynx canadensis*) that is discussed in more detail in the threatened and endangered species section of this report. Lastly, the American black bear (*Ursus americana*) is relatively common in the area and are expected to utilize all of the habitats in the area. As noted, the clearcut areas provide the poorest quality habitat for this and most other species of vertebrates in the area.

Wetland habitats for fauna are relatively diverse and common within the Station Footprint and Buffer Land. The Type 8 bog habitat is the most unique and is potential habitat for rare species of fauna, primarily birds and small mammals, but is not the most common or abundant wetland habitat within the Station Footprint and Buffer Land. All other types of wetlands (Types 2-7) onsite, but not connected to lakes, are the most important for amphibians. These wetlands provide optimum amphibian breeding habitats that lack fish (predators) populations. Adult Anurans (frogs) were observed during the field reconnaissance and included American toad (*Bufo americanus*), grey treefrog (*Hyla versicolor*), northern leopard frog (*Rana pipiens*), and wood frog (*Rana sylvatica*). Potential habitats were also observed for the spring peeper (*Psuedacris crucifer*), western chorus frog (*Psuedacris triserata*), and green frog (*Rana clamitans*), all species common to the area. These wetlands also provide potential habitat for the eastern newt (*Notopthalmus viredescens*) and the blue-spotted salamander. Several of these species require upland habitats for some portion of their life histories. In some of these cases, clearcutting may have provided more or improved upland habitats for herpetofaunal species that

require open upland habitats on sandy soils. For other herpetofaunal species, clearcutting represents a negative impact on upland habitat.

Types 6, 7, and 8 wetlands also provide nesting and foraging habitat for songbirds and raptors. Type 8 wetlands provide habitat for insect fauna that are exclusive to bog habitats. Types 3 and 4 marsh wetlands provide foraging habitats for wading birds, rails, and waterfowl. No colonial waterbird nest colonies were observed within the Station Footprint and Buffer Land. Fish habitats are restricted to the creek and deeper wetlands that occur within the central portion of the site. Small fish (Notropids, darters) were observed in these open water habitats. Based on the field observations, small fish are most likely the only fish assemblages present. Lakes and larger waterbodies that might serve as sources of larger fish and game fish were absent in the immediate proximity of the Station Footprint and Buffer Land. Furthermore, the extensive beaver dams and activity that is widespread throughout the stream system could serve as formidable barriers for the movement of larger fish into the upstream portions of the system, in particular the spring spawning migrations of northern pike (*Esox lucius*).

Habitat quality varies but overall habitat quality for the Station Footprint and Buffer Land would rank as medium to medium-low on a scale from poor to high quality. Wetlands are the highest quality habitats on the Station Footprint and Buffer Land and the Type 8 wetlands rank as high quality due to their uniqueness and lack of disturbance. Disturbed habitats from recent clearcutting are widespread, prevalent, and the primary reason for the diminished habitat quality.

Projects that receive any federal funds are subject to the Fish and Wildlife Coordination Act, requiring that federal agencies consider the project effects on fish and wildlife and their habitats. Fish and game species are protected through hunting, fishing, and trapping regulations enforced by the MDNR and USFWS. Birds and their nests, including the songbirds and raptors found within the site, are protected under the federal Migratory Bird Treaty Act.

Given that the Station Footprint and Buffer Land is located within a timber production area subject to frequent clear cutting, comprised entirely of secondary growth, and within the forest setting of northern Minnesota, trees are not rare and no significant impacts to trees are anticipated. No tree mitigation will occur nor will any mitigation for impacts to vegetative communities, all of which are abundant throughout the region.

8.8.3 Threatened, Endangered and Other Rare Species

8.8.3.1 Federally Listed Species

The federal Endangered Species Act is regulated by the USFWS and the Station Footprint and Buffer Land is within USFWS Region 3. The Region 3 list of federally protected species describes St. Louis County MN as occurring within the breeding range of bald eagle (*Haliaeetus leucocephalus* – federal status, de-listed threatened), within distributional range of the grey wolf (*Canis lupus* – federal status, threatened); within the breeding range of the peregrine falcon (*Falco peregrinus* – federal status, Threatened); and within range of the Canada lynx (*Lynx canadensis* – federal status, threatened).

The USFWS will be contacted by the Department of Energy to request Endangered Species Act Section 7 Formal Consultation for these species for the East Range Site. The USFWS Region 3

Endangered Species Biologist was contacted by consultant SEH on October 18, 2005 to discuss the potential for Formal Consultation (USFWS Record of Conversation, 2005). The USFWS indicated that it will request Formal Consultation to be initiated when the DOE request is submitted. The USFWS recommended that Formal Consultation be requested as soon as possible so that it can be coupled with combined Formal Consultation that the USFWS is providing for several other large projects proposed for the Iron Range region. The USFWS intends to provide Formal Consultation collectively and concurrently for all of these projects, and have invited the Applicant to participate in this combined effort.

In particular, Section 7 Formal Consultation will need to occur for the Canada lynx and grey wolf. Currently, population studies are being conducted on these species in conjunction with the Formal Consultation that has been initiated by other projects in the area. In a telephone conversation with the USFWS Endangered Species Biologist (October 10, 2005), the USFWS invited the Applicant to participate in this comprehensive Formal Consultation process and expand these surveys to include the East Range and the West Range Sites as they are both in close proximity to the other projects that are under current consultation. The projects that are currently partnering on this comprehensive Formal Consultation effort include the proposed PolyMet mine expansion, the Minnesota Steel Industry facility, and the ISPAT Mine Expansion.

Grey wolf sign, including tracks and scat, was observed at scattered locations within the Station Footprint and Buffer Land. Suitable snowshoe hare habitat (the primary prey item for Canada Lynx) is present, but is relatively poor or marginal due to the extensive and recent clearcutting. No bald eagle nests were observed in the vicinity of the East Range Site and the MDNR Natural Heritage Information System (NHIS) database shows no nesting areas within the Station Footprint and Buffer Land or within a 2-mile radius of the East Range Site project area. The NHIS does document five bald eagle nesting areas within a one-mile radius of the various proposed and existing utility and transportation corridors. There are no NHIS occurrences for the peregrine falcon within or adjacent to the Station Footprint and Buffer Land.

According to MDNR data (MDNR Online Data, 2005), there have been “verified with evidence of breeding,” “verified without evidence of breeding,” and “unverified” sightings of Canada lynx within St. Louis County through 2005. The request for Formal Consultation under Section 7 of the Endangered Species Act will determine the need for additional studies and coordination for this species.

8.8.3.2 Minnesota Endangered Species Act

State listed species of special concern (SpC), threatened (T), or endangered (E) species are protected under the Minnesota Endangered Species Act. The MDNR Natural Heritage Information System (NHIS) database contains documented occurrences of SpC, T, and E species; sensitive ecological and natural resources; and results of the Minnesota County Biological Survey (MCBS). The MDNR was contacted to request a review of the NHIS for occurrences within the site boundaries and the areas surrounding the various utility and transportation corridors. At the request of the MDNR, the specific locations of these occurrences are not provided in this report to protect the integrity of these rare or protected species. The following subsections describe the known occurrences of state-listed protected, rare, or otherwise unique natural features that are within the vicinity of the East Range Site.

8.8.3.3 East Range Site IGCC Facility

According to the MDNR NHIS, there are no known occurrences of state-listed protected, rare, or otherwise unique natural features within the immediate vicinity of the Station Footprint and Buffer Land. The closest recorded occurrences of an NHIS feature are 2.5 miles or greater from the Station Footprint and Buffer Land. Although the MDNR NHIS is the most comprehensive database for known occurrences of rare natural features in the state, it does not preclude the discovery of undocumented occurrences within the Station Footprint and Buffer Land. Further coordination with the MDNR regarding habitat conditions at the Station Footprint and Buffer Land will be completed to determine if further review is required for protected or otherwise rare species.

8.8.3.4 Associated Facility Corridors

Since access was not available for nearly all the transportation and utility corridors during the 2004 and 2005 field surveys, potential project implications on state-listed species could only be assessed through review of species locations within approximately one mile of the corridors. A summary of this review is provided in the following corridor sections. Federally listed species are addressed in the ES.

According to the MDNR NHIS, a total of nine listed species in 27 different occurrences have been recorded in the general vicinity of Aurora, Biwabik, Eveleth, and Virginia within one mile of a proposed transportation or utility corridor. Table 8.8-2 describes all of the occurrences that were documented by the MDNR NHIS in the vicinity of the corridors. Since access in the field to these corridors was not available, this summary suggests that further coordination with the MDNR will be completed to determine if more detailed surveys are needed if the East Range Site is selected as the preferred location for the IGCC project. At the request of the MDNR, these locations of occurrences are not provided in order to protect these rare species.

Table 8.8-2
MNDNR NHIS Plant Species Occurrences within One Mile of East Range Site Transportation or Utility Corridors

Scientific Name	Common Name	Protection Status	NHIS Records in Area	Associated Habitat Near Project Area
Flora				
Arethusa bulbosa	Dragon's mouth	Non-status	1	Creek shoreline
Caltha natans	Floating marsh-marigold	Endangered	1	Pond Outlet
Poa sylvenstris	Woodland bluegrass	Non-status	1	Mixed hardwood forest
Waldsteinia fragarioides	Barren strawberry	Special concern	3	Jack pine forest
Botrychium matricariifolium	Matricary grapefern	Non-status	1	Mine tailings
Botrychium simplex	Least moonwort	Special concern	2	Mine tailings
Fauna				
Clemmys insculpta	Wood Turtle	Threatened	13	Partridge and St. Louis Rivers
Haliaeetus leucocephalus	Bald Eagle	Special Concern	4	Various nesting areas, some in management areas
Ligumia recta	Black Sandshell Mussel	Special Concern	1	Lake shoreline

Habitats listed above as associated with a state listed species may require further investigation, which should be determined by the MDNR if the East Range Site is selected. It is likely that the MDNR has not yet completed a thorough review of this area through the County Biological Survey program and there is therefore the potential to encounter other state or federally-listed species not previously identified in the MDNR NHIS database.

8.9 NOISE

Noise will be generated by the IGCC Power Station and by associated rail and roadway traffic. Overall, because the nearest receptor is over one and one-half miles (8300 feet) from the IGGC Power station, L_{10} and L_{50} noise levels are predicted to be below state standards and nearly imperceptible at the nearest residences. Likewise, predicted noise levels along the existing county access road and the proposed railway are well below state standards because the nearest residences and other receptors are located over one mile from the existing road and the proposed rail route. Details on the noise modeling inputs, methods and results are in Section 2.11.5 of the ES.

8.9.1 Noise Standards

The Minnesota Pollution Control Agency (MPCA) has set noise standards for both residential and industrial zones. These standards, measured in dBA, are stipulated in the form of L_{10} and L_{50}. L_{10} means that the measured SPL (in dBA) must not exceed a certain threshold more than 10% of the time (for a one hour survey), with L_{50} being a level that must not be exceeded more than 50% of the time (for a one hour survey). These thresholds are listed as SPL (dBA) maximums by the MPCA, as shown in Table 8.9-1. The Noise Area Classification (NAC) has 4 classes. NAC – 1 includes household units, including farmhouses, as well as religious activities. NAC – 2 applies to more commercial development such as retail, businesses, government services and parks. NAC – 3 and 4 (not considered here) are less stringent, and are composed primarily of industrial uses. Applicable limits for NAC – 1 areas (where there is no overnight lodging) are daytime values only.

Table 8.9-1
7030.0050 Noise Area Classification (NAC)

	NAC - 1		**NAC – 2**	
	L_{50}	**L_{10}**	**L_{50}**	**L_{10}**
Daytime	60dBA	65dBA	65dBA	70dBA
Nighttime (10:00PM-7:00AM)	50dBA	55dBA	65dBA	70dBA

8.9.2 Site Setting and Receptors

The East Range Site and associated utility corridors are located in rural, generally forested areas, with sparsely populated residences. A total of 7 residences or other receptors are located near the Site. These receptor locations are detailed in Section 2.11.5 of the ES and are summarized in Table 8.9-2 below.

Table 8.9-2
East Range Site Receptor Locations

Location	Approximate Distance from the nearest edge of West Range Site	Used for Analyses Type(s)
2. Boat landing and Park	9,200' southwest of plant	Ambient Monitoring; Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
3. Colby Ridge Developments	8,300’ ' south of plant	Ambient Monitoring; Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
4. 321 Kent St, Hoyt Lakes	11,500' south of plant	Ambient Monitoring; Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
5. Faith Lutheran Church, Hoyt Lakes	10,000’ from plant; northwest corner of Dorchester Dr. and Kennedy Memorial Dr.	Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
6. Queen of Peace Catholic Church, Hoyt Lakes	10,200’ from plant; northwest corner of Hampshire Rd. and Kennedy Memorial Dr	Plant Operations Modeling; Construction Impacts; Rail Operations Impacts
7. Trinity Methodist Church, Hoyt Lakes	18,000' to the southeast northeast corner of Hampshire Rd. and Kennedy Memorial Dr.	Plant Operations Modeling

8.9.3 Existing Noise Levels

Noise monitoring was completed at four locations near the East Range Site: receptors 2, 3, and 4, described above, as well as at an uninhabited site located 2000 feet southwest of the IGCC Power Station (receptor 1). All monitoring was completed using a Type II, ANSI approved noise level meter with calibration performed before and after each monitoring cycle.

A windscreen was also used to counter wind effects, with no monitoring performed during times when winds greater than 15 mph were measured, or when precipitation was occurring. Monitoring at each location was performed for no less than one hour and during both times specified as “night” and “day” by MPCA classification.

Tables 8.9-3 and 8.9-4 summarize ambient noise conditions for the time frames measured in and around the East Range Site, and for daytime and nighttime conditions.

Table 8.9-3
Monitored levels at all Receptors, Daytime, dBA.

Daytime Receptors	L_{10}	L_{50}	L_{10} dB over State Compliance	L_{50} dB over State Compliance
Receptor 1, TH 6401, (8:23AM-9:23AM)	50dBA	50dBA	0dB	0dB
Receptor 2, Boat Landing (9:50AM-10: 50AM)	52dBA	51dBA	0dB	0dB
Receptor 3, Colby Ridge, (10:23AM-11:23AM)	53dBA	51dBA	0dB	0dB
Receptor 4, 321 Kent St. (12:30PM-1:30PM)	52dBA	50dBA	0dB	0dB

Table 8.9-4
Monitored levels at all Receptors, Nighttime, dBA.

Nighttime Receptors	L_{10}	L_{50}	L_{10} dB over State Compliance	L_{50} dB over State Compliance
Receptor 1, TH 6401, (10:12AM-11:13PM)	49dBA	49dBA	0dB	0dB
Receptor 2, Boat Landing (11:15PM-12:16AM)	50dBA	49dBA	0dB	0dB
Receptor 3, Colby Ridge, (12:40AM-1:40AM)	50dBA	49dBA	0dB	0dB
Receptor 4, 321 Kent St. (1:45AM-2:45AM)	49dBA	48dBA	0dB	0dB

Hoyt Lakes and the surrounding areas are, in general, very quiet places. During daytime hours there is little or no manufacturing noise other than that from MP's Laskin power plant located across Colby Lake. Little traffic passes along Kennedy Memorial Drive proceeding through town, and there are very few school related noise sources such as busses and playgrounds. Most of the daytime noise was related to lawn mowers in the distance, a small amount of light plane traffic, and distant noise from the Laskin power plant near Colby Lake.

Detailed descriptions of the monitoring locations, conditions, and results are provided in Section 2.11.5 of the ES.

8.9.4 Construction Noise Impacts

As at the West Range Site, the construction process for the proposed IGCC Power Station and associated utilities would be expected to generate noise at the East Range Site during the following phases:

- Site Preparation
- Excavation
- Foundation Placement
- Plant and Building Construction
- Exterior Finish and Cleanup

Equipment utilized during the construction process would differ from phase to phase. In general, heavy equipment (bulldozers, dump trucks, and concrete mixers) would be used during excavation and concrete pouring activities. Most other phases involve the delivery and erection of building and equipment components.

Because there are no residences or other receptors within a mile of the proposed IGCC Power Station site, noise during construction is predicted to be more than 10 dBA below state standards at the nearest residence. Because of the nature of construction noise and common fluctuations in background noise levels, construction activity would be occasionally discernable at the nearest receptors. For example, during final construction, a method used to clean piping and testing called "steam blows" creates substantial noise. A steam blow results when high-pressure steam is allowed to escape into the atmosphere through the steam piping to clean the piping. A series of short steam blows, lasting two or three minutes each, would be performed several times daily over a period of two or three weeks. Steam blows can produce noise as loud as 130 dBA at a distance of 100 feet. The resultant sound level at the nearby receptors would range from 86 to 103 dBA. To minimize these short-term, temporary noise impacts, the piping would be equipped with silencers that would reduce noise levels by 20 dBA to 30 dBA at each receptor location. Detailed construction period predicted noise levels can be found in Section 3.10 of the ES.

Rail line construction would occur within about 6,100 feet from the nearest residence (R3)and noise during rail line construction is predicted to only range from 41-44 dBA. These levels are far below the applicable state standards and rail construction noise would only occasionally be audible to nearby residents.

8.9.5 Operating Noise Impacts: Methodology

The noise modeling methods and resulting contours at the West Range Site (see Section 7.9) were used to estimate noise levels at receptors near the East Range Site. For the East Range Site, however, "base case" noise levels were assumed. That is, no additional noise mitigation was included as part of IGCC Power Station. Unlike the West Range Site, no noise mitigation is required at the East Range Site in order to meet state noise standards at the nearest receptors—all of which are located over one-mile from the IGCC Power Station site itself.

Noise due to the proposed facility was modeled at a total of 7 receptor locations – including R1, an open field site modeled for illustrative purposes only.

8.9.5.1 Power Station Operating Noise Impacts: Results

Noise modeling was completed for the base case plant configuration for Phase I only and for the combined Phase I and Phase II operating scenario. As at the West Range Site, the dominant noise sources at the East Range Site included HRSG and ASU stack exits, large buildings with major process equipment inside (including the GTG and STG buildings, the ASU buildings, Rod Mill buildings, and the Slurry Feed buildings) Acid and Tail Gas burners, the Power Block and ASU cooling towers, and several large water-handling pumps.

The modeling results at the seven nearest receptors are shown below in Table 8.9-5. Under base case assumptions, the facility is predicted to meet state standards (both L_{50} and L_{10}) at all receptors. The predicted noise increases would not be perceptible at the listed receptors.

Details of the noise mitigation measures and the levels resulting from the modeling can be found in Section 3.10 of the ES.

Table 8.9-5
Estimated Operational Noise Levels at Receptors

Receptor	Existing L_{10} /L_{50} Day (dBA)	Existing L_{10} /L_{50} Night (dBA)	Projected Plant Noise L_{10} /L_{50} (dBA)[2]	Decibel Increase L_{10} /L_{50} Day (dBA)	Decibel Increase L_{10} /L_{50} Night (dBA)	Resultant L_{10} /L_{50} Day (dBA)	Resultant L_{10} /L_{50} Night (dBA)
R1, TH6401	50/50	49/49	55/55	1.2/1.2	1/1	51.2/51.2	50/50
R2, Boat Landing	52/52	50/49	40/40	.2/.2	.4/.5	52.2/52.2	50.4/49.5
R3, Colby Ridge	53/53	50/49	40/40	.2/.2	.4/.5	53.2/53.2	50.4/49.5
R4, 321 Kent St.	52/50	49/48	35/35	0/0	0/.2	52/50	49/48.2
R5, Faith Lutheran[1]	53/50	50/49	38/38	0/.2	.2/.3	53/50.2	50.2/49.3
R6, Queen of Peace[1]	53/50	50/49	38/38	0/.2	.2/.3	53/50.2	50.2/49.3
R7, Trinity Methodist[1]	53/50	50/49	38/38	0/.2	.2/.3	53/50.2	50.2/49.3

8.9.6 Vehicular Traffic Noise Impacts

At the East Range Site, the only required roadway upgrade consists of grading and resurfacing Kensington Drive (Co. Hwy 110). The roadway does not need to be realigned. Kensington Drive will be the primary route for truck and employee traffic accessing the IGCC Power Station, but will not increase traffic levels significantly (see Section 8.10.1). Additionally, since Kensington Drive is a county highway without full control of access, it is exempt from Minnesota Noise Standards, pursuant to Minn. Stat. § 116.07, subd. 2a. Potential traffic noise impacts will therefore be evaluated using federal noise criteria.

The proposed highway project is not on a new location, does not represent a significant change in horizontal or vertical alignment, and will not increase the number of through lanes. Therefore, the requirements of 23 C.F.R. 772, federal procedures for abatement of highway traffic noise and construction noise, do not apply.

The slight increase in truck and other vehicle traffic are not predicted to affect nearby noise levels primarily because there are no significant receptors along or near the roadway in the area of the East Range Site.

8.9.7 Railroad Noise and Vibration Impacts

Freight train noise levels would range from 41 to 44 dBA at the receptor locations during a train pass-by. Typical daytime background noise levels were measured to be in the low 50's dBA (L_{50}). While the predicted freight train noise would be below applicable state standards, passing

freight trains or rail yard noise could be noticeable at the boat landing (R2), and in the City of Hoyt Lakes itself (R5, and R7). However, given the relatively small amount of future train operations, the L_{dn} generated by such operations would not be considered significant when compared to background noise levels. Further, the maximum noise levels generated by freight train operations would be clearly below the ATPA guideline of 70 dBA at each residential receptor location, and would not be considered significant. Horn soundings would be expected to be clearly audible to the nearest residential receptors. Because train horns are a requirement of the FRA, such noise impacts are an unavoidable adverse impact. For detailed analysis of rail impacts, see Section 3.10.4.

8.10 TRANSPORTATION AND TRAFFIC

8.10.1 Traffic Impacts

A traffic volume forecast was completed for the East Range Site and shows existing and forecast traffic volumes during construction (2008) and 20 years after construction (2028).

Historic data showed growth rates on the existing system ranging from 1.0% per year to 3.4% per year. The 2008 (Build) volumes were developed by applying these historical growth rates to the existing traffic volumes to expand them out to year 2008 (2008 No-Build), and then adding the construction related traffic volumes.

The 2028 (Build) volumes added the East Range Project's plant related traffic to the 2028 No-Build scenario traffic volume forecast. The 2028 No-Build volumes were developed by applying the same historic annual growth rates to the existing volumes. The forecast volumes are shown in Table 8.10-1.

Table 8.10-1

FORECAST TRAFFIC VOLUMES
WITH EXCELSIOR/MESABA ENERGY EAST RANGE PROJECT -- 2008 & 2028

	CSAH 110		County Road 666		Hampshire Road
Year	West of County Road 666	East of Hampshire Road	North of CSAH 110	East of Hampshire Road	Between CSAH 110 and County Road 666
1995	4,400	520	N/A	N/A	N/A
1999	2,950	650	930	830	N/A
2003	2,950	710	750	520	N/A
Forecast 2008 Build	4,375	2,125	2,175	3,150	1,575
Forecast 2028 Build	3,925	1,525	1,625	1,400	675

Note: N/A = Traffic volume data not available.

The two primary roadways in the area of the East Range Site are County Highways 666 and 110. The volume of traffic on County Highway 666 will peak during plant construction at about 3,150 trips per day and will be lower thereafter. The volume on County Road 110 will also peak during construction at 4,375 trips per day to the west and 2,125 to the east. These levels of traffic on rural two-lane highways should not cause travel delays.

The only problem area identified was the intersection of County Highways 666 and 110 in Hoyt Lakes, which was predicted to have some back-ups at peak hours (such as shift changes) during peak construction periods. This problem was addressed by reconstructing Hampshire Drive and encouraging traffic to/from the east to use this as a cut-through route between County Highways 666 and 110.

None of the roadway segments show forecasted volumes that could not be comfortably handled by the type of roadway proposed (or currently in place, as the case may be).

8.10.2 Railroad

The proposed railroad will be a single track using a 100 wide permanent right-of-way. Railroad tracks, especially tracks designed for unit coal trains, have precise parameters for curvature and grades. These criteria made it difficult to avoid impacts to the environment, as described in Section 8.1, above.

The grading impacts, based on cut and fill sections, will vary from 60 feet to 760 feet wide. Except for the track bed, the disturbed areas will be revegetated.

Current and expected train traffic on the CN rail line that would serve the IGCC Power Station at the East Range Site is discussed in Section 3.5.2.3. About 10 trains per day currently travel on the CN line through Biwabik and Aurora west of the East Range Site at speeds up to 30 miles per hour. Approximately 25 grade crossings (a location where a public highway, road, street, or private roadway, including associated sidewalks and pathways, crosses one or more railroad tracks at grade) are located between Virginia and Hoyt Lakes.

Traveling at 25 miles per hour, a unit coal train would take about four minutes to clear a grade crossing. The IGCC Power Station is expected to require fuel delivery unit trains at a maximum of about 9 round trips per week for both phases, or less than an average of three additional trains traveling through cities along the line each day.

8.11 CULTURAL, HISTORIC AND ECONOMIC IMPACTS

This section of the application addresses the following project impacts:

Public Services
Archaeological and Historical Resources
Economic Impacts
Demographics and Environmental Justice

8.11.1 Public Services

This section describes existing local government services in the City of Hoyt Lakes (East Range Site) and surrounding communities that may be affected by the proposed project, including fire and medical, police, and utilities.

8.11.1.1 Fire and Emergency Medical

Hoyt Lakes operates a volunteer emergency response and fire department with 25 EMT and fire fighters, paid by service run. Hoyt Lakes operates this service cooperatively with the surrounding communities of Aurora, Biwabik and White Township, all of which contribute "cooperative" payments to cover administrative expenses, keep cost of service low, and build reserves for capital purchases. Hoyt Lakes has mutual aid agreements with different combinations of these communities for police, fire and ambulance, and charges a yearly maintenance fee for its building. The nearest hospitals are the White Community Hospital in Aurora (9 miles), and hospitals in Eveleth (27 miles) and Virginia (28 miles).

The St. Louis County Sheriff is the appointed emergency management director for the County, and the County assists municipalities when emergency response needs exceed local capabilities. Likewise, state government may supplement county resources as needed. The St. Louis County Sheriff's Office Emergency Management Division coordinates emergency management plans and has jurisdiction throughout the county unless cities have established their own emergency management organizations.

In an extreme emergency or disaster situation within the county, the Chairman of the County Board, the County Administrator, or the Sheriff has the authority to activate the St. Louis County Emergency Operations Center (EOC). Response activities are coordinated through the EOC to assure effective response and recovery. Mutual aid agreements also exist between local units of government.

8.11.1.2 Police

Hoyt Lakes has a police department consisting of five full time and five part-time officers, with backup provided by the St. Louis County Sheriff's department. St. Louis County has 94 full time and 23 part time police on staff.

8.11.1.3 Utilities

Utilities in the rural areas primarily consist of on-site wells and septic systems. The City of Hoyt Lakes treats surface water for drinking water and has a wastewater collection system that conveys wastewater to the Hoyt Lakes WWTF located in the city. Potable water service and sanitary sewer collection will be extended from the city of Hoyt Lakes existing systems to the IGCC facility site. The utility corridor will be approximately 12,500 feet in length and will affect about 14 acres.

8.11.2 Archaeological and Historical

Section 106 of the National Historic Preservation Act requires consideration of impacts on historic, archaeological and cultural properties determined eligible for listing on the National Register of Historic Places (NRHP). The area of potential effects (APE) for archaeological resources is defined as all areas of potential effects from aspects of direct, physical impacts through the construction of the facility site itself, and its associated transportation systems (road and railroads), HVTL (high voltage transmission lines), gas pipelines and other utilities. The potential area of impact due to transmission line construction includes the area within the right-of-way and nearby areas used during project construction. The recommended APE for the architectural history resources extends to 0.25 miles from the centerline of proposed HVTLs, for routes along existing or new proposed rights of way.

8.11.2.1 Archaeological

In September and October of 2004, the Applicant conducted a Cultural Resources assessment and Phase I Survey for the areas determined to have a high potential for containing archaeological sites. All work was conducted in accordance with the *SHPO Guidelines for Archaeological Projects in Minnesota* (Anfinson 2001) and *The Secretary of the Interior's Standards and Guidelines for Archeology and Historic Preservation* [48 Federal Register 44716-44740] (National Park Service 1983). A November, 2004 report summarizing the results was updated in July 2005. A copy of this report is provided in the ES. The survey area for the Station Footprint and Buffer Land, located in Sections 32 and 33, T59N, R14W within St. Louis County, Minnesota, includes approximately 640 acres (259 hectares).

The archaeological assessment included background research, a visual reconnaissance, and assessment of archaeological potentials within the survey area. Minnesota SHPO site files were researched for information on previously identified archaeological sites and architectural history properties within one mile (1.6 kilometer [km]) of the survey area, and on cultural resource surveys previously conducted within the survey area. A database query was also submitted to SHPO staff to identify any previously identified properties within the survey area, including any that are listed or determined eligible for listing on the NRHP. Archaeologists conducted a visual reconnaissance of the study area to identify locations with moderate to high archaeological potential. In most of Minnesota, such areas are defined as sites:

- Within 500 feet (150 m) of an existing or former water source of 40 acres (19 Hectares) or greater in extent, or within 500 feet (150 m) of a former or existing perennial stream
- Located on topographically prominent landscape features
- Located within 300 feet (100 m) of a previously reported site
- Located within 300 feet (100 m) of a former or existing historic structure or feature (such as a building foundation or cellar depression)

Areas defined as having a relatively low potential for containing intact archaeological resources included inundated areas, former or existing wetland areas, poorly drained areas, and locations with a 20 percent or greater slope. Archaeologists also compared current field conditions with

those from historical documents to assess the potential within the survey area for intact post-contact archaeological sites. The Phase I archaeological survey included shovel testing of those areas identified during the assessment as having greater potential for containing intact archaeological deposits. Most of the site and nearby areas were determined to have a low probability of containing archeological artifacts.

The areas near the Station Footprint and Buffer Land with greater precontact archaeological potential include:

- Topographically prominent areas in proximity to the tributary of the Partridge River
- Topographically prominent areas in proximity to the larger wetlands

Due to surface visibility being less than 25 percent throughout the survey area, shovel testing was necessary to examine those portions of the survey area identified during the assessment as having a greater potential to contain intact archaeological deposits. Shovel testing in those areas encountered no archaeological materials. Based on the low potential of much of the survey areas for intact archaeological resources, combined with the absence of archaeological material, no additional archaeological work at the Station Footprint and Buffer Land was recommended.

Detailed investigations were not performed for the East Range HVTL, natural gas pipeline, water pipeline, transportation corridors (rail line and roadways) and alternate HVTL routes. An architectural, archaeological, historical and cultural resource investigation will be conducted within the corridor selected for the project should the MPUC select the East Range Site as the Project site. These reports will be submitted to the SHPO prior to the commencement of construction activities.

8.11.2.2 Programmatic Agreement

The cultural assessments of the East Range Site do not address traditional Native American tribal boundaries or current religious practices. All federally-recognized tribes with historic or current affiliation to Minnesota and the project area have been invited to participate in the consultation process, and to become a signatory to a Programmatic Agreement. Initial consultation letters were sent in September 2005 from the Department of Energy to all federally recognized tribes that have expressed a cultural and historical interest in Minnesota. One tribe has indicated an interest in following the environmental review process associated with the Project.

8.11.2.3 Architectural Resources

A Phase I architectural history survey was conducted within the East Range Site boundaries to identify previous studies conducted in or near the project area, and to identify expected resource types. As described below, for the HVTL, rail, and road corridors, the recommended APE for the architectural history resources extends to 0.25 miles from the centerline. For underground pipelines, the APE for is limited to the width of the corridor itself.

Two architectural history resources were identified that are listed or may be eligible for inclusion into the NRHP. These include the Two Harbors to Tower Junction Segment of the DM&IR railroad, and the NRHP-listed E.J. Longyear First Diamond Drill Site (Longyear Site, SL-HLC-

001). Further evaluation of the Two Harbors to Tower Junction is needed to determine its eligibility for listing on the NRHP. A determination of effects of the proposed project on the Longyear Site and the potentially historic DM&IR line would be necessary prior to commencement of construction activities.

8.11.3 Population Trends and Demographics

The regional benefits of Mesaba One are described above in Section 6. The economic impacts specific to the local area near the East Range Site are summarized below.

8.11.3.1 Hoyt Lakes Population Trends

The population trends for Hoyt Lakes are shown below in Table 8.11-1. The data show that the population of Hoyt Lakes has declined by nearly 40%, from 3,186 in 1980 to 1,961 in 2004.

Table 8.11-1
Population Trends Since 1980 for Hoyt Lakes

Municipality	1980	1990	2000	2004
Hoyt Lakes	3186	2348	2082	1,961

The area receives a relatively large influx of temporary residents and visitors at lake cabins, resorts and campgrounds in the summer. These temporary residents are not counted in these population statistics, but they do impact the capacity of local government services to meet local needs.

The median housing value of homes in Hoyt Lakes is about $40,000 (2000 Census). Nearby lake front property likely has a considerably higher property value due to the demand for seasonal and recreational housing in the area. There are no residences within one and one-half miles of the East Range Site, and the project is not likely to adversely affect values of nearby residences. The Project would likely increase housing demand and income, which may lead to increased real estate values in the area. The influx of temporary and permanent workers for the facility would likely increase housing demand and property values.

In the case of Hoyt Lakes, many city services were originally built to serve a much larger city than now exists. The current population of about 2000 is 40% less than it was in 1980, and far below the 5,000 to 10,000 population expected at one time. The city's wastewater treatment plant, for example, was originally designed for a population of 5,000 to 10,000 people. There is no longer enough population to support a local high school hockey team for the existing ice arena, nor is there any longer an operating local grade school, although the buildings still exist. Currently, the number of EMT and fire calls for the cooperative 25-person regional EMT and fire department is just enough to support the cost of the service (about 400 runs per year). The Hoyt Lakes city manager estimates that the city could easily absorb up to five hundred new residents without requiring a new dedicated Hoyt Lakes EMT or fire department, or requiring an increase

in the number of personnel to meet the requirements of the existing cooperative agreement with neighboring communities.

A new industrial facility of the magnitude of Mesaba One and Mesaba Two is expected to result in a positive impact on the area population. The addition of over 100 direct new skilled jobs and indirect employment would be expected to attract new residents.

As noted earlier, the most recent example of a baseload power plant's impact on rural Minnesota population trends comes from the Sherburne County Generating Plant's (Sherco) in the City of Becker, Minnesota. Prior to construction and operation of Sherco, Becker was a small rural community with few businesses. Now, after more than twenty years of operation of Sherco, the City has changed dramatically. Many businesses operate within the community and the City has become a magnet for commuters, increasing its population nearly four-fold since 1980. One would expect a similar positive impact from the construction and operation of Mesaba One and Two.

8.11.3.2 Demographics and Environmental Justice

Regional population trends and demographics are summarized in Section 6.1.

The median household income in Hoyt Lakes is $39,490 and 8.9% live below the poverty level. In Hoyt Lakes, income is slightly higher and unemployment is lower than that in the City of Taconite, where median income is $30,250, with 17% reported to live below the poverty level.

The population of Hoyt Lakes is over 97% white. Along the transmission routes and other associated facility routes that are part of the East Range Site, the population is similarly overwhelmingly white. A review of census block data indicates that there is one census block adjacent to the Forbes Substation with a minority population of over 20% of the total population. However, with the low population density in the area, this could consist of only one or two minority families. There are, therefore, no concentrations of minority or poor populations along the proposed transmission routes at the East Range Site.

As described in Section 7.4 above and in the AERA Report attached as an appendix to the West Range IGCC Power Station Application for a Part 70 New Source Review Construction Authorization Permit (attached as Appendix 5), the Project's mercury emissions will not contribute appreciably to mercury concentrations in fish in nearby lakes. Therefore, the IGCC Power Station is not likely to disproportionately affect Native Americans or others in the area who may rely upon locally caught fish as a regular part of their diet.

9. REFERENCES

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10. AGENCY CONTACTS

The following list contains the names, addresses, and contact information for agencies and individuals that were contacted to obtain environmental information.

NAME	TOPIC	PHONE	E-MAIL
Air Quality			
Minnesota Pollution Control Agency			
Sandberg, Rich	Air Permitting	(651) 296-7769	Rich.Sandberg@pca.state.mn.us
Reinertsen, Jenny	Air Permitting	(218) 734-4760	Jenny.Reinertsen@pca.state.mn.us
Smith, Don	Air Permitting	(651) 296-7625	Don.Smith@pca.state.mn.us
Becker, Dennis	Air Dispersion Modeling and AERA	(651) 297-7364	Dennis.Becker@pca.state.mn.us
Nelson, Chris	Air Dispersion Modeling	(651) 296-7750	Christopher.Nelson@pca.state.mn.us
Kessler, Katrina	Water Quality	(651) 296-7376	Katrina.Kessler@pca.state.mn.us
Dymond, Mary	Human Health Risk and AERA	(651) 296-7992	Mary.Dymond@pca.state.mn.us
Ranck, Vanessa	Human Health Risk and AERA		
Monson, Bruce	Mercury and Fish Data	(651) 296-7607	Bruce.Monson@pca.state.mn.us
National Park Service			
Shepard, Don	Air quality in National Parks	(303) 969-2075	Don_Shepherd@nps.gov
Pohlman, David	Air quality in National Parks	(651) 290-3801	David_Pohlman@nps.gov
Notar, John	Air quality in National Parks	(303) 969-2079	John_Notar@nps.gov
Holbeck, Chris	Air quality in Nation Parks (Voyageurs NP)	(218) 283-9107 ext 6148	Chris_Holbeck@nps.gov
Romanski, Mark	Air quality in National Parks (Isle Royale NP)	(906) 487-9080 ext 23	Mark_Romanski@nps.gov
US Forest Service			
Wickman, Trent	Air Quality in Superior National Forest and the BWCA	(218) 626-4372	TWickman@fs.fed.us
OTHER – Contractor to National Park Service and US Forest Service			
Gebhart, Howard	Air quality consultant to NPS and Forest Service	(970) 484-7941	HGebhart@air-resource.com

NAME	TOPIC	PHONE	E-MAIL
Cultural Resources			
U.S. Department of Energy			
Hargis, Richard	Programmatic and Agreement & Cultural Resources	(412) 386-6065	Richard.Hargis@netl.doe.gov
Pukanic, George	Programmatic and Agreement & Cultural Resources	(412) 386-6068	George.Pukanic@netl.doe.gov
U.S. Forest Service			
Okstad, Walt	ARPA Permit	(218) 626-4321	Wokstad@fs.fed.us
Office of the State Archaeologist			
Koenen, Bruce	Archaeological License	(612) 725-2729	Bruce.Koenen@state.mn.us
Anfinson, Scott	Archaeological License	(612) 725-2411	Scott.Anfinson@state.mn.us
Environmental			
Minnesota Department of Natural Resources			
Kavanaugh, Chris	Biological Assessment Issues (West Range)	(218) 999-7821	Chris.Kavanaugh@dnr.state.mn.us
U.S. Army Corps. Of Engineers			
Peterson, Tim	Wetland Permitting (West Range)	(218) 834-6630	Timothy.W.Peterson@mvp02.usace.army.mil
U.S. Fish and Wildlife Service			
Burke, Paul	Threatened and Endangered Species on the West Range Site	(612) 725-3548 ext 205	Paul_Burke@fws.gov
Natural Resources			
Minnesota Department of Natural Resources			
Kavanaugh, Chris	Fisheries Concerns and 316B Guidance (West Range)	(218) 999-7821	Chris.Kavanaugh@dnr.state.mn.us
U.S. Department of Agriculture			
Rokus, Mike	Preliminary Soil Survey (East Range)	(218) 749-8343	Mike.Rokus@mn.usda.gov
Kroll, Jeff	Prime Farmland (East Range)	(218) 749-8343	Jeff.Kroll2@mn.usda.gov
Itasca County National Resources Conservation Service			
Oja, Mike	Prime Farmland (West Range)	(218) 326-6595	Mike.Oja@mn.usda.gov
Railroad			

NAME	TOPIC	PHONE	E-MAIL
BNSF Railway			
Dinkle, James		(312) 850-5699	James.Dinkle@bnsf.com
Canadian National Railroad			
Stein, Dan		(218) 628-4135	Daniel.Stein@cn.ca
St. Louis County Regional Rail Authority			
Manzoline, Bob		(218) 254-0086	Bob.Manzoline@ironworld.com
Transportation			
Itasca County Highway Department (West Range)			
Christy, Dave	Roadway Alignments, Ownership, Design Standards	(218) 327-2853	Dave.Christy@co.itasca.mn.us
Carter, Tony	Roadway Alignments, Ownership, Design Standards	(218) 327-2853	Tony.Carter@co.itasca.mn.us
Minnesota Department of Transportation			
Larson, Brian	Plans for TH 169, Access Point for New Roadway (West Range)	(218) 723-4960 ext 3322	Brian.Larson@dot.state.mn.us
Erickson, Daniel	Plans for TH 169, Access Point for New Roadway (West Range)	(218) 723-4960 ext 3305	Daniel.Erickson@dot.state.mn.us
Levenson, Mark	Existing Traffic Volumes and Forecasts (West and East Range)	(651) 296-8535	Mark.Levenson@dot.state.mn.us
Water Appropriation/Surface Water			
Minnesota Department of Natural Resources			
Peloquin, Mike		(218) 327-4417	Mike.Peloquin@dnr.state.mn.us
Christman, Howard	Floodplains	(218) 327-4106	Howard.Christman@dnr.state.mn.us
Liebfried, Bob	Canisteo Mine Pit	(218) 327-4232	Bob.Liebfried@dnr.state.mn.us
Japs, Jim		(651) 297-2835	Jim.Japs@dnr.state.mn.us
Adams, John	Hill Annex Complex	(218) 327-4110	John.Adams@dnr.state.mn.us
Railson, Steve	Hill Annex State Park	(218) 247-7215	Steve.Railson@dnr.state.mn.us
Kavanaugh, Chris	Fisheries	(218) 999-7821	Chris.Kavanaugh@dnr.state.mn.us
Maurer, Paul	Hill Annex State Park	(218) 327-4388	

NAME	TOPIC	PHONE	E-MAIL
Minnesota Pollution Control Agency			
Dexter, Jim	Construction Stormwater	(218) 529-6253	James.Dexter@pca.state.mn.us
Thomas, John	Industrial Stormwater	(218) 723-4928	John.Thomas@pca.state.mn.us
Estabrooks, Tom		(218) 725-7763	Tom.Estabrooks@pca.state.mn.us
Kessler, Katrina	EPA CWA 316(b)	(651) 296-7376	

APPENDIX 1:

APPENDIX 1 UNUSED

APPENDIX 2:

APPENDIX 2 UNUSED

APPENDIX 3:

Appendix 3 Unused

APPENDIX 4:

NATURAL GAS MATERIAL SAFETY DATA SHEETS

APPENDIX 5:

Application for Part 70/New Source Review Construction Authorization Permit: West Range IGCC Power Station

See attached MPUC Joint Permit Application Appendices CD

APPENDIX 6:

Application for NPDES/State Disposal System Permit: West Range IGCC Power Station

See attached MPUC Joint Permit Application Appendices CD

APPENDIX 7:

East Range Water Quality Data

APPENDIX 8:

MARKET ANALYSIS FOR SLAG AND SULFER PRODUCED BY THE IGCC POWER STATION

APPENDIX 9:

Application for a Water Appropriation Permit: West Range IGCC Power Station

See attached MPUC Joint Permit Application Appendices CD

APPALACHIAN POWER
A unit of American Electric Power

News&Issues

News Releases

1/12/2006

APPALACHIAN POWER FILES APPLICATION TO BUILD IGCC POWER PLANT IN WEST VIRGINIA

Appalachian Power yesterday filed an application with the Public Service Commission of West Virginia seeking authority to construct a 600-megawatt Integrated Gasification Combined Cycle (IGCC) electric generating unit in West Virginia. The proposed power plant would be located next to the company's Mountaineer Plant near New Haven in Mason County.

Gov. Joe Manchin announced the filing in his State of the State address, saying that progress to site an IGCC plant in West Virginia is good economic development news.

"As one of the first commercial scale gasification projects, this proposed plant will offer the state an opportunity to lead the nation in the development of clean coal technology for power generation," Gov. Manchin said. "Gasification technology also offers future opportunities to produce clean liquid fuels and chemical feedstock for other industries."

An IGCC power plant efficiently reduces and removes sulfur dioxide, nitrogen oxides, particulates and mercury from plant emissions. IGCC plants also offer the opportunity for more efficient, less costly carbon capture for disposal in deep geologic formations.

The filing for a Certificate of Public Convenience and Necessity reflects Appalachian Power's need for new generating capacity to meet its customers' growing demand for electricity. Although Appalachian Power has not made a final decision about whether it will build a plant in West Virginia, the certificate is the first step required before the company could proceed with construction of a plant, according to Dana Waldo, Appalachian Power president and COO.

"We're pleased to be able to take this next step in the regulatory process to site an IGCC plant in West Virginia," Waldo said. "Looking beyond this plant, we also have committed to work with the Governor to address policy and technical issues related to his Coal Conversion Initiative."

Appalachian Power's parent company, American Electric Power (NYSE: AEP), announced in 2004 that it plans to build up to 1,200 megawatts of new generation using IGCC technology somewhere in its eastern service territory, and last year narrowed its possible sites to one each in West Virginia, Ohio and Kentucky. The company has since filed a cost recovery plan for a plant in Ohio with the Public Utilities Commission of Ohio, and has stated its intention to build the first 600-megawatt plant along the Ohio River in Meigs County, if cost recovery is ensured.

General Information
- News releases
- Storms & Outages
- Media contacts

Rates
- West Virginia
- Virginia

Transmission Projects
- Pending approval
- In construction

Generation Projects
- IGCC
- Scrubbers & Environmental controls
- Smith Mountain Relicensing

Advanced search

Appalachian Power provides electricity to 1 million customers in Virginia, West Virginia and Tennessee. It is a unit of American Electric Power, the nation's largest electricity generator. AEP owns more than 36,000 megawatts of generating capacity and is one of the nation's largest electric utilities, with more than 5 million customers in 11 states.

This report made by AEP and certain of its subsidiaries contains forward-looking statements within the meaning of Section 21E of the Securities Exchange Act of 1934. Although AEP and each of its registrant subsidiaries believe that their expectations are based on reasonable assumptions, any such statements may be influenced by factors that could cause actual outcomes and results to be materially different from those projected. Among the factors that could cause actual results to differ materially from those in the forward-looking statements are: electric load and customer growth; weather conditions, including storms; available sources and costs of, and transportation for, fuels and the creditworthiness of fuel suppliers and transporters; availability of generating capacity and the performance of AEP's generating plants; the ability to recover regulatory assets and stranded costs in connection with deregulation; the ability to recover increases in fuel and other energy costs through regulated or competitive electric rates; new legislation, litigation and government regulation including requirements for reduced emissions of sulfur, nitrogen, mercury, carbon and other substances; timing and resolution of pending and future rate cases, negotiations and other regulatory decisions (including rate or other recovery for new investments, transmission service and environmental compliance);resolution of litigation (including pending Clean Air Act enforcement actions and disputes arising from the bankruptcy of Enron Corp.); AEP´s ability to constrain its operation and maintenance costs; AEP´s ability to sell assets at acceptable prices and on other acceptable terms, including rights to share in earnings derived from the assets subsequent to their sale; the economic climate and growth in AEP´s service territory and changes in market demand and demographic patterns; inflationary trends; AEP´s ability to develop and execute a strategy based on a view regarding prices of electricity, natural gas, and other energy-related commodities; changes in the creditworthiness and number of participants in the energy trading market; changes in the financial markets, particularly those affecting the availability of capital and AEP´s ability to refinance existing debt at attractive rates; actions of rating agencies, including changes in the ratings of debt; volatility and changes in markets for electricity, natural gas, and other energy-related commodities; changes in utility regulation, including membership in regional transmission structures; accounting pronouncements periodically issued by accounting standard-setting bodies; the performance of AEP´s pension and other postretirement benefit plans; prices for power that AEP generates and sells at wholesale; changes in technology and other risks and unforeseen events, including wars, the effects of terrorism (including increased security costs), embargoes and other catastrophic events.

Jeri Matheney
(304) 348-4130
Cell: (304) 543-1377
jhmatheney@aep.com

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United States Department of Energy
Office of Public Affairs
Washington, D.C. 20585

News Media Contact(s):
Craig Stevens, 202/586-4940

For Immediate Release
February 22, 2006

DOE Awards $235 Million to Southern Company to Build Clean Coal Plant

WASHINGTON , DC - The U.S. Department of Energy awarded $235 million to Southern Company, in partnership with the Orlando Utilities Commission and Kellogg, Brown and Root, to develop one of the cleanest coal-fired power plants in the world.

Representatives of the Energy Department and Southern Company signed a cooperative agreement that launches the design, construction, and demonstration of an integrated gasification combined cycle (IGCC) power generation system at the Orlando Utilities Commission's Stanton Energy Center. The system will produce 285 megawatts of electricity for the Orlando area – which will power approximately 285,000 households – and is scheduled to begin operations in 2010.

"The funding for this new Southern Company facility allows for greater availability of more clean coal technology in the U.S. By harnessing our coal resources in an environmentally sound manner, we will help power generations to come with this clean, safe energy source," Assistant Secretary for Fossil Energy Jeffrey Jarrett said.

The project is one of four selected in October 2004 under the President's Clean Coal Power Initiative, a $2 billion, 10-year effort to advance technologies that can help meet the Nation's growing demand for low-cost electricity while protecting the environment.

IGCC technology, which produces a coal-derived synthesis gas for power generation, is considered today's environmentally preferred source of electricity from coal. The system being developed by Southern Company and its partners will add advanced emission controls to make it one of the cleanest, most energy-efficient coal power plants built to date.

In addition, the technology to be used in Orlando is unique in that it will cost-effectively use low-rank, high-moisture, and high-ash content coals at lower costs. These coals, which include lignite and sub-bituminous (e.g., Powder River Basin) coals, make up half of the proven reserves in the United States and the world.

The Office of Fossil Energy's National Energy Technology Laboratory will manage the project for the Energy Department. Initial funding of $13.8 million will support project start-up activities through March 2007. The total cost of the 10-year project is $557 million, of which the Energy Department will contribute $235 million.

For more information on the Department of Energy's Office of Fossil Energy programs and initiatives, visit http://www.fossil.energy.gov/.

U.S. Department of Energy, Office of Public Affairs, Washington, D.C.

Press Release

NRG ENERGY, INC. (NYSE - NRG)

NRG Announces Comprehensive Repowering Initiative

Company Release - 06/21/2006 07:06

PRINCETON, N.J.--(BUSINESS WIRE)--June 21, 2006--

NRG Energy, Inc. (NYSE:NRG) today announced plans to develop approximately 10,500 megawatts (MW) of new generation capacity over the next decade to help meet the energy needs of its high-demand, capacity-constrained markets and to support NRG's continued growth. This repowering initiative, which will be funded with the support of partners and project finance debt, would represent a total investment of $16 billion.

With this repowering initiative, NRG will:

-- Enhance its dispatch mix with almost 8,000 MW of new baseload capacity - including 2,700 MW of nuclear - and 2,500 MW of new, highly efficient intermediate and peaking capacity;

-- Further diversify its fuel mix and reduce reliance on higher-priced, imported fuels, not only through its solid fuel repowerings, but also through the acquisition of a new wind development company with wind projects in active development in Texas and California;

-- Create thousands of new construction jobs and 1,500 permanent jobs; and

-- Reduce the carbon intensity of NRG's baseload fleet by 20-25 percent.

"NRG is strategically located in domestic markets with high and growing demand for power and an over-reliance on expensive natural gas for their power generation," said David Crane, NRG's President and Chief Executive Officer. "NRG's development program is designed to meet the growing energy needs of these regions, while both reducing their dependence on natural gas for power generation purposes and making meaningful progress towards reducing our carbon profile."

"Our proposed mix of baseload plants--involving two nuclear units, three gasified coal units, two traditional pulverized coal units with full back-end controls, at least one modern combined cycle plant and at least two wind farms--will substantially reduce the carbon intensity of NRG's existing baseload fleet, in particular, and of the nation's baseload coal alternative, in general," said Crane. "And our shareholders will benefit from the economic returns of these investments."

Project Financing Preserves NRG's Financial Strength and Flexibility

"Consistent with NRG's track record of financial discipline and capital allocation, the financing plan for these projects preserves NRG's balance sheet strength and liquidity," said Robert Flexon, NRG's Executive Vice President and Chief Financial Officer. "Investments will be underpinned by long term offtake contracts and hedges that support non-recourse project financing as well as third party equity partners and the Company's existing cash flows."

Focus on Operational Excellence and Active Risk Management to Be Maintained

"This repowering and development program builds on the foundation of operational excellence being advanced through our FORNRG initiatives. Our stakeholders can be confident that we will maintain our focus on aggressive cost controls and superior operating performance," Crane said.

Given the size, capital intensity and long development time for many of these new plants, particularly the baseload plants, NRG intends to contract at least 70 percent of its new output through power purchase agreements, bilateral contracts or hedges with financial firms. NRG's plants are located in regions that currently have significant opportunities for long term offtake agreements. For example, in the Northeast, request for proposals for power purchases have been announced or authorized in Connecticut, Delaware and New York; and bilateral contracts for wholesale power are being pursued by

cooperatives, municipalities, investor-owned utilities and large industrials in California, Louisiana, and Texas. As an example, NRG has secured a significant power purchase agreement with SMEPA for 75 MW for 4.5 years that will carry them until BC II unit 4 goes commercial, at which time they will take equity (and the associated output) in the BC II unit 4 project.

Environmentally Responsible Development

All of NRG's proposed new generation will utilize a variety of state-of-the-art environmental technologies.

Upon completion of the development program, NRG will have increased its US solid-fuel generation capacity by 46 percent(1) while reducing its air emissions and carbon intensity by 67 percent and 20-25 percent, respectively, compared to current levels.

Additionally, the expansions announced today will be built adjacent to existing generating units and use existing infrastructure, including roads and water treatment facilities, minimizing additional environmental impact to the surrounding areas.

Renewables (Wind)

NRG announced yesterday that it has reached a definitive agreement to acquire privately held Padoma Wind Power, LLC, a leading wind energy development and co-development company. NRG's acquisition of Padoma is part of "ecoNRG," the Company's ongoing environmental business effort, targeted at achieving continuous environmental innovation and improvement.

Padoma's principals have over 80 years of combined experience in the development, technical integration, financing, construction and operation of utility-scale wind energy facilities. Together, they have led the development, financing, construction and operation of more than 40 wind farms in the United States and Europe comprising over 1,300 MW of installed capacity. Padoma currently has three projects under active development independently, in addition to a pipeline of over a dozen wind projects which it is developing in conjunction with third parties. The projects under active development include over 500 MW of new wind generation in California, Texas and New Mexico.

The addition of a wind development team with a proven track record of execution is a meaningful step toward building a scaleable renewable energy platform. NRG anticipates future constraints on carbon production, increasing the cost of entry into the renewable energy market in the mid to long term.

"Acquiring Padoma is consistent with NRG's multi-fuel strategy and provides us with immediate access to industry-leading expertise and a robust project pipeline in the growing wind generation market," said Crane. "More than 20 states have passed legislation mandating a renewable portfolio standard as part of their efforts to curb emissions. With Padoma, NRG is well-positioned to meet this demand for renewable energy sources, while also reducing our own carbon intensity and providing financial upside opportunities through the expansion of our energy services offering."

Regional Overview

Texas

Texas's demand growth is among the strongest in the nation and in order to ensure the reliability of electrical service in the region, new plant construction is essential. NRG's development plan incorporates multiple technologies including gas peakers, pulverized coal and nuclear power. Each new plant's permitting and construction schedule varies, enabling NRG to meet expected demand growth as it develops.

NRG's repowering plan for Texas contemplates adding 3,500 MW of new baseload capacity using both coal and nuclear fuel, as well as 500 MW of more efficient, gas-fired peaking and intermediate capacity to serve particularly high-demand, capacity constrained areas around Houston. NRG also anticipates building wind facilities in Texas as part of the Padoma development portfolio.

"Texas has a broad and distinct history of meeting the nation's energy needs," said Texas Lieutenant Governor David Dewhurst, commenting on the nuclear and wind components of NRG's announcement. "This is a direct result of the can-do entrepreneurial spirit that has shaped our great state. Continuing to develop an alternative fuels industry in Texas will help ensure we remain an energy leader nationally for decades to come."

Recent developments in our Texas repowering initiative include:

-- On June 19, 2006, NRG filed a letter of intent with the Nuclear Regulatory Commission to construct 2,700 MW of nuclear power at the existing South Texas Project (STP) nuclear facility

-- On June 12, 2006, NRG filed an air permit application with the Texas Commission on Environmental Quality (TCEQ) for Limestone 3, a new 800 MW pulverized coal unit

-- On June 21, 2006, NRG filed an air permit application with the TCEQ for uprating two W. A. Parish coal units by a total of approximately 100 MW by 2010. This project includes the installation of back-end emission controls (i.e. scrubbers). When the two scrubbers are added, emission of SO2 (inclusive of Limestone 3 and the two uprates), will decline by approximately 30,000 tons annually.

-- On May 1, 2006 NRG provided letters to state leaders in support of the FutureGen Industrial Alliance. One of two of the Texas sites proposed for the FutureGen IGCC test unit would be on NRG-donated property near our Limestone facility

STP Units 3 & 4

Construction of Units 3 and 4 is expected to cost $5.2 billion, creating approximately 3,000 construction jobs per unit during the peak construction period and an additional 500 new operating staff positions per unit. Our development plan for each of the new nuclear units is expected to create over $9.2 billion of economic activity for the State and result in 5,600 new permanent jobs statewide.(2)

NRG will proceed with permitting and development of new nuclear power generation at STP based on ABWR nuclear power plant technology, which is proven in design and construction and has a track record of reliable and safe operation. NRG filed its letter of intent to submit an application with the Nuclear Regulation Commission on June 19, 2006 to construct two new ABWR units at STP. The ABWR technology is the most advanced nuclear technology in operation in the world today with a history of on time, on budget construction in Japan. The General Electric Company's ABWR design has been certified by the U.S. Nuclear Regulatory Commission. It is NRG's intent to work with GE and Hitachi,(which has been involved in developing and constructing four ABWR plants in operation in Japan) as well as GE's other international team of suppliers with experience in successfully constructing ABWR nuclear power plants.

"Nuclear power is an important part of the continued development of our baseload fleet in Texas,." said Steven Winn, NRG's Executive Vice President and President, Texas Region. "We recognize the need for new, low-cost generation and we recognize the importance of reducing the emissions profile of power generators within the growing ERCOT market."

Limestone Unit 3

NRG expects to invest $1.2 billion to construct the new unit at Limestone. With prompt approval of the permit the unit could be online by 2012. Approximately 1,000 construction jobs will be created at peak construction and an additional 100 new permanent operating positions are expected upon commencement of commercial operations. The aggregate development plan for Limestone Unit 3 is expected to create over $4.3 billion of economic activity for the State, and result in 1,800 new permanent jobs statewide, including 1,300 in central Texas.(3)

NRG anticipates that off-take for Limestone will be covered through a blend of bi-lateral negotiated contracts with local municipalities, industrials and coops as well as use of more market-based hedge instruments. NRG is currently in negotiations with a range of potential off-takers.

"The existing Limestone units are consistently among the safest units in the country, and maintain some of the country's highest capacity factors, and we expect the new unit to operate according to the same high standards," said Winn.

Parish Uprate and Associated Scrubbers

Under the plan filed with the TCEQ today, the output of Parish Unit 7 will be increased approximately 40 MWs in 2008, and the output of Parish Unit 6 will be increased by 60 MWs in 2010. Associated with these uprates, NRG will add one new scrubber at Parish in 2010, and a second new scrubber at Parish in 2014. The two scrubbers will result in reduced emissions of SO2 of approximately 30,000 tons annually.

New Gas Capacity

In August, NRG intends to file a multi-site permit application to begin to update its Houston-based gas generation fleet. New, efficient gas units will be added to replace existing capacity. The new, fast-start units will provide better grid support within the Houston zone. These units should provide additional support for periods of high electricity demand, and produce a net reduction in emissions per MWhr generated. The anticipated total increase in capacity is approximately 500 MW.

Net Reduction in Emissions per MW.

NRG remains committed to providing additional generation to Texas in the most environmentally responsible manner possible.

Since 1999, NRG and its predecessor companies have spent in excess of $700 million to add emissions control technologies to its existing generation fleet in Texas. This has resulted in a net reduction in nitrogen oxide (NOx) emissions of 75 percent. By scrubbing the two Parish units, NRG will continue this philosophy of proactive investment in environmental control technology. After the addition of this equipment, fleet sulfur dioxide (SO2) emissions will be reduced by a further 40 percent.

With the completion of two new nuclear units at STP, overall emissions intensity across the NRG Texas fleet will decline by a combined 20-30 percent.

Proposed New Generation Facilities in Texas

Unit Name and Location	Fuel/Technology	Dispatch	Gross MW Additions	Date of Operations
Limestone unit 3	Coal/Pulverized Coal	Baseload	800	2012
STP units 3 and 4	Nuclear/ABWR	Baseload	2,716	2014/2015
Houston gas peakers	Natural Gas/CT, Peaker/ CCGT	Intermediate	500	2008-2010

Northeast

The NRG Northeast redevelopment plan calls for the addition of 2,250 MW of new baseload capacity using IGCC technology and 840 MW of new, dual-fuel oil and gas-fired intermediate and peaking capacity to serve particularly high-demand, capacity constrained areas, such as New York City and southwest Connecticut. As part of this plan, NRG expects to retire 968 net MW of less efficient, higher emitting units.

Recent developments in our Northeast repowering initiatives include:

-- Completed a year long evaluation process to evaluate and choose a technology provider and assess both site feasibility and economic viability;

-- Completed a thorough technological review of the IGCC technology providers resulting in the selection of a preferred coal gasification process;

-- Initiated permitting process for each of the sites NRG plans to repower; and

-- Developed a specific development and action plan for each state.

NRG expects to contract substantially all of its development projects in the Northeast through state administered processes. The contracts will range up to 20 years in length. These processes will commence as early as the fourth quarter of 2006 and are currently anticipated to be completed in the first half of 2007. NRG has performed extensive due diligence to prepare to participate in these processes and has begun the permitting processes.

NRG's Northeast development plan is expected to result in lower emission rates across the board, including a 59 percent reduction in SO2, a 49 percent reduction in NOX, an 84 percent reduction in mercury, and a 4 percent reduction in CO2 intensity.

During peak construction NRG expects to create almost 3,000 construction and support jobs in the Northeast region. NRG expects to add an additional 300 permanent operating staff positions following completion of the development plan.

"Virtually all key stakeholders in the Northeast agree that new investment in power plants is needed to address rising and unstable power prices stemming from tightening of supply and demand and an over-reliance on natural gas as a fuel for power generation. This new investment must also address the need to reduce emission levels," said Curt Morgan, Executive Vice President and President, Northeast Region. "With NRG's Northeast development plan we address these critical issues with proposed investment in state-of-the-art power plant technology while increasing employment and driving additional economic activity throughout the Northeast."

Proposed New Generation Facilities in Northeast Region

Unit Name and Location	Fuel / Technology	Dispatch	Gross MW Additions	Date of Operations
Indian River, DE	Coal/IGCC	Baseload	752	2011/2012
Montville, CT	Coal/IGCC	Baseload	752	2011/2012
Huntley, NY	Coal/IGCC	Baseload	752	2013/2014
Cos Cob, CT	Gas/CT	Peaking	40	2008
Middletown, CT	Gas/CCGT	Peaking	300	2009
Devon, CT	Gas/CCGT	Peaking	200	2009
Astoria, NY	Gas/CCGT	Peaking	200-400	2008-2010

South Central

NRG's development plan for the South Central region adds of 1,000 MW of new baseload capacity. Upon completion of this expansion as well as development projects already underway, NRG will have 2,775 net MW of generating capacity in the South Central region.

Recent developments in our South Central repowering initiative include:

-- Agreed upon key terms with three parties for joint development and co-ownership of Big Cajun II Unit 4. South Mississippi Electric Power Association (SMEPA), East Texas Electric Cooperative and City of North Little Rock, Arkansas will collectively own 260 MW of the project.

-- Bridge contract with SMEPA for 75 MW for 4.5 years

-- Permit for Big Cajun I re-powering filed with LDEQ(4)

The South Central Region will utilize state-of-the-art emissions controls, including selective catalytic reduction (SCR), scrubbers, sorbent injection, and bag houses to meet Best Available Control Technology (BACT) requirements. The total cost of this equipment is projected to be approximately $850 million and will generate net emission reductions of approximately 55 percent for SO2, 40 percent for NOx, and 70 percent for mercury while remaining net neutral on NRG's carbon intensity in the region.

During peak construction NRG expects to create approximately 1,400 construction and support jobs and a permanent operating staff of 70 is expected following completion of the development plans.

"By building coal-fired plants in gas-based markets, NRG will be able to provide consumers with lower-cost, stable and reliable energy solutions," said John Brewster, NRG's Executive Vice President and President, South Central Region. "This is yet another way that NRG will strengthen relationships with stakeholders in Louisiana and distinguish itself from other power producers in the region."

Proposed New Generation Facilities in South Central Region

Unit Name and Location	Fuel / Technology	Dispatch Additions	Gross MW	Date of Operations
Big Cajun II - Unit 4	Coal/Pulverized Coal	Baseload	775	2010
Big Cajun I	Petcoke/Fluidized Bed Boiler	Baseload	230	2009

West

The expansion of NRG's portfolio in the West is predicated on receiving long-term off-take agreements from the incumbent utilities. NRG's development projects lie inside the Los Angeles and San Diego load pockets. Southern California Edison and SDGE are significantly short resources and have announced competitive solicitations for new generation. NRG intends to compete in these solicitations.

NRG has allocated $1.5 billion for the West redevelopment plan, which contemplates adding 647 gross MW of new gas-fired base load capacity and 1,145 gross MW of new gas-fired intermediate and peaking capacity. NRG also anticipates building a new 150 MW wind facility.

Recent developments in our West repowering initiative include:

-- Continued to satisfy the conditions associated with the combined cycle permit at El Segundo;

-- Began the process leading to the dismantling of the retired units at El Segundo; and

-- Initiated a permit process for a combined cycle plant at Encina.

The El Segundo site is currently permitted for a 640 MW combined cycle unit. The Long Beach site is in the process of being permitted. NRG is preparing a submittal for a 339 MW peaking facility at the Long Beach site.

The Encina site is located inside the SDGE service territory. SDGE has indicated a strong interest in immediate peaking capacity and future base load capacity at the Encina site. NRG is preparing a competitive bid of 339 MW of new peaking generation at the site, configured to be converted to 640 MW of combined cycle generation.

NRG has adequate emissions offsets to support the new generation at all of the California sites.

"Wind based energy will add another element of diversity to our fuel mix," said Steve Hoffmann, NRG's Senior Vice President and President, Western Region. "We are pleased to expand our presence in the West with this new wind

capacity and new highly efficient gas-fired baseload as we strive to provide consumers with a more reliable energy supply."

Proposed New Generation Facilities in West Region

Unit Name and Location	Fuel / Technology	Dispatch	Gross MW Additions	Date of Operations
El Segundo	Gas/CCGT	Baseload	647	2011
Long Beach	Gas/CT	Peaker	354	2009
Encina	Gas/CCGT	Intermediate	730	2011
Kearney Mesa	Gas/CT	Peaker	144	2011

Development Principles

The Company reaffirms that this comprehensive repowering initiative will be pursued in accordance with its longstanding commitments to prudent balance sheet management, risk diversification, return of capital to shareholders and construction based on long term contracts.

Webcast Information

NRG will host a live webcast for analysts, investors and the media at 1:30 p.m. eastern today, June 21, 2006, to discuss today's announcement. To listen to the live webcast and view the accompanying slide presentation, log on to NRG's website at http://www.nrgenergy.com and click on "Investors." To participate in the call, dial 877-407-8035. International callers should dial 201-689-8035. The call will be available for replay shortly after completion of the live event on the "Investors" section of the NRG website.

Satellite, C-Band Feeds:

DATE	TIME (all times EASTERN)	COORDINATES
Wednesday, June 21	2:30 - 2:45 p.m.	IA 5, Tr.13, DL 3960V
Thursday, June 22	4:30 - 4:45 a.m.	IA 5, Tr. 13, DL 3960V
Thursday, June 22	4:30 - 4:45 p.m.	IA 5, Tr. 23, DL 4160V

Technical Info DURING FEED ONLY, NBN TOC, 212-684-8910, EXT. 221

About NRG

NRG Energy, Inc. now owns and operates a diverse portfolio of power-generating facilities, primarily in Texas and the Northeast, South Central and Western regions of the United States. Its operations include baseload, intermediate, peaking, and cogeneration facilities, thermal energy production and energy resource recovery facilities. NRG also has ownership interests in generating facilities in Australia, Brazil and Germany.

Safe Harbor Disclosure

This news release contains forward-looking statements within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934. Such forward-looking statements are subject to certain risks, uncertainties and assumptions and include NRG's expectations regarding the timing, completion, costs, financing, environmental impact, job creation and financial success of the development projects described herein, and typically can be identified by the use of words such as "will," "should," "expect," "estimate," "anticipate," "forecast," "plan," "believe" and similar terms. Although NRG believes that its expectations are reasonable, it can give no assurance that these expectations will prove to have been correct, and actual results may vary materially. Factors that could cause actual

results to differ materially from those contemplated above include, among others, general economic conditions, permitting and regulatory obstacles, construction delays, the volatility of energy and fuel prices, changes in the wholesale power markets and related government regulation, the availability of financing and the condition of capital markets generally, our ability to access capital markets, and the inability to implement value enhancing improvements to plant operations and companywide processes.

NRG undertakes no obligation to update or revise any forward-looking statements, whether as a result of new information, future events or otherwise. The foregoing review of factors that could cause NRG's actual results to differ materially from those contemplated in the forward-looking statements included in this news release should be considered in connection with information regarding risks and uncertainties that may affect NRG's future results included in NRG's filings with the Securities and Exchange Commission at www.sec.gov.

More information on NRG is available at www.nrgenergy.com

(1)Based on net MW prior to equity sell down divided by existing coal capacity

(2)June 2006Texas and Matagorda County Economic Impact study prepared for NRG by The Perryman Group

(3)Based on Economic Impact Analysis Report prepared by Perryman Group dated June 2006

(4)Louisiana Department of Environmental Quality

Source: NRG Energy, Inc.

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Energy Facilities Under Review

Cascade Wind Project

Golden Hills Wind Farm Project

Lower Columbia Energy Center

Shepherds Flat Wind Farm

Cascade Wind Project

Proposed Facility:
Wind energy facility with a peak generating capacity of approximately 60 megawatts.

Location: Wasco County

Status:
UPC Oregon Wind LLC has submitted a preliminary application for a site certificate. The Department is reviewing the application for completeness.

On October 9, the applicant submitted its response to the Department's first Request for Additional Information (RAI). The response is posted on the applicant's website.

Applicant:
UPC Oregon Wind LLC
Website: www.cascadewind.com

Contact:
Krista A. Kisch
Vice President
Business Development - West Region
UPC Wind Management, LLC
110 West A Street, Suite 675
San Diego, CA 92101

(619) 320-2010

Department of Energy Staff: Adam Bless

History:

- **July 12, 2006:** Applicant submitted a request for expedited review.
- **July 25, 2006:** Department of Energy approved expedited review.
- **April 10, 2007:** Applicant submitted a preliminary application for a site certificate.

Documents:

(The applicant has posted the preliminary application and additional information on its website.)

- "Incompleteness" Letter to the Applicant [31 kb pdf]
  Attachment 1 (ODFW: deer collaring project) [22 kb pdf]
  Attachment 2 (ODFW: completeness comments) [33 kb pdf]
  Attachment 3 (USFWS comments) [793 kb pdf]
  Attachment 4 (Pacific Habitat Services comments) [17 kb pdf]
  Attachment 5 (RAI #1) [22 kb pdf]
- UPC Letter (requesting additional time) [25 kb pdf]
- October 2007 Update [18 kb pdf]

Golden Hills Wind Farm Project

Proposed Facility:
A wind energy facility with a peak generating capacity of up to 400 megawatts.

Location: Sherman County

Status:
BP Alternative Energy of North America, Inc., has submitted a preliminary application for a site certificate. The preliminary application is posted on the applicant's website. The Department is reviewing the preliminary application for completeness.

Applicant:
BP Alternative Energy of North America, Inc.
Website: www.goldenhillswind.com

Contact:
Kelly O'Brien
BP Alternative Energy North America Inc.
700 Louisiana Street
Suite 3300
Houston, TX 77002

(713) 354-2153

Department of Energy Staff: Adam Bless

History:

- **April 11, 2007:** Applicant submitted a Notice of Intent.
- **July 6, 2007:** Department issued a Project Order.
- **August 10, 2007:** Applicant submitted a preliminary application.

Documents:

- Notice of Intent (main text) [182 kb pdf]
  Project Map [2.2 MB pdf]
- Project Order, July 2007 [94 kb pdf]

Lower Columbia Energy Center

Lower Columbia Clean Energy Center Project

Proposed Facility:
Integrated coal gasification combined cycle power plant with a peak generating capacity of 520 megawatts.

Location:
Columbia County

Status:
The applicant has submitted a notice of intent. The Department has issued a project order.

Applicant:

Westward Energy LLC

Contact:
Eric Gjelde
Senior Vice President
Westward Energy LLC
P.O. Box 11637
Bainbridge Island, WA 98110

(206) 780-7826

Department of Energy Staff: Adam Bless

History:

- **September 18, 2006:** Applicant submitted a notice of intent

Documents:

- Notice of Intent, Exhibits A and B [2.09 MB pdf]
  Notice of Intent, Exhibits C through I [3.09 MB pdf]
  Notice of Intent, Exhibit J [3.86 MB pdf]
  Notice of Intent, Exhibit K [4.11 MB pdf]
  Notice of Intent, Exhibits L through P [926 kb pdf]
- Project Order[101 kb pdf]

Shepherds Flat Wind Farm

Proposed Facility:
Wind energy facility with a peak generating capacity of up to 909 megawatts.

Location: Gilliam and Morrow counties.

Project Area Map (Application Figure C-1) [168 kb pdf]
Site Boundary Map (Application Figure C-2) [2.7 MB pdf]

Status:
The application is complete. The Department has requested comments from reviewing agencies and the public. The Department is preparing a draft proposed order.

Applicant:
Caithness Shepherds Flat, LLC

Contact:
Patricia Pilz
656 San Miguel Way
Sacramento, CA 95819

(916) 456-7651

Department of Energy Staff: John White

History:

- **June 27, 2006:** Applicant submitted a notice of intent.
- **February 1, 2007:** Applicant submitted a preliminary application.

- **February 14, 2007:** Applicant submitted an amended preliminary application.
- **November 19, 2007:** Date of filing (application complete).

Documents:

- Notice of Intent, June 2006.
  Part 1 (main text, without maps, and attachment P1) [333 kb pdf]
  Part 2 (map G2, Project Location) [203 kb pdf]
  Part 3 (map G3, BPA Facilities) [1.46 mb pdf]
  Part 4 (map G4, Preliminary layout, north project area [1.96 mb pdf]
  Part 5 (map G5, Preliminary layout, south project area [2.02 mb pdf]
  Part 6 (map G6, Study Areas) [413 kb pdf]
  Part 7 (Exhibit A attachments) [404 kb pdf]
  Part 8 (Attachment J1) [1.82 mb pdf]
  Part 9 (Attachment J2) [2.87 mb pdf]
- Public Notice of Filing [45 kb pdf]
- Application, November 2007. The application is not available for downloading. The complete application consists of the Amended Preliminary Application submitted in February 2007 and the Application Supplement submitted in November 2007. These documents are available for public review at the Oregon Department of Energy in Salem, at the Gilliam County Library, 310 S. Main in Condon, at the Arlington Public Library, 500 W. First Street in Arlington and at the Boardman Library, 200 South Main Street in Boardman.

GASIFICATION TECHNOLOGIES 2005

San Francisco, CA,

October 11 2005

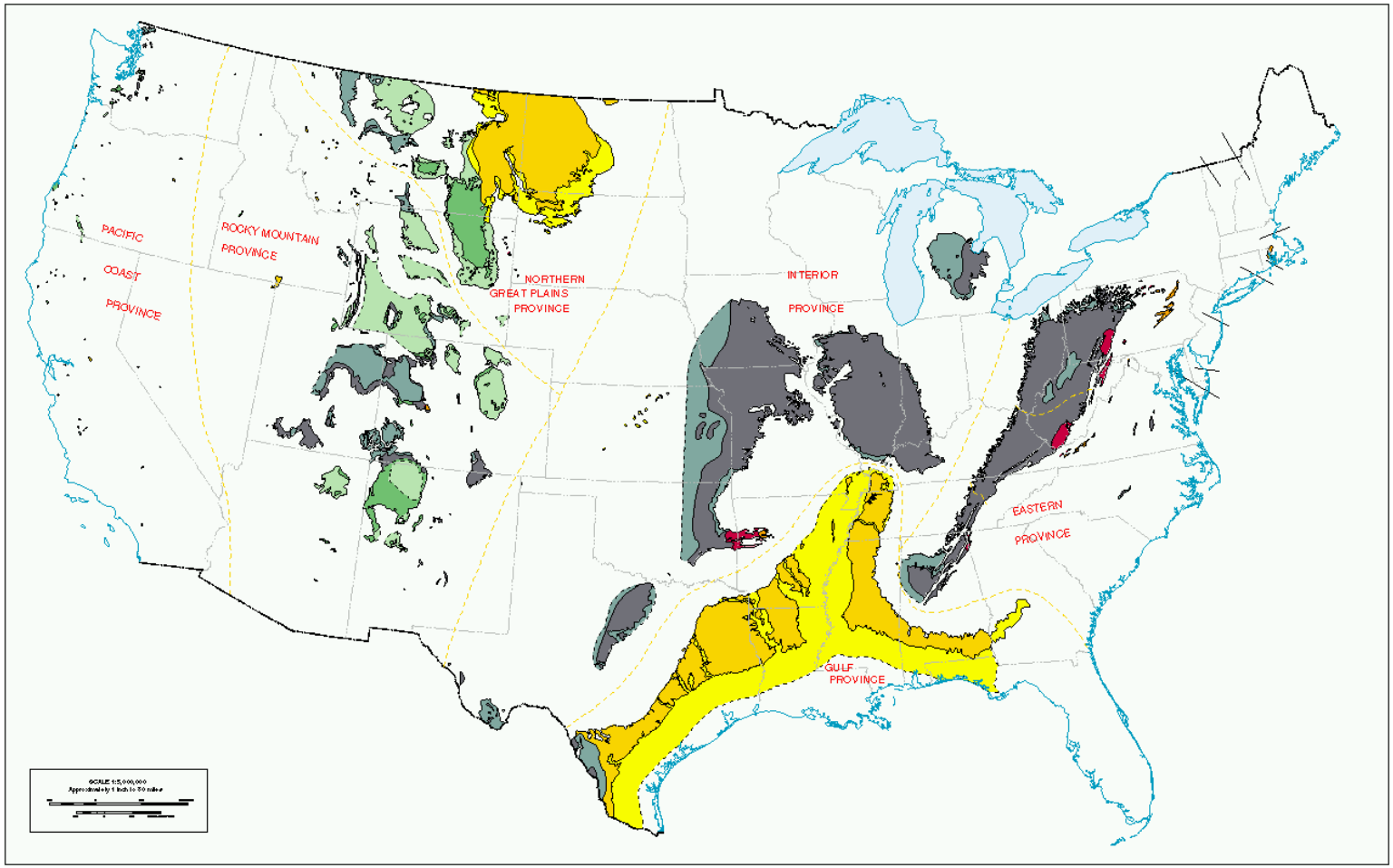
E-Gas Applications for Sub-Bituminous Coal

Ron Herbanek, Mechanical Engineering Director, E-GAS
Thomas A. Lynch, Project Development Manager

ConocoPhillips

E-Gas Technology for Gasification

U.S. Coal Resource Regions (Lower 48)



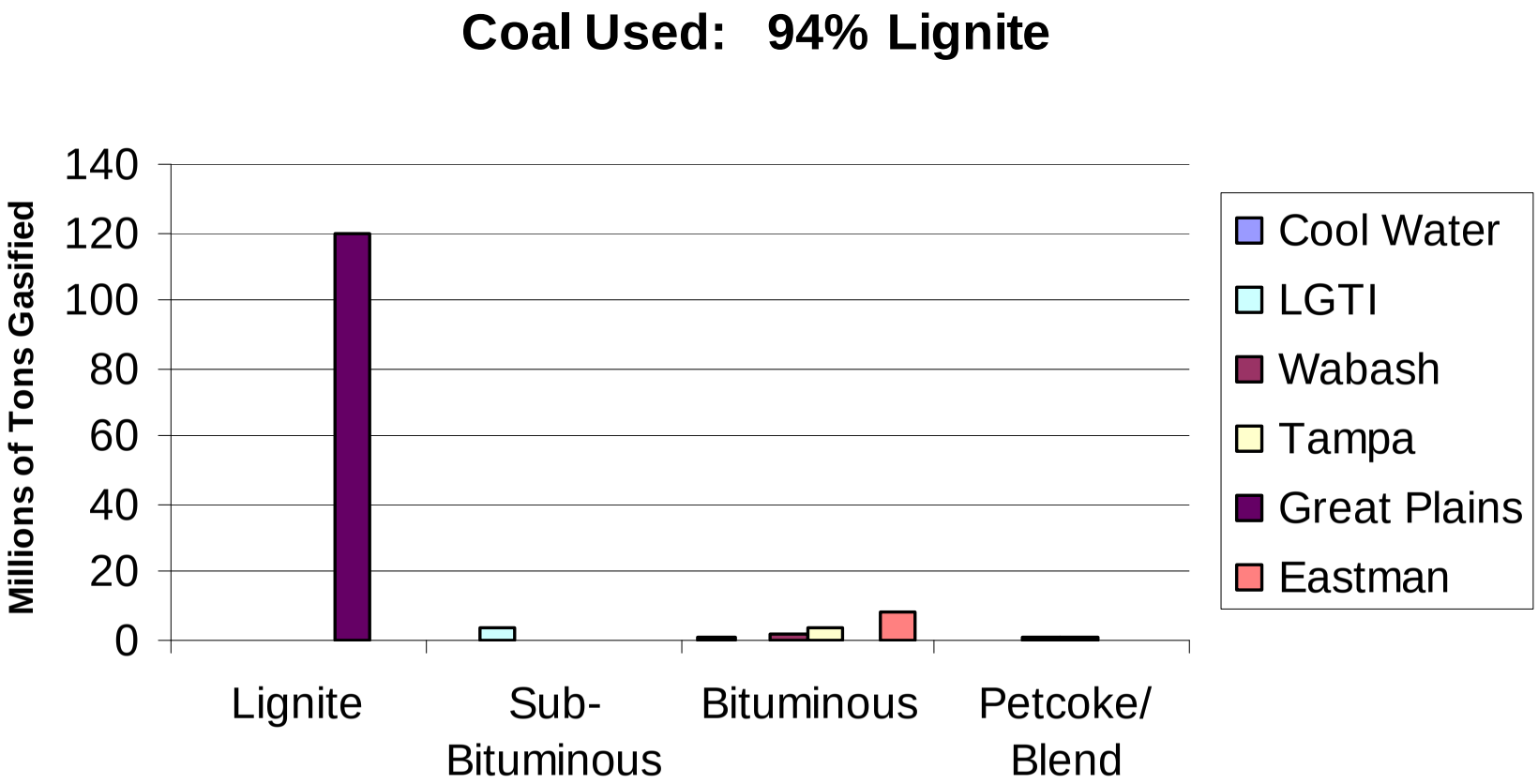
Source: Energy Information Administration

Coal - THE US Energy Resource

- Demonstrated Reserves
  - 508 B tons (275 B tons recoverable)
  - 185 B tons (36%) Sub Bituminous
- Current Annual Production (2004)
  - 1.1 B tons
  - 0.37 B tons (34%) Sub Bituminous
- Electric Utility Consumption (2004)
  - 1.0 B tons (>90%)

Source: Energy Information Administration & National Mining Association

Modern Era Coal Gasification – Power & Industrial



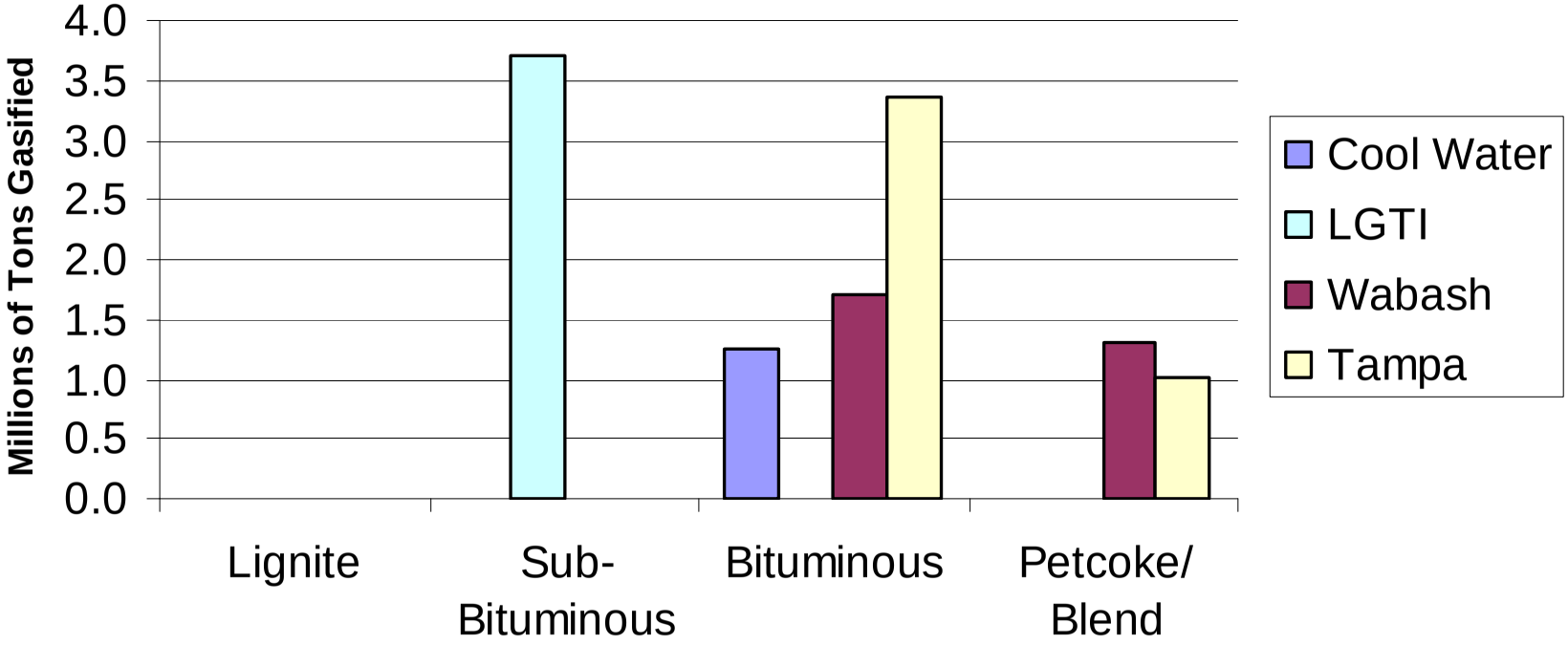
Source: DOE NETL

E-Gas Technology for Gasification

ConocoPhillips

U.S. Coal-to-Power Gasification

Coal Used: 37 % Sub-Bituminous - 63% Bituminous



Source: DOE NETL

E-Gas Technology for Gasification

ConocoPhillips

LGTI – Louisiana Gasification Technology, Inc

One Third of the Coal-to-Power Gasification in U.S.

- 2400 tpd Sub Bituminous coal feed
- Operated 1987 – 1995
- Processed 3.7 MM tons
- Fueled (2) S-W SGT6-3000E GTGs (a.k.a W501D5)
- 75% Availability (1994-1995)

E-Gas Technology for Gasification

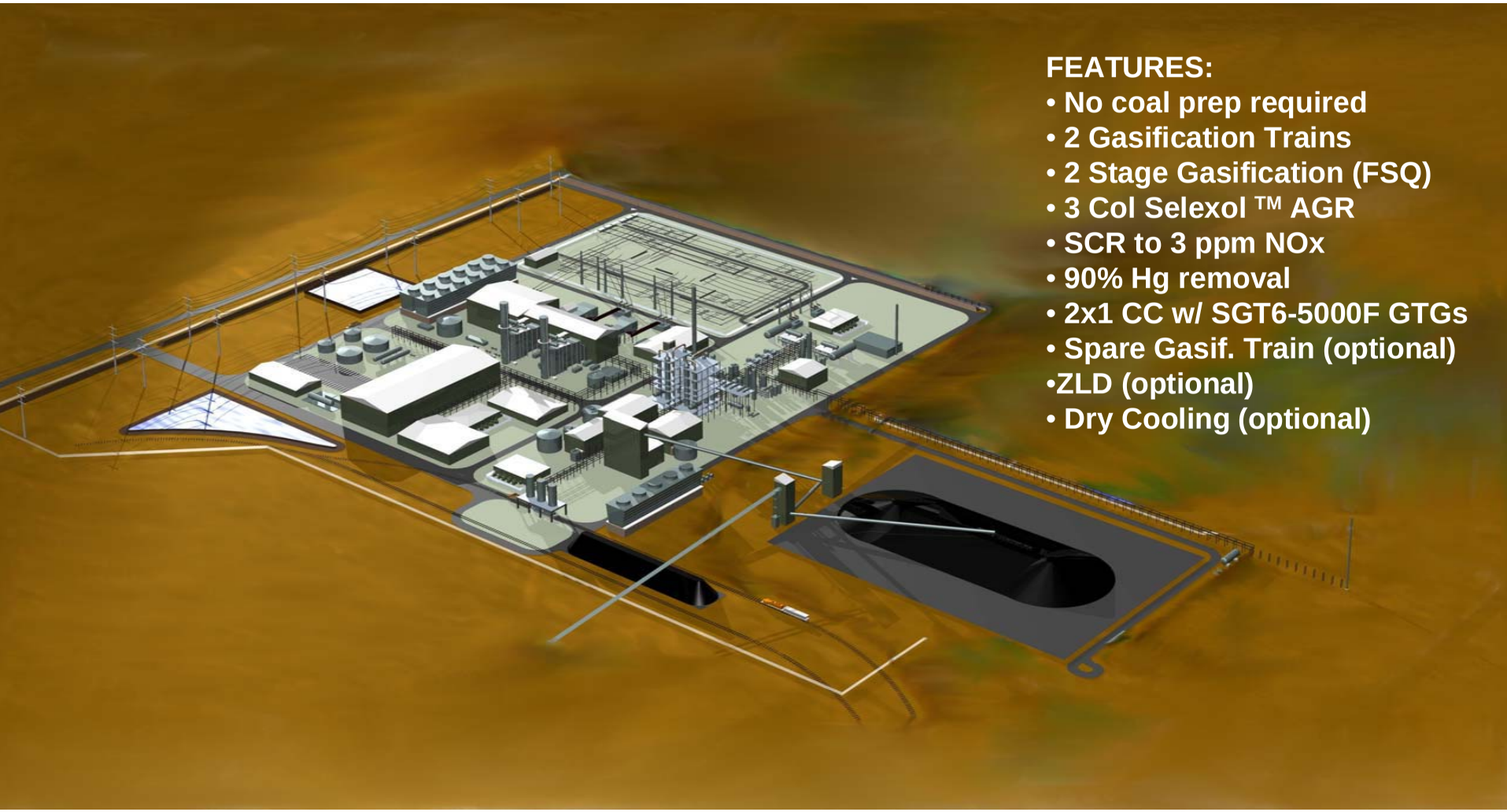
ConocoPhillips

Feed Design Considerations

Attribute	Impact	Mitigation [1]
Moisture	High moisture = lean Slurry (50-55%)	Slurry heating plus FSQ in 2 stage gasifier improves HR
Sulfur	Low Sulfur = lean acid gas	Selexol ™ provides high CO_2 selectivity
Ash	Slag quantity	N/A – high ash degrades HR, low ash requires flux addition
Slurry-ability	Moisture limits slurry concentration	N/A - (ALS and feed drying are not economical)
T_{250}	Determines 1st stage operating temperature	N/A – High value requires flux addition
Fixed Carbon	Determines feed rate & RXR sizing	N/A - "spike" with petcoke
Oxygen	Determines ASU size	N/A – (high O2 reduces ASU size)

Notes:
[1] N/A indicates mitigation not applicable to /required for Sub Bituminous feed

600 MW Sub Bituminous IGCC Design Template



FEATURES:

- No coal prep required
- 2 Gasification Trains
- 2 Stage Gasification (FSQ)
- 3 Col Selexol ™ AGR
- SCR to 3 ppm NOx
- 90% Hg removal
- 2x1 CC w/ SGT6-5000F GTGs
- Spare Gasif. Train (optional)
- ZLD (optional)
- Dry Cooling (optional)

E-Gas Technology for Gasification

3D Rendering Provided by Fluor/Siemens/ConocoPhillips

ConocoPhillips

600 MW Sub Bituminous IGCC Case Description

	Midwest	Mine Mouth
Site Conditions	500 ft, 50 F avg. amb.	5,000 ft, 45 F avg. amb.
Q Coal (AR, HHV), Btu/lb	8,340	
Composition:		
Carbon (dry basis), wt%	69.07	
Sulfur (dry basis), wt%	0.53	
Ash (AR), wt%	5.32	
Moisture (AR), wt%	30.24	
Acid Gas Removal	3 Col. Selexol ™	
Steam Conditions psig/F	1800/1050/1050	
Heat Rejection	Cooling Tower	Air Cooled
GTG Emissions Control	15 ppm NOx (diluent) plus SCR	
Process Wastewater	SW recycle via R.O.	SW recycle + ZLD

E-Gas Technology for Gasification

ConocoPhillips

600 MW Sub Bituminous IGCC Estimated Plant Performance

	Midwest	Mine Mouth
Feed Rate, tpd (AR)	8,341	7,259
Oxygen, tpd (95% vol)	4,732	4,132
Gross Power, MW	778.1	671.4
Aux. Power, MW	133.8	115.9
Net Power, MW	644.3	555.9
Net H.R., Btu/kWh (HHV)	8,996	9,075
Emissions [1]:		
NO_x, lb/MMBtu	0.02	
SO_2, lb/MMBtu	0.01	

Notes:
[1] Target permit levels

E-Gas Technology for Gasification

ConocoPhillips

600 MW Sub Bituminous IGCC Plant – Indicative Economics

ECONOMIC PARAMETERS

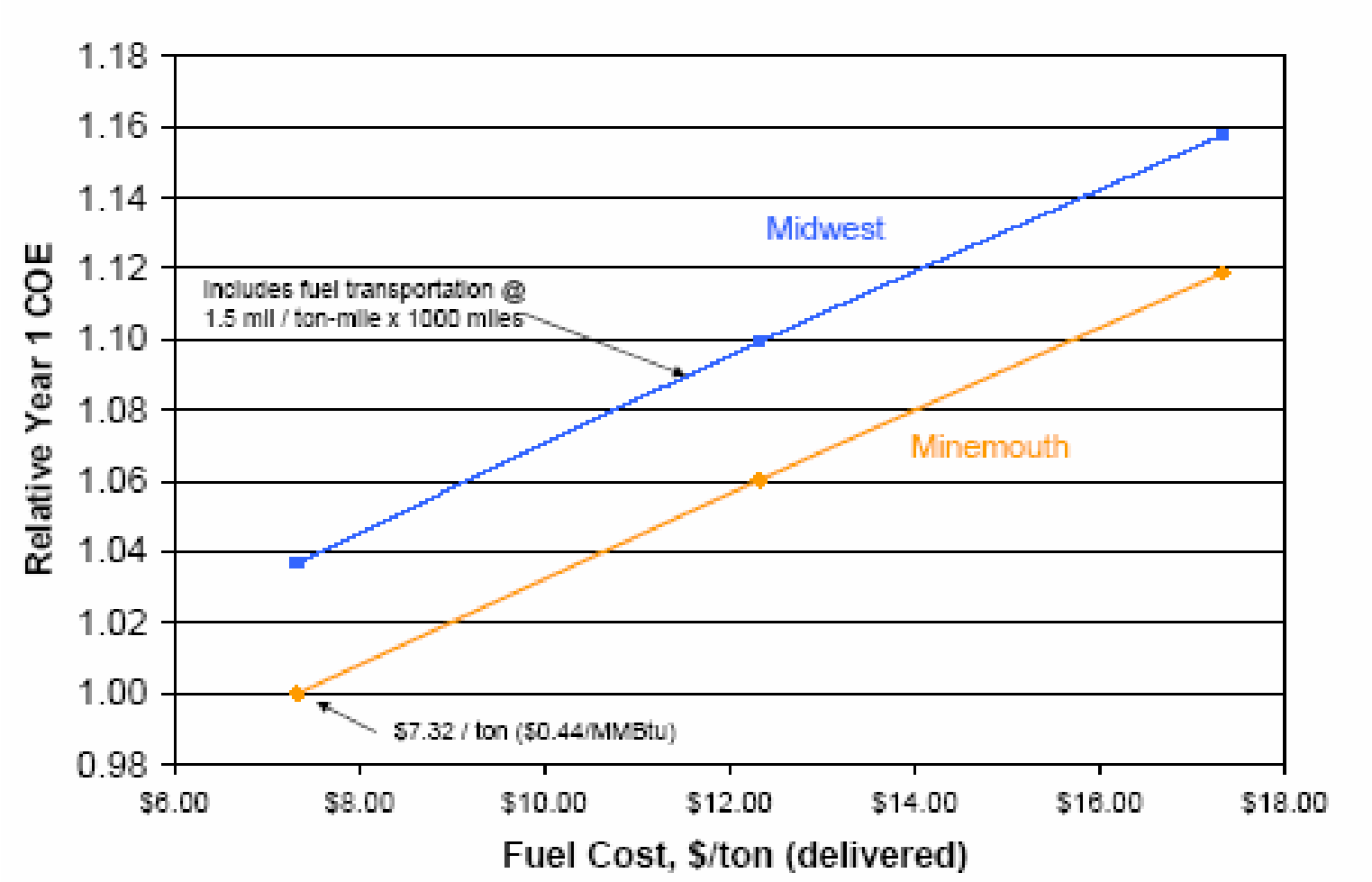
	Midwest	Mine Mouth
EPC Cost, $MM [1]	935 - 1131	868 - 1051
Owner's Costs, $MM [2]	70	150
Ann. O&M, $MM [3]	37	39
Availability, % [4]	80 - 85	80 - 85

Notes:
[1] ISBL, Overnight cost, $3Q05 (incl. 20% P&C)
[2] OSBL costs (transmission), Permitting, FEED, License, Land, etc.
[3] O&M (non-fuel) calculated at ~4% of EPC
[4] Two gasification trains, no spare

E-Gas Technology for Gasification

ConocoPhillips

COE vs. Fuel Cost ($2010)



E-Gas Technology for Gasification

ConocoPhillips

Sub Bituminous Feed Gasification

Feed Flexible
With multi-feed experience

Superior Environmental Performance
NOx
SO2
Hg

Cost Effective
Layout and design

High Efficiency
2 Stage Gasification with Heat Recovery

Water Friendly
Low Consumption
And
Wastewater generation

Available late 2008

E-Gas Technology for Gasification

ConocoPhillips

The IGCC Process: From Coal To Clean Electric Power

Outlook on Integrated Gasification Combined Cycle (IGCC) Technology

Testimony of Edward Lowe
Gas Turbine-Combined Cycle Product Line Manager
General Electric Power Systems
1 River Road
Schenectady, New York

Submitted to the

Subcommittee on
Clean Air, Wetlands and Climate Change
Hearing on Compliance Options for Electric Power Generators
January 29, 2002

Good morning, Mr. Chairman and members of the Subcommittee. My name is Ed Lowe. I am the Gas Turbine-Combined Cycle Product Line Manager for GE Power Systems. I appreciate the opportunity to testify this morning.

I am pleased to be here today to share with you our views about the benefits that Integrated Gasification Combined Cycle (IGCC) technology can deliver. IGCC can cost effectively produce power from solid fuels, such as coal, with substantial environmental benefits over other coal power generation technologies. If IGCC is adopted as the preferred coal based power generation technology, it will help the country and our customers meet the environmental goals of reducing NOx, mercury and other air pollutants, while also advancing sound energy policy goals of retaining a secure and diverse mix of fuels for electric power generation and improving the efficiency of coal based power generation.

OVERVIEW OF IGCC TECHNOLOGY

IGCC is a process that converts low value fuels such as coal, petroleum coke, orimulsion, biomass, and municipal wastes into a high value, low Btu, environmentally friendly natural gas-type fuel, also called “synthesis gas” or simply “syngas”. When used to fuel a combined gas turbine and steam turbine plant, known as a combined cycle system, coal based syngas fuel produces electricity more efficiently and with lower emissions than traditional direct fire coal boilers.

Coal gasification is not new, although there have been many technological improvements over its development cycle. The first mention of using coal gasification in the United States to produce "Town Gas" was by the Baltimore Gas Company in 1842, and by the 1910s,

commercial coal gasification was commonly used in the United States and Europe to provide cities with gas for streetlights and domestic consumption.

However, the combination of gasification with gas turbine power plants – the IGCC concept - had to wait until gas turbine combustion technology had advanced to the point that it was ready to accept the significant technical challenge of combusting low Btu IGCC fuels. Gas turbines for IGCC are markedly different from the vast majority of gas turbines that are fueled by natural gas. IGCC gas turbines must be specifically engineered to achieve highly efficient and reliable service on syngas. These design enhancements relate primarily to the combustion and fuel systems, but also encompass special safety, packaging, and controls modifications.

IGCC's roots trace back to GE's Global Research Center in Schenectady, NY. In the early 1970's pilot testing demonstrated poor fuels could be converted to clean syngas, and that it was possible to integrate a gas turbine and a chemical gasification plant. Further work, at GE's Schenectady laboratories, continued in the early 1980s on gas cleanup and with full-scale combustion development. This work led to the first large commercial coal IGCC Plant, the 120 MW Cool Water Plant located in California. This was a partnership funded project with EPRI and other participants that utilized GE's innovative gas turbine combustion technology. Commissioned in 1984, Cool Water demonstrated the technical feasibility of IGCC.

In the 1990's commercial IGCC plants were successfully built and operated with steady improvements in reliability, efficiency and cost. Two examples of current coal IGCC plants are Tampa Electric Company's Polk 250 MW IGCC plant in Florida, commissioned in 1996, and the Public Service of Indiana's (now Cinergy) Wabash River 250 MW IGCC plant in Indiana, commissioned in 1995. These two plants, utilizing GE gas turbines, have successfully logged over 50,000 operating hours on coal synthesis gas.

Since GE pioneered IGCC nearly three decades ago, we have developed a broad IGCC product line of gas turbines with matching steam turbines spanning the 100 to 400 MW module range. GE has sold over 23 IGCC gas turbines and attained over 400,000 gas turbine operating hours on syngas. GE is committed to developing new and improving existing IGCC gas turbine designs. New York continues to serve as the central hub of our efforts to advance this technology. The development of concepts for further improvement in emissions is continuing at our Global Research Center in Schenectady, and we recently strengthened our commitment to advance IGCC technology with the commissioning of a new combustion development facility in Greenville, South Carolina.

ENVIRONMENTAL ADVANTAGES OF IGCC

IGCC is inherently less polluting and more efficient than any other coal power generation technology. In IGCC, harmful pollutants are removed from the syngas before they reach the gas turbine; therefore, end-of-pipe/stack cleanup is not necessary. IGCC efficiently removes ash, sulfur compounds, ammonia, mercury, other metals, and any particulate matter to reduce air pollution. Emissions of SO_x, NO_x, mercury, heavy metals, and particulate from an IGCC plant are fractions of the emissions from conventional, coal power plants.

For example:

- IGCC NO_x emissions are approximately half those of modern pulverized coal steam-boiler plants. About 0.07 lb/million Btu NO_x emissions can be achieved through IGCC. This is approximately a 60% reduction in NO_x emissions from the average coal plants operating today. Since 1980, the can-annular combustors employed by GE have been continuously improved to handle a wide variety of fuels and to reduce NO_x emissions. Beginning with the Cool Water Coal IGCC test program, NO_x emission performance was demonstrated at less than 0.125 lb/million Btu using "E" class gas turbine technology. The recent TECO Polk and PSI Wabash plants, have achieved similar NO_x values (less than 0.1 lb/million Btu), using higher efficiency "F" class technology. Similarly, full pressure and temperature laboratory test programs using various process diluents, including N_2, H_2O, and CO_2, lead us to believe that the challenging target of single digit NO_x emissions (0.04 lb/million Btu) may be possible. GE is evaluating whether to implement a development program with the goal of achieving this challenging target, and the support of EPA, or legislative changes, would encourage our initiation of such a program.

- 95% mercury removal is being achieved by a gasification plant in Kingsport, Tennessee. Similar mercury removal systems can be used to economically and reliably remove mercury for new IGCC plants.

- Sulfur can be recovered from the syngas either as elemental sulfur or sulfuric acid in pre-combustion cleanup. Both elemental sulfur and sulfuric acid are marketable industrial by-products depending on local economics. With little sulfur remaining in the syngas stream that enters the gas turbine, the emissions of SO_x for an IGCC plant are less than half of those of even state-of-the-art direct combustion coal boiler plants.

GE's emphasis on improving turbine and combined cycle efficiencies has directly benefited IGCC emissions performance. High IGCC efficiencies yield CO_2 greenhouse gas emissions that are 12% lower than those of state-of-the-art coal steam-boiler plants. These emissions are approximately 30% lower than those of average coal plants

operating today, for comparison purposes. Additionally, in the gasification process carbon can be removed from the syngas to create a hydrogen-rich fuel that can further reduce CO_2 greenhouse gas emissions. In our combustion development programs, GE has successfully demonstrated combustion of 90% hydrogen syngas fuel to demonstrate the technical feasibility of power plants with ultra low CO_2 emissions.

Let me emphasize this key point: In the IGCC process harmful pollutants are removed from the syngas stream before combustion, rather than in post combustion flue gas treatment. The pressurized syngas stream represents less than 1/100 of the volume of flue gas from direct coal combustion and the contaminants in syngas are concentrated. Therefore, IGCC pre-combustion clean-up is far more effective and much lower cost than the post-combustion clean-up employed in direct combustion coal steam-boiler plants.

And there is another important environmental benefit: In IGCC coal ash is converted in the gasifier into a solid, vitreous slag which is chemically inert. This non-leaching slag can be employed in the construction industry as road fill or as strengthening aggregate for building concrete. IGCC does not require secure landfill sites for ash storage and ash-landfill pollutant leaching into the groundwater is not an issue.

ENVIRONMENTAL REGULATION SHOULD NOT BE A BARRIER TO IGCC DEPLOYMENT

In spite of these significant environmental benefits, we are concerned that permitting bodies may burden IGCC with duplicative and reliability reducing end-of-pipe controls for NO_x, such as SCR (selective catalytic reduction). These systems cannot work as reliably on IGCC as they do on natural gas fired units. The pollution prevention combustion technology on GE's IGCC gas turbines delivers NO_x emissions below that of alternative coal technologies and we strongly believe that IGCC must be evaluated as a coal technology with consideration given for its total environmental benefits when setting emission targets.

OPTIMAL USES OF IGCC

Gasification is a steady state chemical process and therefore IGCC plants perform best in base-load applications. IGCC gas turbines require natural gas or distillate as a start-up fuel; so that all IGCC gas turbines must be dual fuel capable. As a consequence, IGCC plants can switch to the backup fuel when syngas is unavailable or co-fire when syngas is limited. With the availability of backup fuels and combustion

design flexibility, IGCC plant power availability can approach that of natural gas combined cycle plants.

IGCC must be optimized based on the design requirements, which is primarily defined by the fuel characteristics — there is no universal IGCC design that will satisfactorily meet all expectations. A myriad of technical possibilities must be balanced for each gasifier type and each syngas fuel to optimize IGCC systems for specific fuel type and site conditions. Through cycle optimization studies and by incorporating lessons learned from successful operation of many IGCC units, GE has optimized system configurations for all major gasifier types and most GE heavy-duty industrial gas turbine models.

GE is conducting continuous improvement programs, which endeavor to further enhance the overall performance level of IGCC plant designs. Working with various process technology suppliers, GE is helping to facilitate, define and develop lower cost and higher efficiency IGCC plant designs.

FAVORABLE ECONOMICS

The cost to build large IGCC plants has steadily decreased over the last 25 years; the installed turnkey Engineer Procurement Construct (EPC) price is now projected to be $1200 per kW. This makes the superior IGCC technology cost competitive with other modern coal power plant options such as Circulating Fluid Bed, or super critical and ultra-super critical pulverized coal boiler plants with state of the art emission control systems.

Continuous gas turbine technology improvements raise the prospect for further economic improvements as output power and plant efficiencies increase. As additional IGCC plants go operational, improvements in system performance and plant design cost can be expected from a growing and maturing technology experience base.

CONCLUSION

IGCC clearly becomes the superior coal technology option when its higher plant efficiency – 5 percentage points above other coal technologies – and significant environmental advantages are considered.

Coal IGCC offers superior environmental performance while projected to produce electricity at prices competitive with modern direct-fired coal power plants. IGCC also provides an inherent capability to cost effectively meet future environmental needs because contaminates are removed in a low volume, high

concentration, pre-combustion fuel gas steam. We look forward to exploring options with you and with regulatory agencies to ensure that appropriate laws and policies are in place to allow IGCC's environmental and efficiency benefits to be achieved.

U.S. EPA's Clean Air Gasification Activities

Presentation at the
Gasification Technologies Council
Winter Meeting
January 26, 2006
Tucson, Arizona

Robert J. Wayland, Ph.D.
U.S. Environmental Protection Agency
Office of Air and Radiation
Office of Air Quality Planning and Standards
Research Triangle Park, N.C.

Environmental Technology Initiative

- Key leaders at the Agency understand that innovative technology has been and will continue to be a key element in meeting our environmental needs in a economical, cost-effective manner
- EPA Senior management challenged Agency staff to figure out ways to facilitate and incentivize the development and deployment of such technologies
  - Created the Environmental Technology Council (ETC)
- Solicited topics for consideration Agency-wide
  - 47 possible technologies and environmental problems in need of technology solutions were identified
  - 14 projects were selected as “priorities” for the Agency, based on Agency-wide voting across all EPA offices and Regions.

Two Gasification Projects Selected

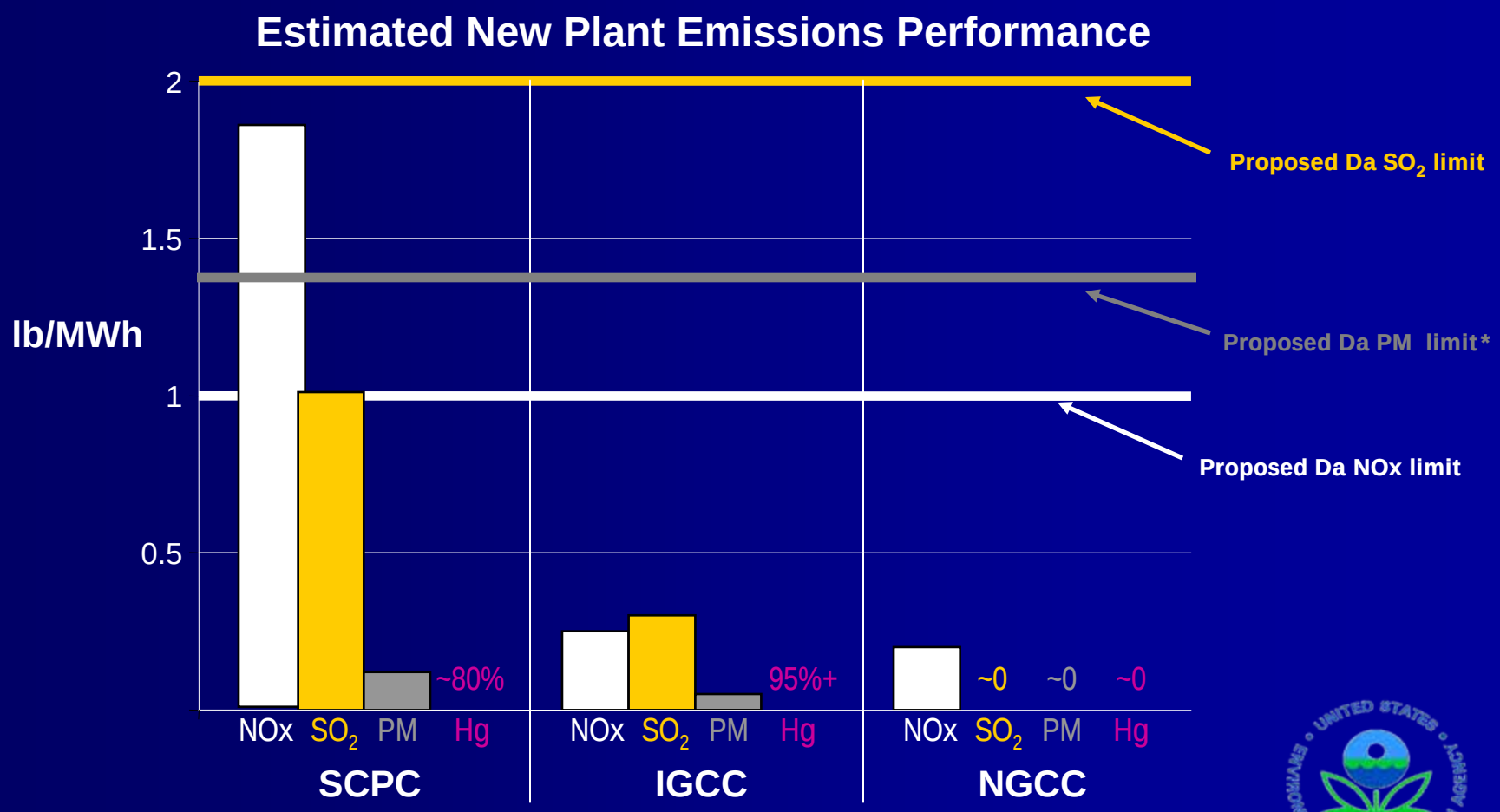
- Integrated Gasification Combined Cycle (IGCC)
  - Generating electricity from the gasification of coal and other fossil fuel byproducts
  - Office of Air and Radiation is lead office on development and deployment of IGCC technology
- Waste-to-Energy
  - Utilization of biomass, petroleum residuals, petroleum coke, secondary materials
  - Office of Research and Development in conjunction with the Office of Solid Waste are the leads on the waste-to-energy effort
  - OAR, ORD and OSWER are working together as a cross-Agency team to promote these technologies for deployment

Coal – The Future of Electricity Generation

- The world needs to make electricity from coal in an environmentally and economically sustainable way
  - IGCC has fundamental advantages from both environmental and efficiency perspectives relative to conventional coal-fired power generation technologies
    - Inherently lower emissions of NO_X, SO_2 and Hg
    - Requires less fresh water – special issue in the drier, water-limited Western regions of the U.S.
    - Considerably more commercially useful byproducts (and thus, less waste materials)
  - High potential for reducing Greenhouse Gas (GHG) emissions by allowing for carbon capture and sequestration at costs significantly below conventional PC generation costs

CINERGY's Wabash River Facility

Gasification Offers Clean Alternative



* - Taking comment on the adoption of PM-CEMS; other alternative is 0.015 lb/MMBtu limit

EPA's Role in Deployment

- The Environmental Technology Initiative's purpose is to
  - Achieve improved, real-world environmental results through the design, development and deployment of innovative technologies
    - **Identify** short- and long-term priority environmental problems with attainable technological solutions
    - **Coordinate** efforts within EPA and other Federal agencies to identify and implement such technological advancements and solution
    - **Create** partnerships with other Federal agencies, State governments, Tribal governments, non-profit groups and industry to incentivize technology enhancements and deployment
      - Creation of joint EPA/DOE team to promote deployment of IGCC

EPA/DOE Team Activities

- Objective is to facilitate a critical number of commercial plants to address both environmental and operational concerns
- EPA Air Permitting Initiatives
  - Identification and quick resolution of novel air permitting issues
  - Help in expediting the air permit process
- DOE/EPA developing a model to assess the economic viability of IGCC plants under different conditions
- EPA/DOE conducting a technical study to establish the environmental footprint of the IGCC technology relative to conventional PC plants

EPA Actions to Date – Progress Report

- Regulatory Issues
  - Current issue for IGCC facilities is New Source Review (NSR) and Prevention of Significant Deterioration (PSD) permitting
    - December 13, 2005 – Steve Page memo (IGCC and BACT)
      - EPA's interpretation of when IGCC should be considered in NSR and PSD permitting
      - In the case of pulverized coal boilers and similar conventional coal-fired technologies, IGCC should not be considered as control technology candidate under BACT
  - Selective Catalytic Reduction (SCR) as BACT for IGCC units
- Headquarters and Regional offices want to work with companies interested in developing IGCC technology in the near future
- EPA is committed to working with State permitting authorities
  - States are the primary permitting authority under NSR/PSD – often can be more stringent than Federal regulations
  - Agency is attempting to be "upfront" and let States know "where we stand" on IGCC permitting issues
    - Anticipate this may help expedite and streamline the NSR & PSD permitting process considerably

Potential Regulatory Hurdles

- **Should Selective Catalytic Reduction (SCR) be required as best achievable control technology (BACT) for IGCC?**
  - **TECO's Polk Power Station, Tampa, Florida**
    - Florida DEP ultimately decided in conjunction with Region IV that SCR was **not** required as BACT – a position supported by Headquarters
  - **BACT is a case-by-case determination**
    - "One Size Doesn't Fit All"
      - Circumstances at a new plant may not be the same as what drove our decision at Polk Power Station
    - Regardless, SCR as BACT is a decision that merits our attention and resolution sooner as opposed to later

SCR Technical Issues

- Currently reviewing request from U.S. power company for guidance on SCR as BACT
- Issues under review:
  - SCR not demonstrated on plants utilizing coal-derived syngas
    - Lack of U.S. experience
    - One plant operational in Japan
  - SCR feasibility, high cost and risk issues vary between IGCC plants, PC plants and NGCC facilities
  - Ability to obtain meaningful performance guarantees for SCR and/or HRSG systems
  - SO_2 BACT analysis and its impact on SCR costs and feasibility
    - MDEA, Rectisol or Selexol

Potential Regulatory Incentives

- **Final New Source Performance Standards (NSPS) for Subpart Da**
  - IGCC Units constructed on/after February 9, 2005 would be subject to the same emission limits as a coal-fired boiler
    - Given current IGCC technology, this should not pose any regulatory burden on new, planned IGCC facilities
  - Duct burners moved into KKKK
- **Final Clean Air Mercury Rule (CAMR)**
  - Created separate source category for IGCC units
    - Hg emission limit of 20 x 10^{-6} lb/MWh
    - Comparable to a bituminous PC-fired power generation system

Future Plans and Needs

- To incentivize the commercial deployment of IGCC technology EPA needs to better understand the environmental footprint of these facilities relative to conventional power generation technologies
  - EPA/DOE Environmental Footprint Study
- EPA is working on models to assess the economic viability of IGCC plants under different conditions
  - Working closely with DOE on these economic and environmental efforts
- One remaining barrier is the cost of IGCC technology
  - EPA is working in conjunction with DOE to evaluate various proposals to address this economic barrier
  - Energy Policy Act of 2005
    - Exploring options and incentives

Draft Results of EPA's IGCC vs. PC Study

- Nexant, Incorporated contracted to perform study in conjunction with EPA and DOE input
  - Solicited comment/input on draft report from numerous stakeholder groups
- Final results targeted for discussion at the GTC meeting in Tampa, Florida in March 2006
  - Public release March 2006
- Aspects of the Study
  - Thermal performance of IGCC and PC units
  - Estimated air emissions
  - Water use requirements and solid waste output
  - CO_2 capture and sequestration potential

Barriers to IGCC Deployment

- Concerns of higher costs
  - Nominally considered to be approximately 20%
- Concerns of novel environmental permit issues delaying construction and increasing costs
  - NSR and PSD issues
  - BACT analyses
- Concerns of plant reliability
  - Need for dual-train gasifier
  - HRSG fouling downstream of the SCR
  - Power block reliability
- Cultural resistance to a facility with a large chemical plant component
  - Chemical Engineers vs. Mechanical Engineers

Technical Study Scope

- IGCC and PC plant comparisons provided, using bituminous/subbituminous coals and lignite
- Plant size: 500 MW
- Plant configurations:
  - Oxygen-blown IGCC, 1,800 psig / 1,000° F / 1,000° F
  - Subcritical PC, 2,400 psig / 1,000° F / 1,000° F
  - Supercritical PC, 3,500 psig / 1,000° F / 1,000° F
  - Ultra-supercritical PC, 4,500 psig / 1,100° F / 1,100° F (double reheat)

Technical Study Scope, (Cont'd)

- IGCC plant environmental controls:
  - NOx: Diluents (SCR evaluated)
  - SO_2: MDEA (Selexol evaluated)
  - Particulate: scrubber
  - Mercury: carbon bed
- PC plant environmental controls:
  - NOx: SCR
  - SO_2: wet FGD for bituminous coal (BC) and lignite (LIG) and spray dryer for subbituminous coal (SBC)
  - Particulate: ESP for BC and LIG, Baghouse for SBC
  - Mercury: sorbent injection (activated carbon) for SBC

Thermal Performance Bituminous Coal*

Plant Type	IGCC	PC Sub-Critical	PC Super-Critical	PC Ultra Super-Critical
Net output, MW	500	500	500	500
Thermal Efficiency, % HHV	41.8	35.9	38.3	42.7
Heat Rate, Btu/kWh	8,167	9,500	8,900	8,000

* Preliminary/draft results.

Thermal Performance Subbituminous Coal*

Plant Type	IGCC	PC Sub-Critical	PC Super-Critical	PC Ultra Super-Critical
Net output, MW	500	500	500	500
Thermal Efficiency, % HHV	40.0	34.8	37.9	42.1
Heat Rate, Btu/kWh	8,520	9,800	9,000	8,100

* Preliminary/draft results.

Thermal Performance Lignite*

Plant Type	IGCC	PC Sub-Critical	PC Super-Critical	PC Ultra Super-Critical
Net output, MW	500	500	500	500
Thermal Efficiency, % HHV	38.4	33.1	35.9	37.9
Heat Rate, Btu/kWh	8,897	10,300	9,500	9,000

* Preliminary/draft results.

Air Emission Comparisons*

Pollutant	IGCC Project	PC Project	PC Project
NOx	0.07*	0.07	0.06
SO_2	0.03	0.09	0.09
PM/PM_{10}	0.011	0.013/0.012	0.018
VOC	0.004	0.0027	0.0036
CO	0.03	0.15	0.15
Hg	0.5	0.66	1.7
Coal Type	Bituminous	Bituminous/Sub-bituminous	Sub-bituminous

* Preliminary/draft results. All emissions in lb/MMBtu, except for Hg, which is in lb/TBtu. NOx for IGCC is based on 15 ppmvd at 15% O_2.

Water Use and Solid Waste Comparisons**

Parameter	IGCC Plant*	PC Plant*
Cooling water, gpm	99,500	167,300
Makeup water, gpm	141	325
Solid waste, tpd	224	616

* Each plant is approximately 290 MW in size.

** Preliminary/draft results.

CO_2 Capture and Sequestration Potential*

Parameter	IGCC Plant	PC Plant
CO_2 capture, %	91	90
Plant output derating, %	14	29
Heat rate increase, %	16.5	40
Capital cost increase, %	47	73
COE increase, %	38	66

* Preliminary/draft results.

Main Study Areas Still Under Development

- Capabilities of air pollution control technologies used in IGCC and PC plants
  - Efficiency of sulfur removal processes (IGCC)
  - Feasibility of SCR (IGCC)
  - Evolution of Hg removal technologies (PC)
  - Carbon capture efficiency (IGCC and PC)
- Comparison of mercury emission control capabilities between IGCC and PC plants
  - Sorbent injection (PC)
  - Activated carbon beds (IGCC)
- Comparison of water consumption and waste water and solid waste generation rates between IGCC and PC plants
- Potential of CO_2 capture within PC plants

Cost Comparisons Bituminous Coal Applications

Costs	IGCC	Subcritical PC	Supercritical PC	Ultra Supercritical PC
Total Capital Requirement, $/kW	1,670	1,347	1,431	1,529
Operating Cost, $1,000s	27,310	27,700	29,000	30,400

1. All costs are in 2004 dollars.
2. Costs are based on published data. The actual costs may be different due to site specific factors. IGCC costs do not account for possible increases for items such as performance guarantees, warranties and availability.
3. Operating costs include fixed and variable O&M costs.

Cost Comparisons Subbituminous Coal Applications

Costs	IGCC	Subcritical PC	Supercritical PC	Ultra Supercritical PC
Total Capital Requirement, $/kW	1,910	1,387	1,473	1,575
Operating Cost, $1,000s	29,700	28,300	29,600	31,100

1. All costs are in 2004 dollars.
2. Costs are based on published data. The actual costs may be different due to site specific factors. IGCC costs do not account for possible increases for items such as performance guarantees, warranties and availability.
3. Operating costs include fixed and variable O&M costs.

Cost Comparisons Lignite Applications

Costs	IGCC	Subcritical PC	Supercritical PC	Ultra Supercritical PC
Total Capital Requirement, $/kW	2,350	1,424	1,511	1,617
Operating Cost, $1,000s	34,000	29,640	30,940	32,440

1. All costs are in 2004 dollars.
2. Costs are based on published data. The actual costs may be different due to site specific factors. IGCC costs do not account for possible increases for items such as performance guarantees, warranties and availability.
3. Operating costs include fixed and variable O&M costs.

Conclusions

- EPA has undertaken several initiatives to facilitate and incentivize IGCC technology
  - Environmental Study (release: March 2006)
  - December 13, 2005 Steve Page Memo – IGCC and BACT
  - Future guidance on SCR as BACT for IGCC facilities
- Preliminary IGCC vs. PC Study Results:
  - IGCC thermal performance significantly better than current PC technologies
  - Overall better environmental performance for IGCC
  - IGCC has potential advantage in capturing and sequestrating CO_2 at lower costs
- EPA is not trying to pick a technology winner, but trying to ensure that IGCC has a chance to prove itself commercially

For more information contact:

Dr. Robert J. Wayland
Office of Air Quality Planning & Standards
Research Triangle Park, NC
(919) 541-1045
Wayland.robertj@epa.gov

Testimony of James E. Rogers
Chairman, CEO and President
Cinergy, Corp.
House Science Committee
Business Actions Reducing Greenhouse Gas Emissions
Wednesday, June 8, 2005

Good Morning. My name is Jim Rogers, I am Chairman, CEO and President of Cinergy. Cinergy was formed ten years ago by the combination of PSI Energy in Indiana and Cincinnati Gas & Electric. As you know, Cinergy also recently announced a merger with Duke Energy based in Charlotte, North Carolina.

Let me tell you a bit about our company before I explore our interest in greenhouse gas emission policies.

Cinergy serves approximately 1.5 million customers in Ohio, northern Kentucky and much of Indiana. We operate nine coal-fired generating stations that burn almost 30 million tons of coal per year.

As a Midwest utility Cinergy has ample access to coal. And with rising natural gas prices, coal is the most economical choice for supplying our customers with electricity. Despite our generating choice, in 2003 Cinergy committed to reducing our greenhouse gas emissions to five percent below 2000 levels during the period of 2010 and 2012. To reach that goal we are spending $21 million to fund projects through the remainder of the decade. We plan on reaching the goal despite a growing demand for electricity in our region, and taking into account the electricity penalty we will realize when the bulk of our generating units are outfitted with pollution control equipment to meet new Environmental Protection Agency regulatory requirements.

All in all we expect that we will need to cut greenhouse gas emissions by a total of 30 million tons.

While electric rates in the Midwest are likely to increase as a result of pollution control expenditures to reduce sulfur dioxide, nitrogen oxide and mercury, no increases will be due to our carbon commitment. We made our decision to reduce GHG emissions despite the fact that there currently is no commercially viable method of capturing and sequestering carbon from coal fired power plants. However there are new technologies on the horizon and research on carbon capture and sequestration applications will and must continue to keep coal a viable and necessary fuel for the future.

In fact, Cinergy is completing a feasibility study on the construction of an Integrated Gasification Combined Cycle power plant (IGCC) -- the state of the art coal plant technology available to us today. It is relatively easier and less energy intensive to capture CO2 from an IRC's high pressure synthesis gas than from conventional pulverized coal flue gas. In addition, sulfur dioxide, nitrogen oxide and mercury emissions are substantially reduced with IGCC technology and because it is more efficient even without carbon capture components, it does reduce carbon emissions.

According to industry analysts' estimates, the cost of IGCC is 10 – 20% more then traditional pulverized coal. Those costs will come down, however, if the appropriate incentives are made available and we are able to deploy five or more facilities over the next decade. It is also a technology that is a necessary component of any international technology transfer program. Developing countries that today operate plants without even the simplest of pollution control equipment can with technologies such as IGCC begin reducing all emissions more efficiently and completely.

Let me turn to the subject at hand. Why has Cinergy taken on this commitment and why expend so much attention on greenhouse gas emissions?

I spend a good deal of my time, not just in running the company – but also in researching and participating in domestic and international economic and environmental conferences so that I can appropriate the wisdom from those venues back to our shareholders and all of our stakeholders. Over the past several years I have developed a better understanding of climate change and I see the debate in the

scientific world honing in on a few basic facts: that the world is warming and that human activities have contributed to the warming. What the impacts will be I don't think we yet fully understand.

Because of this, I believe people increasingly will believe that greenhouse gas emissions should be reduced and that actions should begin today to prepare for that eventuality.

But what if I and the multitude of scientists and industries agreeing with that premise are wrong? If we approach this issue appropriately, then we will have worked to create new environmentally friendly technologies, pursued methods of saving energy far more efficiently and work to lower our dependence on foreign oil. We will have advanced to a multitude of fuel sources and technological configurations that will help move our economy into a cleaner and more self reliant future. And I don't know anyone that can argue effectively against that outcome.

Let me share with you some of what I call signposts that I have observed over the past several years which helped guide me to the development of our position today. We published these signposts in our 2004 annual report because we chose not to ignore the issue of greenhouse gases but to address it in a positive manner.

Signpost #1
The states are taking action

Four states have an overall cap on GHG emissions and two have a cap on power plant CO2 emissions. Eight states regulate GHG emissions. And, eight states have filed suits against Cinergy and four other utilities to curb their GHG emissions, while others are involved in suits with EPA over the need to regulate carbon.

A coalition of nine northeast states has initiated the regional greenhouse gas initiative which would create a regional market based cap and trade program.

Governor **Schwarzenegger** of California an executive order identifying a goal to reduce emissions including:

- By the year 2010, to reduce California's GHG emissions to less than those produced in 2000.
- By 2020, to reduce GHG emissions below 1990 levels.
- By 2050, reduce overall emissions a full 80 percent below 1990 levels.

He noted that the state is going to accelerate the timetable to get more energy from renewable sources 20 percent by 2010 and a third by 2020.

These sources include solar, wind, geothermal, and biomass from agriculture and other waste.

The state's fleet of government vehicles, all 70,000 of them, will be replaced with hybrids.

Signpost #2

An increasing number of Members of Congress are expressing concern about global warming.

While in 1997 the US Senate voted to reject the Kyoto Protocol, that did not mean they were rejecting the issue. I think that it is important to remember that the ratified 1992 Agreement of the Parties has an objective of stabilizing atmospheric greenhouse gas concentrations "at a level that prevents dangerous anthropogenic interference with the climate system" has never been refuted.

Senators McCain and Lieberman have introduced and modified their climate reduction proposal and those voting favorably have increased even though there are still not enough votes to pass the Senate.

Multi-Emissions legislation which I have championed for years sadly can't move past the Senate Environmental and Public Works committee because the issue of climate remains unresolved. That hurts the utility industry and its customers because also unresolved are rules that regulate sulfur dioxide, nitrogen oxide and mercury as well – all which will undoubtedly cost ratepayers millions in unneeded expenditures because the roadmap for an eventual solution to those issues will be tied up in courts for years.

Signpost #3
Kyoto has been approved by 38 Industrial nations this year.

Europe wants to accelerate GHG mitigation and some countries, including Tony Blair with whom I met yesterday, are interested in exploring what lies beyond Kyoto's 2012 expiration.

I think that it is also important to consider that while industry in Europe is mandated to deal with emissions reductions, that issue could become increasingly confrontational in trade discussions as the lack of a U.S. policy could possibly be considered a trade subsidy.

Signpost #4

A growing number of shareholders are asking companies to quantify the risks associated with GHG emissions.

Increasingly investor groups are asking utilities and other companies to quantify their GHG emission risks and to determine what steps are being taken to manage those risks.

The assets of socially responsible mutual funds are growing faster then the mutual fund industry as a whole.

And, the California Public Employee Retirement System (CalPERS) announced that it will sign onto the Global Carbon Disclosure Project, an international effort to improve the transparency of business risks associated with climate change.

Signpost #5

CO2 and GHG emissions trading markets are developing in Europe and the US.

The EU initiated its emissions trading scheme this year and facilitates the trading of CO2 allowances among 12,000 EU industrial installations.

The Chicago Climate Exchange established in 2003, has grown from 13 to 85 members.

Signpost #6
Global Warming is becoming part of our everyday consciousness

The issue served as cover stories for Business Week and National Geographic in 2004.

And this past Saturday's New York Times included a front page story discussing world-wide technology advances in energy efficiency. The story highlighted countries that have outperformed the U.S., from Japan with its newly manufactured kilowatt saving refrigerators and air conditioners to Germany with its impressive new fuel efficient homes and to Singapore which is placing new restrictions on autos to encourage increased bus and rail usage.

Increasingly, U.S. businesses are stepping up to take action. Not just in the utility industry but if you look at the President's voluntary climate reduction program, numerous business have made commitments to reduce emissions. Even Exxon-Mobile is now advertising voluntary actions it is taking to reduce its impact on climate trapping emissions.

But what does all of this domestic and international activity mean for the U.S.? While other countries are incentivizng new technologies in a comprehensive fashion, we are arguing about what to do. And where will those other countries take those technologies? To China which according to the New York Times consumes 11.5 times the energy of Japan to produce the same industrial output.

Despite the fact that Japan is far from meeting its Kyoto target --- it is already moving from industry to home and automobile – in attempts to dramatically increase efficiency and alternative vehicle use. And who is dominating the world market on hybrid vehicle sales? The Japanese car manufacturers. Meanwhile Japan has nearly tripled its industrial output from 1973 to today while keeping its overall energy consumption roughly flat.

While the world is deploying leapfrogging technology in an effort to deal with climate change, the U.S. lags sorely behind; concerned that movement to address the climate issue might create some kind of economic instability.

So how do you and we in industry alter the climate paradigm?
I think that it will require a number of steps – smaller steps then embracing Kyoto that will set us on the right path.

First, again, there are aspects of climate science that are indisputable even thought significant additional scientific work remains to refine the unknowns.

- CO_2 is at its highest concentrations in the past 400,000 years
- The earth is getting warmer
- The warming is caused by a combination of human and natural processes

Second, I think that we have all recognized that Kyoto was a 10,000 pound gorilla, and too much for the U.S. to tackle. As a result, I think that it is important to eliminate the linkage between any kind of carbon reduction policy and Kyoto.

While I believe that the best approach to climate is an economy wide approach – I think the path there may need to be more creative and perhaps even incremental in order to demonstrate the ability to control emissions in an economically viable way.

Whatever emission reduction approach is adopted, I believe that coupling it with legitimate methods of advancing technology is crucial. I know that this Committee focuses on Research and Development. I believe that taking a hard look at what programs are funded and what can be jumpstarted in order to bring them closer to commercial adaptation is important. Much of the discussion on R&D tends to focus on the R and not so much on the D --- development or what I think we need to see is Deployment. Getting these technologies into the marketplace earlier and more effectively is an issue that I believe is often overlooked.

And, I think that beyond traditional government programs, the development of technology funds to help offset the costs of meeting emission reduction targets can work, not only by spreading out the cost of those targets throughout the entire economy but by also helping the U.S. regain the lost momentum to lead the energy efficiency technology race. Ideas abound about how to fund these off budget – and they may not be practical right now – however beginning the discussion is important if optimal solutions to meeting a greenhouse gas reduction target are to be utilized.

Third, I also think it's important to greatly simplify the implementation of taking on emission reduction commitments. As a Utility company executive I am mystified each and every time the issue of meeting climate reduction programs or even the development of a voluntary registration of emissions arises, with it surfaces the host of issues that makes a solution all but impossible. How do you deal with past actions, additionality, every household and homeowner taking on a commitment? The Answer: Don't.
Let's not follow the complicated example of our friends across the "pond" that have developed hundreds of varying allocation rules for every industry or fuel type. Keep it simple. Make a forward looking commitment, meet it and if you go below it – allow those tons to be used to trade with others.

And finally as the Committee continues to examine greenhouse gas emissions I would urge you to be creative. This commitment in my own company has empowered our employees to creatively address how best to meet that commitment. The Acid Rain Program reforms to command and control regulations helped minimize the role of the government in business decisions and unleashed the power of the market by making reductions a good investment. This is the one of the economically efficient paths forward. To take another approach provides naysayers with the unwavering momentum that challenges the possibility of forward movement.

I believe that the country needs leadership in this area. I don't believe that I am being disloyal to the President whom I support, to Congress or to my shareholders when I say that the time is now to move positively toward reachable goals that will not only put us on

track to operate in a greenhouse constrained environment, but on a track that will also make this country the technological leader it once was and can be again.

IGCC Technology Status, Economics and Needs

Neville Holt –Technical Fellow

EPRI Science & Technology Development Division

Presented at the

International Energy Agency (IEA) Zero Emission Technologies (ZET) Technical Workshop

February 17, 2004

Gold Coast,Queensland, Australia

EPRI

IGCC Briefing for IEA ZET Workshop Presentation Structure

- US Incentives for IGCC. DOE Programs.
- IGCC Technology. Environmental Attributes.CO_2 Capture
- Current IGCC Status. Lessons Learned.
- IGCC Economics. Comparison to PC. Effect of Coal type on Technology Economics. Not all Gasification Technologies are the same. Costs of CO_2 Capture.
- IGCC with Co-production of Hydrogen
- Phased Construction of IGCC for later CO_2 Capture
- Barriers to IGCC Commercial Deployment.
- IGCC RD&D Needs
- Issues/Priorities

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U.S. Government Support for Coal Gasification

- **Huge domestic Coal Reserves available at low cost sufficient for several hundred years**
- **Coal Gasification is regarded as Key Technology for many aspects of U.S. Energy supply and Security. U.S Government has supported Coal Gasification RD&D continuously since WW II**
- **DOE Clean Coal Demo program 1990-2000 supported 250 MW IGCC plants at Tampa and Wabash**
- **Currently DOE Vision 21 program and Clean Coal Power Initiative (CCPI) have the objectives of:**

  - Retention of Coal for electricity production

  - Reduced dependence on energy imports (primarily oil but increasingly natural gas)

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Recent U.S. IGCC Initiatives

- 1.5 Billion $ Program for Hydrogen Fuel Cell Car
- Increased Program for CO_2 Sequestration. Regional Partnerships formed to examine sources & sinks.
- February 27, 2003 FutureGen announcement "A Coal-fueled Prototype for a Hydrogen Production/Carbon Sequestration Power Plant".
- Clean Coal Power Initiative (CCPI) 2nd Solicitation due in January 2004 specifically encourages IGCC
- Major Universities (Harvard,MIT,Princeton), IGCC Coalition, Gasification Technologies Council (GTC) evaluating IGCC Incentives
- Tax Incentives for IGCC and Advanced Coal were included in some Drafts of the Energy Bill

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FutureGen Initiative

- A 1 Billion $ Coal based IGCC program is proposed to provide ~ 275 MW of power, ~1 million tons/year of CO_2 for Sequestration in a Geologic formation and Hydrogen for Fuel Cells and later transportation.
- DOE has asked the Power Industry to form a ("CoolWater type") Consortium with Coal Companies to manage the project and provide 20% cost sharing
- DOE envisages the project as a "large scale engineering laboratory for testing new technologies"

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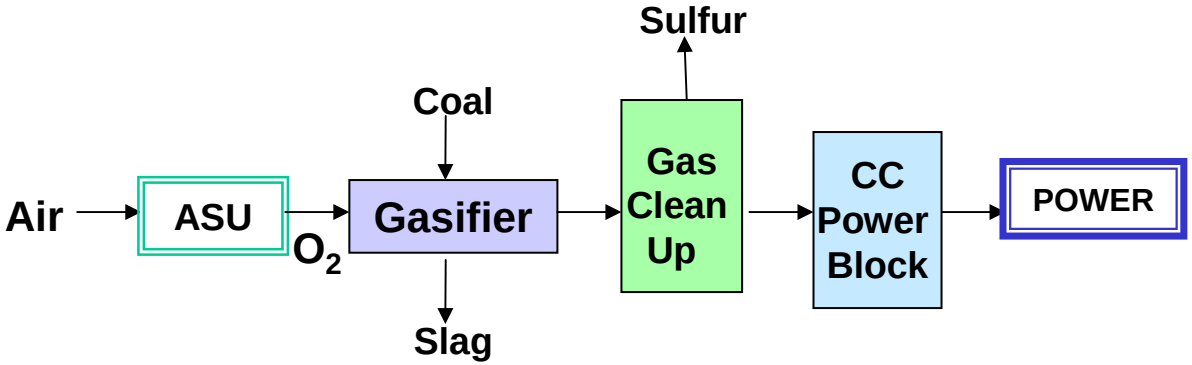
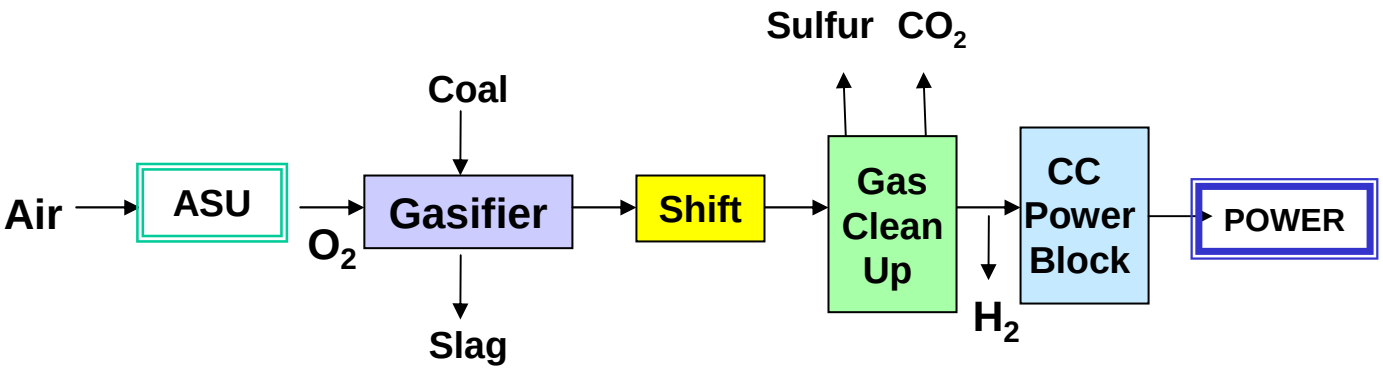
IGCC with and without CO_2 Removal





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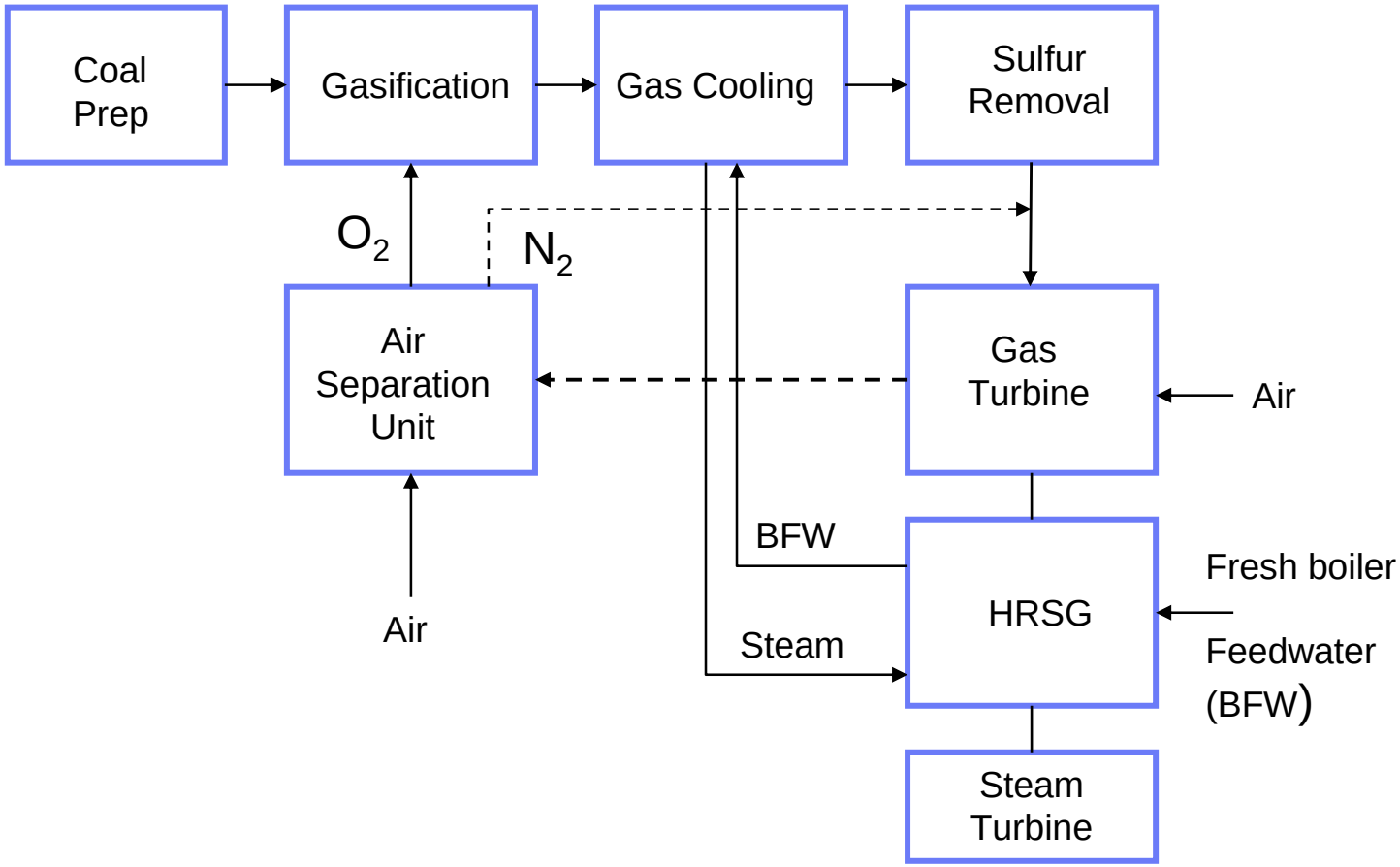

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IGCC Environmental Attributes

- Sulfur is removed (99.5-99.99%) from syngas and produced as sulfur or sulfuric acid for sale.
- NOx emissions are controlled by firing temperature modulation in the gas turbine.
- Particulates are removed from the syngas by filters and water wash prior to combustion so emissions are negligible.
- Current IGCC design studies plan <3ppmv each of SOx, NOx and CO.
- Chlorine removed from syngas by water wash and subsequent waste water treatment. Salt or Ammonium Chloride can be produced if zero water discharge is required.
- Mercury and other HAP's removed from the syngas by absorption on activated carbon bed –18 yrs. Experience at Eastman Chemical.
- Coal ash is produced as a dense slag with several potential uses(no limestone scrubber sludge or large volume solid waste as there would be with PC or CFBC).
- CO_2 removal from syngas under pressure is much less expensive than post combustion removal from flue gas of PC or NGCC plants.

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Coal based IGCC Plants

Project/ Location	Combustion Turbine	Gasification Technology	Net Output MW	Start-Up Date
Wabash River, IN	GE 7 FA	E gas (Now Conoco Phillips)	262	Oct 1995
Tampa Electric, FL	GE 7 F	Texaco (Now Chevron Texaco)	250	Sept 1996
Demkolec (now Nuon), Buggenum Netherlands	Siemens V 94.2	Shell (Now offered jointly with Krupp Uhde as Shell)	253	Jan 1994
ELCOGAS Puertollano Spain	Siemens V 94.3	Prenflo (Now joint with Shell)	310	Dec 1997

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Gasification - Major Candidates (N.B. All are Entrained Flow)

Technology Name/Owner	Texaco/ ChevronTexaco	E Gas/ ConocoPhillips	Shell/Shell
Feed	Coal in Water Slurry	Coal in Water Slurry	Dry coal
Configuration	Single Stage Downflow	Two Stage Upflow	Single Stage Upflow
Gasifier Wall	Refractory	Refractory	Membrane Wall
Pressure PSIG	500-1000	Up to 500	Up to 600
Notes	Offered as Quench or with Heat Recovery	Only offered with Heat Recovery	Only offered with Heat Recovery . Jointly with Krupp Uhde

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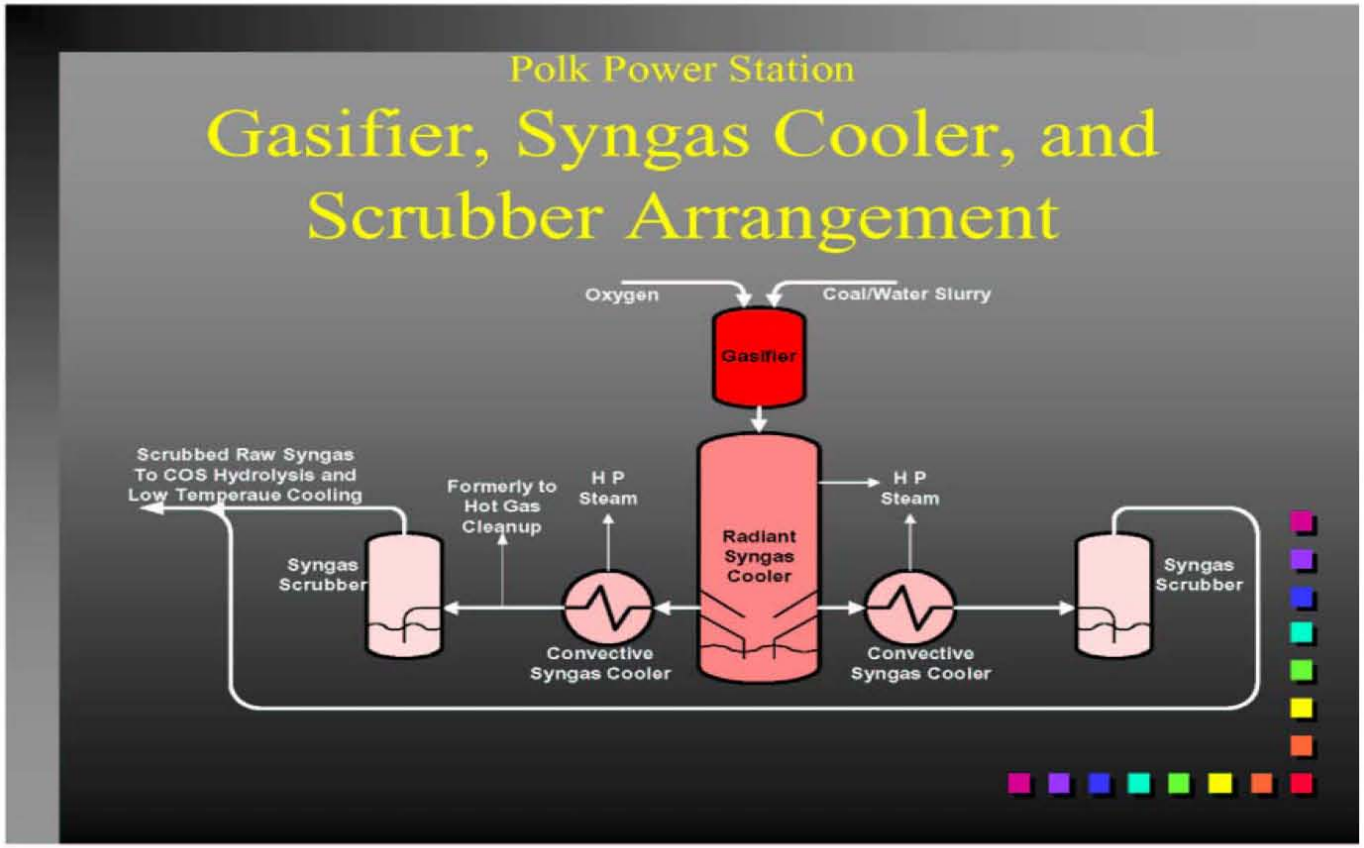
Tampa Electric – Gasification Arrangement



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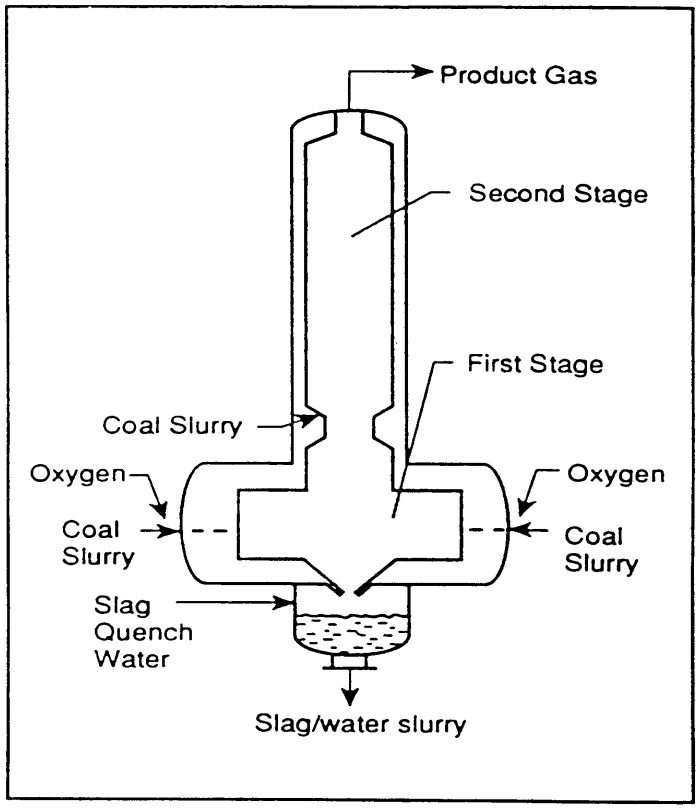
E Gas Gasifier – As used at Wabash IGCC (Technology now owned by ConocoPhillips)



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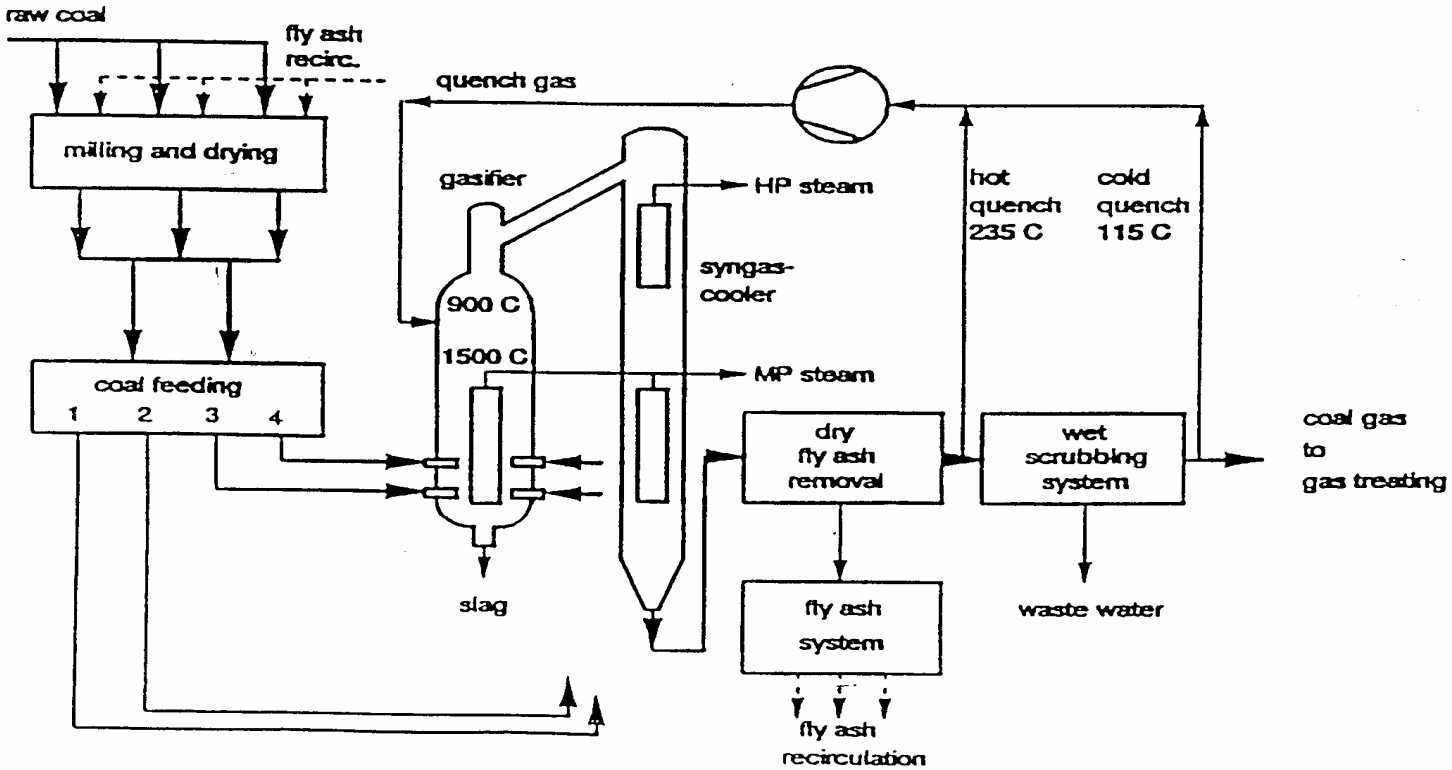
Shell Gasification as used at the 250 MW IGCC at Buggenum



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Coal IGCC - Lessons Learned

- **Very low SO_2, NOx, and Particulate Emissions below recent PC plants permit limits**
- **Global E Gas, Texaco, Shell and Prenflo gasifiers successfully demonstrated at commercial size**
- **Existing single train IGCC coal plants have not yet achieved their yearly availability targets of 85%– although on a quarterly basis the targets have been achieved. Commercial multi-train plants with spare gasifiers will achieve >90% availability.**
- **The high degree of Integration used in the European IGCC plants is not recommended for new IGCC plant designs.**
- **IGCC is currently being commercially used in many plants worldwide based on the gasification of petroleum residuals providing power, steam and hydrogen.**
- **Future advances in ASU's, gasification, gas clean up, gas turbine and fuel cell technologies will improve efficiency and lower cost**

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IGCC Design Studies – Notes on Sparing of Gasifiers

- **Texaco and E Gas refractory lined gasifiers need planned outage of 25-30 days for refractory replacement every 2-3 years. If a 90% Overall IGCC Equivalent Availability is required then, based on experience and lessons learned at the commercial demonstration plants, a spare gasifier would be required. The spare reduces the Scheduled Outage time and some of the Forced Outage time.**
- **Shell gasifiers do not need such extended outages and have a higher Availability. However Shell would also need a spare if 90% Availability was needed.**
- **E Gas paper at 2002 conference made a case for no spare for those instances where Spring and Fall power demand is lower so that planned outages could be taken at such times.**
- **Careful consideration of the needed IGCC plant Equivalent Availability and annual power demand profile should be made to decide on sparing.**

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EPRI Economic Estimates for Clean Coal Technologies without CO_2 Capture- 2003. 500 MW Plants Bituminous Coal

Fuel	Pittsburgh #8 Coal	Pittsburgh #8 Coal	Natural Gas	Natural Gas
Technology	Ultra Supercritical PC	IGCC E Gas No Spare/ With Spare	NGCC	NGCC
Capacity Factor	80	80	80/65/40	80
Fuel Cost $/MBtu HHV	1.5	1.5	3.5	4.9
TPC $/kW	1286	1210/1305	440	440
COE $/MWh	46.6	45.8/48.3	36.5/40/47.5	46.5

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Coal Technology Evaluations with CO_2 Capture – Preferred Technology depends on the Coal

- **Without Capture IGCC has higher COE than PC + FGD with bituminous coals, but with Capture the IGCC COE is markedly lower than PC + FGD.**
- **However the IGCC advantage over PC with Capture depends on the Gasification technology with High Pressure (55-70 bar) gasifiers being most advantageous.e.g. The estimated $/metric ton(mt) avoided cost of CO_2 at the plant gate is 19 from Texaco Quench IGCC, 29 from E Gas and Shell IGCC and 48 from USC PC. Sequestration at 5$/mt adds 5-6$/mt to these avoided costs.**
- **At the current State of Commercial Development IGCC without Capture and using Low Rank Coals is not competitive with PC. With Capture IGCC using Sub-bituminous Coal (e.g. PRB) has slightly lower COE than PC but with Lignite the PC COE is lower than IGCC.**

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EPRI Economic Estimates for IGCC & PC Plants without CO_2 Capture – 500 MW with Low rank Coals

Technology	IGCC E Gas	IGCC Shell No Spare/ Spare	PC Sub	IGCC E Gas	PC Sub
Coal	Wyo. PRB	Wyo. PRB	Wyo. PRB	Lignite	Lignite
TPC $/kW	1640	1480/1690	1330	1830	1340
Coal Cost $/MBtu HHV	1.0	1.0	1.0	0.5	0.5
COE $/MWh at 80% Capacity Factor	54	48/54	44	55	43

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IGCC for Low Rank Coals - Improvements Needed

- Although entrained flow gasifiers can process all ranks of coal the existing commercial gasifiers all show a marked increase in cost and reduced performance with low rank and high ash coals.
- For slurry fed gasifiers (Texaco, E Gas) the energy density of high moisture and/or high ash coal slurries is markedly reduced which increases the oxygen consumption and reduces the gasification efficiency
- For dry coal fed gasifiers (Shell) there is an energy penalty (and therefore reduced steam turbine output) for drying the high moisture coals to the low moisture content necessary for reliable feeding via lock hoppers and pneumatic conveying
- Although IGCC is closely competitive with PC for bituminous coals the IGCC–PC capital cost and COE gap widens for low rank coals. For PRB ~ 200-300$/kW and ~ 400 $/kW for US lignites
- Potential improvements include slurry preheating & flashing, Coal/CO_2 slurry, coal pump (e.g.Stamet) or other device to deliver as received (AR) coal reliably at pressure, Transport gasifier etc

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EPRI Economic Estimates Clean Coal Technologies with CO_2 Capture – 2003

450 MW Plants Pittsburgh #8 Bituminous Coal, All IGCC with spare gasifiers

Technology	IGCC Texaco Quench	IGCC Texaco Radiant SGC	IGCC E Gas	IGCC Shell	PC Ultra Supercritical
MW no Capture	512	546	520	528	600
TPC $/KW no capture	1268	1500	1305	1620	1235
COE $/MWh no capture	48.6	53.6	48.3	55.5	45
MW with capture	452	486	442	465	460
TPC $/kW with capture	1620	1900	1870	2190	2110
COE $/MWh with capture	61	67	67	73.6	75.4
Avoided Cost of CO_2 $/mt	18	21	29	29	45

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EPRI

Canadian Clean Power Coalition (CCPC) Study for Low Rank Coals with CO_2 Capture

(as reported by CCPC- except PC sub bit EPRI interpolation)

Coal	Alberta Sub bituminous	Alberta Sub bituminous	Saskatch. Lignite	Saskatch. Lignite	Saskatch. Lignite
Technology	USC PC Amine Scrubbing	Texaco quench IGCC	USC PC Amine Scrubbing	Shell IGCC	Oxyfuel O_2/CO_2 Combustion
Net MW	316	437	311	361	373
Coal Cost $/GJ $/MBtu	0.45/0.48	0.45/0.48	0.80/0.85	0.80/0.85	0.80/0.85
TPC US $/kW	2585	2205	2826	2847	3960
COE $/MWh	64	61	74	82	97

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EPRI

IGCC Co-Production of Hydrogen

- **1 kg of Hydrogen is the same LHV as I US gallon of gasoline (113,700 Btus)**
- **If just 1% of the produced syngas from a 500 MW IGCC plant is used to produce hydrogen this is enough hydrogen to provide the fuel needs of 10,000 vehicles. This corresponds to ~4 filling stations each with 12 pumps and 350 fill-ups per day (total 1400 fill-ups/day).**
- **Hydrogen produced in this manner could well be the lowest cost hydrogen because of the economies of scale provided by the large gasifiers in an IGCC plant. Preliminary results from Parsons study showed Hydrogen costs of 7-8$/MBtu LHV when using 10-1% of the Hydrogen (60,000-6000 kg/d) from a 500 MW IGCC plant with CO_2 Capture. This compares with 9$/MBtu from a SMR with Natural Gas at $4/MBtu.**
- **Biomass gasification would potentially use the same technology but suffers from the diseconomies of smaller scale. Biomass is probably best used as a partial feed to coal based IGCC**

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Phased Construction of IGCC

- Concept 1. NGCC to 2. IGCC to 3. IGCC with CO_2 Capture
- Most NGCC's are at sites unlikely to have future coal capability
- Adding IGCC will probably need major change to HRSG and ST
- Shift Reactors, CO_2 Removal & Compression needed for Capture
- Parsons study for EPRI evaluated Phased IGCC Concept with Pre-investment for subsequent Capture and its effect on COE .

  - IGCC without Capture with and without Pre-investment

  - IGCC with Capture with and without Pre-investment
- These Phased IGCC results provide initial inputs for:
  - Utility specific trade-off studies on pre-investment, Scenario and Present worth Analyses
- Decisions regarding the value of pre-investment depend on:
  - Timing of the various phases
  - Relative natural gas and coal prices
  - Cost and performance of competing technologies

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ChevronTexaco Quench Phased IGCC Results

Case	Base	Base Retrofitted for CO_2 capture	Pre-Investment Oversized	Pre-Investment Retrofitted for CO_2 Capture
Net Power MW	509	435	509	449
Heat Rate Btu/kWh HHV	9653	11,569	9653	11,550
TPC M$ $/kW	590 1158	678 1596	620 1217	683 1522
Delta Cost of Retrofit M$		88		63
COE $/MWh	45.7	59.3	47.1	57.2

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Barriers to IGCC commercial Deployment

- PC can meet current standards. (No incentive to be ultra-clean)
- IGCC Financing costs higher than PC
- No reward for risk taking (FOAK etc)
- Excess capacity in many regions. (NGCC overbuild)
- IGCC needs more project development than NGCC or PC. FEED followed by EPC bids. No standard IGCC design.
- Lack of familiarity with IGCC in the Power Industry. Perception of Cultural differences in Design, Construction and Operations

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IGCC RD&D Needs

- **Higher pressure designs for more cost effective CO_2 capture and improved performance with low rank coals.**
- **Improved reliability of major components. Mostly related to Gasification section. Better refractory life and design, better fuel injectors (for slurry fed gasifiers). Continuous slag removal (except E GAS).**
- **High pressure feeding for dry coal fed gasifiers. Lock hoppers become less attractive as pressure rises. Mechanical Pumps (e.g. Stamet)? Use slurry to pump to high pressure then flash? Use Coal in CO_2 slurries?**
- **Cost reduction. When CO_2 capture is needed consider Quench to reduce capital cost and steam needed for Shift.**
- **Gas turbine designs to avoid performance derate with Syngas and Hydrogen**

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IGCC Key RD&D Areas

- Air Separation Unit (ASU)
- Coal Feeding at High Pressure
- Entrained Gasifier Design
- Fluid Bed Gasification
- Syngas Coolers (SGC)
- Hot Gas Filters
- Desulfurization
- Gas Separation Membranes
- Mercury etc. Removal
- ATS Gas Turbines
- Advanced Power Cycles (FC etc.)

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IGCC RD&D Candidates

- **Ion Transfer Membranes (ITM) for ASU. Any ideas for an ASU at closer to ambient temperatures?**
- **Transport gasifier particularly for low rank and higher ash coals**
- **Syngas coolers. Fire tube lower cost than water tube. Prevention of fouling. Better materials.**
- **Hot gas filter design. Higher temperatures (but below alkali condensation). Better materials, Fouling prevention.**
- **Warm gas desulfurization. Must also remove Cl, Hg, NH_3etc**
- **Membrane separation of gas components e.g. CO_2 and H_2 However would prefer to have both produced at reasonably high pressure to avoid the severe energy penalties of additional compression.**

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IGCC Commercial Development Issues and Priorities for Issue Resolution

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The Cost of Mercury Removal in an IGCC Plant

Final Report
September 2002

Prepared for:

The United States Department of Energy
National Energy Technology Laboratory

By:

Parsons Infrastructure and Technology Group Inc.

Reading, Pennsylvania
Pittsburgh, Pennsylvania

DOE Product Manager:
Gary J. Stiegel

DOE Task Manager:
James R. Longanbach

Principal Investigators:
Michael G. Klett
Russell C. Maxwell
Michael D. Rutkowski

PARSONS

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LIST OF ACRONYMS AND ABBREVIATIONS

acf	actual cubic feet
acfm	actual cubic feet per minute
AGR	acid gas removal
CCT	clean coal technology
COS	carbonyl sulfide
EPA	U.S. Environmental Protection Agency
°F	degree Fahrenheit
ft	foot, feet
h	hour
Hg	mercury
IGCC	integrated gasification combined cycle
kW	kilowatt
kWh	kilowatt-hour
lb	pound
lb/ft^3	pound per cubic foot
MACT	maximum achievable control technologies
MSW	municipal solid waste
MW	megawatt
MWe	megawatts electric
MWh	megawatt-hour
O&M	operations and maintenance
PC	pulverized coal
PE	purchased equipment
ppb	parts per billion
ppbw	parts per billion by weight
psi	pounds per square inch
psia	pounds per square inch absolute
S	sulfur
scf	standard cubic feet
sec	second
TCC	total capital costs

1. SUMMARY

The U.S. Environmental Protection Agency (EPA) has decided to regulate mercury emissions from coal-burning utilities. Mercury has been identified by the EPA as the toxic substance of greatest concern among all the air toxics emitted from power plants. The EPA must propose regulations to control mercury emissions from coal- and oil-fired power plants by December 15, 2003 and issue final regulations by December 15, 2004. Meeting these regulatory requirements without excessive costs is a significant concern. Integrated gasification combined cycle (IGCC) plants offer the capability of removing the mercury from the compressed syngas prior to combustion where the gas volume treated is much less than the low pressure, post-combustion flow volume; thus the process is much less costly. In addition, the technology of removal of mercury in an IGCC plant has already been commercially demonstrated to remove greater than 90 percent of the mercury. This study was to determine the cost of such a removal system as applied to commercial-scale IGCC plants and show that the performance and cost levels are acceptable.

Currently, there is no single proven technology that can uniformly control mercury from power plant flue gas emissions in a cost-effective manner, while consistently achieving mercury removal levels of 90 percent. The effectiveness of existing flue gas emission controls in removing mercury can vary considerably from plant to plant, or even from boiler to boiler. With today's technologies, mercury removal can range from essentially no control to as high as 90 percent. This variability in control costs and performance expectations has led to uncertainty in control strategies and concerns for the operational and cost impacts on coal-fired power generation with mercury controls.

However, an IGCC power plant has the potential of achieving very high mercury removal performance with established technology. IGCC plants have the potential of removing mercury from the syngas upstream of the gas turbine. Syngas volumes are much smaller at this point. Thus, mercury removal in an IGCC has the potential to be both less complex and less expensive. As a result, mercury removal in an IGCC power plant can be expected to be very high in removal effectiveness, low in cost, and reliable in design.

Parsons developed a conceptual design and cost estimate for applying a carbon bed adsorption system to estimate the cost of mercury control in an IGCC plant. The IGCC plant design was based on a reference design developed by Parsons. Input on the performance of carbon bed systems was obtained from Eastman Chemical Company, based on their substantial commercial operating experience with this technology. This conceptual design and cost estimate is specifically applicable to gasification systems using high-temperature slagging gasifiers and bituminous coal, which includes most of the coal gasification plants in the U.S. that are currently operating or are in various planning stages.

The cost format was based on the methodology used in the EPA Mercury Study Report to Congress, while the cost estimate (capital and operations and maintenance, or O&M) was based on Parsons' in-house data and experience. The purchased equipment costs were scaled from Parsons' in-house data for pressure vessels used in a syngas application. The total capital cost is $3.34 per kilowatt.

O&M costs were based on factors for labor, material, and overhead. Carbon costs were by far the largest O&M cost factor, amounting to over 67 percent of the O&M costs. The total O&M is $0.183/MWh (or mills/kWh). Based on a 15 percent capital recovery factor, the total cost per year is $0.254/MWh or $3,412 per pound of mercury.

The study evaluated the sensitivity of the costs on five key parameters including mercury concentration, capital costs, carbon costs, carbon replacement time, and increasing mercury capture.

The cost of removal of mercury by a carbon bed in an IGCC plant is lower than in a pulverized coal (PC) plant. The mercury is removed from the compressed syngas in an IGCC plant, which greatly reduces the acfm (actual cubic feet per minute) and thus the size of the equipment and the number of beds. The estimated cost of $0.254 per MWh and $3,412 per pound of mercury can be compared to estimates of costs of mercury removal from PC power plants. In the EPA Mercury Study Report to Congress, the cost for 90 percent mercury removal from a 975 MW utility boiler using carbon beds was reported at $3.10 per MWh and $37,800 per pound of mercury.

The costs for mercury removal in PC plants can be an order of magnitude higher than the removal costs in an IGCC plant. However, the additional capital cost increment to add 90 percent mercury removal to an IGCC plant is less than 0.3 percent and the increase in the cost of electricity is less than one percent.

2. INTRODUCTION

The EPA has decided to regulate mercury emissions from coal burning utilities. A proposed regulation will be due no later than December 15, 2003, and promulgated the following year. Currently, no single technology has been proven that can uniformly control mercury from power plant flue gas emissions in a cost-effective manner, while consistently achieving mercury removal levels of 90 percent. The effectiveness of existing flue gas emission controls in removing mercury can vary considerably from plant to plant, or even from boiler to boiler. With today's technologies, mercury removal can range from essentially no control to as high as 90 percent. This variability in control costs and performance expectations has led to uncertainty in control strategies and concerns for the operational and cost impacts on coal-fired power generation with mercury controls.

However, an integrated gasification combined cycle (IGCC) power plant has the potential of achieving very high mercury removal performance with established technology. IGCC plants have the potential of removing mercury from the syngas upstream of the gas turbine. Syngas volumes are much smaller at this point. Thus, mercury removal in an IGCC has the potential to be both less complex and less expensive. As a result, mercury removal in an IGCC power plant can be expected to be very high in removal effectiveness, low in cost, and reliable in design.

The objective of this study was to prepare a conceptual design and a cost estimate of a carbon bed adsorption system for application to an IGCC plant configuration typical of coal-based gasification plants that would be subject to the proposed mercury regulations. This study showed a cost advantage of mercury removal in an IGCC plant, based on the following two attributes:

- There is an existing operating experience base on which the design and cost estimate can be reliably based; thus, the application to future plants can be made with low uncertainty.
- The mercury removal step in an IGCC plant can be located at a point in the process where the volumetric flow rate is much smaller than would be for post-combustion applications; thus, smaller equipment can be used.

Parsons developed a conceptual design and cost estimate for applying a carbon bed adsorption system to estimate the cost of mercury control in an IGCC plant. The IGCC plant was based on a reference IGCC plant developed by Parsons with input from Tampa Electric Company and Texaco Power.[1] The basis of the reference plant is the clean coal technology (CCT) demonstration plant installed at Polk County, Florida. Input on the performance of carbon bed systems was obtained from the Eastman Chemical Company, which uses carbon beds at its syngas facility in Kingsport, Tennessee.[2]

[1] "Clean Coal Reference Plants: IGCC Texaco," Parsons, December 2001.
[2] Telephone communication with Dave Denton, Eastman Chemical Company.

3. BACKGROUND

3.1 REGULATORY INITIATIVES

The EPA has announced that it will regulate emissions of mercury and other air toxics from coal- and oil-fired electric utility steam generating units (power plants). Mercury has been identified by the EPA as the toxic substance of greatest concern among all the air toxics emitted from power plants. Coal-fired power plants are the nation's largest source of mercury air emissions in the United States – about 43 tons of mercury each year.

The EPA must propose regulations to control mercury emissions from coal- and oil-fired power plants by December 15, 2003 and issue final regulations by December 15, 2004. Under this timetable, regulations would require utility compliance by December 2007 because the Clean Air Act requires sources to install maximum achievable control technologies (MACT) three years after regulations are promulgated.

3.2 MERCURY REMOVAL FOR CONVENTIONAL COAL-FIRED PLANTS

The ability to achieve 90 percent removal with established reliability and reasonable cost has not yet been established for coal-fired plants and continues in development and evaluation. As a result, there are concerns and uncertainties as to how effectively mercury removal can be achieved and the potential cost impacts on power generation. For conventional coal-fired power plants, the mercury removal step will likely be applied post-combustion, where the full quantity of combustion oxygen and the accompanying diluent nitrogen from the air supply increases the volume of the gas stream.

In the EPA Mercury Study Report to Congress, the costs for 90 percent mercury removal from a 975 MW utility boiler were reported for three technologies including carbon beds, activated carbon injection, and activated carbon injection with spray cooling.[3] The costs ranged from $1.43 per MWh and $17,400 per pound of mercury for activated carbon injection with spray cooling to $3.10 per MWh and $37,800 per pound of mercury for carbon beds.

EPRI has recently summarized the costs of mercury removal in power plants.[4] Their average baseline costs for various carbon injection systems at 90 percent mercury removal ranged from $2.80 per MWh to $3.30 per MWh.

As a frame of reference, the cost of electricity without a mercury removal process is typically about $35/MWh. The "Market-Based Advanced Coal Power Systems – Final Report, May 1999," prepared by Parsons for the DOE[5], used the same calculation methodology as this study and determined the cost of electricity for ten power plant systems (including IGCC, PC, and fluidized-bed coal, and natural gas combined cycle plants) to range from $31 to $39/MWh.

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[3] "Mercury Study Report to Congress: Volume VIII, An Evaluation of Mercury Control Technologies and Costs," EPA-452/R-97-010, December 1997, page 3-6.
[4] Chang, R. and Offen, G., "Mercury Control Options," Modern Power Systems, November 2001.
[5] http://www.fe.doe.gov/coal_power/special_rpts/market_systems/market_sys.shtml

Thus, the above estimated cost of mercury removal would represent an increase of as much as 5 to 10 percent in the cost of electricity.

3.3 MERCURY REMOVAL EXPERIENCE IN GASIFICATION

3.3.1 Activated Carbon

One of the principal suppliers and developers of activated carbon adsorbents for mercury removal, as well as for other gas components, is Calgon Carbon Corporation in Pittsburgh, Pennsylvania. The Calgon Type-HGR carbon has been used for low-pressure drop adsorption of mercury from natural gas since the early 1970s. There are many other activated carbon suppliers worldwide. The carbon is impregnated with sulfur at a concentration of about 10 to 15 wt%, and mercury reacts with sulfur as the gas goes through the sulfur-impregnated carbon bed to form mercuric sulfide (HgS). After the sulfur on the carbon is exhausted, the spent adsorbent is shipped to a hazardous chemicals disposal site. HgS is a very stable compound and its long-term storage presents no problems. The spent carbon can also be incinerated and the mercury recovered from the incinerator gas via cooling and condensation. In this case, a complex and expensive cooling/condensation method would be used, followed by trim gas phase carbon beds for residual mercury removal, and flue gas scrubbing for the resulting SO_2.

The Eastman Chemical Company's chemicals from coal facility began operations in 1983 and was the first use of a Texaco quench gasifier to provide feed gas for the production of acetyl chemicals. This facility also employed carbon beds to remove mercury from the syngas. The syngas production area produces raw gases, which are split into two process streams. About one-third of the raw gas is routed to the shift reactor. Both gas streams are cooled and then sent to individual carbon beds for mercury and other heavy metal removal. Each stream is then sent to a Rectisol sulfur recovery unit.

The purpose of the mercury removal is to protect the acetyl chemical product from any mercury contamination. Sulfur-impregnated activated carbon is used as the adsorbent in the packed beds operated at 86°F and 900 psi. A 90 to 95 percent mercury removal has been reported with a bed life of 18 to 24 months. Eastman has yet to experience any mercury contamination in its product.[2]

Higher levels of mercury removal from synthesis gas have not yet been verified. However, there is commercial experience in nearly total mercury removal from natural gas. Calgon has supplied activated carbon to a Texas pipeline company that achieves well over 99.99 percent mercury removal from high-pressure natural gas. In that case, the mercury in the inlet gas is about 50 $\mu g/Nm^3$ (approximately 70 ppbw), and the mercury in the outlet gas is 0.001 $\mu g/Nm^3$ (below detectable limits). The inlet mercury concentration of this natural gas case is similar to what one would expect in the synthesis gas from gasification of bituminous coal. There is reason to expect that carbon beds could also remove mercury from synthesis gas to below detectable levels. However, if carbon beds are designed for mercury removal, attention also has to be paid to any other trace components that can be adsorbed by carbon. The mercury-carrying capacity of activated carbon can be significantly compromised by the presence of other trace compounds.

3.4 VARIABILITY OF MERCURY CONTENT IN COAL

In evaluating the design and cost of a mercury removal system, it is important to understand the applicability of the estimate to a variety of gasification feedstock materials and to make sure that the basis for the estimate can be considered to be representative. Table 1 shows a summary of test results of the mercury content of various coals.[6] Data on the average mercury content of petroleum coke and tires are also included in this table. The data show a wide variety of mercury content as measured, but the averages are within a range of ±50 percent of the overall average. They are also within the designed capabilities of the carbon bed systems estimated in this study as shown by the sensitivity study. The mercury content of the coal is also not an important factor as explained by the limited influence of the mercury content of the coal (and thus the syngas stream) on the lifetime of the carbon bed. While the mercury content of coals can vary greatly, 100 ppbw is typical of the mercury content of high-sulfur bituminous coal, and is close to the overall average. This level of mercury content is also consistent with the measured value at the Polk County IGCC plant on which the reference plant design was based. With the exception of some of the waste coals analyzed, the average mercury content of the categories lies within the range of the design, cost, and sensitivity studies of this assessment.

Table 1
Mercury Content of Coals

Fuel Type	Number of Analyses	Average Mercury Content (ppbw, dry)	Range (ppbw, dry)
Anthracite	65	113	60 – 230
Bituminous coal	27,355	137	1 – 1,300
Bituminous coal, high S	512	99	10 – 557
Bituminous coal, low S	563	89	10 – 912
Lignite	1,047	106	20 – 750
Petcoke	1,171	50	0.9 – 500
Subbituminous coal	8,614	71	8 – 900
Tires	149	56	10 – 328
Waste anthracite	426	190	40 – 540
Waste bituminous	572	464	33 – 1,180
Waste subbituminous	53	119	65 – 347
Overall	**40,527**	**107**	**0.9 – 1,300**

[6] http://www.epa.gov/ttn/atw/combust/utiltox/utoxpg.html

4. DESIGN CONSIDERATIONS

The syngas flow rate, composition, and properties were taken from the IGCC plant design, based on the Tampa Electric IGCC Demonstration Project, which utilizes an entrained-flow, oxygen-blown Texaco gasification process.[1] Figure 1 is a block flow diagram of the plant. Table 2 shows the detailed compositions at each state point. The plant configuration is based on the radiant cooler gasifier mode. The power generation technology is based on selection of a gas turbine derived from the General Electric 7FA machine. The plant is configured with one gasifier including processes to progressively cool and clean the gas, making it suitable for combustion in the gas turbines. The resulting plant produces a net output of 250 MWe at an efficiency of 37.6 percent on an HHV basis. Performance is based on the properties of Pittsburgh No. 8 coal.

The raw synthesis gas exiting the radiant syngas cooler is cooled in the series of heat exchangers before entering the fuel gas scrubber. The cooled syngas at 450°F then enters the scrubber for particulate removal. The quench scrubber washes the syngas in a counter-current flow in two packed beds. After leaving the scrubber at a temperature of about 290ºF, the gas is suitable for feeding to the COS hydrolysis reactor. The quench scrubber removes essentially all traces of entrained particles, principally unconverted carbon, slag, and metals. Following the syngas scrubber, the gas is reheated to 410°F and fed to the COS hydrolysis reactor. The COS is hydrolized with steam in the gas over a catalyst bed to H_2S, which is more easily removed by the acid gas removal (AGR) solvent. Before the raw fuel gas can be treated in the sulfur removal process, it must be cooled to 103°F. During this cooling, most of the water vapor condenses. The promoted monodiethanolamine (MDEA) process for AGR was chosen because of its high selectivity toward H_2S and because of the low partial pressure of H_2S in the fuel gas. The AGR process utilizes an MDEA sorbent and several design features to effectively remove and recover H_2S from the fuel gas stream. The MDEA solution is relatively expensive, and measures are taken to conserve the solution during operations. As the presence of CO causes amine degradation in the form of heat stable salts, an amine reclaimer is included in the process. Also, additional water wash trays are included in the absorber tower to prevent excessive solvent loss due to vaporization. Fuel gas enters the absorber tower at 103°F and 378 psia. Approximately 99 percent of the H_2S is removed from the fuel gas stream. The resulting clean fuel gas stream exits the absorber and is heated in a regenerative heater to 310°F. H_2S is regenerated and sent in a concentrated stream to the Claus plant.

Figure 1
Modified Block Flow Diagram
Texaco Gasifier-Based IGCC Reference Plant

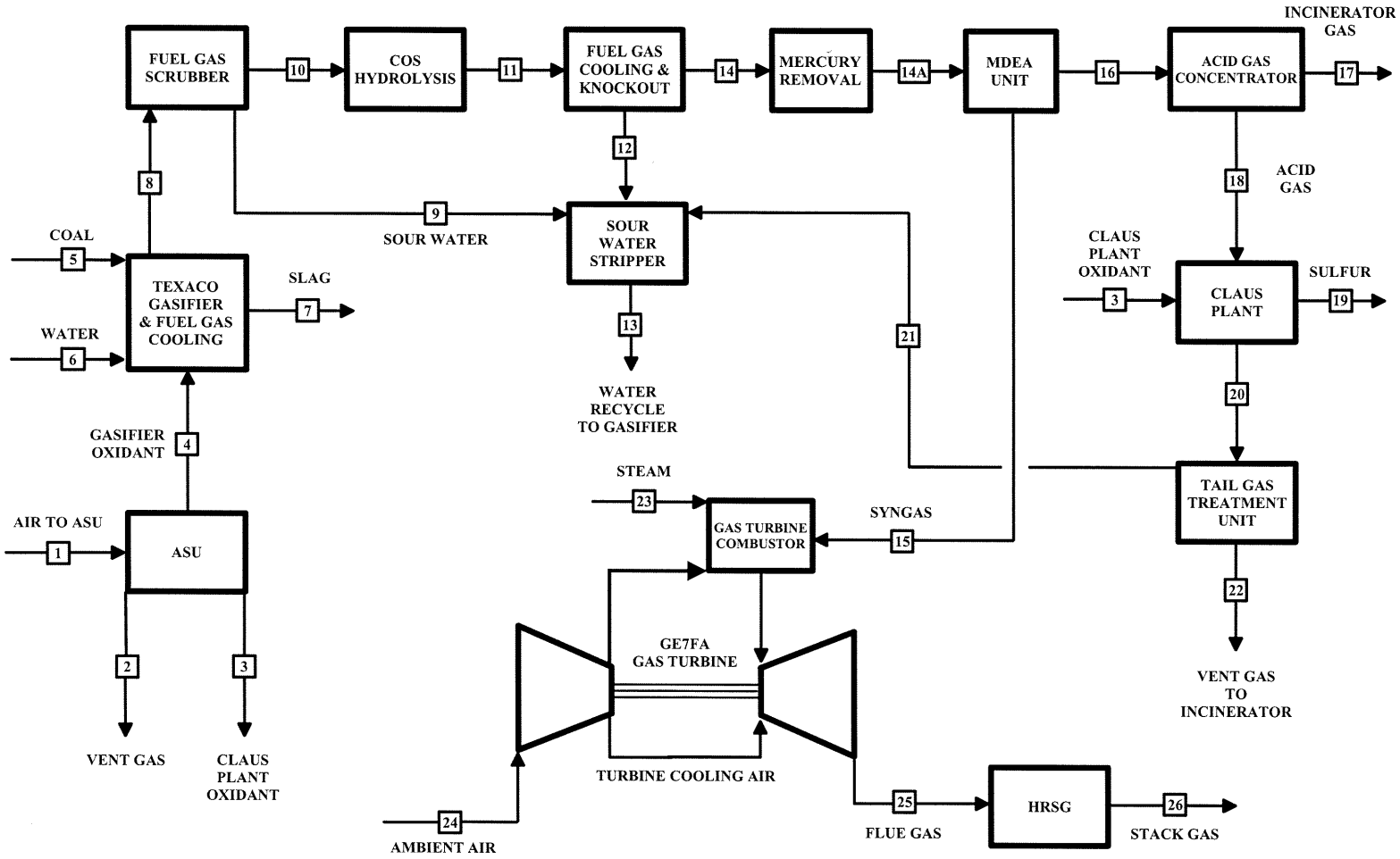


Table 2
Texaco IGCC Plant – Detailed Composition (page 1 of 3)

Stream Number	1	2	3	4	5	6	7	8	9
Vapor - Liquid									
Mole Fraction									
Ar	0.0094	0.0029	0.0360	0.0360	0.0000	0.0000	0.0000	0.0094	0.0012
CH_4	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0006	0.0001
CO	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.4160	0.0074
CO_2	0.0003	0.0004	0.0000	0.0000	0.0000	0.0000	0.0000	0.1078	0.0302
H_2	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.3002	0.0067
H_2O	0.0104	0.0131	0.0000	0.0000	0.0000	1.0000	0.0000	0.1488	0.7782
N_2	0.7722	0.9645	0.0140	0.0140	0.0000	0.0000	0.0000	0.0070	0.0002
O_2	0.2077	0.0191	0.9500	0.9500	0.0000	0.0000	0.0000	0.0000	0.0000
COS (ppm)	0	0	0	0	0	0	0	763	734
H_2S (ppm)	0	0	0	0	0	0	0	7,764	20,512
NH_3 (ppm)	0	0	0	0	0	0	0	1,570	154,789
SO_2 (ppm)	0	0	0	0	0	0	0	0	0
Total V-L Flow (lbmol/h)	28,975	23,105	89	5,779	0	4,778	0	22,081	148
Total V-L Flow (lb/h)	836,054	646,943	2,870	186,247	0	86,071	0	454,926	2,809
Solids (lb/h)									
Coal	0	0	0	0	209,208	0	0	0	0
Slag	0	0	0	0	0	0	26,322	266	266
Sulfur	0	0	0	0	0	0	0	0	0
Temperature (°F)	59	60	90	227	59	59	200	450	314
Pressure (psia)	14.7	20.0	30.0	650.0	14.7	14.7	15.0	430.0	430.0

Table 2
Texaco IGCC Plant – Detailed Composition (page 2 of 3)

Stream Number	10	11	12	13	14	15	16	17	18
Vapor - Liquid									
Mole Fraction									
Ar	0.0095	0.0095	0.0000	0.0000	0.0110	0.0116	0.0000	0.0000	0.0000
CH_4	0.0006	0.0006	0.0000	0.0000	0.0007	0.0008	0.0000	0.0000	0.0000
CO	0.4188	0.4188	0.0000	0.0000	0.4880	0.5127	0.0040	0.0068	0.0000
CO_2	0.1083	0.1091	0.0001	0.0000	0.1271	0.0935	0.7906	0.9883	0.4994
H_2	0.3022	0.3022	0.0000	0.0000	0.3521	0.3699	0.0029	0.0049	0.0000
H_2O	0.1446	0.1438	0.9960	1.0000	0.0027	0.0029	0.0000	0.0000	0.0000
N_2	0.0070	0.0070	0.0000	0.0000	0.0082	0.0086	0.0000	0.0000	0.0000
O_2	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
COS (ppm)	763	8	0	0	9	9	0	0	0
H_2S (ppm)	7,678	8,433	114	0	9,807	52	202,395	3	500,565
NH_3 (ppm)	538	538	3,785	0	324	0	0	0	0
SO_2 (ppm)	0	0	0	0	0	0	0	0	0
Total V-L Flow (lbmol/h)	21,933	21,933	3,115	4,778	18,824	17,867	908	541	367
Total V-L Flow (lb/h)	452,117	452,117	56,122	86,071	396,099	357,167	37,949	23,623	14,326
Solids (lb/h)									
Coal	0	0	0	0	0	0	0	0	0
Slag	0	0	0	0	0	0	0	0	0
Sulfur	0	0	0	0	0	0	0	0	0
Temperature (°F)	410	411	111	59	103	310	120	123	127
Pressure (psia)	415.0	402.0	378.0	14.7	378.0	362.5	35.0	30.0	30.0

Table 2
Texaco IGCC Plant – Detailed Composition (page 3 of 3)

Stream Number	19	20	21	22	23	24	25	26
Vapor - Liquid								
Mole Fraction								
Ar	0.0000	0.0066	0.0000	0.0109	0.0000	0.0094	0.0093	0.0093
CH_4	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
CO	0.0000	0.1045	0.0000	0.0139	0.0000	0.0000	0.0000	0.0000
CO_2	0.0000	0.3290	0.0000	0.5957	0.0000	0.0003	0.0803	0.0803
H_2	0.0000	0.0555	0.0000	0.2184	0.0000	0.0000	0.0000	0.0000
H_2O	0.0000	0.4556	1.0000	0.1009	1.0000	0.0104	0.1608	0.1608
N_2	0.0000	0.0364	0.0000	0.0600	0.0000	0.7722	0.6370	0.6370
O_2	0.0000	0.0000	0.0000	0.0000	0.0000	0.2077	0.1126	0.1126
COS (ppm)	0	521	0	95	0	0	0	0
H_2S (ppm)	0	5,505	10	103	0	0	0	0
NH_3 (ppm)	0	0	0	0	0	0	0	0
SO_2 (ppm)	0	6,221	0	0	0	0	8	8
Total V-L Flow (lbmol/h)	0	511	159	310	13,937	111,584	135,502	135,502
Total V-L Flow (lb/h)	0	14,119	2,856	9,616	251,080	3,219,670	3,827,920	3,827,920
Solids (lb/h)								
Coal	0	0	0	0	0	0	0	0
Slag	0	0	0	0	0	0	0	0
Sulfur	6,001	5	0	0	0	0	0	0
Temperature (°F)	347	280	120	123	500	59	1,131	280
Pressure (psia)	23.6	23.6	16.5	14.9	350.0	14.7	14.8	14.7

The operation of the combined cycle unit in conjunction with oxygen-blown IGCC technology is projected to result in very low levels of emissions of NO_x, SO_2, and particulate (slag). A salable byproduct is produced in the form of elemental sulfur. The low level of SO_2 in the plant emissions is achieved by capture of the sulfur in the gas by the amine-based MDEA acid gas removal (AGR) process.

NO_x emissions are limited to approximately 15 ppm by the use of steam injection. The ammonia is removed with process condensate prior to the low-temperature AGR process. This helps lower NO_x levels as well. The techniques of selective catalytic reduction (SCR) or selective non-catalytic reduction (SNCR) can reduce emissions further, but were not necessary. Particulate discharge to the atmosphere is limited to low values by the gas-washing effect of the syngas scrubber and the AGR absorber.

4.1 CARBON BED LOCATION

Calgon Carbon has product bulletins providing process design guidance information[7] that is helpful in the selection of the carbon bed location. The information was available for elevated pressures consistent with the IGCC reference plant design. Based on their curves for pressure drop through beds of either their granular 4x10 U.S. mesh carbon or their 4 mm diameter pellets, estimates of the pressure drop and sizing of the beds could be made. The pressure drop through the bed of pellets selected as the basis for this design and estimate is about 20 percent less than with the granular carbon.

The Calgon information also addressed the temperature effects on the effectiveness of mercury removal. At temperatures of around 100°F and lower, the concentration of mercury can be reduced readily to levels of 0.01 $\mu g/Nm^3$ (less than 1 ppbw) and substantially lower. At temperatures of 160°F and above, removal to below 0.01 $\mu g/Nm^3$ cannot be consistently achieved. The mercury removal rate and removal limit are relatively insensitive to pressure in the applicable range and are adversely affected by moisture level and the presence of water adsorbed on the carbon.

As seen in Figure 1, there are numerous syngas sites from which mercury can be removed, but the Calgon removal guidelines tend to narrow the choices.

Stream 8 contains most of the contaminants from the gasified coal. However, attempting to remove the mercury from this stream would entail removing residual particulate matter from the syngas. Also, the 450°F temperature is significantly higher than the optimum operating temperature for carbon. Stream 10, located upstream of the COS hydrolysis unit, is at a temperature of 410°F and a pressure of 415 psia. Again, this stream is at high temperature for the carbon bed operation. Stream 15 is a clean stream following the AGR process; locating the mercury removal process there would subject the AGR process to mercury and other heavy metal contamination. Removing the mercury and other contaminants before the sulfur recovery unit should enhance the performance of the unit and increase the life of the solvent.

[7] Calgon Carbon Corporation Product Bulletin AB-742-06/94

Several of the more likely choices for locating the carbon bed adsorption system are summarized in Table 3. This table shows that the volumetric flow of treated gas is substantially lower as a low-temperature, high-pressure syngas stream prior to combustion and expansion of the syngas. This represents a significant advantage in the sizing and cost of equipment and the amount of carbon needed in the treatment system.

Table 3
Volumetric Flow Comparison

Stream Number	14	15	25	26
Location	Syngas cooled, before sulfur removal	Syngas cooled, after sulfur removal	Flue gas, before HRSG	Stack gas
Flow Rate: **lbmol/h** **10^3 lb/h**	 18,824 396	 17,867 357	 135,502 3,828	 135,502 3,828
Temperature, °F	103	310	1131	280
Pressure, psia	378	362.5	14.8	14.7
Volumetric Flow Ratio*	1.00	1.35	520	244

* *Ratio of volumetric flow rate compared to the selected location after the syngas cooler and prior to the sulfur removal system. Volumetric flow rates adjusted for temperature and pressure.*

Locating the carbon beds downstream of the syngas coolers substantially reduces the level of moisture in the syngas as shown in comparing Streams 11 and 14 in Table 2, where the moisture content is reduced from 0.1438 mole fraction to 0.0027.

Accordingly, the packed carbon bed vessels were located upstream of the AGR process in Stream 14 at a temperature of 103°F and a pressure of 378 psia. Figure 1 has the location of the mercury removal process identified. A separate stream table, Table 4, shows the relative change in the properties and composition of Streams 14 and 14-A as a result of installing the mercury removal vessel. The stream pressure drops 10 psi and the mercury concentration drops from 52 ppbw to 5 ppbw.

Table 4
Effect of Mercury Removal on Stream Properties

Stream Number	14 Before Hg Removal	14-A After Hg Removal
	Mole Fraction	Mole Fraction
Ar	0.0110	0.0110
CH_4	0.0007	0.0007
CO	0.4880	0.4880
CO_2	0.1271	0.1271
H_2	0.3521	0.3521
H_2O	0.0027	0.0027
N_2	0.0082	0.0082
O_2	0.0000	0.0000
COS (ppm)	9	9
H_2S (ppm)	9,807	9,807
NH_3 (ppm)	324	324
SO_2 (ppm)	0	0
Mercury (ppbw)	**52**	**5**
Total V-L Flow (lbmol/h)	18,824	18,824
Total V-L Flow (lb/h)	396,099	396,099
Temperature (°F)	103	103
Pressure (psia)	**378.0**	**368.0**

Eastman Chemical also locates beds downstream of the gas cooling and ahead of the sulfur recovery unit. Our temperature of 103°F is close to the temperature of 86°F at which Eastman Chemical operates.[2]

4.2 MERCURY CONCENTRATION

The mercury input to the packed bed was based on mercury in the coal as measured at the Polk County IGCC plant of approximately 100 ppbw (0.0207 lb/hour[8]). This mercury content is consistent with the average levels reported for high-sulfur bituminous coal and the overall average as shown in the preceding Table 2. A concentration of 100 ppbw in the coal is equivalent to 52 ppbw in the syngas if all the mercury appears in the syngas. It is likely that

[8] NETL, Ohio State Meeting-GJS-07/10/01.

some mercury may be removed in the fuel gas scrubber as well as captured in the slag before it reaches the bed. At the Polk County plant, approximately 40 percent of the mercury in the coal was unaccounted for when measuring mercury emissions in the stack. It is reasonable to expect that some portion of this difference can be attributed to collection in the slag and the scrubber effluent. The performance estimate in this study is total mercury capture: that is, it assumes no mercury reduction upstream of the carbon beds.

4.3 PROCESS PARAMETERS

An empty vessel basis gas residence time of approximately 20 seconds was used based on Eastman Chemical's experience.[2] Allowable gas velocities are limited by considerations of particle entrainment, bed agitation, and pressure drop. One-foot-per-second superficial velocity is in the middle of the range normally encountered[8] and was selected for this application. The density of 30 lb/ft^3 was based on the Calgon Carbon Corporation HGR-P sulfur-impregnated pelleted activated carbon.[9]

These parameters determined the amount of carbon needed, the size of the vessels, and the space velocity (the ratio of volumetric flow rate of gas to the volume of catalyst: 4,000 $hour^{-1}$ based on standard cubic feet or 200 $hour^{-1}$ based on actual cubic feet. While a single vessel of 10½-foot diameter was feasible, it was decided to use two smaller diameter vessels of 7½-foot diameter to add flexibility to the plant operations. The small number of vessels and the small size can be attributed to the reduced volumetric flow for the cooled syngas treated prior to the AGR system and the gas turbine combustion as noted in the earlier table.

The total bed pressure drop of about 10 psia was converted into a power cost penalty and included in the O&M cost estimate. In the sensitivities study, the addition of a second bed in series to achieve much higher mercury removal percentages added additional pressure drop and power penalties.

4.4 MERCURY REMOVAL

Packed beds of sulfur-impregnated carbon have been applied to hydrogen streams of chlor-alkali plants and typically remove about 90 percent of the mercury content of the stream.[10] Eastman Chemical also uses sulfur-impregnated carbon in its bed and has experienced removals of 90 to 95 percent.[2] Carbon removals of greater than 99 percent can be achieved by the use of dual beds, i.e., two beds in series.

This study assumes that the use of sulfur-impregnated carbon in a carbon bed achieves 90 percent reduction of mercury emissions. Use of the pelletized form of the carbon was assumed. A cost sensitivity of increasing the removal to 99+ percent by adding a second bed to

[9] http://www.calgoncarbon.com/bulletins/HGR-P.htm
[10] "Mercury Study Report to Congress: Volume VIII, An Evaluation of Mercury Control Technologies and Costs," EPA-452/R-97-010, December 1997, page 2-24.

each train is also presented. This includes the effect of additional pressure drop and power penalty due to the addition of the second bed.

4.5 CARBON REPLACEMENT TIME

Eastman Chemical replaces its bed every 18 to 24 months.[2] However, it is not because of mercury loadings that the bed is replaced, but for other reasons including:

- A buildup in pressure drop.
- A buildup in water in the bed.
- A buildup of other contaminants.

For this study an 18-month carbon replacement cycle has been assumed. Under these assumptions, the mercury loading in the bed would build up to 0.4 weight percent. Mercury capacity of sulfur-impregnated carbon can be as high as 20 weight percent.[11] Even with the highest mercury content found in various coals as reported in Table 1 (1,300 ppbw), the loading after 18 months would be only 9.6 percent. The moisture content reduction by condensation in the fuel gas cooling and knockout section before the AGR also aids the carbon bed lifetime. Thus, under most conditions, mercury loading should not be a factor in carbon replacement time.

[11] http://www.calgoncarbon.com/bulletins/TYPE_HGR.htm

5. DESIGN BASIS

Table 5 summarizes the design basis used for this study.

Table 5
Design Basis
Mercury Removal by a Carbon Bed – Fixed-Bed Option

Syngas, lb mol/h	18,824
Syngas, lb/h	396,099
Molecular weight	21.0
Temperature, °F	103
Pressure, psia	378
Mercury concentration, ppbw	52
Carbon field packed density, lb/ft^3	30
Carbon loading, lb mercury/lb carbon	0.39%
Space velocity, h^{-1}	4,000
Syngas, scf/h	6,757,816
Syngas, acf/h	300,729
Absorbent carbon, scf	1,689
Cycle time absorption, h	10,515
Superficial velocity, ft/sec	1.00
Number of vessels	2
Vessel ID, ft	7.3
Bed height, ft	20.2
Carbon life, cycles	1
Carbon replacement time, year	1.50
Carbon replacement rate, ton/year	16.9
Initial carbon charge, ton	25

6. COST ESTIMATE

Table 6 presents estimated costs for applying this packed bed carbon adsorption system to an IGCC plant. The cost format was based on the methodology used in the EPA Mercury Study Report to Congress,[12] while the cost estimate (capital and O&M) was based on Parsons' in-house data and experience.

The purchased equipment costs were scaled from Parsons' in-house data for pressure vessels used in a syngas application. The installation cost, which includes foundations and piping, was estimated to be 50 percent of the purchased equipment costs. The total capital cost came to $834,350 or $3.34 per kilowatt. The ratio of equipment costs to total costs of a little more than two is consistent with the recent Reference Design.[1]

O&M costs were based on factors for labor, material, and overhead. Carbon costs were based on Calgon Carbon Corporation's list price for pelletized, sulfur-impregnated carbon of $6.43/lb.[13] Carbon costs are by far the largest O&M cost factor, amounting to over 67 percent of the O&M costs.

Disposal costs of $500/ton were estimated assuming hazardous waste disposal. The total O&M is $320,683. Based on a 15 percent capital recovery factor, which is typical for a power plant, the total cost per year is $445,836 or $0.254/MWh (or mills/kWh). The cost of mercury reduction is $3,412 per pound.

[12] "Mercury Study Report to Congress: Volume VIII, An Evaluation of Mercury Control Technologies and Costs," EPA-452/R-97-010, December 1997, Appendix B.
[13] Telephone communication with Calgon Carbon Corporation.

Table 6
Cost Estimate
Mercury Removal by a Carbon Bed – Fixed-Bed Option

PLANT PARAMETER	
Plant net capacity, MWe	250
Hg level before fixed bed, ppbw	52
Hg level after fixed bed, ppbw	5
Capacity factor, %	80
CAPITAL COST ($)	
Purchased equipment (PE)[1]	$407,000
Installation[2]	$203,500
Indirects[3]	$122,100
Contingency[4]	$101,750
Total capital costs (TCC)	$834,350
TCC, $/kW	$3.34
OPERATING & MAINTENANCE COST ($/year)	
Operating labor[5]	$4,380
Supervision[6]	$657
Maintenance labor[7]	$2,409
Maintenance material[8]	$8,344
Carbon[9]	$217,203
Power[10]	$36,792
Disposal[11]	$8,445
Overhead[12]	$9,080
Taxes, insurance, administration[13]	$33,374
Total O&M	$320,683
Capital recovery[14]	$125,153
Total, $/year	$445,836
Total, $/MWh	$0.254
Mercury reduction, lb/year	131
Mercury, $/lb	$3,412

Notes:

1 *Scaled from Parsons' in-house data*
2 *50 percent of PE costs*
3 *30 percent of PE costs*
4 *25 percent of PE costs*
5 *1 hour/shift @ $20/h*
6 *15 percent of operating labor costs*
7 *0.5 hour/shift @ 10% wage rate premium over labor wage*
8 *1 percent of TCC*
9 *Based on Calgon Carbon Corporation list price of $6.43/lb for sulfur-impregnated carbon, pelletized*
10 *Using a pressure drop of 10 psi and 35 mills per kWh*
11 *Based on hazardous waste disposal of $500/ton*
12 *60 percent of labor and maintenance costs*
13 *4 percent of TCC*
14 *Capital recovery factor of 15%*

7. SENSITIVITIES

Table 7 shows the results of a sensitivity analysis to determine the impact of increased mercury concentration if one, for example, co-fired municipal solid waste (MSW) with coal. Co-firing 10 percent MSW would increase the mercury concentration in the syngas from 52 ppbw to 100 ppbw; co-firing 25 percent MSW would increase it to 150 ppbw.

The mercury concentration does not affect the size of the filter or the amount of carbon used. Since the mercury loadings, even at these increased levels, are well below the carbon capacity, they would not impact the carbon replacement rate. Thus, none of the capital and the O&M costs would be impacted. However, the cost per pound of mercury removed decreases because of the increased amount of mercury removed annually by the carbon bed.

Table 7
Sensitivity of Increased Mercury Concentration

Mercury concentration in the syngas, ppbw	52	100	150
Mercury reduction, lb/year	131	250	375
Reduction costs, $/lb	$3,412	$1,785	$1,190

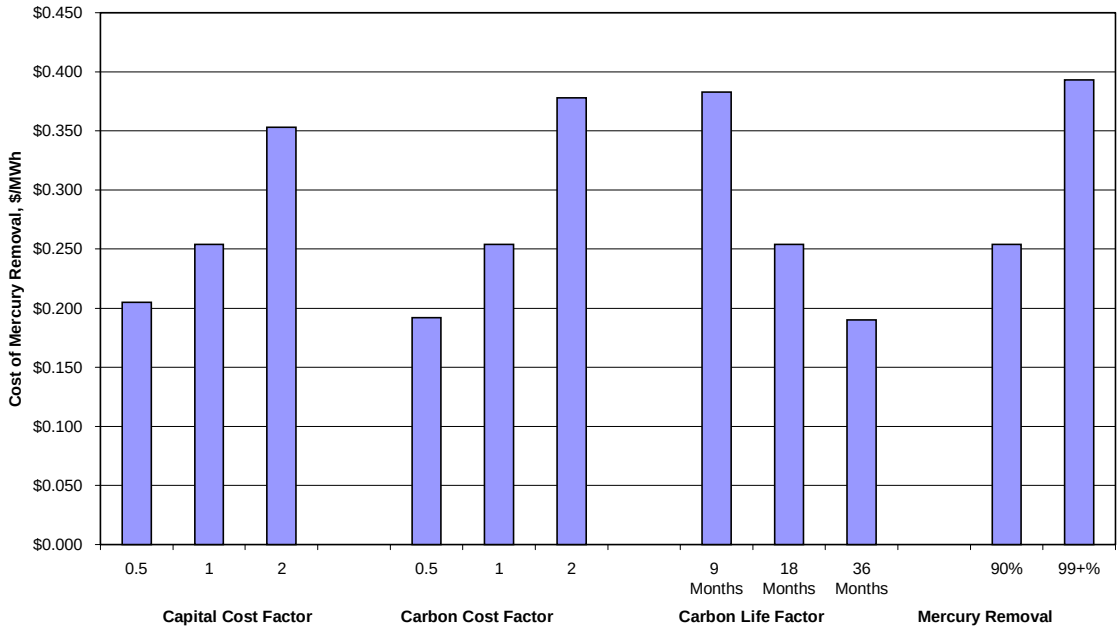
The sensitivity of the costs on four other key parameters including capital costs, carbon costs, carbon replacement time, and increasing mercury removal levels was evaluated. Increasing the mercury removal level from 90 to 99 percent was based on doubling the number of beds and having two beds in series. Table 8 shows the results of these sensitivity studies.

Table 8
Sensitivity of Key Parameters

Capital costs factor	0.50	1.0	2.0
Capital Costs, $/kW	1.67	3.34	6.67
Total cost, $/MWh	0.205	0.254	0.353
Reduction costs, $/lb	$2,754	$3,412	$4,728
Carbon costs, $/lb	$3.22	$6.43	$12.86
Carbon cost factor	0.50	1.0	2.0
Total cost, $/MWh	0.192	0.254	0.378
Reduction costs, $/lb	$2,581	$3,412	$5,075
Carbon replacement time, months	9	18	36
Carbon replacement factor	0.50	1.0	2.0
Total cost, $/MWh	0.383	0.254	0.190
Reduction costs, $/lb	$5,139	$3,412	$2,549
Mercury removal, %		90	99+
Capital Costs, $/kW		3.34	6.67
Total cost, $/MWh		0.254	0.393
Reduction costs, $/lb		$3,412	$4,791

Figure 2 shows a column chart for these same sensitivities.

Figure 2
Sensitivity of Key Parameters on Mercury Removal Costs



Doubling the capital costs increases the mercury reduction costs by a little more than one-third since these costs are only about one-third of the cost of mercury removal. Changes in the cost of carbon and the replacement time for carbon have a slightly larger impact on the cost of mercury removal than changes in the capital costs. The cost of sulfur-impregnated carbon is by far the largest cost item, amounting to over half of the total costs.

Increasing the mercury capture by using dual beds effectively doubles the capital costs but increases the cost of removal by only about one-half. The O&M costs increase by approximately 30 percent since the carbon replacement rate, which depends on the amount of contaminants removed, remains essentially the same.

8. APPLICABILITY OF ESTIMATE

This particular study is based on mercury removal in an IGCC plant using a high-temperature slagging gasifier with bituminous coal and a low-temperature acid gas removal system. This is consistent with the low temperature constraints of the carbon beds for achieving very high levels of mercury removal. With this basis, the experience at Eastman Chemical in their coal gasification system is directly applicable and provides a level of confidence in the performance expectations. It is expected that this performance and cost estimate will also be reasonable when using low rank coals, such as sub-bituminous coal, as the mercury content is within the applicable range as shown in Table 1 and the gas composition would be expected to be somewhat similar to this case. Extensions of these results to low-temperature gasifiers, drastically different feedstocks, and flow schematics with higher temperature acid gas removal systems have not been addressed in this study and would likely need to be evaluated with experimental data. Of these, the strong interest in using "warm-gas" cleanup systems in the range of 350 to 700°F will be the most important process parameter to address in the future. For that case, a different adsorbent will need to be developed and demonstrated. The study case in this report represents the major portion of the existing and planned gasification plant base.

9. CONCLUSIONS

The cost of mercury removal from an IGCC plant is reduced to very reasonable and acceptable levels by locating the carbon bed in the pressurized syngas stream. This stream is characterized as having a greatly reduced acfm (actual cubic feet per minute) of gas flow relative to that of stack gas by a factor of nearly 200 for the same size plant. Thus, the size of the equipment, the number of beds, and the consequent costs are much less than treating for mercury removal in the stack gas of either a PC plant or an IGCC plant. The estimated cost of $0.254 per MWh and $3,412 per pound of mercury from an IGCC plant can be compared to estimates of costs of mercury removal from PC power plants. For example, in the EPA Mercury Study Report to Congress, the cost for 90 percent mercury removal from a 975 MW utility boiler using carbon beds was $3.10 per MWh and $37,800 per pound of mercury.[3]

The capital cost estimate of $3.34 per kW for removal from the IGCC plant represents less than 0.3 percent of the capital for the total IGCC plant. The small increase in the cost of electricity of $0.254 per MWh due to adding the mercury removal system represents an increase in the overall cost of electricity from the plant of less than 1 percent. These measures of cost are substantially lower than that which might be expected of conventional coal power plant options.

FILE

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PUCO

EXHIBIT NO. _____

BEFORE
THE PUBLIC UTILITIES COMMISSION OF OHIO

In the Matter of the Application of Columbus Southern Power Company and Ohio Power Company for Authority to Recover Costs Associated with the Construction and Ultimate Operation of an Integrated Gasification Combined Cycle Electric Generating Facility	)	Case No. 05-376-EL-UNC

DIRECT TESTIMONY
OF
BRUCE H. BRAINE
ON BEHALF OF
COLUMBUS SOUTHERN POWER COMPANY
AND
OHIO POWER COMPANY

Filed: May 5, 2005

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BRUCE H. BRAINE
CASE NO. 05-376-EL-UNC

Page No.

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2. Purpose of Testimony 2

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BEFORE
THE PUBLIC UTILITIES COMMISSION OF OHIO
DIRECT TESTIMONY OF
BRUCE H. BRAINE
ON BEHALF OF
COLUMBUS SOUTHERN POWER COMPANY
AND
OHIO POWER COMPANY
CASE NO. 05-376-EL-UNC

PERSONAL DATA

Q. What is your name and business address?

A. My name is Bruce H. Braine and my business address is 1 Riverside Plaza, Columbus, Ohio 43215.

Q. By whom are you employed and in what capacity?

A. I am employed by American Electric Power Service Corporation. My title is Vice President – Strategic Policy Analysis.

Q. What are your responsibilities as Vice President – Strategic Policy Analysis?

A. At AEP, I am responsible for the analysis of federal and state energy and environmental policies, as well as analyzing and assisting in the development of long-term environmental, energy and technology strategy for AEP.

Q. What is your educational and professional background?

A. I received my bachelor's degree from Brown University in 1976. I received my master's degree in business administration from Stanford University in 1980 where I concentrated in economics and finance as well as graduating from Stanford's Public Management Program.

Previously at AEP, (1997-2000), I served as senior vice president - analysis for AEP Energy Services. In this role, I directed the analyses of the electricity,

natural gas, coal, and emission markets, as well as AEP investment analyses. Prior to joining AEP in 1997, I spent 17 years as an energy and environmental consultant. I served as a senior vice president at ICF, where I directed ICF's $6 million electric utility business consulting unit. I also was a principal in the Washington, D.C., economic and management consulting firm of Putnam, Hayes and Bartlett.

Q. What are your current professional affiliations outside of AEP?

A. I serve as a member of Electric Power Research Institute's (EPRI) Global Climate Change Committee, on the Board of Directors of the Chicago Climate Exchange, on the Board of Directors of the International Emissions Trading Association (IETA), on the Edison Electric Institute's (EEI) Strategic Issues Committee and EEI Economics and Public Policy Executive Advisory Committee.

PURPOSE OF TESTIMONY

Q. What is the purpose of your testimony in this proceeding?

A. The purpose of my testimony is to sponsor the results of an economic analysis of a new integrated gasification combined cycle (IGCC) generating facility to determine whether this is the most economic option for providing baseload generation in Ohio. The analysis that I have conducted and I am testifying about today is presented in detail in Part IV of a White Paper prepared under my direction as well as the direction of the Companies' witness, Mr. Mudd. (MJM/BHB Exhibit 1).

1 **SUMMARY OF ECONOMIC ANALYSIS**

2 Q. Do you sponsor as part of your testimony Part IV of the White Paper?

3 A. Yes, I do.

4 Q. Please summarize the conclusions of your economic analysis in Part IV of the

5 White Paper.

6 A. In order to determine the relative economics of IGCC technology, I have

7 compared the costs of building a new IGCC plant today with a pulverized coal

8 (PC) plant and a new combined cycle gas plant, which are other viable baseload

9 generation alternatives. While determination of new plant economics often

10 dictates a comparison of the total costs to market, in this instance, there is little to

11 no liquidity in the forward market for power during the relevant time frame

12 (2010-2039). As such market prices obtained from market quotes are generally

13 not available and if available are not particularly meaningful. A sounder

14 approach in this instance is to look at power generation alternatives to IGCC such

15 as pulverized coal and combined cycle gas with the view that market in the long

16 run will be set by the lowest cost alternative to produce power.

17 I have found that IGCC has lower economic costs than PC (pulverized

18 coal) and significantly lower economic costs than NGCC (natural gas combined

19 cycle) ---even though IGCC has higher capital and operating costs today

20 compared to PC or NGCC. In reaching this conclusion. I have not only

21 considered the emission costs of the current environmental regulations (and the

22 new Clean Air Interstate and Mercury rules) and the inherent advantage of IGCC

23 vs PC in this regard, but also have taken into account possible future greenhouse

gas legislation. In this latter case, IGCC has much more significant "option" value than PC coal in that it provides an economically viable alternative for retrofit of carbon capture and sequestration (CCS) systems.

Further, an IGCC power plant is a superior choice for Ohio when considering a number of factors that were not quantified in this assessment. These include fuel flexibility, by-product and product flexibility, as well as furthering the commercialization and lowering the long-run costs of the technology for future IGCC applications.

Q. Does this conclude your testimony?

A. Yes, it does.

BHB/MJM Exhibit 1

Integrated Gasification Combined Cycle Technology

An American Electric Power Service Corporation
White Paper

Prepared by Bruce H. Braine,
Vice President – Strategic Policy & Analysis

and

Michael J. Mudd
Program Manager, Technology Development

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I. Introduction

More than half of the electricity generated in the United States is fueled by coal. And roughly 90 percent of all coal mined in the U.S. goes toward generating electricity. Meanwhile, political instability around the world adds urgency to the goal of a nation and an economy that is domestically powered. The U.S. already produces 25 percent of carbon dioxide in the world, and 82 percent of the greenhouse gases produced in this country come from fossil fuels, including coal. AEP is the largest consumer of coal in the United States. We are in an environmental conundrum – living with coal is a challenge, yet we can't live without it.

Technological innovations are critical to the future of the coal-fired electric industry in the United States. Clean coal technologies must be embraced by the electric utility industry across the country, and at this point in history, Integrated Gasification Combined Cycle (IGCC) technology is the premier clean coal technology. This is a public responsibility AEP takes very seriously. Earlier this week, the Company was honored by the EPA with a 2005 Climate Protection Award for demonstrating ingenuity, leadership and public purpose in its efforts to reduce greenhouse gases.

AEP, as the nation's largest consumer of coal, is committed to leading the industry in the technology destined to become the standard. The Company already has taken a great many strides toward clean coal – through retrofits of emission control technologies on our power plants, and through carbon sequestration efforts across the globe.

According to the EPA's 2003 statistics, Ohio led the nation in NOx and SO_2 emissions, and ranked only behind Texas in CO_2. It is AEP's opinion that taking this first

step into the new era of electric generation using IGCC technology is both fiscally responsible and the right thing to do as a matter of public policy, both for AEP and for Ohio.

No single technology will allow the U.S. electric utility industry to continue producing low cost, reliable electricity. A diverse array of technologies and strategies will ensure the security and sustainability of the U.S. electric grid. But key to any successful strategy will be the expansion of commercial IGCC technology.

With this background in mind, Columbus Southern Power Company and Ohio Power Company, subsidiaries of AEP, filed an application March 18, 2005, with the Public Utilities Commission of Ohio seeking authority to recover costs related to building and operating a new clean-coal technology power plant.

AEP has announced its intent to build up to 1,200 megawatts of new generation using IGCC clean-coal technology, the largest commercial-scale use of the technology for power generation in the United States, and the largest IGCC power project announced to date. AEP has identified properties in Kentucky, Ohio and West Virginia as sites under consideration. IGCC technology represents an advanced form of coal-based generation that offers enhanced environmental performance.

This white paper is being submitted to the Public Utilities Commission of Ohio to discuss the issues surrounding AEP's intention to construct a commercial 600-MW IGCC plant in Meigs County, Ohio. This paper includes three main topics:

- A description of IGCC and other generating technologies,
- Carbon capture and sequestration considerations, and
- The economics of IGCC.

II. Generating Technology Options

In considering options for new investments to meet growing electricity demand and to replace retiring generation capacity, several technologies merit consideration.

Fossil fuels will continue to play a role in our nation and – owing to the company's location near an abundant source of low-cost coal – at AEP. Advanced technologies using coal and natural gas for power generation in an efficient, sustainable manner comprise a key portion of AEP's portfolio of technology options.

Coal-based technologies being considered include pulverized coal (PC) combustion designs; circulating fluidized bed (CFB) combustion designs; and integrated gasification combined cycle (IGCC) designs. PC and CFB systems are options already in widespread use for reliable and affordable baseload electricity production.

IGCC has been proclaimed by some as *the* clean-coal technology. Precluded by natural gas combined cycle (NGCC) operations during the low-price heyday of natural gas, coal-fired IGCC has only recently become the favored option, although the technology has existed for decades.

In addition, NGCC plants, which require the least up-front capital but are vulnerable to gas-price volatility, also must be considered. The evaluation of each of these fossil fuel technologies takes place in the context of current suitability for reduction of carbon and other emissions, as well as their respective potentials for future retrofits to remove carbon emissions.

Non-fossil fuel options include nuclear and renewable energy. New reactor designs and ongoing improvements in safety systems make nuclear power an increasingly viable option as an emission-free power source, but concerns about public acceptance,

waste storage and capital costs continue to temper AEP's interest in new nuclear power. Renewable energy, especially wind and biomass, represents another approach to emission-free power generation, and AEP continues to aggressively pursue cost-effective opportunities in this area.

Finally, distributed resources and energy storage technologies hold potential in complementing new generation options by optimizing the operation of existing electric power infrastructures.

It must be recognized that there are many variables that impact the capital cost of a power plant. Some of those factors are:

- The cost to transport material to the site,
- The impact of ambient temperatures on the design and performance of a power plant,
- The marketplace itself, which can impact the prices of critical commodities including steel and concrete,
- The final design of the plant, which will impact the type of equipment and the cost of the equipment selected,
- The performance requirements such as emission limits and design efficiency, and
- The structure of the contracts, which will impact the risk premium (contingency) included in the cost of the facility.

Integrated Gasification Combined Cycle

The first patent for a gasifier was granted in Germany in 1887. Widespread as a chemical plant technology from that point through the 1950s, IGCC took off as a commercially feasible technology for electricity generation following key studies begun

by the U.S. Department of Energy in 1970. In 1980, Texaco Gasification (now owned by General Electric) was contracted to build the Cool Water pilot plant in southern California. That plant was commissioned in 1984.

The basic IGCC concept was first successfully demonstrated at commercial scale at the Cool Water Project from 1984 to 1989. There are currently two commercial-size, coal-based IGCC plants in the United States and two in Europe. The two U.S. projects were supported initially under the DOE's Clean Coal Technology demonstration program, but are now operating without DOE support. The 262-MW Wabash River IGCC re-powering project in Indiana started up in October 1995 and uses the E-Gas gasification technology (which was acquired by ConocoPhillips in 2003). The 250-MW Tampa Electric Co. Polk Power Station IGCC project in Florida started up in September 1996 and is based on Texaco gasification technology.

The first of the large European IGCC plants was the NUON (formerly SEP/Demkolec) project in Buggenum, the Netherlands, using Shell gasification technology. It began operation in early 1994. The second European project, the 335-MW ELCOGAS project in Puertollano, Spain, uses the Prenflo (Krupp-Uhde) gasification technology and started coal-based operations in early 1998. In 2002, Shell and Krupp-Uhde announced that henceforth their technologies would be merged and marketed as the Shell gasification technology.

The IGCC process employs a gasifier in which coal is partially combusted with oxygen and steam to form what is commonly called "syngas" – primarily a combination of carbon dioxide, carbon monoxide, water vapor and hydrogen. The sulfur in the fuel forms hydrogen sulfide in the gasifier, and the ash is converted to a glassy slag. The

syngas is then cleaned to remove the particulate and sulfur compounds. Mercury can also be removed in a bed of activated carbon. The syngas then is fired in a gas turbine to generate electricity. The hot exhaust from the gas turbine passes to a heat recovery steam generator (HRSG), where it produces steam that drives the steam turbine generator. Power is produced from both the gas and steam turbines. Appendix A provides an illustrated schematic of the process.

Among the three major types of gasifier systems used today, entrained-flow gasifiers have been selected for the majority of IGCC project applications. Gasifiers of this design operate at temperatures above the slagging temperature of the fuel, and as a result, the formation of tars and methane is avoided. Entrained flow designs include the coal/water-slurry-fed processes of GE and ConocoPhillips; and the dry-coal-fed Shell process. A major advantage of the high-temperature entrained-flow gasifiers is that they avoid tar formation and its related problems. The high reaction rate also allows single gasifiers to be built with large gas outputs that are of sufficient size to fuel large commercial gas turbines. AEP believes this technology is capable of achieving the environmental benefits of a natural gas-fired plant, while capitalizing on the relatively low and stable fuel costs associated with coal.

Because gasification operates in a low-oxygen environment (unlike pulverized coal-firing, which is oxygen-rich for combustion), the sulfur in the fuel converts to hydrogen sulfide (H_2S), instead of sulfur dioxide (SO_2). The H_2S can be more easily captured and removed before the fuel is combusted. By reducing the volume of gas to be treated, it is possible to economically remove sulfur at high rates. The amount of waste product is also minimized compared to the sorbent technologies used by PC plants.

1 Removal rates of 99 percent and higher are common using technologies proven in the
2 petrochemical industry. That removal rate is transferable to the electric generation
3 industry. IGCC units also can be configured to operate with very low NOx emissions
4 without the need for Selective Catalytic Reduction (SCR). NOx emissions typically fall
5 in the 15-20 ppmv ranges, just above those from NGCC units, and are similar to those
6 from pulverized coal-fired boilers equipped with low NOx burners and SCR systems.
7 Integrated gasification combined cycle is of particular interest to AEP, in light of
8 the abundance, accessibility, and affordability of high-rank bituminous coals that are
9 abundant in the Midwest. An IGCC plant also is capable of operating on other coal types,
10 such as sub-bituminous coals and lignite coals, as well as other feedstocks such as
11 petroleum coke; although some of the IGCC technologies are better suited for bituminous
12 coals. IGCC also is well positioned for integration of carbon capture and sequestration
13 technologies, which could become a critical approach in mitigating greenhouse gas
14 emissions.

15 The AEP IGCC plant will be designed to burn Eastern bituminous coal, similar to
16 that of the Pittsburgh-8 seam. It also will have the ability to blend petroleum coke with
17 the coal in order to provide the flexibility to take advantage of lower-cost fuel should the
18 opportunity arise. The fuel specifications were established to allow the use of much of
19 Ohio's indigenous coal, providing a significant market potential for Ohio coal. However
20 it is important to note that the Ohio River location of the plant – providing barge access –
21 will allow AEP to obtain the lowest-cost coal available that meets our specifications,
22 whether or not the coal is produced in Ohio. This will result in lower electricity costs to
23 our consumers.

1 AEP entered into an agreement with General Electric and Bechtel in the early part
2 of 2005 to conduct a scoping study for an AEP-specific IGCC plant. This work by GE
3 and Bechtel has been conducted in parallel with their efforts to develop the scope and
4 cost of a standard GE/Bechtel plant. The design proposed by the GE/Bechtel team uses
5 GE's proprietary ChevronTexaco gasifier design.

6 AEP's scoping study provides for a number of technical deliverables. These
7 deliverables will provide the basis for selecting the configuration of the proposed IGCC
8 plant. To facilitate the development of this basic scope definition, a number of studies
9 have considered the internal processes of the plant. This allows AEP to determine those
10 options that offer the best fit to AEP's needs in terms of balancing capital costs and the
11 benefits derived from certain options. Additionally, the scoping study provides for the
12 development of high-level project schedules and an indicative cost estimate.

13 AEP will develop the scope for certain parts of the plant. The portions of scope
14 being developed by AEP include those site-specific items with which AEP is most
15 familiar. These include fuel and material unloading and handling, switchyard and
16 transmission interconnection, river frontage improvements and development. The
17 GE/Bechtel IGCC offering is based on the use of two GE 7FB combustion turbines. Each
18 of these will exhaust into a heat recovery steam generator (HRSG). The steam produced
19 by these HRSGs and from elsewhere in the process will be used to drive a steam turbine.
20 The two gas turbines and the steam turbine will produce a net electrical output of 600
21 MW.

22 One issue with IGCC technology is whether it will have the same or better
23 availability compared to conventional PC plants. Most industry data on the gasifiers

currently in operation point to two significant design features that adversely impact availability: the fuel nozzle and the refractory. The environment under which the fuel nozzle must operate is a zone where the coal, water and oxygen react, but in a high-temperature reducing atmosphere. With the GE systems, the high degree of stress and corrosion on the nozzles historically has meant the nozzles need to be changed every 30-90 days, in a process that requires the gasifier to be shut down. The gasifier vessel is constructed of abrasive-resistant and thermal insulating bricks and cast material. The refractory that lines the inside of the vessel needs to be replaced every 18-24 months. This requires an outage that can take several weeks.

For these reasons, several economic studies indicate improved economics (lower life-cycle cost of electricity) when a spare gasifier is installed to keep the unit running when the above maintenance is performed. This decision to spend the additional $50-$75 million for the spare gasifier will be based on the expected improvement in availability. AEP has not yet decided whether to install the spare gasifier.

Other Factors – Commercialization and Technology Development

IGCC investment also furthers the commercialization of the technology. As such, it moves IGCC further along the technology learning curve, resulting in lower plant costs sooner than would be the case otherwise. The effect is difficult to measure, not to mention the specific share of this effect that is due to the construction of the 600-MW plant in Ohio.

However, most experts generally maintain that base IGCC costs could fall to levels similar to PC over the next decade as commercialization occurs. Thus, the impact

of the first IGCC coal plants (such as the AEP-Ohio plant) could be significant and would result in even greater long-term benefits.

Discussion of Alternative Options

Pulverized Coal

Pulverized coal-fired plants are often considered to be the workhorse of the U.S. electric power generation infrastructure. In a PC plant, the coal is ground into fine particles that are blown into a furnace where combustion takes place. The heat from the coal combustion generates steam to drive a turbine that drives a generator to make electricity. Major byproducts of combustion include sulfur dioxide, nitrogen oxide, carbon dioxide, and ash, as well as various forms of elements in the coal ash, including mercury. Several of the combustion byproducts must be removed from the system before the flue gas leaves the stack.

The steam cycle for the pulverized coal-fired units, which determines the efficiency of the generating unit, falls into one of two categories, *subcritical* and *supercritical*. Subcritical main steam conditions are typically 2,400 psig/1,000°F, with a single reheat to 1,000°F, while supercritical steam cycles typically operate at main steam pressures of 3,600 psig, with 1,050-1,100°F main steam and reheat steam temperatures. Some designs are being developed above 1,100°F, called *ultrasupercritical* cycles, but they still are in the development stage and are not commercially available.

Subcritical PC designs are generally preferred for load-following or cycling operation, where they are used to change their output as the electricity demand fluctuates, since subcritical systems can achieve higher efficiencies during reduced load operation

than comparable supercritical units. The initial capital costs of subcritical units are lower than a comparable supercritical unit by up to about 4-6 percent, but the overall efficiency of the subcritical design is lower than the supercritical design, by about 3-4 percent. Since the supercritical design achieves high efficiency at full load, supercritical units are generally superior choices for baseload operation.

The selection between supercritical versus subcritical design still depends on many other site-specific factors including fuel cost, emission control requirements, capital cost, load factor, and expected reliability and availability. AEP has recognized the benefits of the supercritical design for many years. All 18 of the units in the AEP East system built since 1964 have used the supercritical design.

Circulating Fluidized Bed

A Circulating Fluidized Bed (CFB) plant is similar to a PC plant except that the coal is crushed rather than pulverized, and the coal is combusted in a reaction chamber rather than the furnace of a PC boiler. Because CFB boilers are generally more suited for low-rank high-ash coals such as the lignite coals common in the western states, the economics of CFB boilers is not included in this paper.

Natural Gas Combined Cycle

An NGCC plant combines a steam cycle and a gas cycle to produce power. Hot gases (~1,100°F) from a combustion turbine exhaust pass through a heat recovery steam generator, where they are cooled to about 250°F and produce steam as a result. The steam

drives a turbine generator that produces about one-third of the power, with the combustion turbine producing the other two-thirds.

The main features of the NGCC plant are high reliability, lower capital costs, excellent operating efficiency, low emission levels, and shorter construction period than coal-based plants. In the past 8-10 years, NGCC plants were the most widely selected to meet new intermediate and baseload needs due to these features and very favorable natural gas prices. However, as gas prices have risen the cost of electricity from NGCC plants has been very high, and attention has been re-directed to coal-fired alternatives for baseload generation.

III. Carbon Capture and Sequestration Impact on Generation Technologies

Carbon capture technologies available in IGCC facilities are critical factors supporting reliance on IGCC. In its own right, IGCC technology is superior in terms of emissions mitigation. But today's political and natural environments all indicate the high likelihood of future carbon capture requirements legislated by federal laws or regulations, and possibly additional state requirements as well. And it is in this area that, absent revolutionary improvements in technology, IGCC leaves the other technologies far behind.

Reducing carbon dioxide emissions from a fossil-fuel technology can be accomplished in three ways: reducing the carbon content of the fuel, removing the carbon dioxide from the flue gas or increasing generating efficiency.

Reducing the carbon content of fuel can be accomplished by either switching from coal to natural gas (since natural gas has approximately 20 percent less carbon than coal, and correspondingly greater hydrogen content), or by removing the carbon from the relatively low volume of synthetic gas before it is combusted, as would be the case for CO_2 removal in an IGCC system.

Removing the CO_2 from the flue gas is a very expensive process. Currently, the most likely technology to be used to "scrub" the CO_2 from the flue gas would be by using a monoethanolamine (MEA) or methyldiethanolamine (MDEA) absorption process. This process has a high capital cost (approximately $800/kW to $1,000/kW) and a high efficiency penalty of more than 30 percent.

Increasing the generating efficiency of a coal-based plant has its practical limitations. Efficiency improvements will not result in significant CO_2 emission reductions.

Studies have indicated that the energy penalty for CO_2 removal from an IGCC system would likely be in the order of a 20 percent efficiency penalty, and a 35 percent capital cost penalty. Other technologies are being developed for carbon capture, however these technologies remain in the early stages of development. Significant breakthroughs will be required before we can see the benefit of many of these other innovative technologies for the capture of CO_2.

Comparison of Technology Costs and Technical Parameters

Tables 1a and 1b below compare the capital costs and technical parameters of IGCC, PC and NGCC units. (Because CFB combustion technology is generally not deemed suitable for Eastern bituminous coals, it is not an appropriate consideration for this region of the country. Therefore, it is not considered in any of the data provided by AEP in relation to this project.)

Table 1a
Comparisons of IGCC, PC and NGCC
Without CO_2 Capture

Technology	IGCC	PC	NGCC
Net MW	600	600	600
Heat Rate Btu/kWh	8,700	8,690	7,200
Total Plant Cost, $/kW	1,550	1,290	440

Table 1b
Comparisons of IGCC, PC and NGCC
With CO_2 Capture

Technology	IGCC	PC
Net MW	530	460
Heat Rate Btu/kWh	10,700	11,300
Total Plant Cost, $/kW	1,950	2,150

IV. Economic Analysis of IGCC Coal Compared to Power Generation Alternatives

A central tenet of the economic evaluation of new power plant investment typically is a comparison of the total cost of electricity to the market price for electricity. There is little to no liquidity in the forward market for power in the year 2010 and after. As such, market prices obtained from market quotes are generally not available, and where available, are not particularly meaningful. Therefore, the cost of new generation is the proxy for the market.

A sounder approach, therefore, in this instance is to look at power generation alternatives to IGCC, such as pulverized coal and NGCC, with the view that market will be set by the lowest cost alternative to produce power in the long run. This is a reasonable assessment since, in the long run, power plants will not be built to meet demand unless prices provide a return on investment and cover the costs of building and operating the plant over time.

Further, the Company believes that by 2010, the present oversupply of generating capacity will be brought into balance with growing demand in the AEP-East region. This supply/demand balance should result in prices being set approximately by long run costs of generating power after 2010.

Comparative Economics of Coal Technologies, Including Air Emission Costs

Therefore, determining the relative economics of IGCC requires a comparison with the alternative least-cost baseload generating options.

Table 2 below shows the cost comparison of IGCC levelized costs compared to PC and NGCC in Ohio. While IGCC today is somewhat higher in levelized cost than PC ($56.2/MWh vs. $52.2/MWh), it is significantly lower in cost than NGCC, based on AEP's forecasts of natural gas costs. The levelized busbar costs not only include levelized capital, O&M and fuel, but also include the impact of new air emission regulations on the costs of the technologies.

Table 2
Comparative Economics of New Generation Options
(In Levelized Nominal Dollars, Beginning in 2010)

	IGCC*	PC**	Gas CC***	IGCC w/ CCS****	PC w/ CCS*****
Total Plant Cost ($/kW)	1,550	1,290	440	1,950	2150
Capital – Levelized ($/MWh)	31.7	26.4	8.2	45.1	57.3
Total Levelized O&M ($/MWh)	9.1	8.9	3.5	16.6	19.9
Levelized Fuel ($/mmBtu)	1.61	1.61	7.34	1.61	1.61
Levelized Fuel ($/MWh)	14	14	52.8	17.2	18.2
Levelized Emission Cost ($/MWh)	1.5	2.9	.1	.6	.6
Total Levelized Cost ($/MWh)	56.2	52.2	64.7	79.4	95.9

* IGCC: Integrated gasification combined cycle
** PC: Pulverized coal
*** Gas CC: Gas combined cycle
**** IGCC w/CCS: IGCC with carbon capture/sequestration
***** PC w/CCS: PC with carbon capture/sequestration

Under EPA's recently promulgated Clean Air Interstate Rule (CAIR) and the Clean Air Mercury Rule (CAMR), which cover SO_2, NOx and mercury emissions, most fossil fuel power plants will be subject to a cap on their overall annual emissions of SO_2,

NOx and mercury, with emissions trading permitted. Therefore, additional emissions costs resulting from plant operations also are included in the levelized cost calculations. Because the IGCC plant's SO_2 and mercury emissions are generally lower than the PC plant's, its emissions costs are also lower. This narrows the cost difference between today's technologies. Emission costs reflect market SO_2 values and estimated values for the annual NOx and mercury markets.

Other Economic Factors That Could Lower IGCC Costs

In addition to the economic analysis above, there are other factors that could lower the relative costs of IGCC and improve the differential between IGCC and PC. These factors have not been quantified in the analysis above but should be considered in the overall assessment of IGCC:

- **Fuel flexibility:** IGCC provides some advantages over PC with regard to fuel flexibility. For example, it is possible that petroleum coke may be produced in Ohio in the not-too-distant future. Blends of petroleum coke would lower overall fuel costs and could be easier to use in an IGCC plant than in a PC plant.
- **Marketable by-products:** IGCC can produce marketable by-products when the coal is gasified, such as sulfur or sulfuric acid and slag. These potential by-products were not included in the economics of IGCC.
- **Product flexibility:** Owing to the IGCC plant's low variable costs, AEP anticipates operating its IGCC plant whenever it is available to meet electricity demand. However, it is possible during some periods when demand and prices both are low that the plant may not be called upon to produce power in PJM. AEP is currently

studying whether production of other marketable products such as methanol and diesel during idle generation periods might enhance the overall economics of the plant.

Long-term Economics Including CO_2 Option Value

IGCC has long run costs similar to PC, when taking into account potential costs associated with possible future greenhouse gas legislation. Because an IGCC plant provides AEP with the option to capture and sequester carbon, an IGCC plant has an inherent "option" value compared to PC or NGCC, where these costs are prohibitive. While an option also exists to potentially capture and sequester carbon from a PC plant, its value is considerably lower in a PC plant, owing to its very high costs within that technological framework.

While the prospects of passage of greenhouse gas legislation in the United States are not imminent in the next four years, there is a greater likelihood that passage will occur after that time. If and when there is legislation, it is likely to include some form of greenhouse gas/carbon dioxide constraints or cap, with emissions trading permitted. Thus, similar to SO_2 and NOx, as well as mercury in the future, there is likely to be a market for CO_2 emission allowances and a value associated with CO_2 emission reductions or offsets at power plants, as well as other sources of greenhouse gases.

This analysis used a range of carbon dioxide allowance prices reflecting the potential stringency and timing of possible future legislation. Using these prices and the costs of the new generating technology options, we have conducted a probabilistic decision analysis.

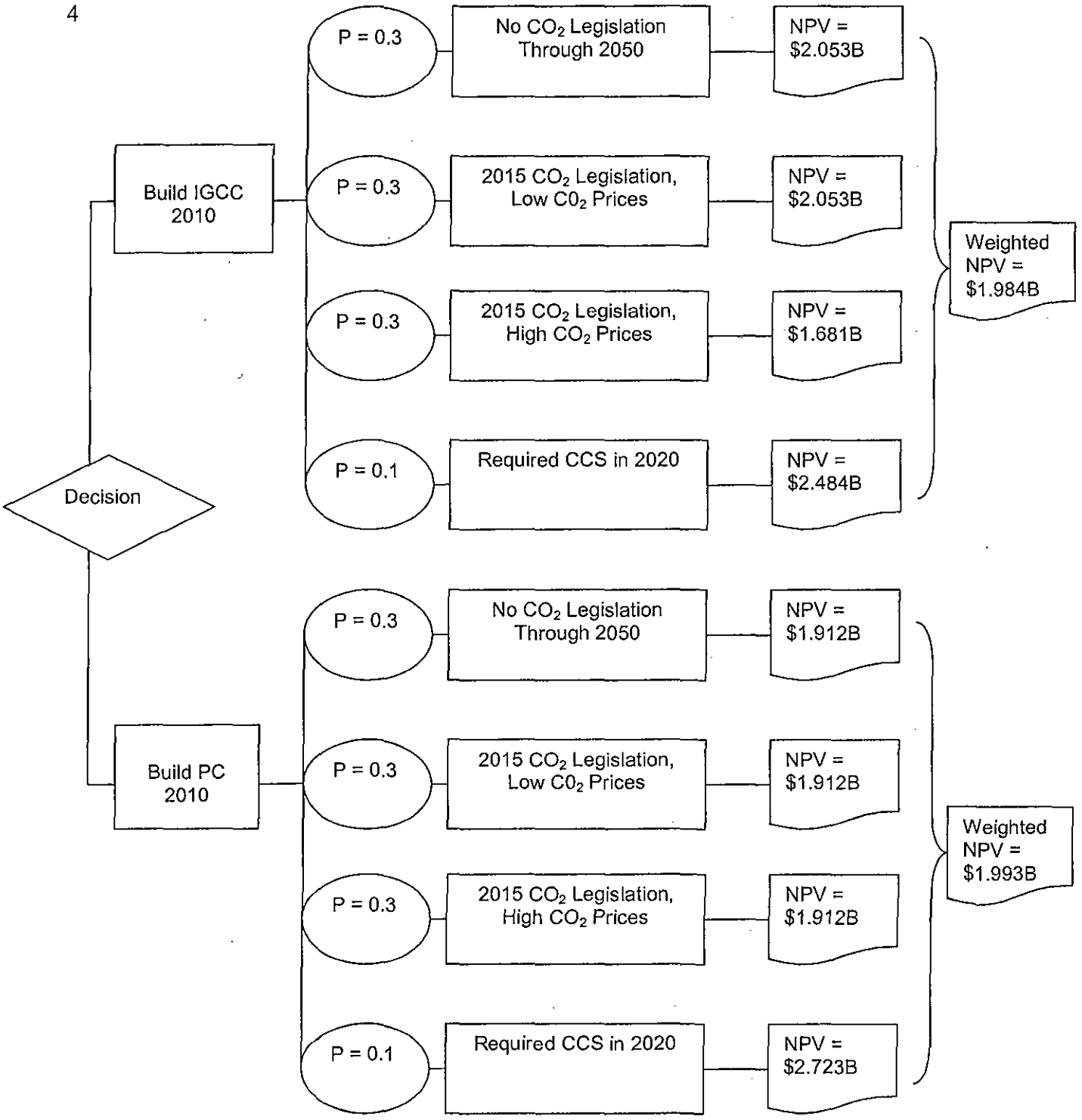
We have assumed three potential future states of the world with equal probabilities (30 percent each) and one additional very stringent scenario (with a 10 percent probability.)

1. No CO_2 legislation,
2. CO_2 legislation with low carbon prices, and
3. CO_2 legislation with high carbon prices.
4. CO_2 legislation requiring carbon capture and sequestration on all new coal plants in 2010 or later by 2020.

The results of the evaluation, shown in Fig. 1, indicate that the long run costs (in net present value [NPV] terms) of an IGCC power plant are similar to those of a PC plant.

Thus while today's costs and today's environmental requirements yield higher costs for an IGCC plant than a PC plant, factoring in the option value of carbon capture and sequestration if the IGCC plant is built would result in the net costs of the IGCC being similar to a pulverized coal unit. Fig. 1 shows the calculation of the net present value costs under the alternative future scenarios. This simple analysis indicates that if future climate change legislation is factored into the analysis, IGCC is a more economic choice than PC by $9 million in NPV terms.

Fig. 1
Effect of Potential CO_2 Costs on NPV Cost of New Power Plants



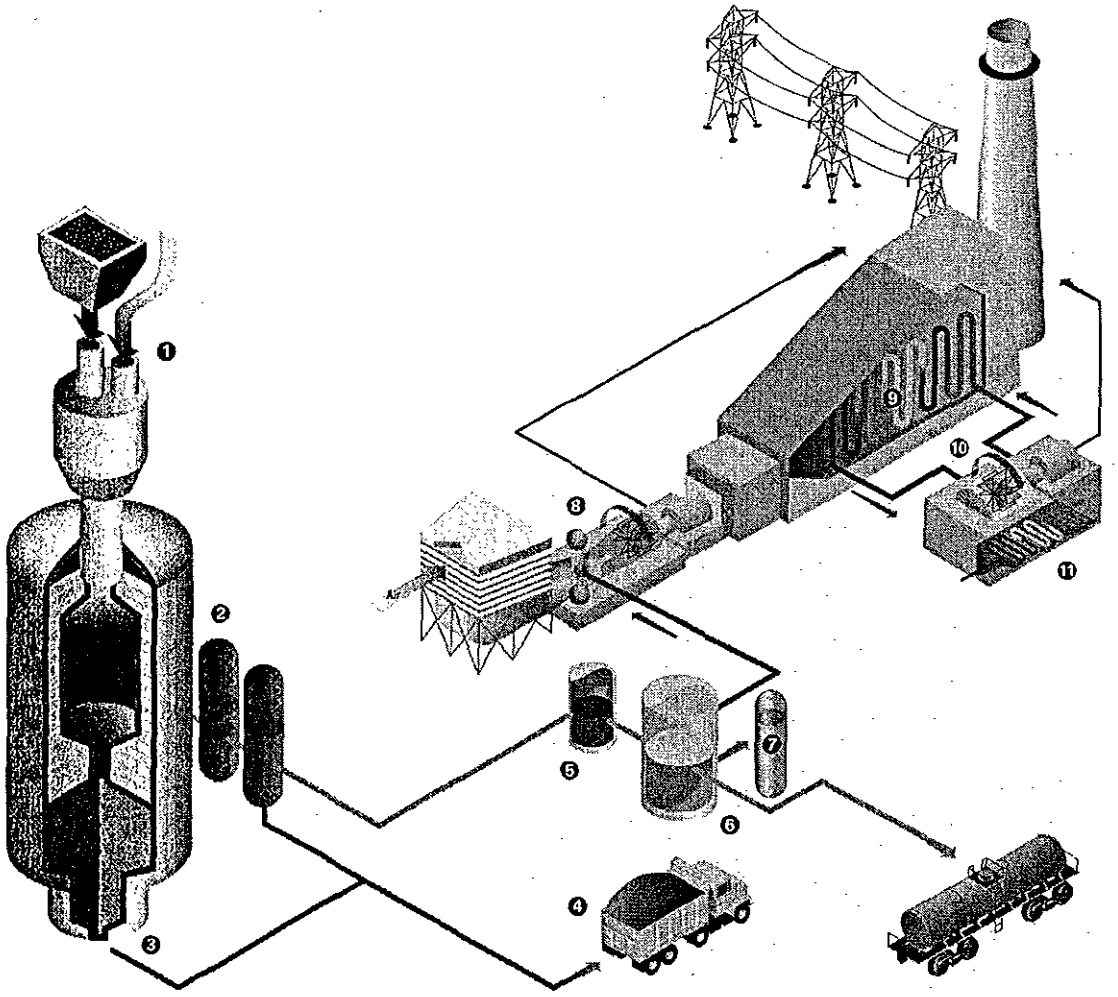
Economic Summary

When considering only current environmental regulations, the cost of electricity for IGCC is somewhat more expensive than PC and less expensive than NGCC. However, when the CO_2 option value of IGCC and PC are considered, IGCC and PC have very similar economics.

Further, an IGCC power plant is a superior choice for Ohio when considering a number of other factors that were not quantified in the assessment. These include fuel flexibility, by-products and product flexibility, as well as furthering the commercialization and lowering the long run costs of the technology for future IGCC applications.

Appendix A

IGCC Process Schematic



Legend:

1. Coal, water and oxygen are fed into a high-pressure gasifier, where the coal is partially combusted and converted into syngas.

2. The ash in the coal is converted to inert, glassy slag.

3. The syngas produced in the gasifier is cooled and cleaned of particles.

4. The slag and other inert material may be used to produce other products or may be safely managed in a landfill.

5. Next, the syngas passed through a bed of activated charcoal, which captures the mercury.

6. The sulfur is removed from the syngas and converted to either elemental sulfur or sulfuric acid for sale to chemical companies or fertilizer companies.

7. The syngas can either be burned in a combustion turbine or used as a feedback for other marketable chemical products.

8. The syngas is fired in a combustion turbine that produced electricity.

9. The hot exhaust from the gas turbine passes to a Heat Recovery Steam Generator (HRSG).

10. Steam produced in the HRSG, along with additional steam that has been generated throughout the process, drives a steam turbine, which also produces electricity.

11. The steam from the turbine cools and then condenses back into water, which is then pumped back into the steam generation cycle.

EASTMAN CHEMICAL COMPANY

KINGSPORT PLANT

CHEMICALS FROM COAL OPERATIONS

1983 – 2000

Presented at the

2000 Gasification Technologies Conference

San Francisco, California

October 8 – 11, 2000

Presented by

R. G. Smith, Gasification Department, Eastman Chemical Company

ABSTRACT:

The Chemicals from Coal Facility of Eastman Chemical Company began operation in 1983 using various purchased technologies such as Texaco gasification and Linde AG Rectisol gas clean-up as well as Eastman developed technologies for chemical production. Initially, the plant was designed to produce approximately 500 million pounds per year of acetic anhydride and acetic acid to supply half of Eastman's acetyl raw material needs. The facility was expanded in 1991 and additional debottlenecking brought the capacity to the current level of approximately 1.14 billion pounds per year. Two 450 cubic foot gasifiers are campaigned to give gasifier system uptimes in excess of 98% with a maximum rate of approximately 1350 tons per day of coal. The gasifiers are operated at 150% the original design basis. The facility is now the sole source of raw materials for Eastman's profitable acetyl stream and is operationally and economically a proven means of producing acetyl chemicals from coal.

INTRODUCTION

This paper reviews the progress of the Chemicals from Coal Facility of Eastman Chemical Company. Eastman Chemical Company has nearly $5 billion in annual sales and currently has approximately 15,000 people working to produce over 400 different chemicals, fibers, and plastics. The corporate headquarters are located in Kingsport, Tennessee also the site of Eastman's largest manufacturing unit, Tennessee Eastman Division.

The Chemicals from Coal Facility is also located in Kingsport on a 55-acre site adjoining Eastman's existing chemical complex. The facility began operation in 1983 after four years of engineering and construction and more than eight years of work to identify, develop, and assemble the technologies necessary to make the operation viable.

The Chemicals from Coal Facility was the first use of a commercial Texaco coal gasifier to provide feed gas for the production of acetyl chemicals. In addition to the first Texaco coal gasifier, the project was the first use of new technologies developed by Eastman to produce methyl acetate and the final product acetic anhydride.

For this significant advancement in acetyl chemicals production, in 1985, Tennessee Eastman was awarded the prestigious Chemical Engineering Kirkpatrick Award.

In November 1995, the American Chemical Society recognized the Chemicals from Coal Facility as a National Historic Chemical Landmark.

Acetyl chemicals are an important part of Eastman Chemical Company's overall portfolio of chemicals, fibers, and plastics, but they are particularly important to Tennessee Eastman Division. Five of the seven manufacturing divisions at the Kingsport site depend on acetyl raw materials. Approximately 3.2 million pounds per day of "new" acetic anhydride and acetic acid are produced in the Chemicals from Coal Facility and are used in the production of cellulosic plastics and fibers that end up in consumer products like photographic film, tool handles, paints, and cigarette filters. Direct sales of anhydride and acid are used in a wide variety of industrial and pharmaceutical applications (Figure 1).

HISTORY

Prior to the completion of the Chemicals from Coal complex, all of Eastman's acetyl raw materials were petroleum and natural gas based. Ethane, propane, and naptha were cracked to form propylene and ethylene. The ethylene was converted to acetaldehyde at Texas Eastman Division. The acetaldehyde was transported to Kingsport and further oxidized to acetic acid and then converted to acetic anhydride via natural gas fired, ketene cracking furnaces.

The oil shortages of the 1970's and specifically the oil embargo of 1973 provided the incentive for Eastman to begin to explore the possibility of supplying the acetyl stream from coal mined in nearby Southwest Virginia and Eastern Kentucky, instead of oil.

Eastman engineers along with Bechtel as the contractor began to identify existing technologies as well as developing new ones to make the dream a reality. The first gasifier start-up occurred on June 19, 1983 and by April of 1984, the entire complex was in full production. The original capacity of the plant was needed to supply one half of Eastman's acetyl demand. After several years of successful operation, the decision was made to expand the facilities to meet the entire acetyl demand from coal (Figure 2).

This expansion was completed in 1991 and was accomplished by building essentially duplicate downstream chemical plants and clean-up equipment. However, the gasification plant was not significantly expanded. Increased rates through the gasifiers were achieved by debottlenecking key parts of the gasification process. In addition, the percentage of raw gas processed in the shift reactor to make methanol synthesis gas was reduced and diverted to the higher value acetic anhydride production. In this way, the acetic anhydride production was doubled without a two-fold increase in raw gas produced. However, approximately 25% of the internal methanol demand is now met via outside purchases.

After this "Phase II" expansion, the gasifiers ran at about 128% of the original design capacity. Today, through years of continual debottlenecking, the gasifiers run at a maximum of 150% of the original design (approximately 1300 tons per day of coal).

Table 1 is a chronological listing of the historical milestones of the complex.

DESCRIPTION OF PLANT

The Chemicals-from-Coal complex is divided into two major operating areas – synthesis gas production and chemicals production. There were nine plants in the original complex. Four more plants were added in the Phase II expansion.

The synthesis gas production area contains the coal handling/slurry preparation plant, the gasification plants, the Rectisol gas clean-up plant, the CO/H2 separation plant, and the sulfur recovery plant. Oxygen is supplied by Air Products and Chemicals, Inc., from an air separation plant adjacent to the Eastman plant.

Oxygen and coal are supplied to either of two 450 cubic foot Texaco quench gasifiers (Figure 3). Raw gases produced are split into two process streams. About one third of the raw gas is routed to the shift reactor to produce enough hydrogen to make the correct stoichiometric composition for methanol production. Both gas streams are cooled in the cooling trains and sent to the Rectisol plant. Steam, at a rate of approximately 200,000 pounds per hour, is produced in the cooling trains and utilized in downstream processes.

Carbon dioxide and hydrogen sulfide are removed from the gas streams in chilled methanol absorbers (Figure 4). The hydrogen sulfide is sent to a Claus/SCOT recovery unit for recovery of sulfur. Carbon Dioxide is vented to the atmosphere. The process gas stream is sent to the CO/H2 cryogenic separation plants to produce the carbon monoxide used in the acetic anhydride plant.

The CO/H2 gas mixtures received from the cryogenic separation units are mixed with the gas stream from the shifted gas train and sent to the methanol plants.

The chemicals production area contains the methanol plants, methyl acetate plants, acetic anhydride plants, and catalyst recovery plants. Methanol is produced in a Lurgi unit as well as a new "Liquid Phase" unit, which is a joint venture between Eastman, Air Products and Chemicals Inc., and the Department of Energy. The methanol is reacted with acetic acid to produce methyl acetate in a proprietary Eastman process. Methyl acetate is reacted with carbon monoxide in a proprietary catalytic process to produce acetic anhydride with co-production of acetic acid.

KEY OPERATIONAL ISSUES

Production rate, reliability, maintenance cost, and safety are of primary importance to the operation of the Chemicals-from-Coal facility. Of course there are many other important areas to consider, but the success in these four areas is key to the acetyl business.

Production Rate

Because the acetyl stream is such an important part of Eastman's overall business and the demand for these products is very high, essentially every pound produced can be sold in the market place. So naturally, the pressure to increase production rate is always present. Also, the incremental cost to make additional product is much lower than the fully allocated cost, so the profit margins for incremental production are large. By debottlenecking key areas of the gasification process, the production rate has been increased from 128% of original design (basis for the Phase II expansion) to recent levels as high as 150% of the original design. Figure 5 gives the total gas production for the last several years. The steady trend upward indicates that the accomplishments that have been made by continual debottlenecking.

The challenge for the future will be to continue this trend. However, this will be increasingly difficult as we begin to reach bottlenecks in all the plants simultaneously.

Reliability

Perhaps more important than production rate is reliability. Outages of the gasification complex result in downtime in the chemicals production areas, and depending on the duration, can drastically affect production in the majority of the operating divisions at Tennessee Eastman. The primary measure of reliability is percent uptime (or gas availability to the chemical plants). Since 1986, the gasifier uptime (not including planned complex shutdowns every two years) has consistently been above 98% (Figure 6). To achieve this high level of reliability, a two-gasifier design is key. Each time a gasifier is taken off-line, it is "turned around" (i.e., made ready for the next run) typically within 7 to 14 days. Then, if a problem develops on the operating gasifier which doesn't cause immediate shutdown, the spare gasifier can be started and put online without interruption of the gas supply to the downstream plants.

There are a number of operating and maintenance problems that can cause a gasifier to shutdown. See Figures 7 and 8 for historical and recent shutdown causes. In past years, the fuel injector (often referred to as “burner”) has been the primary cause for shutdown. However, Eastman experts have had recent breakthroughs in feed injector design, which have effectively prolonged the life of a burner far beyond the run life of other system components. At the time of this writing, the run length is 75 days and counting.

It appears that the next run length limit will be due to plugging in the quench and black water systems, specifically the steam generators. Projects have been identified to alleviate this bottleneck.

Key to this continual improvement is a “run review” held after each shutdown. Essentially, experts perform an autopsy of the run, identify key problems, analyze critical data, and generate projects to improve the process performance.

Maintenance Cost

Of the operating cost factors, maintenance cost is the highest controllable cost. However, due to reliability, capacity, and safety demands, there has always been a struggle to find the right level of maintenance support. The Chemicals from Coal facility has historically been supported by around the clock maintenance including 10 to 12 mechanical, electrical, and instrumentation mechanics and an expanded day shift crew. However, Eastman has recently cut back on the level of off-hour support in an effort to reduce overall maintenance costs. Eastman has adopted a philosophy of performing a higher percentage of “planned maintenance” and continually assessing the maintenance need while working to identify and implement reliability projects to take work out of the system.

As a result, turnaround times have increased but because of the lower number of failures (a product of reliability projects) 2000 has been the most productive year on record. Additionally, maintenance costs have been reduced by 10 – 15%.

One of the most expensive items in the annual maintenance budget is refractory replacement. Refractory wear is obviously related to temperature and slag properties, but our experience has shown that it is also proportional to gasifier rate. Even though the refractories have improved over the years, the run time per liner has gone down due to the increased throughput. However, the amount of production per liner has remained relatively constant. Currently, the approximate run time per liner is 7,000 hours. Due to the cooler operating temperatures allowed by some of the newer coals, the refractory life is expected to improve in the future.

Safety

Eastman Chemical Company is committed to providing a safe work environment for its employees. Considering the potential hazards that exist in the complex (high pressure, toxic gases, rotating equipment, high level of maintenance activity, extreme high and low

temperatures), this is a considerable challenge and is always a consideration in everything we do. The past safety record has been excellent. Currently, the Acid Division, including the Gasification Complex, has an OSHA recordable rate less than 1.0.

The gasifier system, as well as other critical downstream processes, is equipped with a well instrumented safety system that will automatically shut off the feed if any unsafe conditions are detected. In order to maintain the safety of the operation without having false shutdowns, redundant instrumentation is typically used.

Area on-line CO and H2S monitors are located throughout the operating area to warn of leaks that could create hazardous conditions. In addition, personnel entering the plants wear portable personal CO and H2S detectors equipped with alarms.

MAJOR PROCESS IMPROVEMENTS AND ENHANCEMENTS

Over the seventeen years that the complex has been in operation, many company resources have been poured into efforts to improve the process. The improvements indicated by the previous charts do not tell the full story. Some of the major process improvements and enhancements are discussed briefly below.

Burner Design Improvements

The major cause of shutdown has historically been failure of the feed injector. High temperature sulfidation corrosion has been blamed for the failures. Recent breakthroughs in injector design combined with rigorous inspection and checkout of various feed system components have resulted in greatly improved fuel injector life. Eastman Chemical Company has received 6 patents dealing with fuel injector improvements and is continuing to develop know-how in this important area of process reliability. The most recent campaign utilized a gasifier that operated continuously for 77 days on the same feed injector (Figure 9). This is more than twice the historical average run life. It is expected that runs in excess of 100 days will be achieved.

Complex Shutdown Planning and Execution

The entire Chemicals-from-Coal complex is shutdown for maintenance once every two years. During the 1999 shutdown, approximately 560 jobs were completed on the synthesis gas areas alone. Through careful planning and execution, these outages have been minimized to approximately 9 days in duration. The industry standard for acetyl plants for such major turnarounds is usually 2 to 3 times this amount of time and typically does not include the complexity of a Chemicals from Coal process.

Alternate Feedstocks

Eastman has successfully operated gasifiers feeding petroleum coke, numerous liquid organics, and a wide variety of coals. Sulfurless startups are accomplished using liquid organic feedstocks. A unique system for qualifying coals for use has been developed. In-house slag viscosity testing has been key to this process. As a result, Eastman has been able to select better performing, lower cost coals from the numerous varieties of coals available. Coals allowing more product gas per ton, lower ash fusion temperatures, and forgiving slag viscosity profiles have been identified and are currently in use.

State of the Art Distributive Control Systems

The gasification complex has been retrofitted with a state of the art Honeywell distributive control system (GUS). This system has allowed complex algorithms to be added to the feed controls system as well as other systems in the plant. Additionally, Eastman's Controls Technology group has developed and implemented Model Predictive Control (MPC) techniques to fully utilize the plant capacity and maximize gas production without operator intervention.

Switching Gasifiers

When it is necessary to switch from one gasifier to another and a complete shutdown is not required, gas flow from the "new" gasifier is valved into the cooing train while the gas from the "old" gasifier is valved out. This has historically been a slow, careful process taking a few hours. A "quick switch" procedure has been developed which allows this transition in less than one hour. This improved process also minimizes impact to downstream plants allowing production to continue and reduces the waste gas produced during switches. Figure 10 shows the number of business interruptions (loss of gas supply to downstream plants) since startup. When compared to the number of gasifier shutdowns, Figure 9, the benefit of skilled "switching" techniques is evident.

SUMMARY AND CONCLUSIONS

The Chemicals from Coal Facility continues to be a commercially viable operation for the production of acetyl chemicals from coal. A testament to its reliability and cost efficiency is Eastman's reliance on this facility as the sole source of raw materials for one of the largest streams within the company (in terms of earnings and pounds). Needless to say, Eastman Chemical Company's success depends to a great degree on the profitability of the Gasification complex.

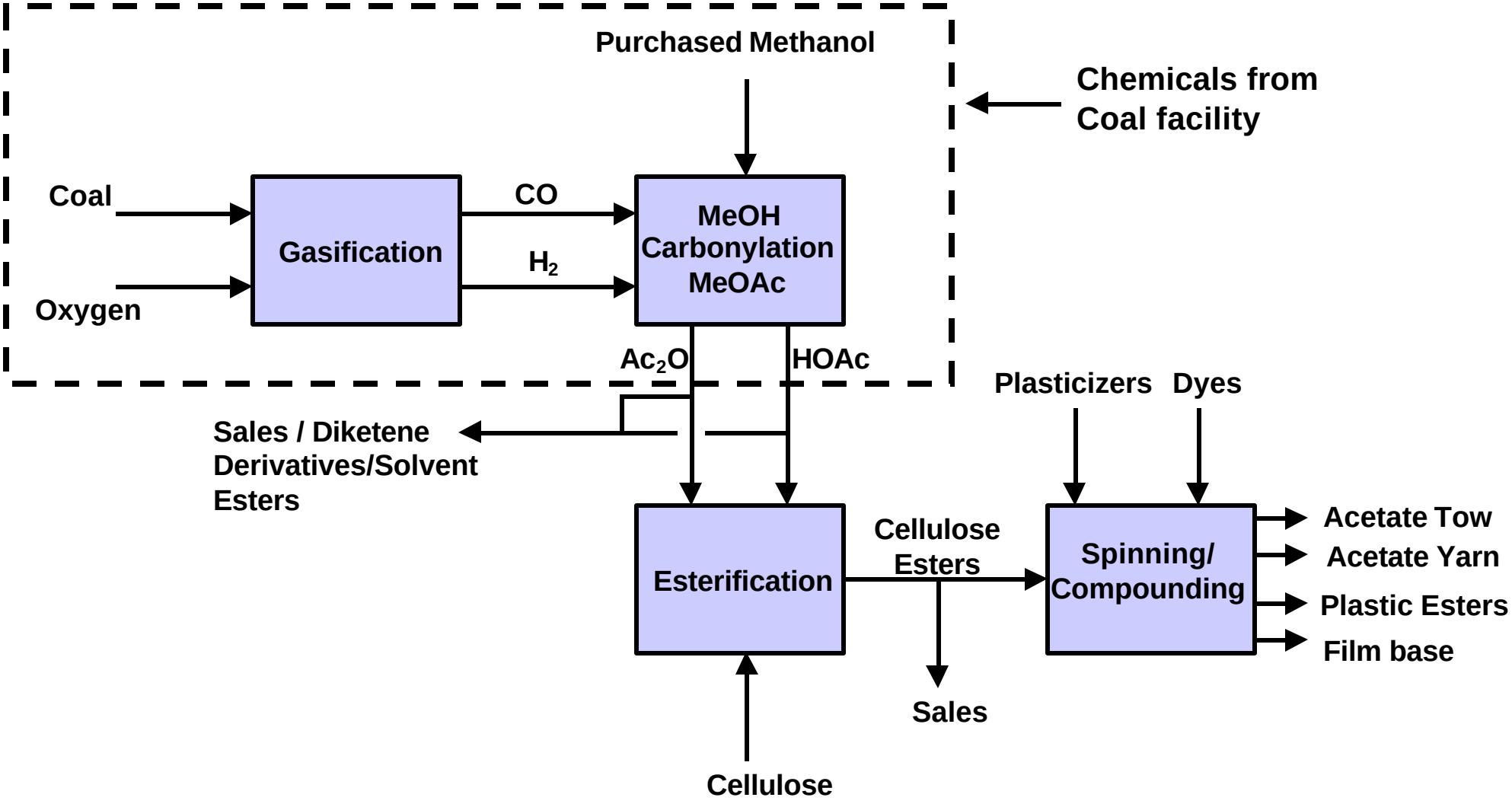
The operation (particularly the gasification process) is maintenance intensive, but can be managed to provide the proper balance between cost and reliability. Eastman has, through years of work, greatly improved the reliability, production, and thus the profitability of this process.

Eastman's experience and expertise in Gasification and Chemical Synthesis technology have made the Chemicals-from-Coal Complex a world class operation.

References:

1. L. T. Arms, “Eastman Chemical Company Gasification Plant Expansion and Operation Summary,” Paper presented at the Eleventh Worldwide Texaco Licensee Symposium, New York, NY (October 1994).
2. T. L. Mitchell, W. L. Trapp, “Eastman Chemical Kingsport Plant – Chemicals form Coal Operations 1983-1997,” (October 1997)

Figure 1: Acetyl Flow at Tennessee Eastman Division



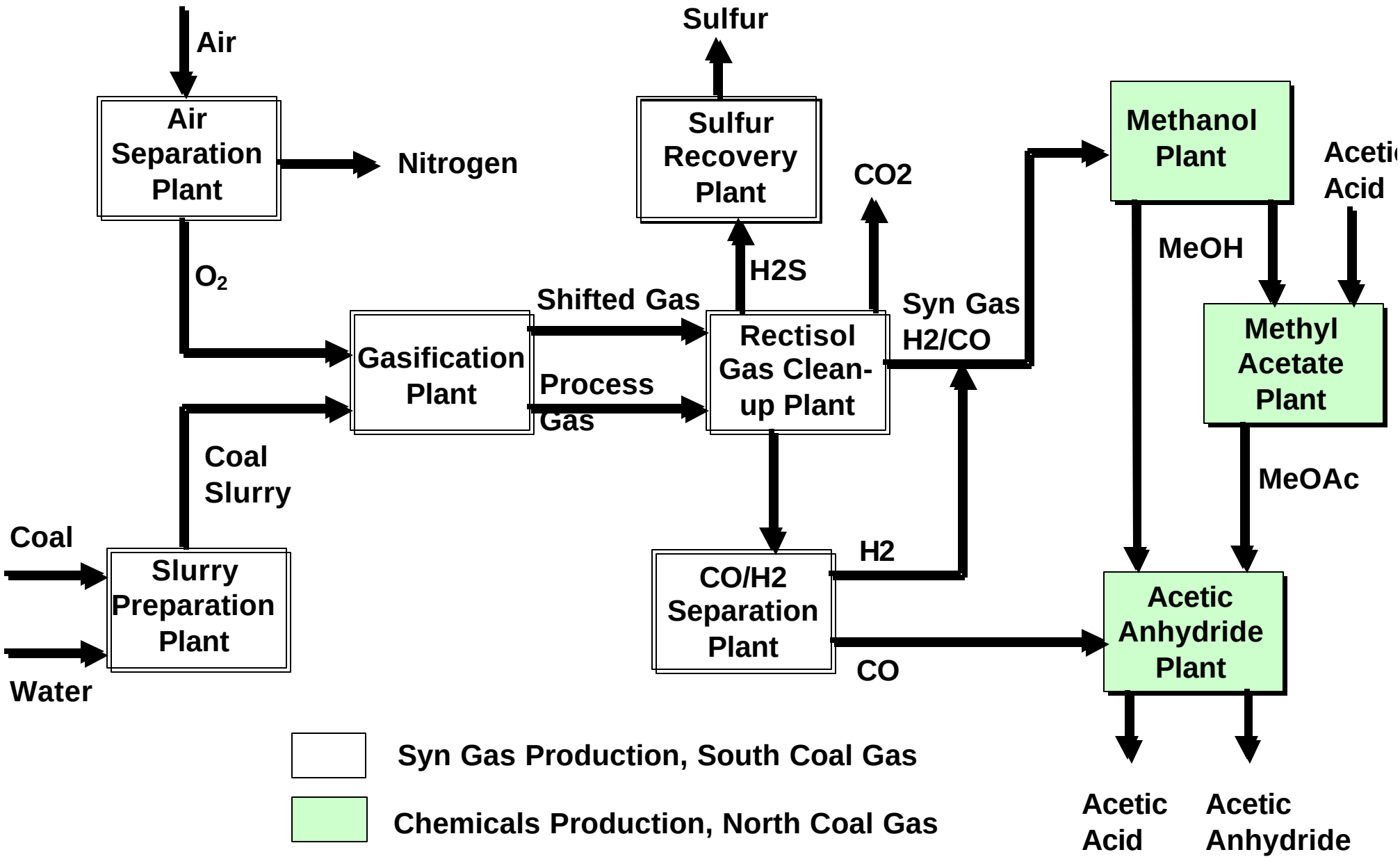


Figure 2: Chemicals from Coal - Block Flow

Table 1

History of the Chemicals from Coal Facility

1970 Eastman scientists conducted a prescient planning study for chemical feedstocks.

1973 Oil embargo: Oil prices increase (acetic anhydride produced from oil based chemicals).

1975 Eastman started R&D on the use of coal based anhydride process.

1977 First small pilot plants began operation.

1978 Bechtel Corporation awarded contract for EPCM for the Chemicals from Coal Facility

1983 Construction Complete

March	Coal grinding and slurry operation
May	Methyl Acetate plant startup
June 19	Startup of Gasifier #1
July 19	Methanol Production started
Aug 12	Startup of Gasifier #2
Oct 6	First production of acetic acid

Oct 83 – July 84 Gasifiers onstream 85% of the time and acetic anhydride operated 75% of the time

1984 July – 2 week shutdown to install improvements necessary for full capacity
July – December – Gasification plant on stream 97% while acetic anhydride plant on stream 95.5%

1988 Bechtel started the EPC work to double the acetic anhydride production

1991 Construction of the Phase II expansion completed (1.2 billion pounds per year production; 1200 tpd coal)

2000 Rates reach 150% of original design; Gasifier run lengths exceed 75 days. (1300 tpd coal -> chemicals)

Figure 3: Simplified Flow Diagram - Gasification

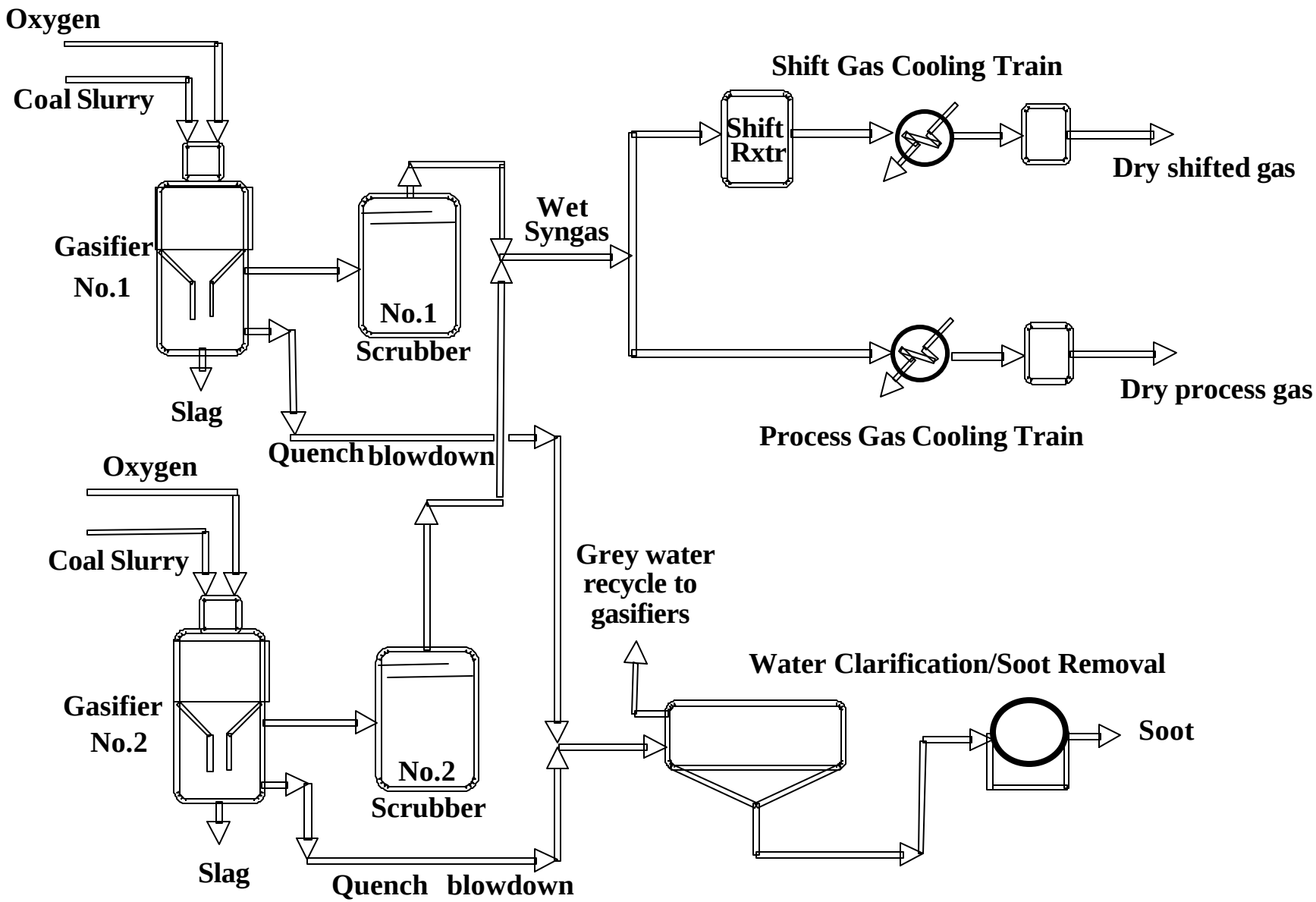


Figure 4: Gas Clean-up, Recovery

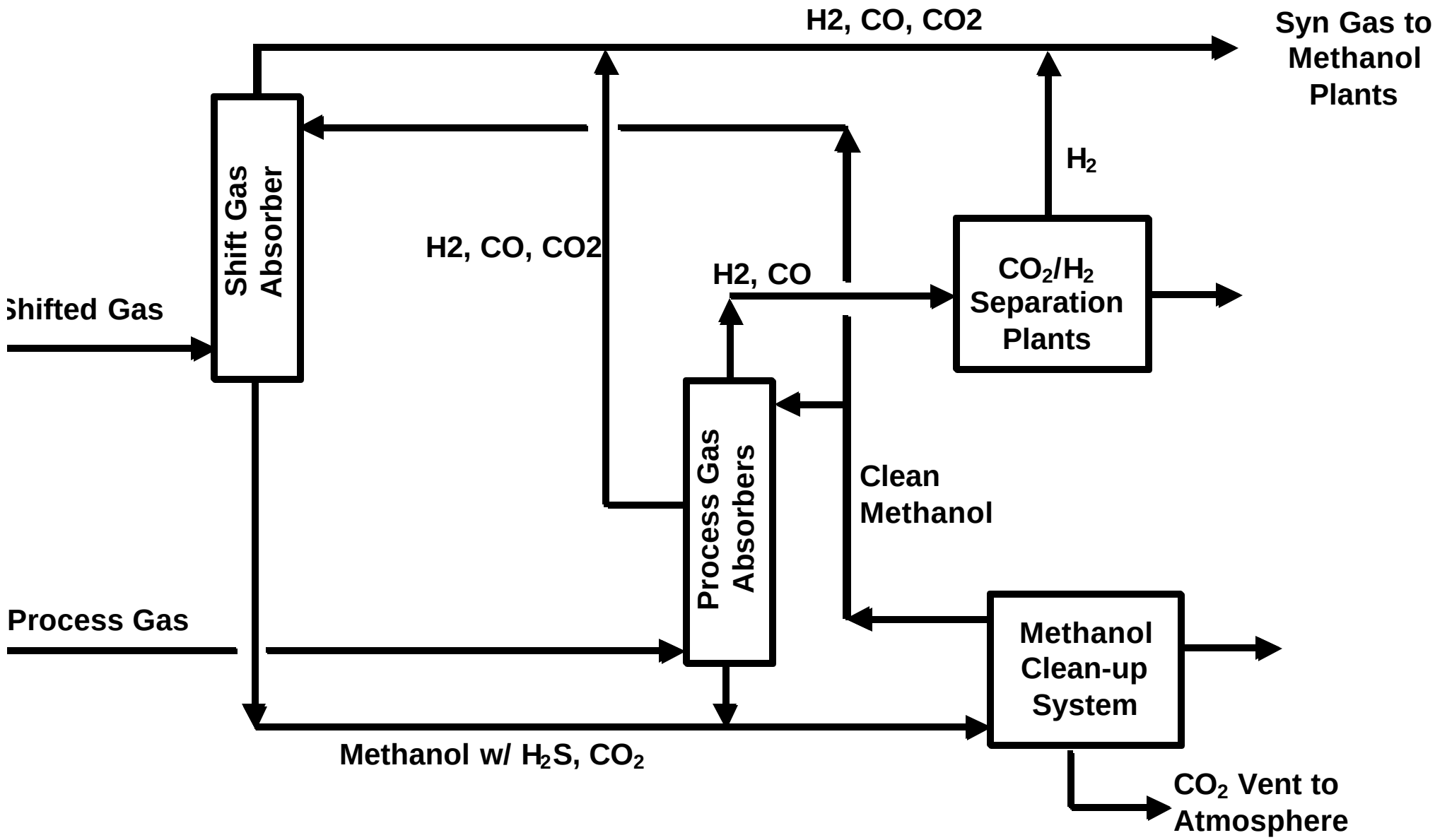


Figure 5: Yearly Gasifier Production Rate

Figure 6: % Uptime
(w/o Planned Biannual Shutdown or Power Failures)

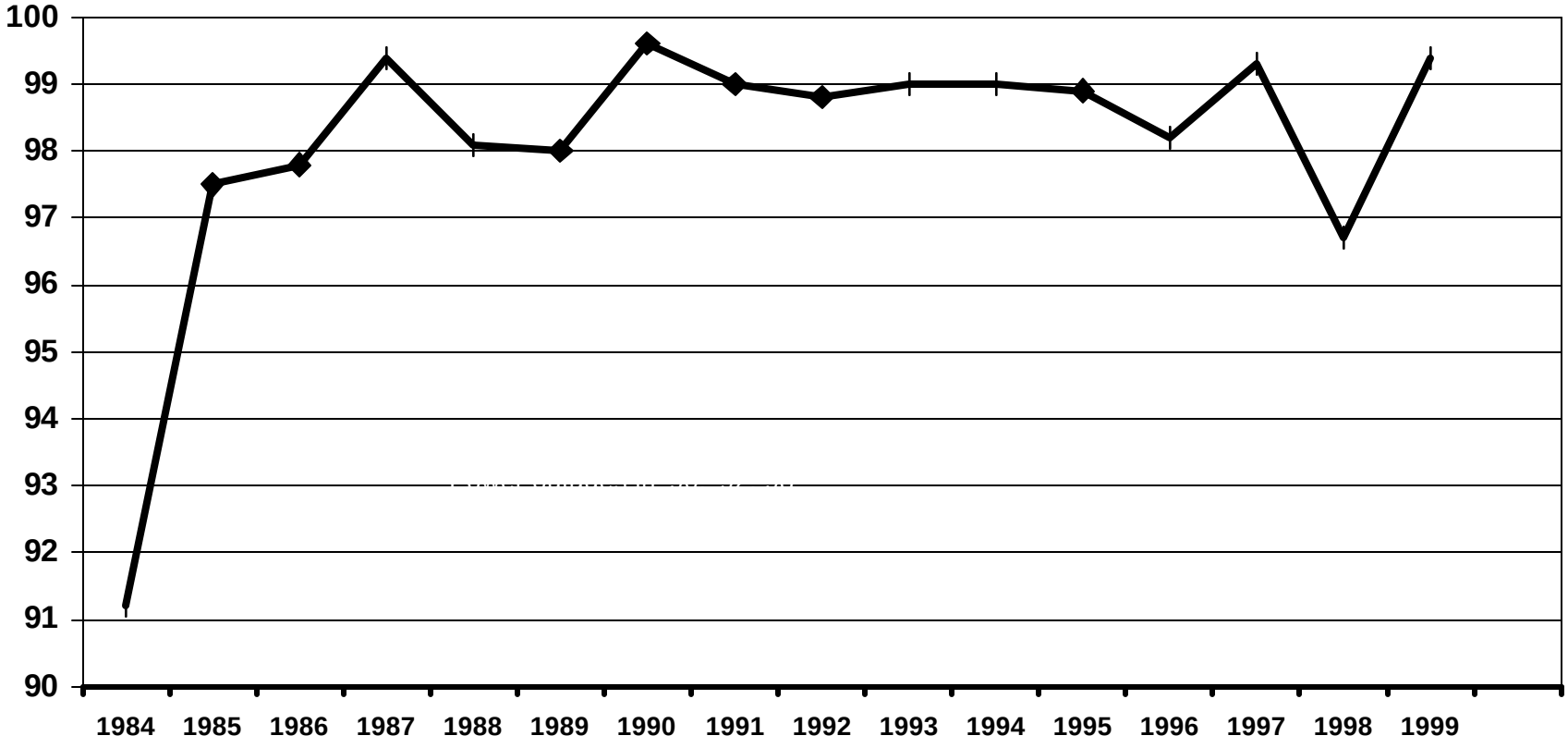


Figure 7:
Top 10 Shutdown Causes
1983 – 2000

Rank	Shutdown Cause	% of total
1	Feed Injector Failure	18.7%
2	Slurry Feed Pump	15.2%
3	Planned Switch	10.9%
4	Low Quench H2O Flow	6.6%
5	Low Slurry Flow	5.8%
6	Low level in Gasifier	4.8%
7	O2 Leak	4.1%
8	PSV failure	2.3%
9	Dip/Draft Tube	1.8%
10	DCS - PLC failure	1.8%

Figure 8: Shutdown Causes
1999 – 2000

1999

Description	Count
Planned switch	3
Seal water supply pump	2
Slag drag failure	1
Relief valve failure	1
Plugged quench ring	1
Lockhopper valve problems	1
High temp on injector jacket	1
Feed Injector Failure	1

2000 through September

Description	Count
Feed injector failure (prior to new design)	2
Low quench flow (pump failure)	1
Plant wide power failure	1
Black Water exchanger fouling	1
Oxygen Plant Shutdown (Lightning)	1

Figure 9: Gasifier Run Length (Days)

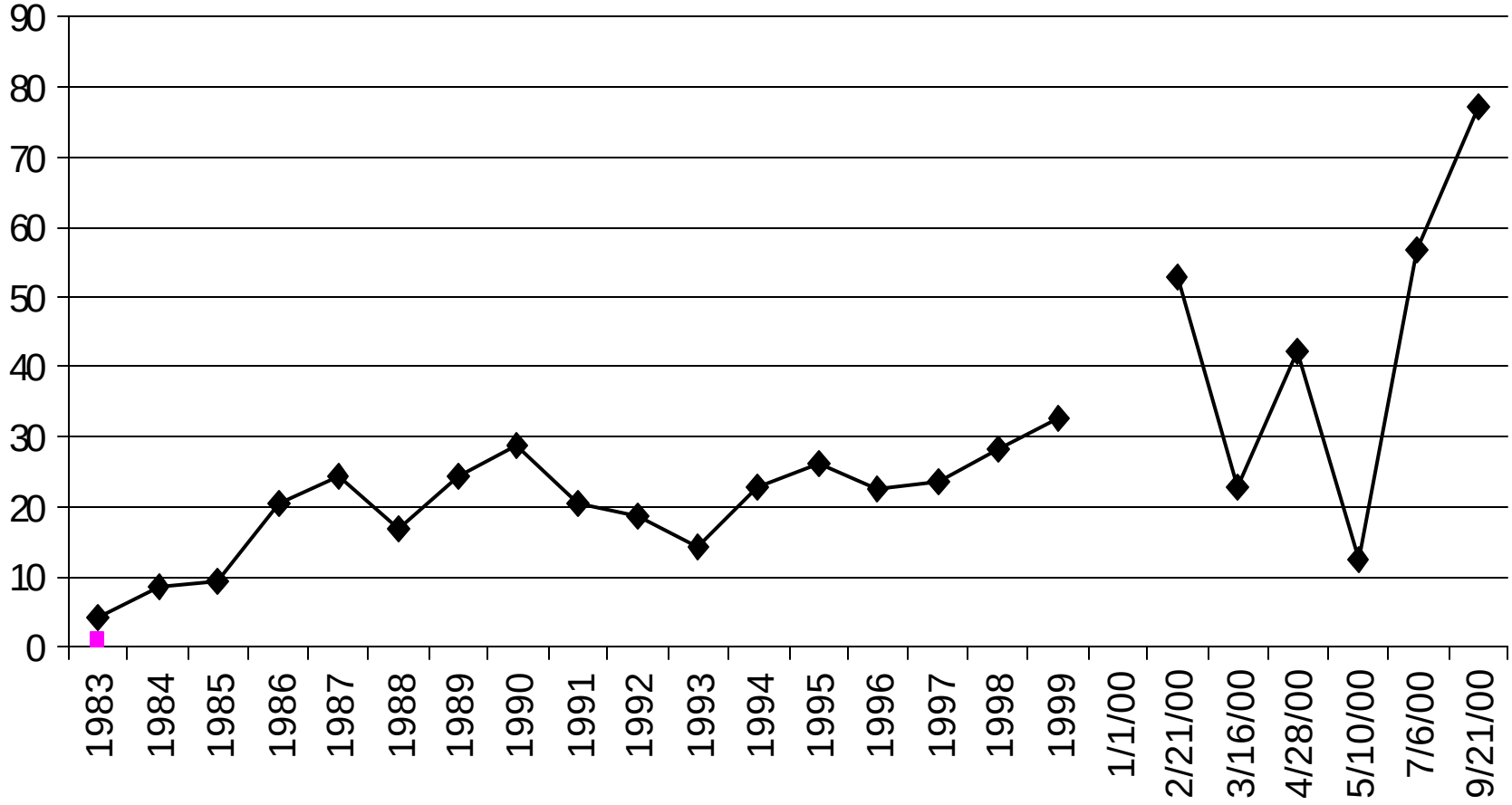
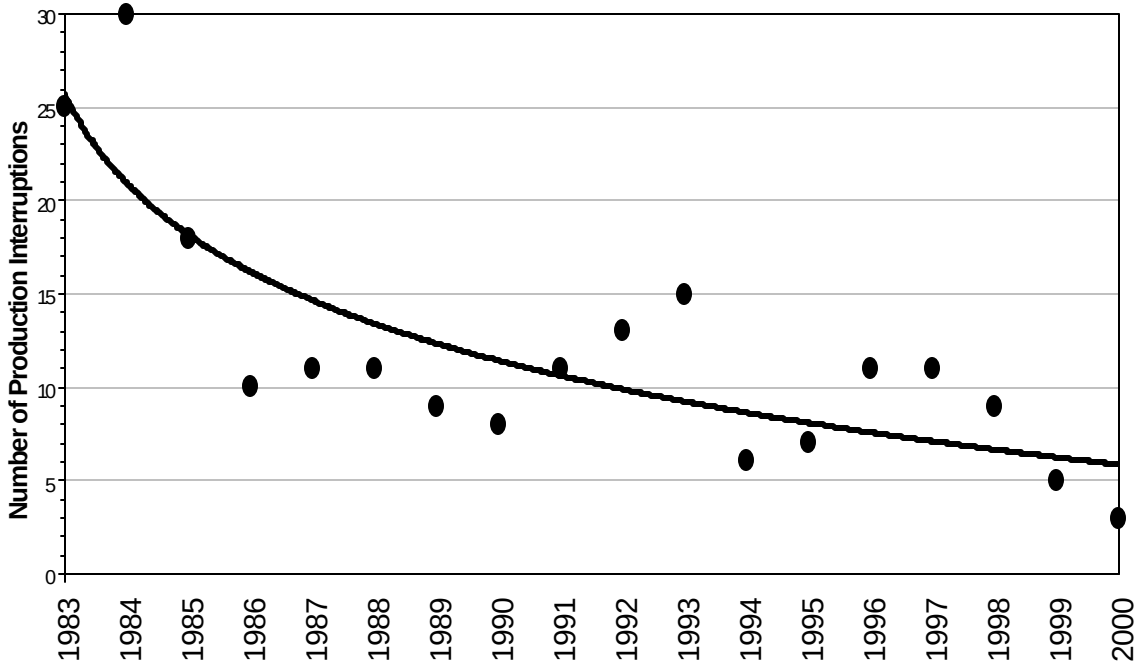


Figure 10: Production Interruptions

1983 to Present



*2000 data is YTD

http://www.epa.gov/climatechange/effects/index.html
Last updated on Thursday, December 20th, 2007.

Climate Change - Health and Environmental Effects

You are here: EPA Home Climate Change Health and Environmental Effects

Health and Environmental Effects

Many elements of human society and the environment are sensitive to climate variability and change. Human health, agriculture, natural ecosystems, coastal areas, and heating and cooling requirements are examples of climate-sensitive systems.

Rising average temperatures are already affecting the environment. Some observed changes include shrinking of glaciers, thawing of permafrost, later freezing and earlier break-up of ice on rivers and lakes, lengthening of growing seasons, shifts in plant and animal ranges and earlier flowering of trees (IPCC, 2007).

Global temperatures are expected to continue to rise as human activities continue to add carbon dioxide, methane, nitrous oxide, and other greenhouse (or heat-trapping) gases to the atmosphere. Most of the United States is expected to experience an increase in average temperature (IPCC, 2007). Precipitation changes, which are also very important to consider when assessing climate change effects, are more difficult to predict. Whether or not rainfall will increase or decrease remains difficult to project for specific regions.

The extent of climate change effects, and whether these effects prove harmful or beneficial, will vary by region, over time, and with the ability of different societal and environmental systems to adapt to or cope with the change.

The Intergovernmental Panel on Climate Change (IPCC, 2007) concludes that “impacts of climate change will vary regionally but, aggregated and discounted to the present, they are very likely to impose net annual costs which will increase over time as global temperatures increase.” The IPCC estimates that for increases in global mean temperature of less than 1-3°C (1.8-5.4°F) above 1990 levels, some places and sectors will see beneficial impacts while others will experience harmful ones. Some low-latitude and polar regions are expected to experience net costs even for small increases in temperature. For increases in temperature greater than 2-3°C (3.6-5.4°F), the IPCC says it is very likely that all regions will experience either declines in net benefits or increases in net costs. “Taken as a whole,” the IPCC concludes, “the range of published evidence indicates that the net damage costs of climate change are likely to be significant and to increase over time.”

The following links provide more detailed information on the human health and environmental effects of climate change:

Related Links

EPA Global Change Research Program

U.S. Climate Change Science Program

Climate Change Impacts and Adaptation in California

IPCC Working Group II
EXIT Disclaimer

- Climate Change Impacts, Adaptation and Vulnerability
- Fourth Assessment Report, Chapter 14, North America (PDF, 36 pp., 895 kb, About PDF)

Pew Center Report
EXIT Disclaimer

- A Synthesis of Potential Climate Change Impacts on the U.S.
- U.S. Market Consequences of Global Climate Change

World Health Organization - Climate Change and Health
EXIT Disclaimer

- Health
- Agriculture and Food Supply
- Forests
- Ecosystems and Biodiversity
- Coastal Zones and Sea Level Rise
- Water Resources
- Energy Production and Use
- Public Lands and Recreation
- U.S. Regions
- Polar Regions
- International
- Extreme Events
- Adaptation

References

- IPCC, 2007: Climate Change 2007: Impacts, Adaptation, and Vulnerability. EXIT Disclaimer Contribution of Working Group II to the Third Assessment Report of the Intergovernmental Panel on Climate Change [Parry, Martin L., Canziani, Osvaldo F., Palutikof, Jean P., van der Linden, Paul J., and Hanson, Clair E. (eds.)]. Cambridge University Press, Cambridge, United Kingdom, 1000 pp.

http://www.epa.gov/air/ozonepollution/health.html
Last updated on Tuesday, March 6th, 2007.

Ground-level Ozone

Health and Environment

Breathing ozone can trigger a variety of health problems including chest pain, coughing, throat irritation, and congestion. It can worsen bronchitis, emphysema, and asthma. Ground-level ozone also can reduce lung function and inflame the linings of the lungs. Repeated exposure may permanently scar lung tissue.

The Clean Air Act requires EPA to set air quality standards to protect both public health and the public welfare (e.g. crops and vegetation). Ground-level ozone affects both.

Health Effects

People with lung disease, children, older adults, and people who are active can be affected when ozone levels are unhealthy. Numerous scientific studies have linked ground-level ozone exposure to a variety of problems, including:

- lung irritation that can cause inflammation much like a sunburn;
- wheezing, coughing, pain when taking a deep breathe, and breathing difficulties during exercise or outdoor activities;
- permanent lung damage to those with repeated exposure to ozone pollution; and
- aggravated asthma, reduced lung capacity, and increased susceptibility to respiratory illnesses like pneumonia and bronchitis.

See the Air Quality Guide for Ozone for ways to protect your family's health when ozone levels reach the unhealthy range and ways you can help reduce ozone air pollution.

Learn more about asthma.

Environmental Effects

Ground-level ozone can have detrimental effects on plants and ecosystems. These effects include:

- interfering with the ability of sensitive plants to produce and store food, making them more susceptible to certain diseases, insects, other pollutants, competition and harsh weather;
- damaging the leaves of trees and other plants, negatively impacting the appearance of urban vegetation, national parks, and recreation areas; and
- reducing crop yields and forest growth, potentially impacting species diversity in ecosystems.

You will need Adobe Acrobat Reader to view the Adobe PDF files on this page.
See EPA's PDF page for more information about getting and using the free Acrobat Reader.

For more information on ground-level ozone, health and the environment, visit:

Ozone and Your Health (PDF, 2 pp, 449 KB) This short, colorful pamphlet tells who is at risk from exposure to ozone, what health effects are caused by ozone, and simple measures that can be taken to reduce health risk.

Ozone: Good Up High, Bad Nearby (PDF, 2 pp, 1391 KB) Ozone acts as a protective layer high above the earth, but it can be harmful to breathe. This publication provides basic information about ground-level and high-altitude ozone.

Smog - Who Does it Hurt? (PDF, 10 pp, 819 KB) This 8-page booklet provides more detailed information than "Ozone and Your Health" about ozone health effects and how to avoid them.

Summertime Safety: Keeping Kids Safe from Sun and Smog (PDF, 2 pp, 314 KB) This document discusses summer health hazards that pertain particularly to children and includes information about EPA's Air Quality Index and UV Index tools.

Air Quality Criteria Document for Ozone (February 2006): This comprehensive assessment of scientific data about the health and environmental effects of ground-level ozone is an important part of EPA's review of its ozone standards.

http://www.epa.gov/air/particlepollution/health.html
Last updated on Tuesday, November 27th, 2007.

Particulate Matter

You are here: EPA Home Air & Radiation Particulate Matter Health and Environment

Health and Environment

The size of particles is directly linked to their potential for causing health problems. Small particles less than10 micrometers in diameter pose the greatest problems, because they can get deep into your lungs, and some may even get into your bloodstream.

Exposure to such particles can affect both your lungs and your heart. Small particles of concern include "inhalable coarse particles" (such as those found near roadways and dusty industries), which are larger than 2.5 micrometers and smaller than 10 micrometers in diameter; and "fine particles" (such as those found in smoke and haze), which are 2.5 micrometers in diameter and smaller.

The Clean Air Act requires EPA to set air quality standards to protect both public health and the public welfare (e.g. crops and vegetation). Particle pollution affects both.

Health Effects

Particle pollution - especially fine particles - contains microscopic solids or liquid droplets that are so small that they can get deep into the lungs and cause serious health problems. Numerous scientific studies have linked particle pollution exposure to a variety of problems, including:

- increased respiratory symptoms, such as irritation of the airways, coughing, or difficulty breathing, for example;
- decreased lung function;
- aggravated asthma;
- development of chronic bronchitis;
- irregular heartbeat;
- nonfatal heart attacks; and
- premature death in people with heart or lung disease.

People with heart or lung diseases, children and older adults are the most likely to be affected by particle pollution exposure. However, even if you are healthy, you may experience temporary symptoms from exposure to elevated levels of particle pollution. For more information about asthma, visit www.epa.gov/asthma.

Environmental Effects

Visibility reduction
Fine particles ($PM_{2.5}$) are the major cause of reduced visibility (haze) in parts of the United States, including many of our treasured national parks and wilderness areas. For more information about visibility, visit www.epa.gov/visibility.

Environmental damage
Particles can be carried over long distances by wind and then settle on ground or water. The

effects of this settling include: making lakes and streams acidic; changing the nutrient balance in coastal waters and large river basins; depleting the nutrients in soil; damaging sensitive forests and farm crops; and affecting the diversity of ecosystems. More information about the effects of particle pollution and acid rain.

Aesthetic damage

Particle pollution can stain and damage stone and other materials, including culturally important objects such as statues and monuments. More information about the effects of particle pollution and acid rain.

You will need Adobe Acrobat Reader to view the Adobe PDF files on this page. See EPA's PDF page for more information about getting and using the free Acrobat Reader.

For more information on particle pollution, health and the environment, visit:

Particle Pollution and Your Health: Learn who is at risk from exposure to particle pollution, what health effects you may experience as a result of particle exposure, and simple measures you can take to reduce your risk. (PDF, 2 pp, 320 KB)

How Smoke From Fires Can Affect Your Health: It's important to limit your exposure to smoke -- especially if you may be susceptible. This publication provides steps you can take to protect your health.

Air Quality Criteria Document for Particulate Matter (October 2004): This comprehensive assessment of scientific data about the health and environmental effects of particulate matter is an important part of EPA's review of its particle pollution standards.

RESPONSE TO PUBLIC COMMENTS

ON

Draft
Air Pollution Control
Prevention of Significant Deterioration (PSD)
Permit to Construct

Permit No. PSD-OU-0002-04.00

<u>Permittee</u>:
Deseret Power Electric Cooperative
10714 South Jordan Gateway
South Jordan, Utah 84095

<u>Permitted Facility</u>:
110-Megawatt Waste Coal Fired Unit
at Bonanza Power Plant

United States Environmental Protection Agency
Region 8
Air & Radiation Program
Denver, Colorado
August 30, 2007

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A. INTRODUCTION

On April 14, 2004, Deseret Power submitted a Prevention of Significant Deterioration (PSD) permit application to the United States Environmental Protection Agency, Region 8 (EPA), to approve construction of a new coal-fired electric utility unit at Deseret's existing Bonanza power plant. The application was updated and re-submitted to EPA on November 1, 2004. Several amendments to the application were submitted over the following year and a half. The application, amendments, draft PSD permit, draft Statement of Basis, and all related correspondence between EPA and Deseret Power are contained in the Administrative Record of this permit action, which was made available for 30-day public comment in late June of 2006.

The existing Bonanza power plant is located in eastern Utah, on the Uintah & Ouray Indian Reservation, and consists of a single bituminous coal fired electric utility unit ("Unit 1"), rated at 500 megawatts electrical output. The fuel for Unit 1 is supplied by the Deserado coal mine, located about 35 miles east of the plant. Unit 1 was constructed in the early 1980's and is operating under a Federal PSD permit originally issued by EPA on February 4, 1981, then updated and re-issued on February 7, 2001.

The new unit at Bonanza plant would consist of a Circulating Fluidized Bed (CFB) boiler and associated equipment, rated at 110 megawatts electrical output, and designed to be fueled with waste coal from the Deserado mine. The PSD permit for the new unit is proposed to be issued as a separate permit from the PSD permit for Unit 1.

The EPA published a public notice in the following newspapers, on the following dates, soliciting comments on its proposal to issue the permit for the new unit, in accordance with Sections 160-169 of the Clean Air Act (CAA), 40 CFR 52.21, and 40 CFR part 124:

Uintah Basin Standard (Roosevelt, UT)	June 27, 2006
Vernal Express (Vernal, UT)	June 28, 2006
Grand Junction Sentinel (Grand Junction, CO)	June 28, 2006
Rio Blanco Herald Times (Meeker/ Rangely, CO)	June 29, 2006
Salt Lake Tribune (Salt Lake City, UT)	June 29, 2006

The public comment period ended on July 29, 2006.

On June 22, 2006, the EPA mailed copies of the draft PSD permit, draft Statement of Basis, public notice, and Administrative Record for the proposed permit action, consisting of all permit-related correspondence, to the following parties:

Uintah County Clerk's Office
147 East Main Street, Suite 2300
Vernal, Utah 84078

Ute Indian Tribe
Environmental Programs Office
6358 East Highway 40
Fort Duchesne, Utah 84026

EPA sent the documents to these locations specifically to have the documents available locally for public review, during the public comment period. As stated in the public notice, these documents were also available at the EPA office in Denver, Colorado, and on the internet through EPA's website, at:

http://www.epa.gov/region8/air, under the heading "Topics of Interest"

The draft PSD permit would require air pollutant emission controls and restrict emissions of the following pollutants at the CFB boiler and associated pollutant-emitting support equipment: total particulate matter, filterable particulate matter, sulfur dioxide, nitrogen oxide, carbon monoxide, and sulfuric acid.

During the public comment period, one comment letter and one comment e-mail were received by EPA that expressed concerns with the draft permit and/or Statement of Basis. The comment letter, received on July 28, 2006, was from a group of seven environmental organizations: Western Resource Advocates, Environmental Defense, Utah Chapter of the Sierra Club, Southern Utah Wilderness Alliance, Western Colorado Congress, Wasatch Clean Air Coalition, and HEAL Utah. Comments #1 through #11 below are from the letter. The comment e-mail, received on July 26, 2006, was from Kathy Van Dame, representing the Wasatch Clean Air Coalition. Comments #12 through #16 below are from the e-mail.

Comment letters supporting the proposed WCFU project were received from the mayors of seven Utah municipalities: Salem City, Spanish Fork, Provo, Manti City, St. George, Nephi and Levan. Since these letters did not express any concerns with the draft PSD permit, EPA does not consider a response necessary.

After the close of the public comment period, EPA received an e-mail dated April 24, 2007, from Katy Savage of Provo, Utah, expressing concern about pollutants that would be emitted from the WCFU project, and a a letter dated April 25, 2007, from Daniel D. McArthur, Mayor of the City of St. George, Utah, expressing concern about delay in issuing the EPA permit for the WCFU project.

A detailed description of the commenters' concerns, along with EPA's responses to the significant issues raised in the comments, is contained in Section B of this document. Some of the lengthier comments have been paraphrased or generalized to allow direct responses to the concerns raised.

All references in Section B to the "Statement of Basis" mean the draft Statement of Basis dated June 14, 2006, which was made available along with the draft PSD permit for public comment in late June of 2006. All references to the "WCFU" mean Deseret

Power's proposed Waste Coal Fired Unit at Bonanza power plant, the subject of this PSD permit action. All references to "EPA" mean the EPA Region 8 office in Denver, unless otherwise indicated.

Section C of this document describes the specific provisions of the draft permit and draft Statement of Basis that have been changed in the final permit decision as a result of public comment. The final permit and final Statement of Basis include some administrative changes that may not be described in Section C, including renumbering permit conditions due to additional conditions added to the final permit, renumbering sections of the Statement of Basis due to additional explanations added to the Statement of Basis, and rewording as necessary to reflect the fact that the permit and Statement of Basis are final, not draft.

Deseret Power requested meetings with EPA, and met with EPA, on October 16, 2006 and on May 7, 2007, and submitted additional written permit-related material after the close of the public comment period. EPA is including the additional material and a summary of the October 16, 2006 and May 7, 2007 meetings in the Administrative Record for EPA's final permit decision.

Documents upon which EPA relied in reaching the final permit decision, and as referenced in EPA's response to comments, such as the Statement of Basis, the PSD permit application, and supplemental documents, are contained in the Administrative Record. Copies of EPA's response-to-comments document, final permit, and final Statement of Basis, are available on EPA's website at:

http://www.epa.gov/region8/air, under the heading "Topics of Interest"

The website also provides a link to the Administrative Record.

Copies of the response-to-comments document, the final permit, and the final Statement of Basis are also available for public review at the same locations where the draft permit and Statement of Basis were available for review:

Uintah County Clerk's Office
147 East Main Street, Suite 2300
Vernal, Utah 84078

Ute Indian Tribe
Land Use Department
P.O. Box 460
6358 East Highway 40
Fort Duchesne, Utah 84026

All documents in the Administrative Record are available at the EPA office:

US EPA Region 8
Air & Radiation Program
1595 Wynkoop Street
Denver, CO 80202-1129
Contact: Mike Owens, 303-312-6440
owens.mike@epa.gov

B. COMMENTS AND RESPONSES

The descriptions of public comments below are a paraphrasing of the originally submitted comments. The full text of each public comment may be found in the Administrative Record for issuance of the WCFU permit, available at the same locations as the draft permit package was available (the Uintah County Clerk's office in Vernal, Utah, the Ute Indian Tribe office in Fort Duchesne, Utah, and the EPA Region 8 office in Denver, Colorado).

1. CARBON DIOXIDE/GREENHOUSE GAS EMISSIONS

Comment #1:

One group of commenters requested that EPA address carbon dioxide (CO_2) and other greenhouse gas (GHG) emissions from the proposed Deseret Bonanza WCFU. The commenters stated that the Clean Air Act requires EPA to do so in two ways.

Comment #1.a. First, the commenters believe EPA has a legal obligation to regulate CO_2 and other GHGs under the Clean Air Act and thus should set CO_2 emission limits in this permit.

Comment #1.b. Second, the commenters believe that EPA should consider emissions of CO_2 in its BACT analyses for other pollutants at the Bonanza WCFU.

In support, the commenters cited a U.S. Supreme Court case that was pending at the time, an Environmental Appeals Board decision, a draft EPA guidance document, and an article presenting a potential legal rationale for using PSD permits to limit CO_2 emissions.

Response #1:

Response #1.a. Disagree. EPA recognizes the importance of addressing the global challenge of climate change, and in light of the Supreme Court's decision in *Massachusetts v. EPA*, 127 S. Ct. 1438 (2007), the Agency is working diligently to develop an overall strategy for addressing the emissions of CO2 and other GHGs under the Clean Air Act. However, EPA does not currently have the authority to address the challenge of global climate change by imposing limitations on emissions of CO_2 and other greenhouse gases in PSD permits.

It is well established that "EPA lacks the authority to impose [PSD permit] limitations or other restrictions directly on the emission of unregulated pollutants." *North County Resource Recovery Assoc.*, 2 E.A.D. 229, 230 (EAB 1986). The Clean Air Act and EPA's regulations require PSD permits to contain emissions limitations for "each pollutant subject to regulation" under the Act. CAA § 165(a)(4); 40 C.F.R. § 52.21(b)(12). In defining those PSD permit requirements, EPA has historically interpreted the term "subject to regulation under the Act" to describe pollutants that are presently subject to a statutory or regulatory provision that requires actual control of

emissions of that pollutant. *See* 43 Fed. Reg. 26388, 26397 (June 19, 1978) (describing pollutants subject to BACT requirements); 61 Fed. Reg. 38250, 38309-10 (July 23, 1996) (listing pollutants subject to PSD review). In 2002, EPA codified this approach for implementing PSD by defining the term "regulated NSR pollutant" and clarifying that Best Available Control Technology is required "for each regulated NSR pollutant that [a major source] would have the potential to emit in significant amounts." 40 C.F.R. § 52.21(j)(2); 40 CFR 52.21(b)(50).

In defining a "regulated NSR pollutant," EPA identified such pollutants by referencing pollutants regulated in three principal program areas -- NAAQS pollutants, pollutants subject to a section 111 NSPS, and class I or II substance under title VI of the Act-- as well as any pollutant "that otherwise is subject to regulation under the Act." 40 CFR 52.21(b)(50)(i)-(iv). As used in this provision, EPA continues to interpret the phrase "subject to regulation under the Act" to refer to pollutants that are presently subject to a statutory or regulatory provision that requires actual control of emissions of that pollutant. Because EPA has not established a NAAQS or NSPS for CO_2, classified CO_2 as a title VI substance, or otherwise regulated CO_2 under any other provision of the Act, CO_2 is not currently a "regulated NSR pollutant" as defined by EPA regulations.

Although the Supreme Court decided the case cited by commenters and held that CO_2 and other GHGs are air pollutants under the CAA, *see Massachusetts v. EPA*, 127 S. Ct. 1438 (2007), that decision does not require the Agency to set CO_2 emission limits in the PSD permit for the Deseret Bonanza WCFU. Notably, the Court did not hold that EPA was required to regulate CO_2 and other GHG emissions under Section 202, or any other section, of the Clean Air Act. Rather, the Court concluded that these emissions were "air pollutants" under the Act, and, therefore, EPA could regulate them under Section 202 (the provision at issue in the *Massachusetts* case), subject to certain Agency determinations pertaining to mobile sources.

EPA is currently exploring options for addressing GHG emissions in response to the Supreme Court decision. EPA is taking the first steps toward regulating GHG emissions from mobile sources, but the Agency has not yet issued regulations requiring control of CO_2 emissions under the Act generally or the PSD program specifically. Accordingly, EPA cannot include emissions limitations for CO_2 (or other GHGs that are not otherwise regulated NSR pollutants) in the Deseret PSD permit because it has long been established that "EPA lacks the authority to impose [PSD permit] limitations or other restrictions directly on the emission of unregulated pollutants." *North County*, 2 E.A.D. at 230. At this time, we believe that any action EPA might consider taking with respect to regulation of CO_2 or other GHGs in PSD permits or other contexts should be addressed through notice and comment rulemaking, allowing for a process which is public and transparent and based on the best available science.

Response #1.b: Disagree. EPA recognizes the importance of addressing the global challenge of climate change, and in light of the Supreme Court's decision in *Massachusetts v. EPA*, 127 S. Ct. 1438 (2007), the Agency is working diligently to develop an overall strategy for addressing the emissions of CO2 and other GHGs under the Clean Air Act. Nevertheless, with regard to the present permitting decision, the

record before the Agency does not suggest, and commenters have not provided any evidence showing, that the outcome of our BACT analysis for the regulated NSR pollutants emitted by the Deseret Bonanza WFCU would have been resulted in a different choice of control technologies had we considered the potential collateral environmental impacts of CO_2 emissions.

The CAA defines BACT as "an emission limitation based on the maximum degree of reduction of each pollutant subject to regulation under this Act emitted from or which results from any major emitting facility, which the permitting authority, on a case-by-case basis, taking into account energy, environmental, and economic impacts and other costs determines is achievable for such facility through application of production processes and available methods, systems, and techniques, including fuel cleaning, clean fuels, or treatment or innovative fuel combustion techniques for control of such pollutant." CAA § 169(3) (emphasis added); *see also* 40 CFR 52.21(b)(12). EPA has established a five-step, top-down process for determining BACT emission limits for each PSD-regulated pollutant considered in a permitting decision: (1) identify all potentially applicable control options (2) eliminate technically infeasible control options; (3) rank remaining technologies by control effectiveness; (4) eliminate control options from the top down based on energy, environmental, and economic impacts; and (5) select the most effective option not eliminated as BACT. *See Prairie State Generating Co.*, 13 E.A.D. ___, PSD Appeal No. 05-05, slip op. at 14-18 (EAB Aug. 24, 2006) (summarizing and describing steps in the top-down BACT analysis). *Accord Three Mountain Power, L.L.C.,* 10 E.A.D. 39, 42-43 n.3 (EAB 2001); *Knauf Fiber Glass, GmbH,* 8 E.A.D. 121, 129-31 (EAB 1999); *Hawaii Electric Light Co.,* 8 E.A.D. 66, 84 (EAB 1998). Thus, EPA has traditionally considered the collateral impacts (energy, environmental, and economic) of each BACT option at Step 4 of this analysis.

The CAA does not specify how EPA should weigh these collateral impacts when determining BACT for a particular source. The Agency's longstanding interpretation is that "the primary purpose of the collateral impacts clause is to temper the stringency of the technology requirements whenever one or more of the specified collateral impacts – energy, environmental, and economic – renders use of the most effective technique inappropriate." *Columbia Gulf Transmission Co.*, 2 E.A.D. 824, 826 (EAB 1989). Accordingly, the environmental impacts analysis "is generally couched in terms of discussing which available technology, among several, produces less adverse collateral effects, and, if it does, whether that justifies its utilization even if the technology is otherwise less stringent." *Old Dominion Electric Cooperative*, 3 E.A.D. 779, 792 (EAB 1992).

In this case, the commenters have not shown that consideration of the environmental impacts of CO_2 emissions in the collateral impacts step of the EPA's BACT analysis for the regulated NSR pollutants would lead to a different result in our selection of BACT for the Deseret facility. The record before the Agency does not suggest that the Agency should have selected a less stringent option as BACT in order to reduce the potential collateral environmental impacts of CO_2 emissions. Although there may be some differences in the CO_2 emissions resulting from use of the technologies we evaluated at step 4 of the BACT analysis, we do not have information indicating such

differences would be significant enough to necessitate changing our selection of BACT for other pollutants. *See Hillman Power Co., L.L.C.*, PSD Appeal Nos. 02-04 (July 31, 2002) ("collateral environmental impacts analysis need only address those control alternatives with any significant or unusual environmental impacts that have the potential to affect the selection or elimination of a control alternative."). Commenters have not given EPA cause to believe that comparisons of the CO_2 emissions from various control technologies considered in the BACT analysis for the Deseret Bonanza WCFU would render unacceptable any of the options we have identified as BACT for this PSD permit.

Specifically, the comments did not contain any information on CO_2 emissions that would lead EPA to reach a different conclusion in its BACT analysis for this facility. The commenters state only that "EPA must consider emissions of CO_2 in its BACT analysis for the Bonanza WCFU," but they do not address how the particular control technologies considered for the Bonanza WCFU would have resulted in substantially differing CO_2 emissions. Nor do they discuss how any such differences would have resulted in differing impacts that would have necessitated our selecting a different technology as BACT. Such comparisons are at the heart of the BACT analysis, and thus are required by a commenter alleging a deficiency in the analysis. *See Old Dominion*, 3 E.A.D. at 793 (finding no error based on petitioner's lack of "specificity and clarity" because they provided "no specific comparison" of differences in the environmental impacts of the various technologies considered in the BACT analysis). *See also Vermont Yankee Nuclear Power Corp. v. Natural Resources Defense Council, Inc.*, 435 U.S. 519, 553 (U.S. 1978) (explaining that comments regarding an Agency's analysis of environmental impacts "cannot merely state that a particular mistake was made, …[but] must show why the mistake was of possible significance in the results"). Accordingly, commenters have failed to show how consideration of CO_2 emissions in the BACT environmental impacts analysis would have changed the Deseret Bonanza permitting decisions.

Moreover, because EPA has historically interpreted the phrase "environmental impacts" to focus on local environmental impacts that are directly attributable to the proposed facility, the collateral impacts analysis of this BACT determination is not the appropriate mechanism for addressing the potential global impacts of CO_2 emissions from the Deseret Bonanza WCFU. *See Columbia Gulf*, 2 E.A.D. at 829-30 (finding that the environmental impacts analysis "focuses on local impacts that constrain the source from using the most effective technology"). Any predicted impacts in the area surrounding the Deseret facility that are potentially due to global climate change – to which the CO_2 and other GHG emissions from the proposed source may contribute generally – are not the type of local environmental impact that is readily traceable directly back to the particular source subject to PSD review.

EPA's interpretation that the collateral environmental impacts analysis should focus on local impacts that are directly attributed to construction and operation of the proposed source is supported by relevant statutory language, legislative history, EAB decisions, and EPA policies and permitting decisions. Both the "case-by-case" language of the BACT definition and Congress' stated reason for adding the collateral impacts analysis to that definition suggest that a facility-centered, locally-focused analysis is

appropriate. *See Kawaihae Cogeneration Project*, 7 E.A.D. 107, 116-17 (EAB 1997) (describing how the collateral impacts analysis considers factors unique to the specific source); Senate Comm. on Environment And Public Works, A Legislative History of the Clean Air Act Amendments of 1977 (Comm. Print August 1978), vol. 6 at 4723-24 (explaining that the collateral impacts clause was added to provide permitting authorities with flexibility to consider the impact of a specific facility on the character of the community in which it was located). While the EAB's *North County* decision directed permitting authorities to look at the effect of emissions from non-PSD regulated hazardous air pollutants (i.e., HAPs) in the collateral impacts analysis, the Board's opinion did not specify that all emissions not directly regulated under PSD – such as CO_2 – had to be considered as well. *See id.*, 2 E.A.D. at 230 (stating that the "exact form" and "level" of the BACT environmental impacts analysis would depend on the facts of the individual permitting decision). In subsequent policy guidance, EPA did not interpret *North County* to call for consideration of global impacts, *see, e.g.,* Memorandum from Gerald Emison, OAQPS Director entitled *Implementation of North County PSD Remand,* pp. 3-4 (Sept. 22, 1987), and the EAB later determined that EPA did not have to consider CO_2 and other GHG emissions in the BACT environmental impacts analysis. *Interpower of New York*, 5 E.A.D. 130 (EAB 1994); *Kawaihae Cogeneration Project*, 7 E.A.D. 107 (EAB 1997). Consistent with these prior EAB decisions and Agency policy, EPA has not previously considered the environmental impact of CO_2 and other GHG emissions in setting the BACT levels for permits,[1] and for the reasons discussed above, we do not consider it necessary to do so in issuing the PSD permit for the Deseret Bonanza WFCU.

[1] Although one draft of EPA's 1990 *NSR Workshop Manual* referenced "greenhouse gas emissions" as an example of environmental impact that a reviewing authority might consider in the BACT analysis, EPA has not done so in practice. The Agency never finalized the draft guidance cited by commenters, and other drafts of that same document do not include the phrase "greenhouse gas emissions" as an example of the type of environmental impact to be considered in the BACT analysis. *See* http://www.epa.gov/region07/programs/artd/air/nsr/nsrmemos/1990wman.pdf, at B49. Moreover, both of these drafts of the *NSR Workshop Manual* also indicate that the BACT environmental impacts analysis should focus on "consideration of site-specific circumstances," which contrasts with the notion that such analysis should be used to consider the source's impact on what is a global issue. *Id.* at B47.

2. INTEGRATED GASIFICATION COMBINED CYCLE (IGCC)

Comment #2:

One group of commenters asserted that the proposed permit did not adequately evaluate IGCC as an available method to lower air emissions in the BACT analysis. The group of commenters presented four arguments:

Comment #2.a. First, arguing that Federal law requires a thorough evaluation of IGCC as part of the BACT analysis.

Comment #2.b. Second, arguing that recent state actions requiring consideration of cleaner coal technology establish irrefutable precedence for the consideration of IGCC, and validate the commenters' position on the "plain language of the definition of BACT."

Comment #2.c. Third, alleging EPA Region 8 previously determined it was appropriate to evaluate IGCC in the BACT analysis for a CFB coal-fired power plant. Commenters cited EPA Region 8's April 6, 2004 letter to the Utah Division of Air Quality, on Utah's proposed PSD permit for Nevco Energy's Sevier Power Company Project. Commenters also cited EPA's April 28, 2004 request to Deseret Power to provide an explanation of why Deseret ruled out IGCC for the WCFU project.

Comment #2.d. Fourth, pointing out the overall benefit of the alternative IGCC technology, including fewer emissions of criteria and hazardous air pollutants, the opportunity for capturing greenhouse gases, and increases in efficiency over other coal burning technologies.

Response #2:

Response #2.a. Disagree. EPA does not agree that the Clean Air Act requires a detailed evaluation of IGCC for the proposed facility, at or beyond step 1 of the top-down BACT analysis. We evaluated whether IGCC should be listed at step 1 and considered the commenters arguments, but we have not been persuaded to change our view that this alternative process would represent a redefinition of the source proposed by the applicant and thus need not be listed as a potentially applicable control option at step 1 and evaluated further in the BACT analysis for this type of facility. We have, however, evaluated this option as a potential alternative to the proposed source under other parts of our PSD permit review; see discussion below in response #2.d.

The Administrator and EPA's Environmental Appeals Board ("EAB" or "Board") have long maintained a policy against utilizing the BACT requirement as a means to fundamentally redefine the basic design or scope of a proposed project. *See, e.g., Knauf Fiber Glass, GMBH*, 8 E.A.D. 121, 140 (EAB 1998); *Pennsauken County, New Jersey, Resource Recovery Facility*, 2 E.A.D. 667, 673 (Adm'r 1988). EPA has not required applicants proposing to construct coal-fired steam electric generating facilities to evaluate building natural gas-fired combustion turbines as part of a BACT analysis, even though a

gas turbine may be inherently less polluting. *SEI Birchwood Inc*, 5 E.A.D. 25 (1994); *Old Dominion Electric Cooperative Clover, Virginia*, 3 E.A.D. 779, 793 n. 38 (Adm'r 1992). Likewise, in *Hawaii Commercial & Sugar Co.,* the EAB found no error by the permitting authority when the petitioner argued that the BACT analysis for a coal-fired steam electric generator should include the option of constructing an oil-fired combustion turbine. 4 E.A.D. 95, 99-100 (EAB 1992).

EPA's policy reflects the Agency's longstanding judgment that limits should exist on the degree to which permitting authorities can dictate the design and scope of a proposed facility through the BACT analysis. This policy is based on a reasonable interpretation of sections 165 and 169(3) of the CAA, which recognizes that, although the permitting authority must take comment on and may consider alternatives to a proposed facility, the BACT analysis itself is conducted without changing fundamental characteristics of the proposed source.

The EAB recently reiterated and explained EPA's policy against redefining the source through the BACT analysis in *Prairie State Generating Company*, PSD Appeal No. 05-05 (Aug. 24, 2006). In the Prairie State case, involving a permit for an coal-fired electric generating station that was co-located and co-permitted with a new coal mine supplying fuel for the facility, the Board determined that it was consistent with EPA's historic policy and the Clean Air Act for the permitting authority in this case to decline to conduct a detailed BACT review of the option of using lower-sulfur coal from another location. Based on various provisions of the Clean Air Act, including language that requires the "proposed facility" to be "subject to" BACT, the Board concluded that "the statute contemplates that the permit issuer looks to how the permit applicant defines the proposed facility's purpose or basic design" as part of Step 1 of the top-down BACT analysis. *Prairie State*, slip. op. at 28-29. The Board further explained that "the permit issuer must be mindful that BACT, in most cases, should not be applied to regulate the applicant's objective or purpose for the proposed facility." *Prairie State* slip. op. at 30. The Seventh Circuit recently affirmed the EAB's *Prairie State* decision, including the Board's interpretation of the interplay of determining what redefines a source and the required BACT analysis. *See generally Sierra Club v. EPA,* slip op. (7th Cir. Aug. 24, 2007).

As discussed by the Board in the *Prairie State* opinion, affirmed by the Seventh Circuit, and explained more fully below, EPA's policy against redefining the proposed source through the BACT analysis is supported by a permissible and reasonable interpretation of the Clean Air Act. The language in sections 165 and 169 of the CAA distinguishes between the consideration of alternatives to a proposed source on the one hand and permitting and selection of BACT for the proposed source on the other. Alternatives to a proposed source are evaluated through the CAA section 165(a)(2) public hearing process, which requires that, before a permitting authority may issue a permit, interested persons have an opportunity to "submit written or oral presentations on the air quality impact of such source, *alternatives thereto*, control technology requirements, and other appropriate considerations." 42 U.S.C. § 7475(a)(2) (emphasis added). By listing "alternatives" and "control technology requirements" separately in section 165(a)(2),

Congress distinguished "alternatives" to the proposed source that would wholly replace the proposed facility with a different type of facility from the kinds of "production processes and available methods, systems and techniques" that are potentially applicable to a particular type of facility and should be considered in the BACT review. *See* 42 U.S.C. § 7479(3).

In contrast to the requirements of section 165(a)(2), other parts of the PSD permitting process, including the requirement to apply BACT, focus on, and are generally confined by, the project as proposed by the applicant. Sections 165(a)(1) and 165(a)(4) of the CAA provide that no facility may be constructed unless "a permit has been issued for *such proposed facility* in accordance with this part" and "the *proposed facility* is *subject to* best available control technology for each pollutant subject to regulation under the Act." 42 U.S.C. § 7475(a)(1) and (a)(4) (emphasis added). The following definition of BACT in section 169(3) of the Act also makes clear that the BACT review is based on the proposed project, as opposed to something fundamentally different:

> an emission limitation based on the maximum degree of reduction of each pollutant subject to regulation under this Act emitted from or which results from any major emitting facility, which the permitting authority, on a *case-by-case basis*, taking into account energy, environmental, and economic impacts and other costs determines is achievable for *such facility* through application of production processes and available methods, systems, and techniques, including fuel cleaning, clean fuels, or treatment or innovative fuel combustion techniques for control of such pollutant.

42 U.S.C. § 7479(3) (emphasis added). The phrases "proposed facility" and "such facility" in section 165(a)(4) and 169(3) refer to the specific facility proposed by the applicant, which has certain inherent design characteristics. The Act also requires BACT to be determined "on a case-by-case basis." The case-specific nature of the BACT analysis indicates that the particular characteristics of each facility are an important aspect of the BACT determination. Thus, the Act requires that permitting authorities determine BACT for each facility individually, considering the unique characteristics and design of each facility.

As the group of commenters has also pointed out, the statutory definition of BACT also requires permitting authorities in selecting BACT to consider "application of production processes and available methods, systems, and techniques, including fuel cleaning, clean fuels, or treatment or innovative fuel combustion techniques." 42 U.S.C. §7479(3). EPA has interpreted this phrase to require that permitting authorities evaluate both add-on pollution control technologies and lower polluting process in the BACT review. *Prairie State* at 33.

Considering these provisions together, the Act requires that we conduct the BACT analysis on a "case-by-case" basis on the "proposed facility" while concurrently considering the "application of production processes and available methods, systems and

techniques" that could alter the proposed facility. The statute does not provide clear direction on how EPA is to reconcile these concepts and simultaneously consider the particulars of the facility proposed by the applicant while also assessing the use of methods or technology that could modify those particulars. Where a statute is ambiguous and Congress has not spoken to the precise issue, an administrative agency may formulate a policy to resolve the issue, provided that the policy is based on a permissible construction of the statute. *Chevron v. Natural Resources Defense Council*, 104 S.Ct. 2778, 2782 (1984). In this instance, sections 165 and 169(3) of the Clean Air Act are permissibly construed to authorize EPA and permitting authorities to establish some level of balance between the case-by-case nature of a BACT determination and the need to consider available processes, methods, systems, and techniques to reduce emissions. EPA's policy against redefining a source as part of the BACT analysis reasonably harmonizes the competing BACT obligations by requiring the permitting authority to consider potentially applicable processes, methods, systems, or techniques that may reduce pollution from the type of source proposed, provided such processes or techniques do not fundamentally redefine the basic design or scope of the facility proposed by the permit applicant.

EPA does not read the legislative history cited by the commenter to require a detailed evaluation of the IGCC technology in the BACT analysis for every proposed facility that generates electricity from coal. That Senator Huddleston intended for the phrase "innovative fuel combustion techniques" to encompass "gasification" or "low Btu gasification" does not necessarily require EPA or other permitting authorities to identify the IGCC option as a candidate for further analysis at step 1 of a top-down BACT review. The "innovative fuel combustion techniques" phrase appears in the BACT definition among a list of examples of things included in the phrase "production processes and available methods, systems, and techniques." Thus, the "innovative fuel combustion" language, like the phrase it modifies in the definition of BACT, is limited by other language discussed above that requires BACT to be applied to each proposed facility and determined on a case-by-case basis. Thus, even assuming that coal gasification was in all respects an innovative fuel combustion technique for producing electricity from coal, we do not interpret the Clean Air Act to require an "innovative fuel combustion technique" to be subject to a detailed BACT review when application of such a technique would re-design the proposed source to the point that it becomes an alternative type of facility, which, as discussed below, we believe would be the case if the IGCC technology were applied to Deseret's project.

Furthermore, it is not clear from the terms of his statement that Senator Huddleston himself intended to require mandatory review of coal gasification in every case where such an option was not proposed by the permit applicant. Senator Huddleston said the purpose of the amendment was to leave no doubt that "all actions taken by the fuel user are to be taken into account." This phrase suggests the Senator wanted to make sure that, when a fuel user was proposing an innovative fuel combustion technique, such as coal gasification, that such actions by the fuel user would be taken into account and credited in the determination of BACT for the proposed facility. Thus, the Senator's statement could be read to express an intent similar to that expressed in a

subsequent Congress when adding the phrase "clean fuels" to the definition of BACT in the 1990 amendments of the Clean Air Act. Pub. Law No. 101-549, § 403(d), 104 Stat. at 2631 (1990). At the time "clean fuels" was added to the list that includes "innovative fuel combustion techniques," the relevant Senate committee report stated the following in consecutive paragraphs:

> The Administrator may consider the use of clean fuels to meet BACT requirements if a permit applicant proposes to meet such requirements using clean fuel. . . . In no case is the Administrator compelled to require mandatory use of clean fuels by a permit applicant.

S. Rep. 101-228, at 338 (describing section 402(d) of S. 1630). Based on this legislative history, EPA does not interpret the list of examples that appear in the BACT definition after the phrase "production processes, methods, systems, or techniques" to require mandatory evaluation of each of those options at advanced stages of the BACT analysis, regardless of the degree to which such an option would redefine the type of facility proposed by the permit applicant.

Although EPA reads the Act to preclude redefining the source and to draw a distinction between alternatives to the proposed source and lower polluting process that can be applied to the proposed source, EPA does not interpret the Clean Air Act to obligate a PSD permitting authority to accept all elements of a proposed project when determining BACT. To the contrary, EPA recognizes that the Act calls for an evaluation of the "application of production processes and available methods, systems, and techniques." 42 U.S.C. §7479(3).

As the Board observed in *Prairie State*, EPA's policy against redefining the source is only relevant when considering lower polluting processes and would not permit a reviewing authority to rule out "add-on controls" at Step 1 of the BACT analysis. Slip. op. at 33. Further, although EPA does not require a source to consider a totally different design, some design changes to the proposed source are within the scope of the BACT review. *See Knauf Fiber Glass*, 8 E.A.D. at 136. As the Board observed in the *Prairie State* case, the central issue in situations involving a lower polluting process concerns "the proper demarcation between those aspects of a proposed facility that are subject to modification through the application of BACT and those that are not." Slip. Op. at 26. The Board observed that one of the permit issuer's tasks at step 1 of the BACT analysis is to "discern which design elements are inherent to [the applicant's] purpose, articulated for reasons independent of air quality permitting, and which design elements may be changed to achieve pollutant emissions reductions without disrupting the applicant's basic business purpose for the proposed facility." *Prairie State*, slip. op. at 30.

Since this line can be difficult to draw in each case, the Administrator and Environmental Appeals Board have generally recognized that the decision on whether to include a lower polluting process in the list of potentially-applicable control options compiled at Step 1 of the top-down BACT analysis is a matter within the discretion of the PSD permitting authority. *Knauf Fiber Glass,* 8 E.A.D. at 136; *Old Dominion*, 3 E.A.D.

at 793; *Hawaiian Commercial*, 4 E.A.D. at 100 & n.9. The Administrator and the EAB have usually respected the decisions of the permitting authority and only remanded permits in cases where it was clear that the permitting authority abused its discretion by excluding a particular option from consideration in the BACT review. *Knauf Fiber Glass*, 8 E.A.D. at 140. *See, e.g., Hibbing Taconite Company*, 2 E.A.D. 838, 843 (Adm'r 1989). The Seventh Circuit affirmed this view in upholding the EAB's *Prairie State* decision, emphasizing the discretion given the permitting authority in making the technical judgment as to "where control technology ends and a redesign of the 'proposed facility' begins." *Sierra Club v. EPA,* slip op. at 5.

In its review of this issue in *Hibbing*, the Board considered whether the option in question would "require any fundamental change to Hibbing's product, purpose, or equipment." *Hibbing* at 843 n. 12. In *Prairie State*, where the use of the alternative coal source arguably did not significantly affect the power-generating equipment to be used at the proposed source, the Board focused on the applicants "objective or purpose" to the extent that purpose was "articulated for reasons independent of air quality permitting." *Prairie State*, slip. op. at 30.

With respect to the project proposed by Deseret, our assessment is that the application of the IGCC process to the Deseret facility would fundamentally change the nature of the proposed major source. The IGCC option would both fundamentally change the basic design of the equipment that Deseret proposes to install and fundamentally alter the objective and purpose of Deseret to make productive use of a coal supply that was previously considered a waste. Thus, we consider the IGCC process to be an alternative to the proposed source that should be evaluated under section 165(a)(2) of the Clean Air Act rather than as a BACT candidate under section 165(a)(4).

From an equipment perspective, Deseret has proposed a facility that fires pulverized waste coal in a fluidized mixture with limestone and inert materials, in a boiler to generate steam to drive an electric turbine. An IGCC facility uses a chemical process to first convert coal into a synthetic gas and to fire that gas in a combined cycle turbine. "Final Report, Environmental Footprints and Costs of Coal-Based Integrated Gasification Combined Cycle and Pulverized Coal Technologies," EPA-430/R-06/006, July 2006. The combined cycle generation power block of an IGCC process employs the same turbine and heat recovery technology that is used to generate electricity with natural gas at other electric generation facilities. Thus, this portion of the IGCC process is very similar to existing power generation designs that EPA has agreed would redefine the basic design of the source when an applicant proposed to construct a pulverized coal-fired boiler. *SEI Birchwood Inc*, 5 E.A.D. 25 (1994); *Old Dominion Electric Cooperative Clover, Virginia*, 3 E.A.D. 779 (Adm'r 1992). Furthermore, the core process of gasification at an IGCC facility is fundamentally different than a boiler. Coal gasification is more akin to technology employed in the refinery and chemical manufacturing industries than technologies generally in use in power generation (i.e. a controlled chemical reaction versus a true combustion process). Use of coal gasification technology would necessitate different types of expertise on the part of the applicant and employees to produce the desired product (electricity). Thus, these fundamental

differences in equipment design are sufficient to conclude that the IGCC process would redefine the proposed source. Similarly, in *Sierra Club v. EPA*, slip op. at 4 (7th Cir. Aug. 24, 2007), the Court upheld the EAB's decision that use of low-sulfur coal that was available only at a distance from a proposed plant would redefine the source, because the plant was designed to use higher sulfur coal located at a nearby mine. As the Court explained, "to convert the design from that of a mine-mouth plant to one that burned coal obtained from a distance would require that the plant undergo significant modifications – concretely, the half-mile-long conveyor belt, and its interface with the mine and the plant, would be superfluous and instead there would have to be a rail spur and factilities for unloading coal from rail cars and feeding it into the plant." *Id.*

Furthermore, Deseret Power's proposal calls for extracting the remaining heating value of the waste coal that has accumulated over the past 20 years in order to conserve other natural resources. In light of the technical difficulties of using IGCC for waste coal (described in detail below), IGCC would not serve the basic purpose of the project, which is to take advantage of the current waste coal reserves and future waste coal generated from the coal washing operations that provide the existing Bonanza Unit 1 with its coal. *See* Letter from Ed Thatcher, Deseret Power, to Richard R. Long, EPA Region 8, May 10, 2005. Thus, in addition to fundamentally changing the basic design of the source that Deseret proposes to construct, the IGCC option would also have the effect of regulating the applicant's objective or purpose for the proposed facility by precluding the use of the waste coal resource. The record reflects that Deseret is seeking to use waste coal for reasons independent of air quality permitting. See *Prairie State*, slip. op. at 30.

We acknowledge that in the *Prairie State* case, the EAB recognized that IGCC technology could be listed as a potentially applicable option at step 1 of the BACT analysis, as Illinois EPA had elected to do in that case. However, the Board's opinion in *Prairie State* did not interpret the Clean Air Act to require IGCC to be listed as a potentially applicable control option at step 1 for every permit application involving a coal-fired steam electric generating unit. In *Prairie State*, the Board did not directly address the issue raised by the Petitioners comment on the Deseret permit because Illinois EPA chose, in an exercise of its discretion, to list the IGCC option at step 1 of the BACT analysis for the proposed facility and further analyze the option. IEPA ultimately eliminated the option at step 2. *See Prairie State*, slip. op. at 45. In Prairie State, the Board pointed to IEPA's consideration of the IGCC option beyond step 1 to illustrate that there was no question that IEPA had conducted a sufficiently thorough step 1 BACT analysis in that case, because IEPA had even considered an option that "would have required extensive design changes to Prairie State's proposed facility." Slip. op. at 36. The Board did not conclude that IGCC, or any other option involving such extensive design changes, had to be listed as a potentially applicable option at Step 1 in each case or find that it would be an abuse of a permitting authorities discretion to decline to list IGCC at Step 1 of the BACT analysis for the type of facility proposed by Deseret. The Board continued to recognize that the decision of where to draw the line between BACT options listed at step 1 and alternatives to the proposed source is ultimately a matter within the discretion of the permitting authority. *Prairie State* slip. op. at 29 n. 22.

Moreover, even if EPA was to list IGCC as a potentially applicable option at step 1 of the BACT analysis for the facility proposed by Deseret, the IGCC option could also be eliminated at step 2 of the top-down BACT analysis for the facility proposed by Deseret. It is not technically feasible to use Deseret's waste coal in the IGCC process. Based on an analysis of samples, Deseret's waste coal has an average heating value of approximately 4,000 Btu/lb, with a range of 3,051 Btu/lb to 5,326 Btu/lb, and ash content of the waste coal is estimated by Deseret to be in excess of 50 percent by weight on a dry basis. *See* Statement of Basis at 9. As explained below, IGCC units are not designed to operate, nor have they been operated, with coal that has a heating value as low, or ash content as high, as the waste coal that will be utilized for the proposed project.

A recently issued EPA report on IGCC states that "relatively little research or commercial work has been done to investigate gasification of low rank coals, including subbituminous and lignite, for electric generation purposes. The existing IGCC plants use bituminous coal as feedstocks." *See* "Final Report, Environmental Footprints and Costs of Coal-Based Integrated Gasification Combined Cycle and Pulverized Coal Technologies," EPA-430/R-06/006, July 2006, page ES-1, available in the Administrative Record for this permit and through website at:

http://www.epa.gov/air/caaac/coaltech/2007_01_epaigcc.pdf

The report only discusses IGCC units as a possibility for use with bituminous, subbituminous and lignite coals. Deseret's waste coal is a lower rank of coal than subbituminous or lignite, having much lower heat content and much higher ash content than either subbituminous or lignite.

The above-mentioned EPA report states that there are currently two commercial-scale, coal-based IGCC plants in the U.S. and two in Europe. The U.S. projects (Wabash River Repowering Project in Indiana and Tampa Electric Polk Power Station in Florida) were both supported by the DOE's Clean Coal Technology demonstration program. Both plants have operated on bituminous coals and petroleum cokes; no use of low-rank coal at these facilities is known. EPA report at 2-6 and 2-7.

Another publication on IGCC analyzes the impact that various coal parameters have on various gasifiers, based on actual operation of the gasifiers. *See* "Coal Quality Handbook for IGCC," published by Cooperative Research Centre for Coal in Sustainable Development, Technology Assessment Report 8, April 1999, available through website at http://www.ccsd.biz/products/qualitybook.cfm .

Page 14 of the Handbook lists the maximum ash content of the coal that can be handled by various types of gasifiers. For a moving bed gasifier, the ash content has to be less than 15 percent; for an entrained bed gasifier, less than 25 percent; and for a fluidized bed gasifier, less than 40 percent. As mentioned above, Deseret's waste coal will have ash content in excess of 50 percent.

In addition to the Wabash River and Tampa Electric IGCC projects, the above-mentioned Handbook reviews several other IGCC demonstration or pilot projects, utilizing various gasifier designs, and the required characteristics of the coal. These projects include:

BGL IGCC Process, owned/operated by British Gas and Lurgi
Demkolec IGCC plant, owned/operated by Shell
Nedo facility, owned/operated by Engineering Research Associates
Pinon Pine Power Project, owned/operated by Sierra Pacific and MK Kellogg
Prenflow IGCC Process, owned/operated by Krupp Koppers and Siemens AG

However, all of these projects require coal with higher heat content and lower ash content than Deseret's waste coal. Of particular significance is that all of these projects (as well as the Wabash River and Tampa Electric projects) require coal with ash content less than 25 percent by weight on a dry basis. This is less than half the ash content of Deseret's waste coal. The Handbook also indicates that the above-mentioned IGCC projects generally require coal with much higher heat content than Deseret's waste coal, 8,100 to 13,760 Btu/lb, compared to Deseret's range of 3,051 to 5,326 Btu/lb, respectively. *See* Handbook at 22-28.

Inquiries with representatives of IGCC test programs confirmed that IGCC units have not been tested on coal with heat content as low as Deseret's waste coal. The U.S. Department of Energy's Power Systems Development Facility near Wilsonville, Alabama, has only utilized coal as low as 6,000 to 7,000 Btu/lb. The National Energy Technology Institute is also not aware of any IGCC unit utilizing coal with the low heating value that will be used in Deseret Power's proposed WCFU. (Ref: June 9, 2004 letter from Ed Thatcher, Deseret Power, to Richard R. Long, EPA Region 8.)

Response #2.b: Disagree. As was recognized by commenters in the comment letter, state decisions as to how to conduct the BACT analysis do not necessarily set the bar for EPA. As discussed above, the decision of where to draw the line between alternatives to the proposed source is a discretionary matter. The fact that some states have elected to list IGCC at step 1 of the BACT analysis for a coal-fired steam electric generating facility does not require EPA to do so if EPA's reasoned assessment is that the option would redefine the proposed source. EPA does not interpret the Clean Air Act to mandate evaluation of IGCC in a BACT analysis in cases involving proposed coal-fired steam electric generating facilities. We do not read the state examples cited by commenters to be based on a contrary interpretation of the Clean Air Act, but rather to reflect policy decisions in those states to conduct a more extensive analysis. Even if a state were to conclude that evaluation of IGCC was mandatory under its interpretation of the Clean Air Act or state law, such a decision by a state is not binding on EPA. Furthermore, because Illinois administers the Federal PSD program under a delegation agreement with EPA Region V, Illinois must act in a manner consistent with EPA's interpretation of the Clean Air Act and controlling regulations.

Response #2.c: Disagree. Regarding EPA's letter to Utah on Nevco, the commenters incorrectly characterized the letter as a determination on evaluating IGCC. Letters from EPA to states providing comments on proposed state PSD permits are not final EPA actions. *See Public Service Co. of Colorado v. Environmental Protection Agency, 225 F 3d 1144 (10th Cir.2000).*

Regarding EPA's request to Deseret Power to provide information regarding IGCC as an alternative to its planned CFB boiler, EPA's correspondence with Deseret merely explored IGCC as a possibility and made no final determination regarding IGCC. (Ref: Letters from Richard R. Long, EPA Region 8, to Ed Thatcher, Deseret Power, dated November 22, 2004, December 29, 2004, and June 22, 2005.)

Response #2.d. Partially agree. Since EPA's judgment is that use of the IGCC process would redefine the proposed source and thus need not be listed as an option at Step 1 of the BACT analysis for the Deseret facility, EPA is treating this comment as a request that EPA consider IGCC technology as an alternative to the proposed source in accordance with section 165(a)(2). EPA agrees with commenters that IGCC technology has many potential environmental benefits, but EPA is not requiring Deseret to employ this alternative technology for the reasons set forth below.

Under CAA section 165(a)(2), a PSD permit may not be issued unless, among other things, "a public hearing has been held with opportunity for interested persons … to appear and submit written or oral presentations on the air quality impact of such source, alternative thereto, control technology requirements, and other appropriate considerations…." EPA interprets section 165(a)(2) of the CAA to require that EPA consider and provide a reasoned response to comments identifying alternatives to the proposed source. *Prairie State*, slip op. at 38-41.

As EPA has observed in other contexts, EPA considers IGCC to be one of the most promising alternative technologies in reducing the environmental consequence of generating electricity. *See* "Final Report, Environmental Footprints and Costs of Coal-Based Integrated Gasification Combined Cycle and Pulverized Coal Technologies," EPA-430/R-06/006, July 2006, at Forward. EPA has undertaken several initiatives to provide incentives for development and deployment of this technology. This approach is consistent with U.S. policy reflected in the Energy Policy Act of 2005, which established loan guarantees and tax incentives to encourage, but not require, development of IGCC facilities.

As a general matter, assessing whether IGCC is an appropriate alternative may entail a robust analysis of a broad range of factors. Such an analysis is not necessary in this case because there are two specific features of this plant that make IGCC a technically unfeasible option: fuel and plant size. The main fuel for this plant is waste coal, which has an ash content ranging from 40 to 56% and a heating value ranging from 3,000 to 5,400 Btu/lb. There exists no IGCC operating experience with this type of coal. An ash content as high as found in this waste coal would be a major issue for the design and operation of a gasifier (an integral part of an IGCC plant). In addition, the proposed

110 MW size for this plant is too small to be considered viable for an IGCC application. The four operating IGCC installations in the world (two of which are in the U.S.) are each greater than 250 MW in size. In general, the currently proposed IGCC plants by the U.S. power industry are larger than these operating IGCC installations. These plants are being proposed in larger size because they would be relatively less expensive per MW of electricity generation. Thus, even if it were possible to build a 110 MW IGCC plant, it would most likely be too costly to be considered economically viable.

More broadly, EPA believes the environmental and energy security goals of the United States are best served by encouraging the development of all forms of clean coal technology and the development of alternative fuels. Further, providing a reliable and secure supply of electricity to meet growing demand in the United States without adverse affects on air quality will require the use of a diverse array of power producing technologies and innovations in pollution control technology for each type of generating unit. Deseret's proposal to utilize a previously untapped reserve of waste coal with the best pollution control technology available for this type of source is consistent with these goals. In summary, comment #2 has not resulted in any changes to the permit.

3. SUPERCRITICAL CFB BOILER

Comment #3:

One group of commenters asserted that EPA should have required consideration of a supercritical CFB boiler in the BACT analysis for the Bonanza WCFU. Commenters cited discussion in a Western Governors Association Technology Working Group report on advanced clean coal technologies.

Response #3:

Agree. In response to this comment, EPA has evaluated a supercritical CFB boiler as a BACT option and has determined that since there are no known supercritical pressure turbines available in the size needed for the WCFU project, this option should be eliminated at step two of the top-down BACT analysis as technically infeasible, because it is not available and applicable for the WCFU project. *See In re Prairie State Generating Co.,* PSD Appeal No. 05-05, slip op. at 14-18 (EAB Aug. 24, 2006) (summarizing and describing steps in the top-down BACT analysis). *Accord In re Three Mountain Power, L.L.C.,* 10 E.A.D. 39, 42-43 n.3 (EAB 2001); *In re Knauf Fiber Glass, GmbH,* 8 E.A.D. 121, 129-31 (EAB 1999); *In re Hawaii Electric Light Co.,* 8 E.A.D. 66, 84 (EAB 1998).

At the first step of the top-down BACT analysis, all demonstrated and potentially applicable control technology alternatives must be identified. This must include a survey of production processes or innovative technologies that have a practical potential for application to reduce relevant emissions at the source type being evaluated. (*Prairie State*, slip op. at 17.) At the second step, "technically infeasible" options are eliminated. A technology is feasible if either it is demonstrated, i.e. installed and operated

successfully at a similar facility, or it is both "available" and "applicable." *Id.* A technology is considered "available" if it can be obtained by the applicant through commercial channels. An available technology is "applicable" if it can reasonably be installed and operated on the source type under consideration. If a technology is not demonstrated, or is found to be unavailable or not applicable, that technology will be eliminated from BACT consideration as technically infeasible. (*Three Mountain Power*, 10 E.A.D. at 42-43 n.3.)

As described by Babcock & Wilcox, a major boiler supplier, a supercritical boiler (regardless of combustion process, i.e. PC-fired, CFB, gas-fired, etc.) is designed to operate with the working medium, i.e. water, at a pressure above the critical point (3200 psia). At this pressure the medium cannot be separated to liquid and steam thus natural circulation is impossible, and the fluid is pumped through all heat absorbing tubes (called "Once-Through" in the boiler industry, versus natural circulation that the sub-critical pressure WCFU boiler is based on). (Ref: e-mails and attachments from Ed Thatcher, Deseret Power, to Mike Owens, EPA Region 8, November 6, 2006.)

The use of supercritical pressure in a power plant affects the design of all components within the plant cycle, boiler, turbine, pumps, etc. The steam cycle is based on available turbine designs. The boiler and other equipment are designed to meet the steam cycle defined by the turbine. This technology is being deployed currently at pulverized coal utility boilers. As such, EPA agrees with commenters that it is appropriate to consider supercritical technology, as a technology transfer control option under step one of the top-down BACT analysis.

However, according to Babcock & Wilcox and Foster-Wheeler, two major boiler suppliers, supercritical pressure steam turbines are not available in the size needed for the WCFU project. The smallest supercritical pressure turbine currently known to be available is three to four times larger than is needed for the WCFU project, which will operate at approximately 1,500 psia and is thus based on a sub-critical steam cycle. (Ref: e-mails and attachments from Ed Thatcher, Deseret Power, to Mike Owens, EPA Region 8, November 6, 2006 and November 13, 2006.)

In addition, the following information was provided by Siemens Power Systems to Deseret Power (forwarded to EPA Region 8 via e-mail from Deseret Power on November 13, 2006):

> *"To our knowledge, no manufacturer offers supercritical steam turbines in 110-120 MW range. The reason is that you would be unlikely to see any significant performance improvements for units that small. Key reasons are as follows:*
>
> *1. When you go to supercritical steam conditions the specific volume of the steam is reduced because of the higher pressure. That means the blades in the HP section have to be shorter. A major source of inefficiency in steam turbines is due to "flow disruptions" at the top and bottom of the blade where the moving flow meets the stationary rotor or casing. As the blades get shorter the impact of this "end wall" condition increases which in turn increases the flow losses.*

2. The supercritical conditions require a once-through boiler which requires a more powerful feed pump drive (higher pressures). That decreases plant efficiency and if you can't make that difference up with improved cycle performance, supercritical makes no sense.

We generally don't see units less than about 500 MW being built as supercritical because the performance improvement isn't significant and the unit is more expensive than subcritical."

The Western Governors Association report (cited by commenters) states that "no supercritical CFB combustion units have been demonstrated on a commercial scale." (Ref: Western Governors Association Technology Working Group's report on advanced clean coal technologies, second page of section titled "Advanced Clean Coal Technology Descriptions." The report is included in materials provided by Deseret Power to EPA via e-mail of November 6, 2006. Those materials are included in the Administrative Record for issuance of the WCFU permit.) EPA is aware of only one supercritical CFB boiler that has been proposed, designed and/or constructed anywhere in the world. As of January 11, 2006, design of that unit had not yet been completed. The unit is being designed for Poland's Poludniowy Koncern Energetyczny (PKE) for installation at its power plant at Lagisza in southern Poland. The proposed unit will have an output of 460 MW (four times larger than Deseret Power's proposed WCFU) and is being designed to fire bituminous coal. It is currently scheduled to begin operation in 2009. (Ref: Foster-Wheeler press release, January 11, 2006. The press release is included in materials provided by Deseret Power to EPA via e-mail of November 6, 2006. Those materials are included in the Administrative Record for issuance of the WCFU permit.)

Supercritical CFB boilers, while potentially applicable as a BACT option, are not a "demonstrated" technology under the BACT analysis, as the only such boiler EPA is aware of (the PKE boiler planned in Poland) has not been installed and operated successfully. Further, the technology is not "available" under the BACT analysis since, as explained above, it is not commercially available for CFB boilers, and supercritical pressure steam turbines are not available in the size needed for the WCFU project. Therefore, this technology is eliminated at step two of the top-down BACT analysis because it is undemonstrated and is not available.

The Environmental Appeals Board (EAB) has considered the question of whether certain technologies are available. The EAB has stated that "[i]f the technology is not available, the permit applicant is under no duty to consider it in the BACT analysis." *In re Pennsauken County, New Jersey, Resource Recovery Facility*, 2 E.A.D. 667, 671-672 (Nov. 10, 1988). The EAB has recognized that "[t]he question of availability for purposes of BACT is a practical, fact determination, using conventional notions of whether the technology can be put into use." *Id.* See also, *In re Maui Electric*, 8 E.A.D. 1, 13-16 (Sept. 10, 1998). EPA has evaluated a supercritical CFB boiler as a BACT option for Deseret Power's WCFU project and has found that there are no supercritical pressure turbines available in the size needed for the project. Therefore, EPA has

concluded that a supercritical CFB boiler is technically infeasible for this project and has eliminated it at step two of the BACT analysis.

In summary, comment #3 has not resulted in any change to the permit; however, the Statement of Basis has been changed, to add an explanation of why a supercritical CFB boiler was eliminated as a BACT control option at step two of the BACT analysis. Since BACT determinations are case-by-case, EPA's determination regarding a supercritical CFB boiler for the WCFU project should not be construed as a statement about what the determination should be for other projects.

4. PROPOSED BEST AVAILABLE CONTROL TECHNOLOGY (BACT) EMISSION LIMITS

4.a -- Cleaner coals:

Comment #4.a: One group of commenters alleged that EPA's analysis of cleaner coals as a BACT option was inadequate. The commenters indicated that while EPA did provide a cost analysis of using all "run-of-mine" coal from the Deserado mine and the resultant additional pollutant reductions (draft Statement of Basis at 24-28), EPA did not provide a comparison of the cost of using "run-of-mine" coal, either in part or wholly, compared to the cost other coal-fired electric utility CFB boilers in the region are paying for coal.

Commenters further alleged that EPA did not provide any comparative cost analysis for use of coal from other mines in the region, either wholly or in part as a blend with the Deserado waste coal. Commenters argued that such analyses are necessary to give context to this evaluation (e.g., *In re Inter-Power of New York. Inc.,* PSD Appeal Nos. 92-8 and 92-9, Decided March 16, 1994), arguing that in determining whether the cost of a control technology is reasonable, the cost must be compared to what other similar sources have had to bear.

As an example, commenters argued that EPA should have provided a comparison to the recently permitted Sevier Power Company's CFB power plant to be located in Sigurd, Utah. That facility will be burning a higher quality bituminous coal than the waste coal proposed for the Bonanza WCFU, and will be subject to lower permit emission limits than the WCFU for SO_2, total PM/PM_{10}, carbon monoxide and sulfuric acid.

Commenters alleged that EPA must analyze and provide data on the cost and quality of coal that the Sevier Power Company and other recently proposed power plants in the region are required to incur before it can determine that the cost of using "run-of-mine" fuel from the Deserado mine – either wholly or in part – is unreasonable. The commenters also suggested that EPA provide a similar analysis for using other higher quality coal available in the region, either wholly or as a blend with the waste coal.

Response #4.a:

Partially agree. As described below, EPA has supplemented the analysis of alternative coals in the Statement of Basis, to: (1) explain more fully, in terms of cost per ton of additional pollutant removed from the atmosphere, why use of coal from any mine in the region other than the Deserado mine, rather than waste coal from the Deserado mine, would be cost-prohibitive as a BACT option, and (2) explain why the BACT option of using ROM coal from the Deserado mine, as well as the BACT option of using coal from any other mine in the region, is cost-prohibitive when compared to the cost of BACT that other similar sources have to bear.

In presenting the analysis for alternative coal from another mine in the region as a BACT option, EPA is not taking a position on whether the use of a coal supply other than the one proposed by the applicant must be evaluated in the BACT analysis for the WCFU or similarly situated facilities. After EPA issued the draft permit for the WCFU, the EPA Environmental Appeals Board issued its opinion in *In re: Prairie State Generating Company*, PSD Appeal No. 05-05 (Aug. 24, 2006). This opinion established that there may be circumstances under which the permitting authority has the discretion not to list alternative coal supplies as an option at Step 1 of the BACT analysis, because such an option could fundamentally redefine the source.

However, we need not address whether this permit presents a similar circumstance, since the draft Statement of Basis included the use of a cleaner coal as an option and evaluated the economic impact of requiring the applicant to use exclusively mined coal from the Deserado mine rather than waste coal, or alternatively, exclusively mined coal from other mines rather than waste coal. (Draft Statement of Basis at pages 25-29.) Since EPA already started down this path of looking at other coal supplies for this permitted project, EPA has supplemented its analysis to further illustrate why it is appropriate to eliminate this option for this permit. Specifically, as described below, EPA is supplementing its BACT analysis in section VI.D.2 of the Statement of Basis, "Alternative coal from other mines," using a cost methodology in terms of dollars per ton of additional pollutant removed, similar to the cost methodology used in section VI.D.1, "Alternative coal from Deserado mine."

The first step in the alternative coal analysis is to determine what the alternative coal would cost, per ton of coal delivered. EPA asked Deseret Power to provide an estimate of what the total cost would be, per ton of coal delivered, to have coal supplied to the WCFU from mines in the region other than the Deserado mine. EPA asked that the estimate be for the least total cost scenario of the various other mines that could potentially supply coal. EPA further asked for a breakdown of mine-mouth ("Free-On-Board") cost plus transportation cost. (Ref: November 14, 2006 e-mail from Mike Owens, EPA Region 8, to Ed Thatcher, Deseret Power, included in the Administrative Record for issuance of the WCFU permit.)

Deseret Power responded that its letter to EPA dated May 10, 2005, at page 5, provided a cost estimate for coal purchased on the open market and delivered to the WCFU unit. The estimated cost for the coal at that time was $40 to $45 per ton delivered, which included the estimated delivery charge of $15 per ton. The FOB mine cost for the coal was estimated to be $25 to $30 per ton at that time. According to the November 13, 2006 issue of Coal Outlook (copy attached to Deseret Power's November 15, 2006 e-mail), the FOB mine cost for coal in Utah has increased to $37.75 per ton for current purchases of coal.

As mentioned in the draft Statement of Basis, the Bonanza plant is approximately 75 miles from the nearest rail transportation and approximately 100 miles by truck from the nearest alternative source of coal. The cost to construct a rail line to connect to the interstate rail system has been estimated by Deseret Power to exceed $300 million. (EPA has eliminated this option as too expensive.) The cost to truck the coal from the nearest

alternative coal source (i.e., other than the Deserado mine) was estimated by Deseret Power to be at least $15/ton. (Ref: Sept. 13, 2005 letter from Deseret Power to EPA, page 3, footnote 1, included in Administrative Record for issuance of the WCFU permit.)

In more recent correspondence to EPA, Deseret Power stated that it believes the delivery cost to haul the coal from the nearest alternative mines to the Bonanza plant site would still be about $15 per ton. Therefore, the current delivered cost would be $37.75 plus $15.00, or about $52.75 per ton delivered. (Ref: November 15, 2006 e-mail from Ed Thatcher, Deseret Power, to Mike Owens, EPA Region 8, included in the Administrative Record for issuance of the WCFU permit.) Being the cost from the nearest alternative mines, this 'cheapest delivered' cost is a conservative estimate, i.e., yielding the lowest calculated BACT cost to switch to coal from a mine other than the Deserado mine.

The next step in the analysis is to determine the annual cost of switching from Deserado waste coal to alternative coal from another mine. This requires a determination of how much alternative coal is necessary to achieve the equivalent annual boiler heat output as combustion of 1.2 million tons of waste coal per year, which is Deseret Power's projected waste coal usage rate. To make this determination, it is necessary to know the estimated heat content of the alternative coal. The CFB boiler project cited by commenters, Sevier Power Company, would use coal with an estimated heat content range of 10,200 to 12,000 Btu/lb, with average heat content of 11,390 Btu/lb. (Ref: "New Source Plan Review" by Utah Division of Air Quality, dated December 23, 2003, for the Sevier Power Company project, page 13, Table I-2, available online at http://www.airquality.utah.gov/Permits/PmtPowerPlants.htm.)

Rather than rely just on the Sevier project cited by commenters for an estimate of heat content of available coals in the region, EPA also examined a recent Utah Geological Survey (UGS) report, which lists heat content of coal at Utah mines ranging from 11,243 Btu/lb to 13,052 Btu/lb. (Ref: "Annual Review and Forecast of Utah Coal, Production and Distribution - 2005," published August 2006 by Utah Geological Survey, Open-File Report 481, Table A8: "Average Coal Quality at Utah Mines, 2005." Report available online at http://ugs.utah.gov/online/ofr.ofr-481.pdf.) For the sake of this analysis, EPA will use the upper end of this range (13,052 Btu/lb) as a conservative assumption, i.e., yielding the lowest calculated BACT cost to switch to alternative coal.

Since Deseret Power's waste coal has an average heat content of about 4,000 Btu/lb, EPA calculates that it would require about 367,760 tons per year of alternative coal rated at 13,052 Btu/lb heat content, to achieve the equivalent annual WCFU boiler heat output as combustion of 1.2 million tons per year of waste coal. The coal purchase cost of the alternative coal would therefore be:

$52.75/ton x 367,760 tons/year = $19,400,000/year.

EPA stated in the draft Statement of Basis that the cost of waste coal would be about $5 per ton delivered. The annual cost of using the waste coal would be:

$5/ton x 1,200,000 tons/year = $6,000,000/year.

(Note: The draft Statement of Basis indicated $5/ton x 1,200,000 tons/year = $3,405,000. This was an inadvertent mathematical error.) The incremental cost to use entirely alternative coal from another mine in the region, rather than waste coal, would therefore be the difference in cost of the two coals, which is $13,400,000/year.

The next step in the analysis is to determine the potential annual emission reductions that could be achieved by switching from waste coal to alternative coal from another mine. In the draft Statement of Basis, EPA presented its calculation of the reductions that could be achieved for each PSD pollutant, if emissions are reduced from the proposed WCFU permit allowables down to the lowest BACT determination EPA is aware of anywhere for a CFB boiler project (including the Sevier Power Company project cited by commenters). For condensible PM, EPA has since revised its estimate of lowest achievable emission rate down to 0.005 lb/MMBtu, to correspond to the condensible portion of the BACT emission limit for total PM/PM_{10} in the Utah permit for the Sevier Power Company project.

Potential Emission Reductions Due to a Switch From Waste Coal to Alternative Coal from Another Mine For Deseret Power's Proposed WCFU

Pollutant	Proposed Emission Limit for WCFU (lb/MMBtu)	Lowest BACT Determination Anywhere for a CFB Boiler Project (lb/MMBtu)	Equivalent Annual Reduction (tons/year)
NO_x	0.080	0.07	63
SO_2	0.040	0.022	114
CO	0.15	0.10	316
H_2SO_4	0.0035	0.0024	7
Filterable PM	0.012	0.010	13
Condensible PM	0.019	0.005	88

NOTE #1: The Sevier Power Company project cited by commenters is permitted at 0.1 lb/MMBtu for NOx, 0.022 lb/MMBtu for SO2, 0.115 lb/MMBtu for CO, 0.0024 lb/MMBtu for H2SO4, and 0.015 lb/MMBtu. "Lowest BACT Determination" values listed above are at least as low.

NOTE #2: The proposed WCFU permit has no separate BACT emission limit for condensibles. The figure of 0.019 lb/MMBtu above is an estimate based on best information available to EPA and the proposed emission controls for the WCFU, as described in the draft Statement of Basis.

EPA believes it is unlikely that lower emissions than listed above could be achieved on any coal in the Region. As explained in Note #1 above, the figures listed as "Lowest BACT Determination Anywhere for a CFB Boiler Project" are at least as low as the BACT determination for each pollutant at the Sevier project cited by commenters. Further, based on "Average Coal Quality at Utah Mines, 2005," listed in the afore-

mentioned UGS report, it appears to EPA that the proposed coal for the Sevier project is at least as clean, in terms of ash content and sulfur content, as any other coals in the region. The lowest ash content of the coals listed in Table A8 of the UGS report is 8.5%. The ash content of the proposed coal for the Sevier project is lower, at 8.3%. The lowest sulfur content of the coals listed in Table A8 of the UGS report is 0.4%. The sulfur content of the proposed coal for the Sevier project is at least as low, at 0.40%. (Ref: Table A8 of the aforementioned UGS report; Table I-2 of the aforementioned "New Source Plan Review" for the Sevier project.)

The calculated cost and corresponding emission reductions described above lead to the following cost estimates, in dollars per ton of additional pollutant removed annually, to use alternative coal from another mine in the region, rather than waste coal from the Deserado mine:

Annualized Cost of Potential Emission Reductions Due to a Switch from Waste Coal at the Deserado Mine to Alternative Coal from Another Mine for Deseret Power's Proposed WCFU

Pollutant	Potential Emission Reduction (Alternative Coal versus Waste Coal)	Cost ($/ton)
NO_x	63 tons/yr	$ 212,698/ton
SO_2	114 tons/yr	$ 117,543/ton
CO	316 tons/yr	$ 42,405/ton
H_2SO_4	7 tons/yr	$1,914,285/ton
Filterable PM	13 tons/yr	$1,030,769/ton
Condensible PM	88 tons/yr	$ 152,272/ton
All (sum)	651 tons/yr	$ 20,583/ton

As mentioned in the draft Statement of Basis's discussion of alternative coal from other mines, there would also be substantial energy and environmental costs associated with obtaining coal from a mine other than the Deserado mine, due to the large number of truck trips to deliver the coal (more than 20 per day, assuming 50 tons payload per truck), at 200 miles round trip per load. The substantial energy expenditure in terms of diesel fuel, the amount of pollution from truck exhaust, and the increased traffic hazard on public highways, all make this option even more cost-prohibitive.

Based on the analysis above, EPA concludes that use of alternative coal from any other mine in the region, rather than waste coal from the Deserado mine, would be cost-prohibitive as a BACT option for the proposed WCFU, even if reductions of all pollutants are summed together and then the annualized cost in dollars-per-ton for emission reduction is calculated on that basis. (As shown above, summing the pollutants yields $20,583/ton, which is a lower dollar-per-ton BACT cost than looking at any one pollutant individually.)

The same annualized dollar-per-ton costs would be incurred if there was only a partial switch to alternative coal from another mine (i.e., coal blending). This is because a partial switch yields only partial emission reductions.

Regarding comparison to the cost of BACT that other similar sources have to bear (which EPA believes is best evaluated in terms of dollars per ton of additional pollutant removed, not simply in terms of what other sources pay for their coal as commenters have suggested), EPA is not aware of any BACT determination for a CFB boiler project anywhere in the U.S. where incremental cost effectiveness as high as $20,583/ton (the EPA-calculated economic cost for using coal from an alternative mine rather than waste coal from the Deserado mine), or as high as $20,241/ton (the EPA-calculated economic cost for using ROM coal from the Deserado mine rather than waste coal from the Deserado mine; see final Statement of Basis at page 28) has been considered reasonable for BACT for any pollutants, regardless of the type of BACT option being considered.

Although EPA considers the economic, energy and environmental costs associated with use of alternative coal for Deseret Power's project to be clearly excessive for BACT, EPA has nevertheless looked at some recent BACT determinations by other permitting authorities for similar projects, for purposes of comparison. EPA found the following:

1) In a PSD permit action in mid 2006 for Longleaf Energy Associates LLC, Longleaf Energy Station project, Georgia indicated that incremental cost effectiveness of $8,964/ton, comparing dry scrubbing to wet scrubbing for SO_2 control at a pulverized coal fired electric utility boiler, was excessive for BACT. Incremental and average cost effectiveness of the selected BACT option (dry scrubbing) was listed as $724/ton.

(Ref: Georgia's Preliminary Determination for SIP Permit Application #15846, page 62, dated July 2006, available online at: http://www.air.dnr.state.ga.us/airpermit/psd/dockets/longleaf/permitdocs/0990030pd.pdf.

2) In a PSD permit action in early 2005 for Rocky Mountain Power Inc.'s Hardin project, Montana indicated that incremental cost effectiveness of $23,855/ton, comparing dry FGD/spray dry absorber to wet FGD for SO_2 control, at a pulverized coal fired electric utility boiler, was excessive for BACT. Average cost effectiveness of wet FGD was listed as $1,395/ton. Average cost effectiveness of the selected BACT option (dry FGD/spray dry absorber) was listed as $918/ton.

(Ref: Montana's Permit Analysis for Hardin project, Permit #3185-02, pages 15 and 17, dated May 16, 2005, obtained from Montana Air Resources Management Bureau, in the Montana Department of Environmental Quality.)

3) In a PSD permit action in early 2007 for Southern Montana Electric Generation and Transmission Cooperative's CFB boiler project (Highwood Generating Station), Montana indicated that a "cost effective value" of \$27,365/ton for SO_2 control, for a control option employing a combination of limestone injection, low-sulfur coal and wet flue gas desulfurization (FGD), was excessive for BACT. Montana also indicated that a "cost effective value" of \$7,939/ton for SO_2 control, for a control option employing a combination of limestone injection, low-sulfur coal and dry FGD, was excessive for BACT.

The selected BACT option for SO_2 control, with a "cost effective value" of \$4,054/ton, employed a combination of limestone injection, low-sulfur coal, and hydrated ash reinjection. Montana did not indicate whether "cost effective value" means incremental cost effectiveness or total cost effectiveness.

(Ref: Montana's Permit Analysis for Highwood Project, Air Quality Permit #3423-00, page 23, dated May 30, 2007, obtained from the Montana Department of Environmental Quality Air Resources Management Bureau, Helena, Montana.)

4) In a PSD permit action in late 2006 for Cargill's Blair corn milling and ethanol production plant, Nebraska indicated that incremental cost effectiveness of \$5,900/ton, comparing limestone injection alone to limestone injection plus dry FGD, for SO_2 control at a CFB boiler, was excessive for BACT.

(Ref: Nebraska permit action CP06-0008, page 12 of Fact Sheet, dated September 8, 2006, available online at http://www.epa.gov/region07/programs/artd/air/nsr/archives/2006/finalpermits/cargill_blair_final_psd_permit.pdf.)

5) In a PSD permit action in late 2006 for ADM's Columbus corn milling and ethanol production plant, Nebraska indicated that incremental cost effectiveness of \$5,600/ton for NO_x control (comparing Selective Non-Catalytic Reduction (SNCR) at 0.07 lb/MMBtu to SNCR at below 0.07), and incremental cost effectiveness of \$6,700/ton for $SO_2/H_2SO_4/HF$ control (comparing limestone injection to "additional" limestone injection) at a CFB boiler, were excessive for BACT. Nebraska listed incremental cost-effectiveness of \$2,174 for the selected BACT option for NO_x control (SNCR at 0.07 lb/MMBtu).

Nebraska also listed average cost-effectiveness of \$5,200/ton for the selected BACT option for VOC control at the CFB boiler (wet scrubbing/packed tower),

(Ref: Nebraska permit action CPM02-0006, page 14 of Appendix B of Fact Sheet; pages 8, 9, 19 and 20 of Appendix D of Fact Sheet, dated August 4.2006, available online at: http://www.epa.gov/region07/programs/artd/air/nsr/archives/2006/finalpermits/adm_columbus_final_psd_permit.pdf.)

6) In a PSD permit in early 2005 for Montana-Dakota Utilities/Westmoreland Power, Gascoyne Generating Station project, North Dakota indicated that incremental cost effectiveness of $14,339/ton, comparing Selective Catalytic Reduction (SCR) at 0.04 lb/MMBtu to SNCR at 0.09 lb/MMBtu, for NO_x control at a CFB boiler, was excessive for BACT. Average cost effectiveness of SCR was listed as $7,545/ton. Average and incremental cost effectiveness of the selected BACT option (SNCR) was listed as $2,926/ton.

(Ref: North Dakota's Permit Application Analysis for Gascoyne Project, pages 65 and 68, dated March 2005, obtained from the North Dakota Department of Health, Environmental Health Section, Air Quality Division, Bismarck, ND.)

7) In a PSD permit action in early 2004 for Red Trail Energy's Richardton, ND, ethanol production plant, North Dakota listed incremental cost effectiveness of $10,252/ton, comparing wet FGD plus limestone injection to dry FGD (spray dryer absorber) plus limestone injection, for SO_2 control at a CFB boiler. Average cost effectiveness of wet FGD plus limestone injection was listed as $1,041/ton. Average cost effectiveness of dry FGD plus limestone injection was listed as $527/ton. North Dakota rejected wet FGD and determined that BACT is represented by dry FGD plus limestone injection.

(Ref: North Dakota's Permit Application Analysis for Red Trail Energy project, pages 38 and 40, dated May 2004, obtained from the North Dakota Department of Health, Environmental Health Section, Air Quality Division, Bismarck, ND.)

8) In a PSD permit action in early 2005 for River Hill Power Company's CFB boiler project, Pennsylvania indicated that incremental cost effectiveness of $15,975/ton, comparing use of the waste coal proposed by the permit applicant to use of the nearest alternative source of coal with lower sulfur content, for SO_2 control at the CFB boiler, was excessive for BACT.

Pennsylvania also indicated that all SO_2 BACT options involving wet FGD systems "were economically infeasible at an incremental dollar per ton value greater than $5,000 per ton of SO_2 removed."

Pennsylvania concluded that use of a spray dryer absorber or flash dryer absorber (i.e., dry FGD) was "economically feasible for the control of SO_2 at an incremental cost of $1,511.01 per ton of SO_2 removed."

(Ref: Pennsylvania's "Plan Approval Application Review Memo, Plan Approval Application #17-00055A," pages 10-11, dated May 2, 2005, obtained from the Commonwealth of Pennsylvania, Department of Environmental Protection, Northcentral Region, Air Quality Program.)

9) In a PSD permit action in early 2005 for Wellington Development's Greene Energy Resource Recovery Project, Pennsylvania indicated that incremental cost

effectiveness of at least $20,000/ton, comparing use of the waste coal proposed by the applicant to pre-combustion cleaning of the waste coal (excluding additional coal disposal costs after cleaning of the waste coal), for SO_2 control at the CFB boiler, was excessive for BACT. Pennsylvania also indicated that overall cost effectiveness of $5,764/ton, for limestone injection plus wet FGD for SO_2 control at the CFB boiler, was excessive for BACT.

(Ref: Pennsylvania's "Comment and Response Document," Air Quality File PA-30-00150A, page 6, dated June 21, 2005; Table 5-4 of PSD Permit Application, prepared by ENSR International, August 2004, page 5-29. Both documents were obtained from the Commonwealth of Pennsylvania, Department of Environmental Protection, Southwest Regional Office, Air Quality Program.)

10) In a PSD permit action in early 2004 for Intermountain Power's Unit 3 project, Utah indicated that incremental cost effectiveness of about $14,000/ton to $16,350/ton, comparing different types of baghouse fabric filter bags (Ryton-type bags versus specialty coated bags) for PM/PM_{10} control at a pulverized coal fired electric utility boiler, was excessive for BACT. Average cost effectiveness of the selected BACT option for PM_{10} control (a baghouse with Ryton-type bags) was $31/ton.

(Ref: Utah's Modified Source Plan Review for IPP3 project, pages 132-133, dated March 22, 2004, available online at: http://www.airquality.utah.gov/Permits/PmtPowerPlants.htm.)

11) In a PSD permit action in early 2007 for Basin Electric's Dry Fork Station project (a pulverized coal-fired electric utility boiler),Wyoming indicated that incremental cost effectiveness of $23,755/ton for NO_x control (comparing Selective Catalytic Reduction (SCR) at 0.043 lb/MMBtu to SCR at 0.040 lb/MMBtu) was excessive for BACT. Average cost effectiveness for SCR at 0.040 lb/MMBtu was listed as $2,004/ton. Average cost effectiveness for SCR at 0.043 lb/MMBtu was listed at $1,751/ton.

Although Wyoming determined that incremental cost effectiveness of $10,303/ton was reasonable for SCR at 0.043 lb/MMBtu, for other reasons described by Wyoming the selected BACT option for NO_x control was SCR at 0.05 lb/MMBtu, with incremental cost effectiveness of $3,512/ton and average cost effectiveness of $1,511/ton.

Wyoming also indicated that incremental cost effectiveness of $15,299/ton for SO_2 control (comparing dry FGD/spray dry absorber at 0.073 lb/MMBtu to wet FGD at 0.054 lb/MMBtu), was excessive for BACT. Average cost effectiveness of wet FGD at 0.054 lb/MMBtu was listed as $1,595/ton.

Although Wyoming determined that incremental cost effectiveness of $9,296/ton was reasonable for a spray dry absorber at 0.043 lb/MMBtu, for other reasons

described by Wyoming the selected BACT option for SO_2 control was a spray dry absorber at 0.08 lb/MMBtu, with average cost effectiveness of $1,159/ton; no incremental cost effectiveness listed by Wyoming for this BACT option..

(Ref: Wyoming's Permit Application Analysis for the Dry Fork project, NSR-AP-3546, pages 6, 7, 8, 10 and 11, dated February 5, 2007, obtained from Wyoming Air Quality Division, Cheyenne, WY.)

12) In a PSD permit action in early 2002 for Black Hills Power & Light's WYGEN2 project, Wyoming indicated that incremental cost effectiveness of $7,742/ton, comparing low-NO_x burners plus SCR at 0.06 lb/MMBtu to low-NO_x burners plus SCR at 0.08 lb/MMBtu, for NO_x control at a pulverized coal fired electric utility boiler, was reasonable for BACT. However, for other reasons described by Wyoming, the selected BACT option was low-NO_x burners plus SCR at 0.07 lb/MMBtu, with "total" (i.e., average) cost effectiveness somewhere between $4,067/ton (the average cost effectiveness to achieve 0.08 lb/MMBtu) and $4,156/ton (the average cost effectiveness to achieve 0.06 lb/MMBtu).

(Ref: Wyoming's Permit Application Analysis for the WYGEN2 project, NSR-AP-92, page 7, dated April 24, 2002, obtained from Wyoming Air Quality Division, Cheyenne, WY.)

13) In a PSD permit action in late 2006 for Black Hills Power & Light's WYGEN3 project, Wyoming indicated that incremental cost effectiveness of $14,609/ton, comparing a baghouse with fiberglass or polyphenylene sulfide filter bags (listed as capable of achieving 0.012 lb/MMBtu) to a baghouse with specialty filter bags such as Teflon (listed as capable of achieving 0.011 to 0.010 lb/MMBtu), for PM/PM_{10} control at a pulverized coal fired electric utility boiler, was excessive for BACT.

Average cost effectiveness of the selected BACT option (a baghouse with fiberglass or polyphenylene sulfide filter bags) was listed as $130/ton. Average cost effectiveness of a baghouse with specialty filter bags was listed as $134/ton.

(Ref: Wyoming's Permit Application Analysis for the WYGEN3 project, NSR-AP-3934, pages 10 and 11, dated October 9, 2006, obtained from Wyoming Air Quality Division, Cheyenne, WY.)

The pages cited above, for each of the 13 examples, are included in the Administrative Record for issuance of the WCFU permit.

Although this information is only on comparative economic costs of BACT options, not on comparative energy and environmental costs (which were generally not quantified by the permitting authorities), the information does seem to indicate that similar sources have typically not been expected to bear BACT costs, on an incremental cost effectiveness basis, as high as the incremental cost effectiveness for using alternative

sources of coal for Deseret Power's s project, in lieu of waste coal ($20,583/ton for alternative coal from another mine and $20,241/ton for alternative coal from the Deserado mine).

Regarding the Sevier project cited by commenters, the State of Utah presented no data in its "New Source Plan Review" on cost of BACT for any PSD pollutant, and none of the BACT options considered by Utah for that project involved alternative sources of coal. Further, no information was provided on cost of coal for the Sevier project.

This supplemental BACT analysis has not altered EPA's determination that use of alternative coal from the Deserado mine or from another mine, either partially or entirely in place of waste coal from the Deserado mine, should be eliminated as a BACT option, in terms of environmental, economic and energy costs, at Step 4 of the BACT analysis.

In summary, Comment #4.a has not resulted in any change to the permit; however, the Statement of Basis has been changed, to include the supplemental analysis described above.

4.b -- Sulfur Dioxide (SO_2):

Comment #4.b: One group of commenters asserted that the BACT analysis and proposed BACT limit for SO_2 are flawed because they do not reflect the maximum degree of reduction that can be achieved. EPA proposed an SO_2 emission limit of 0.055 lb/MMBtu (30-day average) when the uncontrolled SO_2 emissions are 1.9 lb/MMBtu or greater. EPA also proposed a calculated 30-day average SO_2 limit which is based on a 0.055 lb/MMBtu emission rate for the number of days at which the potential uncontrolled SO_2 emissions are 1.9 lb/MMBtu or higher, and a 0.04 lb/MMBtu emission rate for the number of days at which the potential uncontrolled SO_2 emissions are less than 1.9 lb/MMBtu. Individual supporting arguments from commenters are described below, along with EPA's responses

Comment #4.b.(1): Commenters alleged that EPA's proposed variable BACT limit does not reflect the maximum degree of reduction that can be achieved at a CFB boiler. By comparison, commenters cited two different coal-fired CFB power plants (Nevco and AES-Puerto Rico), with the same proposed SO_2 controls as Deseret's WCFU, that are required to meet an SO_2 BACT limit of 0.022 lb/MMBtu. Commenters calculated that the emission limit for AES-Puerto Rico equates to a 98.6% reduction in SO_2 emissions, which must be met on a three-hour average, despite a potential uncontrolled SO_2 emission rate of 1.6 lb/MMBtu, lower than Deseret's WCFU.

Response #4.b.(1): Partially agree. EPA does not agree with commenters that comparison with the Nevco project should lead to reconsideration of the SO_2 BACT emission limit in the draft WCFU permit. EPA does, however, agree with commenters that comparison with the AES Puerto Rico project should lead to such reconsideration, at least in regard to the "cutpoint" in coal quality. (NOTE: By "cutpoint," EPA means the level of uncontrolled SO_2 emission potential of the coal, in lb/MMBtu, that would trigger a switch from a straight 0.055 lb/MMBtu emission limit to a calculated emission limit of between 0.055 and 0.040 lb/MMBtu. A more detailed mathematical description of the "cutpoint" approach, as well as a description of the rationale for that approach, may be found in Step 5 of the SO_2 BACT analysis in the Statement of Basis.)

Comparison with Nevco: As stated on pages 77-78 of the Statement of Basis, Nevco will only have to achieve a control efficiency of 95.5% to meet its emission limit when burning average coal, and 97.2% when burning worst-case coal. The proposed limits for Deseret would reflect higher control efficiency than Nevco. As also stated in the Statement of Basis, if "average" coal for the WCFU (i.e., coal with uncontrolled SO_2 emission potential of about 1.71 lb/MMBtu) is burned for an extended period of time, such as a month or more, the variable BACT limit in the draft WCFU permit would approach the lower limit of 0.04 lb/MMBtu, which corresponds to a control efficiency of 97.7%. The proposed upper emission limit of 0.055 lb/MMBtu at the WCFU would reflect 98.8% control efficiency for "worst-case" coal (i.e., coal with uncontrolled SO_2 emission potential of 4.73 lb/MMBtu). These are both higher control efficiencies than required at the Nevco project for its average and worst-case coals.

Therefore, EPA does not believe that comparison to Nevco should lead to re-consideration of the proposed SO_2 emission limit for Deseret's WCFU project.

Comparison with AES Puerto Rico: Commenters also cited emission limits and theoretical control efficiencies required for the AES Puerto Rico facility. This project includes two CFB boilers burning Columbian coal that utilize limestone injection and dry scrubbers for SO_2 control, same as Deseret's WCFU project. The SO_2 emission limit for the AES Puerto Rico project is 0.022 lb/MMBtu on a 3-hour average. (Ref: PSD permit issued by EPA Region 2 on October 29, 2001 and revised on August 10, 2004, page 4, condition VIII.4-CFB.a.). However, the AES Puerto Rico permit also says "Emissions in excess of the applicable emission limit listed under Condition VIII of this permit, during periods of startup and shutdown, shall not be considered a violation of the applicable emission limit." (Ref: permit at page 15, condition XIV.7.)

This startup/shutdown exemption language does not appear in the draft WCFU permit. Instead, the draft WCFU permit says "The PSD BACT emission limits in this permit, as well as the modeling limits, apply at all times, including periods of startup, shutdown and malfunction." (Ref: draft WCFU permit at page 16, condition III.I.1.) Therefore, EPA believes that making a direct comparison of the stringency of the SO_2 emission limit in the AES Puerto Rico permit with the SO_2 emission limit in the draft WCFU permit is not entirely meaningful. Nevertheless, EPA has re-compared the theoretical control efficiency requirements of the two permits over the respective range of coal qualities, assuming steady-state operations apply and averaging times do not significantly affect those control requirements. This is explained in the step-by-step process below.

First, using mass balance, EPA calculated an uncontrolled SO_2 emission potential of the coal for the AES Puerto Rico facility, in lb/MMBtu, based on coal quality parameters of 0.8% sulfur content and 12,000 Btu/lb heat content cited by commenters for the 'worst case' coal. The result of EPA's calculation was 1.3 lb/MMBtu:

$$\frac{0.008 \text{ lb sulfur}}{\text{lb coal}} \times \frac{2 \text{ lb } SO_2}{\text{lb sulfur}} \times \frac{\text{lb coal}}{12{,}000 \text{ Btu}} \times \frac{1{,}000{,}000 \text{ Btu}}{\text{MMBtu}} = 1.3 \text{ lb } SO_2/\text{MMBtu}$$

To meet an emission limit of 0.022 lb/MMBtu, the AES Puerto Rico facility would need to achieve about 98.3% SO_2 control efficiency. (NOTE: These results differ from the results cited by commenters, which were 1.6 lb/MMBtu and 98.6% control. EPA therefore finds that the commenters' results were incorrect. Commenters did not provide an explanation of how they calculated 1.6 lb/MMBtu and 98.6% control, therefore EPA is unable to determine why commenters' results were incorrect. EPA finds that its own earlier results of 1.7 lb/MMBtu and 98.7% for 'worst-case' coal at AES Puerto Rico, cited on page 76 of the draft Statement of Basis, were also incorrect. This has been corrected in the final Statement of Basis.)

Second, EPA Region 8 obtained information on the sulfur content and heat content of coal that has been used historically at the AES Puerto Rico facility. EPA

Region 8 learned that the sulfur content varied from 0.49% to 0.75% during the fourth quarter of 2004 and the heat content was about 11,350 Btu/lb. From February of 2002 through June of 2003, the sulfur content varied from 0.53% to 0.85% and the heat content varied from 11,317 Btu/lb to 11,495 Btu/lb. From this information, EPA Region 8 found that the uncontrolled SO_2 emission potential of the actual coal ranges from about 1.3 lb/MMBtu ('worst-case' coal) down to about 0.88 lb/MMBtu ('average' coal). EPA Region 8 calculated that at the low end of this range, the AES Puerto Rico facility would need to achieve about 97.5% SO_2 control efficiency, to meet an emission limit of 0.022 lb/MMBtu. (Ref: Memorandum and attachments to the file by Mike Owens of EPA Region 8, dated August 8, 2007, included in the Administrative Record for issuance of the WCFU permit.)

Third, EPA Region 8 compared the above-mentioned control efficiencies for AES Puerto Rico to those that the WCFU would need to achieve to comply with the SO_2 emission limit in the draft WCFU permit. As noted above and on page 77 of the draft Statement of Basis, the WCFU would need to achieve about 98.8% control efficiency to comply with the upper emission limit of 0.055 lb/MMBtu, when burning 'worst-case' waste coal from the Deserado mine, and a control efficiency of about 97.7% to comply with an emission limit of 0.040 lb/MMBtu, when burning 'average' waste coal from the Deserado mine. Both of these control efficiencies are higher than the control efficiencies cited above for the range of coal at the AES Puerto Rico plant (98.3% for worst-case coal and 97.5% for average coal).

The above-mentioned comparison is somewhat misleading, however, for 'average' coal at the WCFU, because at the "cutpoint" in the draft WCFU permit (i.e., uncontrolled SO_2 emission potential of coal of 1.9 lb/MMBtu, only slightly higher than 1.71 lb/MMBtu for 'average' coal), the applicable emission limit would be the upper limit of 0.055 lb/MMBtu. Condition III.D.1.b.(ii)*(b)* of the draft WCFU permit states that the calculated emission limit of between 0.055 and 0.040 lb/MMBtu only applies below the "cutpoint." Therefore, the statement on page 81 of the draft Statement of Basis, that a control efficiency of 97.9% would need to be achieved to comply with the applicable emission limit at the "cutpoint," is incorrect, because the statement was erroneously based on complying with an emission limit of 0.040 lb/MMBtu. The correct control efficiency that would need to be achieved at the 1.9 lb/MMBtu "cutpoint" is actually 97.1%, based on an applicable emission limit of 0.055 lb/MMBtu. This corrected control efficiency is lower than the 97.5% control efficiency that the AES Puerto Rico facility must achieve to meet its SO_2 emission limit of 0.022 lb/MMBtu when burning 'average' coal.

Based on this correction, EPA re-evaluated the appropriate level to set for the "cutpoint" and determined that, to require a minimum control efficiency of 97.5% across the range of coal qualities described in the permit application for the WCFU, the "cutpoint" would need to be 2.2 lb/MMBtu, rather than 1.9 lb/MMBtu. This would correspond to a control efficiency of 97.5%, to comply with an applicable emission limit of 0.055 lb/MMBtu when burning coal with uncontrolled SO_2 emission potential of 2.2 lb/MMBtu.

When burning coal above the revised "cutpoint," i.e., coal with uncontrolled SO_2 emission potential greater than 2.2 lb/MMBtu, to comply with the applicable emission limit of 0.055 lb/MMBtu the WCFU would need to achieve higher SO_2 control efficiencies than 97.5%, reaching 98.8% when burning worst-case coal. Below the revised "cutpoint," a calculated SO_2 emission limit of between 0.055 and 0.040 lb/MMBtu is applicable and needed control efficiencies range from 98.1% just below the cut-point (2.14 lb/MMBtu) to 97.7% for the average coal.

EPA believes this revised "cutpoint" is an appropriate approach for ensuring that the WCFU maintains a high level of SO_2 control over the wide range of coal quality, and reflects the maximum degree of SO_2 reduction that can be achieved, commensurate with SO_2 BACT determinations for other similar facilities (listed in the two tables in Step 5 of the SO_2 BACT analysis in the Statement of Basis), including Nevco and AES Puerto Rico. Specifically, this revised "cutpoint" ensures a minimum control efficiency of at least 97.5%, over the range of worst-case coal to average coal.

EPA Region 8 also reviewed 30-day average SO_2 CEMS data for the AES Puerto Rico facility, in quarterly CEMS reports from the years 2003 through 2006, and found a very low amount of excess emissions with regard to the emission limit of 0.022 lb/MMBtu on a 3-hour average. The reports seem to EPA Region 8 to indicate that an emission limit of 0.022 lb/MMBtu on a 30-day rolling average (and the corresponding control efficiencies) could consistently be met by the AES Puerto Rico facility, over the range of coal quality cited above. Therefore, EPA concludes that the revised "cutpoint" of 2.2 lb/MMBtu for the WCFU represents an overall SO_2 BACT determination that is achievable for a CFB unit with limestone injection and a dry scrubber for SO_2 controls. (Ref: Memorandum and attachments to the file by Mike Owens of EPA Region 8, dated August 8, 2007, included in the Administrative Record for issuance of the WCFU permit.)

Comment #4.b.(1) has resulted in the following changes to the permit and Statement of Basis: The final permit specifies a "cutpoint" of 2.2 lb/MMBtu, rather than 1.9 lb/MMBtu in the draft permit, for triggering applicability of the lower-tier SO_2 BACT emission limit in the permit. The Statement of Basis has also been revised, to add an explanation of why EPA has chosen a "cutpoint" of 2.2 lb/MMBtu, and to correct EPA's calculations for 'worst-case' coal at AES Puerto Rico, explained above, from 1.7 lb/MMBtu and 98.7% control efficiency to 1.3 lb/MMBtu and 98.3% control efficiency.

Comment #4.b.(2): Commenters alleged that while the draft Statement of Basis indicates a 98.8% SO_2 removal efficiency could be achieved with the CFB boiler and the spray dry absorber (draft Statement of Basis at pages 72-73), the proposed BACT emission limit for SO_2 does not reflect this level of control, because it is based on the absolute worst case uncontrolled SO_2 emission rate. Commenters indicated that the 0.055 lb/MMBtu limit reflects 98.8% SO_2 removal from the worst case design coal of 3,000 Btu/lb and 0.71% sulfur (which thus equates to an uncontrolled SO_2 emission rate of 4.73 lb/MMBtu). However, the expected *average* uncontrolled SO_2 emission rate is 1.71

(EPA's Statement of Basis at 15). Commenters concluded that, based on the average uncontrolled SO_2 emission rate, the 0.040 lb/MMBtu SO_2 limit (which would apply when the uncontrolled emission rate is lower than 1.9 lb/MMBtu) only represents a 97.7% SO_2 removal rate from average uncontrolled SO_2 emissions. Commenters argued that 97.7% is over a percentage point lower than the maximum degree of reduction that can be achieved.

Response #4.b.(2): Disagree. While the figures cited by commenters are correct, EPA does not agree that 97.7% control is inadequate for SO_2 BACT, for combustion of "average" waste coal at Deseret's WCFU. Considering that the worst-case coal for the WCFU has uncontrolled SO_2 emission potential two-and-a-half times higher than average coal (4.73 lb/MMBtu versus 1.71 lb/MMBtu), EPA does not believe an SO_2 control efficiency as high as that for worst-case coal (98.8%) can be achieved when burning average coal, which resulted in only 97.5% SO_2 control efficiency at AES Puerto Rico. As explained in response #4.b.(1) above, the two-tiered SO2 emission limit with the revised "cutpoint" compares favorably to the two projects cited by commenters (Nevco and AES Puerto Rico), in terms of SO_2 control efficiencies needed to comply with applicable SO_2 emission limits.

Comment #4.b.(2) has not resulted in any change to the permit or Statement of Basis.

Comment #4.b.(3): Commenters alleged that EPA Region 8 previously made a "similar" comment to the Montana Department of Environmental Quality regarding the proposed Roundup power plant. Specifically, commenters cited EPA as having stated, in a December 18, 2002 letter to Montana, that "[w]hile use of the worst-case coal scenario might be appropriate for establishing a short-term (3-hour or 24-hour) SO_2 emission limit, we consider it inappropriate for establishing a 30-day average emission limit, especially considering that coal blending can be used at minimal additional cost (and is routinely used in the power plant industry) to eliminate or reduce the effect of coal sulfur 'spikes.'"

Response #4.b.(3): Disagree. By describing the comment to Montana as "similar," commenters appear to be suggesting that EPA only considered the worst-case coal scenario when proposing SO_2 BACT emission limits for Deseret's WCFU. This is not true. The Statement of Basis has a lengthy discussion (on pages 77-81) of how EPA set up a two-tiered limit. EPA's comments on Roundup are consistent with EPA's proposed approach of setting up this two-tiered limit for Deseret's WCFU, rather than setting a single limit based on worst case coal

Comment #4.b.(3) has not resulted in any change to the permit or Statement of Basis.

Comment #4.b.(4): Commenters alleged that the Bonanza WCFU has requested to be authorized to burn washed or run-of-mine coal which will have lower uncontrolled SO_2 emissions than the worst case waste coal and thus could be used to eliminate coal sulfur spikes. Also, commenters stated, Deseret has indicated that the Bonanza WCFU will have continuous SO_2 monitoring at the inlet to the dry scrubber. Thus, commenters

argue, Deseret will know on a fairly instantaneous basis when the coal sulfur content is spiking and thus could adjust the fuel accordingly. Consequently, the 30-day average BACT limit should reflect this level of control off of the average uncontrolled SO_2 emission rate of 1.7 lb/MMBtu, which equates to a BACT emission limit of 0.021 lb/MMBtu.

Response #4.b.(4): Disagree. The authorization to burn washed or run-of-mine coal is not unlimited as implied by commenters, but is restricted in the draft WCFU permit, as follows: Condition III.E.2.b. only allows Deseret Power to burn washed or run-of-mine coal, rather than waste coal, during emergencies when waste coal is not available. For situations other than startup or emergencies, condition III.E.2.c. allows use of run-of-mine coal blended with waste coal in any ratio yielding up to 6,500 Btu/lb heat content. This corresponds to roughly a 50/50 blend. As explained in the draft Statement of Basis at page 10, Deseret Power requested this authorization for operational flexibility, such as in the event of operational difficulties arising from use of waste coal as sole fuel, or in the event of unexpected difficulties in meeting BACT emission limits, even though the WCFU is being designed specifically to burn waste coal, and even though use of run-of-mine or washed coal on a routine basis, in lieu of waste coal, would be prohibitively expensive for BACT. EPA already presented a cost analysis in the draft Statement of Basis demonstrating that use of washed or run-of-mine coal, either partially or entirely in place of waste coal, should be eliminated as a BACT option for cost reasons. (See draft Statement of Basis at pages 25-28.)

At Step 5 of the SO_2 BACT analysis, EPA stated that "Deseret Power will be permitted to use coal from the Deserado mine, consisting of either waste coal alone, or else a blend of waste coal and run-of-mine coal, yielding heat content of up to 6,500 Btu/lb. Based on the SO_2 BACT analysis above, EPA believes that the proposed 'second tier' SO_2 emission limit described above will represent BACT for coal from the Deserado mine with heat content up to at lest 6,500 Btu/lb, and will ensure a continued high degree of SO_2 emission control efficiency." (Draft Statement of Basis at page 82.)

EPA disagrees with commenters' argument that SO_2 CEMS data could be used to adjust fuel and eliminate coal sulfur spikes. The SO_2 monitors at the inlet to the scrubber do not reflect the uncontrolled SO_2 emission rate of the raw coal, since a great deal of SO_2 control occurs upstream of the scrubber inlet, via limestone injection in the CFB boiler itself. EPA does not agree that Deseret has that much ability to control spikes in coal sulfur content (see detailed discussion on page 80 of the Statement of Basis) and the scrubber inlet SO_2 monitor does not help with this problem.

Comment #4.b.(4) has not resulted in any change to the permit or Statement of Basis.

Comment #4.b.(5): Commenters stated that at worst, the 30-day average SO_2 emission limit should reflect the percent reduction required at the AES-Puerto Rico facility, which has a similar level of uncontrolled emissions (albeit, worst case coal at AES-Puerto Rico is similar to average coal at the Bonanza WCFU). That facility's SO_2 emission limit reflects 98.6% reduction from uncontrolled emissions of 1.6 lb/MMBtu,

on a three-hour average basis. Thus, commenters concluded, the Bonanza WCFU SO_2 BACT limit should no higher than 0.024 lb/MMBtu, on a 30-day average to allow for the wide variability in sulfur content of the fuel.

Response #4.b.(5): Partially agree. As explained on pages 78-79 of the Statement of Basis, EPA does not agree that it is appropriate, considering the very high variability in coal quality expected to be encountered with Deseret's waste coal, to set a single SO_2 limit that applies to the entire range of possible fuel inputs at the WCFU. Also, as explained in response #4.b.(1), EPA does not agree with commenters' calculations of 1.6 lb/MMBtu and 98.6% control, for the "worst-case" coal scenario at the AES Puerto Rico plant. EPA calculates 1.3 lb/MMBtu and 98.3% control.

EPA does, however, agree that the WCFU should be expected to achieve a level of SO_2 reduction, which is commensurate with BACT determination at other similar facilities (listed in the two tables in Step 5 of the SO BACT analysis in the Statement of Basis), including AES Puerto Rico. Therefore, as explained in response #4.b.(1), EPA has revised the "cutpoint" that would trigger a change in the applicable emission limit for the WCFU, to ensure that the WCFU maintains a high level of SO_2 control over the wide range of coal quality, and to reflect the maximum degree of SO_2 reduction that can be achieved (97.5% or higher, over the range of worst-case coal to average coal), commensurate with SO_2 BACT determinations for other similar facilities including AES Puerto Rico.

Comment #4.b.(5) has resulted in the same changes to the permit and Statement of Basis that are described at the end of response #4.b.(1).

Comment #4.b.(6): Commenters alleged that EPA must also impose shorter term averaging time BACT limits consistent with the averaging times of the SO_2 NAAQS and PSD increments (i.e., 3-hour and 24-hour). Commenters cited an EPA statement, in a December 18, 2002 letter to Montana on the Roundup coal-fired electric utility project, that it is more appropriate to base shorter term average BACT limits on worst case uncontrolled emissions. (See comment #4.b.(3) above.) Thus, commenters concluded, the proposed BACT limit of 0.055 lb/MMBtu would be appropriate on a shorter term averaging time such as a three-hour average (similar to the AES-Puerto Rico permit).

Response #4.b.(6): Disagree, for three reasons. First, EPA set worst case modeling limits in the permit specifically to protect the short-term NAAQS and PSD increments. Second, the proposed SO_2 emission limits for Deseret's WCFU are two-tiered, unlike Roundup, and are not based solely on worst-case uncontrolled emissions. Third, Federal PSD rules at 40 CFR 52.21 do not require BACT limits for all averaging times of the PSD increments or NAAQS. EPA proposes modeling limits in the permit, separate from the BACT emission limits, to ensure that the assumed emission rates used for modeling PSD increment compliance and NAAQS compliance are not exceeded. See more detailed discussion at response #5.a.(1).

Comment #4.b.(6) has not resulted in any change to the permit or Statement of Basis.

Comment #4.b.(7): Commenters argued that in addition, with a 30-day average SO_2 BACT limit based on average coal quality and a 3-hour average SO_2 BACT limit based on worst case coal quality, this would eliminate the need for EPA's proposed variable SO_2 limit, which commenters say would not result in the maximum degree of SO_2 emission reduction that could be achieved. Commenters stated that this is because EPA allows applicability to the variable SO_2 BACT limit to be based on a 30-day average of the uncontrolled SO_2 emission rate (Condition III.J.2. of the draft permit), which will allow the Bonanza WCFU to only have to comply with the higher SO_2 BACT limit with just a few days of spiked coal sulfur content over a 30-day period

Response #4.b.(7): Disagree. As explained on pages 78-79 of the Statement of Basis, since the quality of coal in the waste coal pile is highly variable and not entirely predictable, the two-tiered SO_2 limit is necessary to accommodate fuel variability while still ensuring that controls are maintained at a high level of efficiency over the entire range of predicted coal quality, in accordance with BACT. A 3-hour limit based solely on worst case coal quality would not ensure that controls are maintained at a high level of efficiency over the entire range of predicted coal quality.

Only in situations where coal quality is consistently above the cut-point level of 1.9 lb/MMBtu for uncontrolled SO_2 emission potential for prolonged periods (unlikely to happen frequently, considering the cut-point is 11% higher than what is predicted to be average coal quality of the waste pile) would the higher-tier emission limit of 0.055 lb/MMBtu be the actual 30-day SO_2 BACT limit. The 30-day limit is a weighted average, so having just a few days of high coal sulfur content over a 30-day period would not necessarily cause the applicable emission limit to revert to 0.055 lb/MMBtu. The applicable emission limit might very well remain closer to the lower limit of 0.04 lb/MMBtu. See Statement of Basis discussion on page 80.

Comment #4.b.(7) has not resulted in any change to the permit or Statement of Basis.

Comment #4.b.(8): Commenters argued that the 5-day lag in comparing 30-day average uncontrolled SO_2 emissions to 30-day average controlled emission rates (Condition III.D.1.b.(ii)*(b)* of the draft permit) means that the proposed BACT emission limits would not ensure maximum SO_2 emission reductions on a continuous basis.

Response #4.b.(8): Disagree. EPA believes that the 5-day lag time, allowed under condition III.D.1.b. of the draft WCFU permit, is justified due to the SO_2 sampling time turnaround. EPA does not believe it is appropriate to retroactively apply a more restrictive limit upon a source once coal sampling results are obtained. As explained on page 80 of the draft Statement of Basis,

> "Deseret Power states that it will not be possible for them to determine the analysis of the fuel being fired, as it is being fired. Average samples of fuel being loaded into the silo will be taken to Deseret's laboratory for analysis. Deseret

states that results will take a minimum of one day and may take up to three days. If there will be a substantial delay in getting the results of the in-house analysis, Deseret states that the coal may have to be sent to an outside laboratory for analysis, which may take up to five days. Results therefore might not be available until three days or more after fuel is loaded to the fuel input silo. The applicable SO_2 tier limit would not be known to the WCFU operator until the coal analysis is received."

Although there will be a scrubber inlet SO_2 monitor in addition to the daily coal sampling, the scrubber inlet monitor will not measure the true uncontrolled SO_2 emission potential of the coal, either in practice or in the permit, due to the SO_2 control that occurs in the CFB boiler via limestone injection, upstream of the scrubber and the scrubber inlet SO_2 monitor. Therefore, the scrubber inlet SO_2 monitor will not eliminate the need for daily coal sampling and the associated lag time. As explained by Deseret Power in a January 9, 2006 e-mail to EPA (included in the Administrative Record for issuance of the WCFU permit), SO_2 measurements at the scrubber inlet monitor "will be used to control limestone flow to the furnace to maintain a selected SO_2 inlet to the dry scrubber." The scrubber inlet SO_2 monitor is not required by the draft WCFU permit, only a scrubber outlet SO_2 monitor, for demonstrating compliance with the SO_2 BACT emission limit.

Comment #4.b.(8) has not resulted in any change to the permit or Statement of Basis.

Comment #4.b.(9): Commenters asserted that the draft permit also fails to address BACT requirements when Deseret is using "run-of-mine" coal, either in lieu of waste coal, or as a blend with waste coal, from the Deserado mine (as allowed by condition III.E.2.c. of the draft permit). Commenters stated that EPA has indicated, in correspondence to Deseret, that BACT needs to be met "for the entire range of operating conditions." Yet, commenters argue, EPA did not provide any review of BACT or propose any emission limits to address BACT when the Bonanza WCFU is burning the much higher quality coal either wholly or in part.

Response #4.b.(9): Disagree. EPA's SO_2 BACT analysis did address this situation. See discussion on page 82 of the draft Statement of Basis. Comment #4.b.(9) has not resulted in any change to the permit or Statement of Basis.

Comment #4.b.(10): Commenters argued that to address the variation expected in uncontrolled SO_2 emissions at the Bonanza WCFU, EPA must include a SO_2 removal efficiency requirement as BACT in addition to the BACT emission limits that reflects the maximum degree of emission reduction that can be achieved given the variability in uncontrolled SO_2 emissions. Commenters note that EPA Region 8 recommended a similar approach in its comments on the proposed Roundup power plant in Montana. Specifically, EPA stated "[a] minimum required SO_2 scrubber efficiency should be included in the permit, to ensure proper operation and maintenance of the scrubber, and to ensure that SO_2 emissions are minimized at all times, regardless of the sulfur content in the coal."

Response #4.b.(10): Disagree. The Roundup facility in Montana is a pulverized coal (PC) fired unit, not a circulating fluidized bed (CFB) unit like Deseret's proposed WCFU. A PC fired unit uses a SO_2 scrubber as the single stage of SO_2 control, hence the overall control efficiency can easily be measured via CEMS at the scrubber inlet and outlet. A CFB unit, however, uses two stages of SO_2 control. As explained on page 12 of the Statement of Basis, the first stage is limestone injection into the CFB combustor unit and the second stage is a dry SO_2 scrubber downstream. For this two-stage system of control, overall control efficiency cannot be easily measured on a real-time basis. The proposed two-tiered SO_2 limit for the WCFU is a means to deal with the high coal quality variability and unpredictability of the waste coal supply and maintain an emission limit that ensures SO_2 emissions are controlled to a BACT level at all times.

Comment #4.b.(10) has not resulted in any change to the permit or Statement of Basis.

Comment #4.b.(11): Commenters asserted that, contrary to EPA's approach in the proposed limits in this permit, the percent reduction BACT requirement must be based on at least a daily average. Given the wide variability of uncontrolled SO_2 emissions allowed by the permit, calculating uncontrolled SO_2 emissions on a 30-day average would not ensure the maximum degree of SO_2 emissions reductions on those days when 100% "run-of-mine" coal is being burned.

Response #4.b.(11): Disagree. For situations other than startup or emergencies as defined in permit conditions III.E.2.a and b, permit condition III.E.2.c. allows use of run-of-mine coal blended with waste coal in any ratio yielding up to 6,500 Btu/lb heat content on a 30-day rolling average. This is roughly equivalent to a 50/50 blend, not 100% run-of-mine coal. As explained on page 82 of the Statement of Basis, EPA believes that the proposed 'second tier' SO_2 emission limit will represent BACT for coal from the Deserado mine with heat content up to at least 6,500 Btu/lb, and will ensure a continued high degree of SO_2 emission control efficiency. With the revised coal "cutpoint" described in response #4.b.(1) above, EPA calculates that control of at least 97.5% will be needed to meet the two-tier SO_2 emission limit for the WCFU, for the range of coal quality from worst-case coal to average coal. As explained in response #4.b.(1), EPA believes this level of control is commensurate with SO_2 BACT determinations at other similar sources cited by commenters.

Comment #4.b.(11) has not resulted in any change to the permit or Statement of Basis.

Comment #4.b.(12): Commenters indicated that a 24-hour average percent SO_2 removal should be required as part of the BACT determination, as it would effectively cover all of the various operating scenarios at the Bonanza WCFU.

Response #4.b.(12): Disagree. In a sense, the two-tiered limits are daily averages, as the 30-day weighted average is determined based on each day's coal quality.

Each “daily average” limit – either 0.055 or 0.040 lb/MMBtu - is given a weight depending on the number of days that daily limit applies over a 30-day period. There is a strong incentive for Deseret to keep controls running at their maximum capacity in order to ensure they meet their two-tiered emission limit, especially given the unpredictability of their coal source. Comment #4.b.(12) has not resulted in any change to the permit or Statement of Basis.

In summary comments #4.b.(1) through (12) have resulted in the changes to the permit and Statement of Basis described at the end of response #4.b.(1).

4.c -- Nitrogen Oxide (NO_x):

Comment #4.c: One group of commenters indicated that EPA did not adequately evaluate all of the technologies that could be employed at the Bonanza WCFU to reduce NO_x emissions and thus, the NO_x BACT determination does not reflect the maximum degree of NO_x reduction that can be achieved at the Bonanza WCFU. Individual arguments from commenters are described below, along with EPA's responses

Comment #4.c.(1): Commenters asserted that EPA eliminated evaluation of several NO_x control options as infeasible for a CFB boiler. Those options eliminated include flue gas recirculation and overfire air. See Statement of Basis at 30. Yet, commenters stated, a 1999 EPA guidance document identifies these two controls as options for NO_x control at CFB boilers. (Ref: Technical Bulletin: Nitrogen Oxides (NO_x), Why and How They Are Controlled, EPA-456/F-99-006R, November 1999.)

Commenters further stated that the Technical Bulletin identifies several other options for NO_x control at fluidized bed boilers that were not evaluated in the Bonanza WCFU NO_x BACT analysis, including: natural gas reburn, low excess air, reduced air preheat, reducing residence time at peak temperature through injection of steam, fuel reburning, non-thermal plasma reactor, and sorbent in combustion chamber/duct. Commenters argue that these technologies should have been evaluated by EPA, possibly in combination with SCR and SNCR, to determine the maximum degree of NO_x reduction that can be achieved.

Response #4.c.(1): Partially agree. The draft Statement of Basis should have mentioned the Technical Bulletin and discussed the control techniques listed in it. EPA has since prepared that discussion, which is presented below and is included in the final Statement of Basis. As presented below, EPA finds that each of the above-listed control techniques should be eliminated from further discussion as a BACT control option, due to one or more of the following reasons:

(1) ineffective or physically impossible at the WCFU,
(2) an inherent part of Deseret's proposed CFB boiler design,
(3) already proposed for the WCFU, or
(4) not commercially available.

This discussion therefore does not alter EPA's NO_x BACT determination for the WCFU. Findings are summarized in the table below, followed by individual explanations.

RESULTS OF NO_X CONTROL TECHNOLOGY EVALUATION ARISING FROM EPA TECHNICAL BULLETIN

	Addressed in Statement of Basis	Not Effective or not Physically Possible	Already Proposed to be Included	Not Commercially Available
Natural Gas Reburn	X	X		
Low Excess Air	X		X	
Reduced Air Preheat		X		
Reducing Residence Time		X		
Fuel Reburning	X	X		
Non-Thermal Plasma Reactor				X
Sorbent Injection	X		X	

Introductory discussion of thermal NO_x: The principal NO_x formation mechanism, thermal NO_x, arises from the thermal dissociation and subsequent reaction of nitrogen (N_2) and oxygen (O_2) molecules in the combustion air. Most thermal NO_x forms in the highest temperature regions of the combustion chamber (i.e. the air/fuel interface). Limiting the combustion temperature below 2,800^{o}F is sufficient to limit thermal NO_x. (Ref #1: R.T. Waibel, *Ultra Low NO_x Burners for Industrial Process Heaters*, Second International Conference on Combustion Technologies for a Clean Environment. Lisbon, Portugal, July 19-22, 1993. Figure 4, p. 5. Ref #2: IBO Industrial Emissions Control Technology III. Milwaukee, Wisconsin, August 1-3, 2005. p. 14.)

Most of control techniques listed in the table above act on thermal NO_x. These include natural gas reburn, low excess air, reduced air preheat, reducing residence time, and fuel reburning. The combustion temperature of a CFB boiler, by nature of its design, is much lower than that of a pulverized coal (PC) boiler (1,500^{o}F versus 3,000^{o}F). (Ref: Western Governors Association Technology Working Group Report, undated, page 10.) This lower combustion temperature results in virtually no thermally-generated NO_x. Because of this, control techniques designed to reduce NO_x emissions by reducing the combustion temperature, and thus reducing thermal NO_x, were not considered to have practical potential for application to coal-fired CFB boilers and thus were eliminated as control options at Step 1 of the BACT analysis. EPA explained this on page 31 of the draft Statement of Basis, in regard to Flue Gas Recirculation. Nevertheless, since the above-mentioned control techniques were listed in the EPA Technical Bulletin specifically in regard to CFB units, EPA has prepared the following explanations of why those techniques were eliminated as control options for the WCFU.

Also discussed below are two control techniques that are already proposed to be included for the WCFU, either as an inherent part of the CFB boiler design (low excess air), or as the chosen control option (sorbent injection, a.k.a. Selective Non-Catalytic Reduction). Also discussed are two control techniques that may have practical potential

for application to coal-fired CFB boilers, but are not known to be commercially available for CFB units (non-thermal plasma reactor and carbon injection into the combustion chamber). These techniques have therefore also been eliminated as control options for the proposed WCFU.

Descriptions of individual techniques below were taken from the above-mentioned EPA Technical Bulletin

Natural gas reburn – This is considered to be the same method as generic "fuel reburning," which was identified by the commenters as a separate control technique. The principles are the same whether the additional fuel reburned is natural gas, fuel oil, or coal. See "Fuel reburning" below.

Low excess air – Excess air flow for combustion has been correlated to the amount of thermal NO_x generated. Limiting the net excess air flow to less than 2% can strongly limit NO_x content of flue gas at pulverized coal fired boilers. Although there are fuel-rich and fuel-lean zones in the combustion region, the overall net excess air is limited when using this approach.

A certain amount of excess air is required to maintain flame stability and provide satisfactory combustion. Limiting excess air to such a low level would also increase emissions of carbon monoxide (CO).

Reducing the amount of excess air may be a valid way to reduce NO_x emissions from an older CFB unit with poor combustion controls. However, the unit proposed by Deseret is a new unit with state-of-the-art combustion controls. One of the goals of those controls is to minimize excess air to maximize boiler efficiency. If one were to consider reducing excess air further than the design rate, it would result in increased CO emissions and disrupt the stable operation of the unit. Further, this control technique acts primarily on thermal NO_x and therefore, while it may have substantial effect on NO_x emissions at pulverized coal fired boilers, it has much less effect on NO_x emissions at combustion sources such as CFBs that operate at low combustion temperatures.

This control technique was addressed on page 31 of the Statement of Basis, through EPA's reference to Table 1.1-2 of AP-42, which indicates it does not have practical potential for application to coal-fired CFB boilers. It has therefore been eliminated at Step 1 of the BACT analysis.

Reduced air preheat – Preheating the combustion air cools the flue gases, reduces the heat losses, and gains efficiency. However, this can raise the temperature of combustion air to a level where NO_x forms more readily. By reducing the amount of air preheat, the combustion temperature is lowered and NO_x formation is suppressed. However, reducing the amount by which the incoming combustion air is preheated carries a significant efficiency penalty of up to 1% per 40°F. (Ref: above-mentioned EPA Technical Bulletin on NO_x control, page 12.) This reduction in efficiency would increase emissions of all criteria pollutants. As mentioned in the "Introductory

discussion of thermal NO_x"above, the combustion temperature of a CFB boiler, by nature of its design, is much lower than that of a pulverized coal (PC) boiler and results in virtually no thermally-generated NO_x. Therefore, reduced air preheat is not considered to be an effective NO_x control option for coal-fired CFB boilers, i.e., it does not have practical potential for application to CFB boilers for NO_x control. It has therefore been eliminated at Step 1 of the BACT analysis.

Reducing residence time at peak temperature through injection of steam – This control technique involves injection of water or steam, which causes the stoichiometry of the mixture to be changed and adds steam to dilute calories generated by combustion. Both of these actions cause combustion temperature to be lower. If temperature is sufficiently reduced, thermal NO_x will not be formed in as great a concentration.

In order to control NO_x, steam is typically injected directly into the flame to reduce the adiabatic flame temperature. In a CFB boiler, this is not physically possible, as combustion occurs throughout the fluidized bed. As with reduced air preheat, injecting steam would reduce boiler efficiency and result in increased emissions of all pollutants.

This control technique is addressed in the introductory discussion of thermal NO_x above and is not considered to be an effective control option for coal-fired CFB boilers, i.e., it does not have practical potential for application to CFB boilers for NO_x control. It has therefore been eliminated at Step 1 of the BACT analysis.

Fuel reburning – This control technique consists of recirculation of cooled flue gas with added fuel, similar to Flue Gas Recirculation (FGR) discussed on page 31 of the Statement of Basis. With fuel reburn, calories are diluted and the primary combustion temperature can be lowered. In other words, the peak flame temperature can be lowered through adsorption of the combustion heat by the relatively inert flue gas. As explained in the Statement of Basis, and in the introductory discussion of thermal NO_x above, this control technique acts on thermal NOx and is not considered to be effective on combustion sources such as CFBs that operate at low combustion temperatures. As such, it does not have practical potential for application to CFB boilers for NO_x control. It has therefore been eliminated at Step 1 of the BACT analysis.

Non-thermal plasma reactor – This control technique involves using methane and hexane as reducing agents. Non-thermal plasma has been shown to remove NO_x in a laboratory setting with a reactor duct only two feet long. The reducing agents were ionized by a transient high voltage that created a non-thermal plasma. The ionized reducing agents reacted with NO_x and achieved a 94% destruction efficiency. There are indications that an even higher destruction efficiency can be achieved. A successful commercial vendor uses ammonia as a reducing agent to react with NO_x in an electron beam generated plasma. Such a short reactor can meet available space requirements for virtually any plant. The non-thermal plasma reactor could also be used without reducing agent to generate ozone and use that ozone to raise the valence of nitrogen for subsequent absorption as nitric acid.

Trinity Consultants investigated the non-thermal plasma reactor as a NO_x control option and advised Deseret Power that it is not known to be commercially available. (Ref: E-mail from Ed Thatcher, Deseret Power, to Mike Owens, EPA Region 8, November 13, 2006.) Therefore, while this control technique might be considered a technology transfer control option at Step 1 of the BACT analysis, it is eliminated at Step 2 as technically infeasible because it is not known to be commercially available for NO_x control at CFB boilers.

Sorbent in combustion chamber/duct.—This control technique involves injection of limestone into the combustion zone. Injection of ammonia (Selective Non-Catalytic Reduction) is also already included in the design of the proposed WCFU.

According to the above–mentioned EPA Technical Bulletin on NO_x control, another version of sorbent injection "uses carbon injected into the air flow to finish the capture of NO_x. The carbon is captured in either the baghouse or the ESP just like other sorbents." (Ref: Bulletin at page 19.) Although carbon injection is an emerging technology used to reduce mercury emissions, Deseret Power is not aware of it having been used anywhere to control NO_x. (Ref: E-mail dated November 13, 2006, from Ed Thatcher of Deseret Power to Mike Owens of EPA Region 8.) EPA is similarly not aware of carbon injection having been used anywhere to control NO_x. (Ref: Memorandum from Mike Owens of EPA Region 8 to Deseret Bonanza WCFU PSD Permit file, dated August 8, 2007.) Carbon injection for NO_x control is therefore eliminated at Step 2 of the BACT analysis as technically infeasible because it is not known to be commercially available for that purpose.

In summary, the evaluation and discussion above does not alter EPA's NO_x BACT determination of 0.080 lb/MMBtu for the WCFU. Comment #4.c.(1) has not resulted in any change to the permit; however, the Statement of Basis has been revised to include the discussion of potential NO_x control options above.

Comment #4.c.(2): Commenters asserted that while EPA required evaluation of selective catalytic reduction (SCR) on the proposed CFB boiler, SCR was improperly eliminated from the BACT review. First, EPA required evaluation of low temperature SCR, but Deseret apparently found that low temperature SCR was only applied to natural gas applications.

Commenters cited a memorandum from Don Shepherd to John Notar (both in the Air Resources Division at the National Park Service) regarding the NEVCO Energy – Sevier Power – Engineering Analysis, in which Mr. Shepherd stated "[w]hen the question of application of SCR to a CFB was raised at the Pittsburgh workshop [on selective catalytic reduction and non-catalytic reduction for NO_x control], one consultant stated that he knew of no reason why it could not be done. (In fact, one presenter in Pittsburgh suggested that addition of limestone, as would be inherent in a CFB, is desirable in counteracting the potential catalyst-poisoning effects of arsenic found in many coals)." Commenters argued that the question which should have been posed is whether SCR

could be applied to coal-fired CFB boilers. Commenters cited a statement in EPA's draft 1990 New Source Review Workshop Manual that opportunities for technology transfer must be identified and evaluated in the BACT analysis.

Response #4.c.(2): Disagree. EPA's draft Statement of Basis did, in fact, evaluate whether or not SCR could be applied to coal-fired CFB boilers.

With regard to statements from the National Park Service (NPS) about application of SCR to CFBs, the draft Statement of Basis (at page 32) explained that EPA asked Deseret Power to contact SCR vendors, based on NPS information about low-temperature SCR as a possible option. Specifically, Deseret Power was requested to find out if low-temperature SCR is commercially available. The answer was no. The vendors cited by the NPS as possible suppliers of low-temperature SCR informed Deseret Power that they actually provide SCR technology only for natural gas applications, not for coal-fired boilers. EPA concluded that low-temperature SCR is not a technically feasible NO_x control option for the WCFU, as it is not commercially available to be applied to this project. (Draft Statement of Basis at page 32.)

With regard to step 2 (technical feasibility) of the top-down BACT analysis, two key concepts are important in determining whether an undemonstrated technology is feasible – "availability" and "applicability." *See Prairie State*, slip op, at 17; Three *Mountain Power*, 10 E.A.D. at 42-43 n.3. A technology is considered "available" if it can be obtained by the applicant through commercial channels or is otherwise available within the common sense meaning of the term. An available technology is "applicable" if it can reasonably be installed and operated on the source type under consideration. A technology that is available and applicable is technically feasible.

The draft Statement of Basis explained that EPA did identify SCR (excluding low-temperature SCR) as a technically feasible control option, and asked Deseret Power to evaluate the possibility of reheating the flue gas downstream of the baghouse to the temperature range known to be effective for SCR use. This evaluation included a detailed cost estimate described in the Statement of Basis. EPA concluded that the economic impacts of reheat, without even considering the higher capital cost of SCR versus SNCR, justified elimination of SCR as a BACT control option. (Draft Statement of Basis, pages 32-35.)

Comment #4.c.(2) has not resulted in any change to the permit or Statement of Basis.

Comment #4.c.(3): In a second argument regarding SCR, commenters asserted, "while EPA did require the evaluation of whether the flue gas downstream of the baghouse could be reheated to the temperature range known to be effective for SCR use (650-750 F) (Statement of Basis at 32), EPA should also have required evaluation of reheating the gas stream to the temperature range at which low temperature SCR could be used." Commenters argued that, according to the Institute of Clean Air Companies, low temperature catalysts can work in the range of 350 – 550 F. (Commenters cited the

ICAC website at http://www.icac.com/i4a/pages/index.cfm?pageid=3399, under NO_x Control Technologies.) Thus, commenters argued, EPA should have required Deseret Power to evaluate heating the gas stream up to 350 F and using low temperature SCR, which would use considerably less fuel than needed to reheat the gas stream to 650 F.

Response #4.c.(3): Disagree, for three reasons:

First, EPA explained in the draft Statement of Basis (at page 32), that low-temperature SCR was eliminated as technically infeasible because it is not commercially available to be applied to this project.

Second, EPA's cost analysis for reheat (on pages 34-35 of the draft Statement of Basis) was based on raising the stack temperature to 480F – as supplied by Deseret. This is within the range of 350-550F, as described by the ICAC noted above. Based on the ICAC website cited by commenters, "(i)n clean, low temperature (350-550F) applications, catalysts containing precious metals such as platinum and palladium are useful." EPA described the high cost results for reheat in the draft Statement of Basis (pages 34-35). The high cost results were only for reheat and did not include any of the substantially higher installation costs for SCR versus SNCR. This additional cost would undoubtedly negate any reduction in cost achieved by lowering the temperature threshold from 480 to 350F.

Third, commenters have not come up with any new evidence that low-temperature SCR could work.

Comment #4.c.(3) has not resulted in any change to the permit or Statement of Basis.

Comment #4.c.(4): In a third argument regarding SCR, commenters asserted that the presumed emission limit that could be met with SCR should have been lower than 0.04 lb/MMBtu used in the draft Statement of Basis at 33. Commenters stated that EPA did not provide any rationale for this presumed NO_x emission rate with SCR, except to cite to the level assumed by North Dakota in its BACT analysis for Gascoyne. Commenters argued that EPA should have instead evaluated a NO_x emission limit based on the maximum degree of emission reduction that can be achieved with SCR. Commenters stated that, according to Babcock & Wilcox, commercial SCR installations have shown that 90% NO_x reductions can be achieved with low ammonia slip, and that Babcock & Wilcox states that up to 95% NO_x control can be achieved with SCR. Thus, commenters concluded, considering the NO_x emission rate without SCR of 0.15 lb/MMBtu, which EPA indicated was an overestimate of NO_x emissions expected from the Bonanza WCFU (Statement of Basis at 34-35), the appropriate NO_x emission rate with SCR to evaluate would be at most 0.015 lb/MMBtu rather than the assumed 0.04 lb/MMBtu.

Response #4.c.(4): Partially agree. EPA has no definitive evidence that 0.015 lb/MMBtu could be achieved with SCR at the proposed Deseret WCFU. Further, EPA

does not agree that 0.04 lb/MMBtu is not a reasonable presumption for lowest emission rate that could be met with SCR at the Deseret WCFU. Nevertheless, EPA does agree it is conceivable a lower emission rate than 0.04 lb/MMBtu could be met with SCR. Since 90% NO_x removal from SCR is believed to be achieved at some facilities, and since Deseret Power has not provided more case-specific information for SCR capabilities for its WCFU project, EPA has revised its cost analysis, based on the Babcock & Wilcox information cited by commenters. The revised analysis reflects the possibility that a lower NO_x emissions rate than 0.04 lb/MMBtu could be achieved with SCR, as detailed in response #4.c.(5) below.

Comment #4.c.(4) has not resulted in any change to the permit; however, the Statement of Basis has been changed, to reflect this revised analysis of SCR described in response #4.c.(5) below.

Comment #4.c.(5): Based on the rationale in comments #4.c.(2) through (4) above, commenters asserted that the analysis for SCR must be re-evaluated to consider whether low temperature SCR could work on the Bonanza CFB boiler, either with or without flue gas reheating, and considering a NO_x emission rate that reflects the maximum degree of emission reduction that can be achieved.

Response #4.c.(5): Partially agree. EPA does not agree that the analysis for SCR must be re-evaluated to consider whether low temperature SCR could work. As explained in the Statement of Basis, and as explained in responses #4.c.(2) and (3) above, EPA concluded that low-temperature SCR is not a technically feasible NO_x control option for the WCFU, as it is not commercially available to be applied to this project.

EPA does agree, however, that the analysis for SCR with flue gas reheating should be revised to reflect a lower NO_x emissions rate achievable with SCR than 0.04 lb/MMBtu. The revised analysis is below. Most of the text is the same as in the draft Statement of Basis, but with revised cost calculations.

In order to be responsive to the commenters' assertion that 90% NO_x reduction could be achieved by installation of SCR on the Deseret WCFU, EPA has re-evaluated the cost-effectiveness of reheating the flue gas, in terms of the possibility of achieving a final NO_x rate of 0.015 lb/MMBtu rather than 0.04 lb/MMBtu. Note that this does not include the other capital and operating costs associated with purchasing, installing, operating, and maintaining the SCR system, so this analysis substantially underestimates the true cost per ton of NO_x reductions that would be incurred by Deseret, if SCR were applied to this project. The SCR analysis below is a modification to the draft Statement of Basis to reflect the final emission rate of 0.015 lb/MMBtu cited by commenters. In addition, consideration was given to the additional NO_x emissions generated by distillate fuel combustion when calculating total cost effectiveness, which should have been done in the draft Statement of Basis analysis.

a. Selective Catalytic Reduction. As noted above, for SCR to be a technically feasible NO_x control option for this project, flue gas reheating would be

required downstream of the particulate controls. This would involve significant additional fuel cost. The cost and environmental impacts are discussed below. Even without flue gas reheating, a SCR system does require some additional energy in order to overcome the pressure drop over the SCR catalyst beds; however, this has not proven to be a significant energy or economic impact for employing SCR technology on coal-fired power plants.

With any SCR installation, there are some commonly noted adverse environmental impacts. These would include ammonia slip emissions, catalyst disposal, and potential ammonia handling hazards. These impacts are usually deemed to be offset by the environmental benefits of significant NO_x reduction from the SCR system. For example, with the SCR system located downstream of the particulate and SO_2 control devices in order to deal with technical problems associated with a CFB application, there may be additional condensible particulate emissions resulting from the conversion of SO_2 to SO_3 and eventually to H_2SO_4 over the catalyst bed.

Another adverse environmental impact is the additional emissions from combustion of distillate fuel oil or propane for flue gas reheating. Deseret Power has calculated a required heat input of 99.2 MMBtu/hr to raise the temperature of the flue gas from 275 F to 480 F. The 480 F used by Deseret Power is on the low end of, or even below, where an SCR can most effectively operate. Thus, the fuel consumption values may actually be higher than calculated by Deseret.

Since there are no natural gas lines into Deseret Power's Bonanza plant, the only reheat options are distillate fuel oil or propane. EPA has calculated the emissions based on AP-42 emission factors. These emissions are presented in the table below. The calculations assume heat rating of the distillate fuel oil to be 0.14 MMBtu/gal, which equals 710 gallons per hour. For propane, the calculations assume 0.0905 MMBtu/gal, which equals 1,100 gallons per hour.

The difference in emission rates between SNCR and SCR would be 0.065 lb/MMBtu (i.e., 0.08 minus 0.015). Assuming CFB operation at 90% of capacity on an annual average, this difference would be equivalent to a NO_x reduction of 370 tons per year:

$$(0.08 \text{ lb/MMBtu} - 0.015 \text{ lb/MMBtu}) \times (1{,}445 \text{ MMBtu/hr}) \times (8{,}760 \text{ hr/yr}) \times (0.9) \times (1 \text{ ton}/2{,}000 \text{ lb}) = 370 \textbf{ tons/year}$$

With distillate reheat, the net NO_x reduction would be 308 tons per year (i.e., 370 minus 62). With propane reheat, the net NO_x reduction would be 278 tons per year (i.e., 370 minus 92). These figures are shown in the table below.

Estimated Emissions From Reheating of CFB Flue Gas
To Accommodate Use of Conventional SCR
At Deseret Power's Proposed WCFU

Pollutant	Distillate Oil Emissions (tons/year)	Propane Emissions (tons/year)
PM (total)	10	3
SO_2	3	Negligible
NO_x	62	92
VOC	1	2
CO	16	15

Even without considering reheat cost, the annualized cost of SCR is several times greater than SNCR, due to higher capital and operating costs. (Example: PSD permit application dated August 2005, for South Heart CFB boiler project in North Dakota, calculates the annualized capital recovery cost for SCR to be about six times as much as for SNCR. Ref: Page 4-16 of the permit application, included in the Administrative Record for issuance of the WCFU permit.) As explained above, SCR installed downstream of particulate controls would also involve reheat cost. Deseret Power provided cost figures for only the supplemental fuel that would be required to reheat the flue gas so that SCR could be used. No additional costs were calculated for capital, installation, or operation of the SCR system or capital, installation, and other non-fuel operational costs for the reheat system. Hence, this is a very conservative cost analysis, since as mentioned above, these additional capital, installation and operational costs for the SCR and reheat system would likely be substantial. The lowest-cost option for reheat fuel was calculated to be distillate oil at $12,411,476 per year, based on 6,205,738 gallons per year at $2.00 per gallon.

Without any add-on controls, EPA estimates that the CFB boiler should be able to achieve a NO_x emission rate of about 0.15 lb/MMBtu or lower. (Actual operational data on existing CFB boilers suggests to EPA that this value could be much lower. The 0.15 value was chosen by EPA only as a conservative estimate in doing this cost analysis.) Using this uncontrolled emission rate as a baseline, the total cost effectiveness for the SCR/reheat system only, considering the cost of reheat fuel, is calculated as follows:

Emission reduction going from baseline to SCR controlled emissions:

(0.15 lb/MMBtu – 0.015 lb/MMBtu) x (1,445 MMBtu/hr) x
(8,760 hr/yr)(0.9) (1 ton/2,000 lb) = **769 tons/year**

The average cost per ton for NO_x reductions, considering only distillate fuel costs when considering the additional NOx that would be generated by burning distillate fuel:

($12,411,476 / yr) / (769 - 62 ton/yr) = **$17,555/ton**

The incremental cost of going from SNCR to SCR, considering only the distillate fuel costs is calculated as follows:

($12,411,476 / yr) / (370 ton/yr) = **$33,545 /ton**

The incremental cost going from SNCR to SCR, considering only the distillate fuel costs, and considering the additional emissions caused by reheat for SCR, is calculated as follows:

($12,411,476 / yr) / (370 - 62 ton/yr) = **$40,297 /ton**

EPA concludes that the economic impacts associated with a cost of more than $40,000 per ton of pollutant removed justify elimination of SCR as the top control option. Both the total cost effectiveness and incremental cost effectiveness can be considered cost-prohibitive for BACT. In addition, if capital, installation, and other operational costs for both the SCR and reheat system were considered, the above cost values would increase significantly.

In summary, comment #4.c.(5) has not resulted in any change to the permit, since SCR has still been eliminated for cost reasons; however, the Statement of Basis has been changed to reflect the revised NO_x analysis for SCR described above.

Comment #4.c.(6): Further, commenters asserted, in determining whether the costs for SCR are reasonable, the costs must be compared to the costs other coal-fired electric utility boilers have had to bear for NO_x control under BACT determinations. Commenters argued that it is not appropriate to compare SCR to the cost of SNCR, which is less effective than SCR in reducing NO_x.

Response #4.c.(6): Disagree. Commenters do not cite specific coal-fired electric utility boilers the Deseret Power WCFU should be compared to, as far as the costs of NO_x BACT; however, EPA assumes the commenters mean those facilities that have had to bear the costs of installing SCR. To EPA's knowledge, the only coal-fired electric utility boilers that have installed, or will be installing, SCR based on a BACT determination are for pulverized-coal units. Presently, EPA does not know of any CFB boilers that have installed SCR, or that have been required to install SCR, based on a BACT determination, nor have commenters provided any such information.

Determination of whether a control alternative can be eliminated in step four of the top-down BACT analysis involves a demonstration that "circumstances exist at the source which distinguish it from other sources where the control alternative may have been required previously." *In re Maui Electric Co.,* 8 E.A.D. 1, 6 (EAB 1998) (internal quotations omitted). Clearly, the fact that Deseret Power's WCFU is a CFB boiler, fired on high-ash waste coal, is a distinguishing feature that creates far different flue gas characteristics and unacceptably high particulate loading to an SCR system that would be installed upstream of the particulate control device, compared to those pulverized coal boilers that have installed SCR systems, or are required to install SCR systems based on a

BACT determination. These differences were explained on page 32 of the draft Statement of Basis.

EPA also disagrees with the commenters' claim that EPA inappropriately compared the cost effectiveness of SCR to SNCR. In step one of the top-down BACT analysis, both the average cost effectiveness of a control option and the incremental cost effectiveness between dominant control options can be calculated. *In re General Motors Inc.,* PSD Appeal No. 01-30, slip op. at 26 (EAB March 6, 2002). While EPA believes it is appropriate to calculate the incremental cost effectiveness of SCR, EPA did not rely on this cost alone. EPA also calculated the average cost effectiveness and found that cost was also high.

In summary, comment #4.c.(6) has not resulted in any change to the permit or Statement of Basis.

Comment #4.c.(7): Commenters asserted that if EPA determines that SCR can be eliminated, after revising the BACT review in light of comments above, then its evaluation of SNCR and the associated NO_x emission limit must be based on the maximum degree of emission reduction achievable with SNCR. Commenters asserted that SNCR should be able to reduce NO_x emissions by at least 50%, while EPA's proposed 0.080 lb/MMBtu NO_x emission limit for SNCR reflects a 47% NO_x reduction. Commenters concluded that a 50% NO_x reduction with SCNR would equate to an emission limit of 0.075 lb/MMBtu, or even lower, considering that EPA believes the 0.15 lb/MMBtu uncontrolled NO_x emission rate is an overestimate. (Commenters cited the draft Statement of Basis at 34-35.)

Response #4.c.(7): Disagree. EPA believes going from 0.075 to 0.08 is justified in order to provide a margin of compliance, and it is consistent with the BACT limits for other sources listed on page 37 of the draft Statement of Basis. The margin of compliance includes a reasonable safety factor that would permit Deseret to achieve compliance on a consistent basis.

The Environmental Appeals Board has recognized that permitting agencies have the discretion to set BACT limits at levels that do not necessarily reflect the highest possible control efficiencies but, rather, will allow permittees to achieve compliance on a consistent basis. (See *In re Three Mountain Power*, 10 E.A.D. 39, 53 (EAB, May 30, 2001) and *In re Masonite Corp.*, 5 E.A.D. 551, 560-61 (EAB 1994). See also *In re Knauf Fiber Glass, GmbH*, 9 E.A.D. 1, 15 (EAB, Mar. 14, 2000). ("There is nothing inherently wrong with setting an emission limitation that takes into account a reasonable safety factor. …The inclusion of a reasonable safety factor in the emission limitation calculation is a legitimate method of deriving a specific emission limitation that may not be exceeded.")

Comment #4.c.(7) has not resulted in any change to the permit or Statement of Basis.

Comment #4.c.(8): Commenters stated that EPA pointed out to Deseret in its July 8, 2005 letter that there are several other proposed CFB boilers using SNCR with proposed NO_x emission limits of 0.07 lb/MMBtu, including the Estill County Energy Partners Project in Kentucky, the Kentucky Mountain Power Project in Kentucky and the River Hill project in Pennsylvania. As EPA commented to Deseret, the Estill County project is most similar to Bonanza in size and coal quality, and thus Deseret should be able to meet a similar limit at the Bonanza WCFU. Although Deseret later pointed out that no PSD permit had been issued for the Estill County project yet, that does not negate the point that the owners/operators proposed a 0.07 lb/MMBtu NO_x limit for their facility. Thus the NO_x BACT analysis for SNCR should be evaluated using a lower NO_x limit, in the range of 0.07 to 0.075 lb/MMBtu to ensure that the limit reflects the maximum degree of NO_x reduction that can be achieved.

Response #4.c.(8): Disagree. See discussion on page 38 of the draft Statement of Basis for our analysis and consideration of permits with limits of 0.07 lb/MMBtu. The Estill County project was eliminated from consideration because the permit application is no longer being actively processed. No draft permit was issued for the Estill County project and no BACT determination for NO_x was proposed by the permitting agency.

EPA inadvertently omitted the Kentucky Mountain Power Project (KMPP) from the table on page 37 of the draft Statement of Basis ("Summary of Recent CFB Projects Permitted or Proposed: NOx Emission Rates Using SNCR"). EPA has added KMPP in the final Statement of Basis.

The KMPP permit, issued on May 4, 2001, specifies a NO_x emission limit of 0.07 lb/MMBtu; however, unlike the permit for Deseret's WCFU, the KMPP permit says, at Section D, Condition 3, "The NO_x emission limit of 0.07 lb/MMBTU is waived for the specific SNCR optimization study activity as detailed in Condition 2 above not to extend more than 365 days after the initial compliance demonstration. However, the nitrogen oxide emissions rate shall never exceed 0.10 lb/MMBTU, during or after the SNCR optimization study." (Ref: Page 26 of KMPP permit, available on website at:

http://www.air.ky.gov/NR/rdonlyres/696A8A04-2F29-4338-AD6A-7F6B29252676/0/Final.pdf)

By contrast, the permit for Deseret's WCFU says the final limit is 0.080 lb/MMBtu, with no waiver or provision for raising the limit later. Since Kentucky is willing to waive the initial NO_x emission limit for up to a year while a study is conducted, and adjust it up to as high as 0.10 lb/MMBtu after the study is conducted, EPA discounts to some degree the significance of KMPP's initial emission limit of 0.07 lb/MMBtu.

Comment #4.c.(8) has not resulted in any change to the permit; however, the KMPP permit has been added to the above-mentioned table in the Statement of Basis, along with the explanation above on why EPA discounts to some degree the significance of KMPP's initial emission limit of 0.07 lb/MMBtu.

Comment #4.c.(9): Commenters asserted that the draft permit fails to address BACT requirements for NO_x when Deseret is using "run-of-mine" coal either in lieu of waste coal, or as a blend with waste coal, from the Deserado mine. (Commenters cited condition III.E.2.c. of the draft permit.) As indicated by EPA in correspondence to Deseret Power, BACT needs to be met "for the entire range of operating conditions." Yet, commenters argued, EPA did not provide any review of BACT or propose any emission limits to address BACT when the Bonanza WCFU is burning the much higher quality coal either wholly or in part. As discussed above, commenters argued, such a BACT limit must be imposed on a 24-hour average basis to ensure the maximum degree of NO_x emission reduction is required when 100% "run-of-mine" coal is being burned.

Response #4.c.(9): Disagree. The authorization to burn run-of-mine coal is not unlimited as implied by commenters, but is restricted in the draft WCFU permit, as follows: Condition III.E.2.b. only allows Deseret Power to burn washed or run-of-mine coal during emergencies when waste coal is not available. For situations other than startup or emergencies, condition III.E.2.c. allows use of run-of-mine coal blended with waste coal in any ratio yielding up to 6,500 Btu/lb heat content. This corresponds to roughly a 50/50 blend. Use of run-of-mine coal for the WCFU, either in lieu of waste coal, or as a blend with waste coal, was evaluated in detail on pages 25-29 of the draft Statement of Basis. The proposed BACT determination for NO_x, as well as for other pollutants, is based on the proposed fuel restrictions in the draft PSD permit, also laid out on page 29 of the draft Statement of Basis.

In summary, comments #4.c.(1) through (9) have not resulted in any changes to the permit. However, the Statement of Basis has been changed as follows:

(1) Added a list of additional NOx control options from the Nov. 1999 EPA Technical Bulletin that were not already addressed in the draft Statement of Basis, along with an explanation of why each option was eliminated in the top-down BACT evaluation for Deseret's WCFU,

(2) Revised the cost analysis for SCR, to reflect a lower NO_x emissions rate achievable with SCR than 0.04 lb/MMBtu, and

(3) Added the Kentucky Mountain Power Project to the list of CFB projects with permitted NOx emission rates of 0.07 lb/MMBtu, along with an explanation of why EPA discounts to some degree the significance of KMPP's initial emission limit of 0.07 lb/MMBtu.

4.d -- Total PM/PM_{10}:

Comment #4.d.(1): One group of commenters asserted that EPA's proposed emission limit for total PM/PM_{10} does not reflect BACT. Commenters noted that EPA has proposed a limit for total PM/PM_{10} of 0.03 lb/MMBtu, on a 30-day rolling average. However, commenters argued, as shown in the data provided by EPA in its Statement of Basis, this limit does not reflect the maximum degree of reduction that can be achieved.

Specifically, commenters noted, EPA identifies several other CFB boilers with similar pollution controls as proposed for the Bonanza WCFU with lower total PM/PM_{10} limits. (draft Statement of Basis at 57.) Six of the eight CFB boiler permits reviewed by EPA had lower total PM limits than the proposed 0.03 lb/MMBtu. Three of the eight permits reviewed had limits on total PM of 0.012 lb/MMBtu. Commenters argued that EPA readily discounted these emission limits, but without any review of the specific details behind these emission limits (such as how the sources calculated these emission limits). (draft Statement of Basis at 58.)

Commenters further stated that while EPA did not discount the total PM emission limits of the three proposed facilities in Region 8 (Highwood, Gascoyne, and South Heart), which ranged from 0.0232 lb/MMBtu – 0.026 lb/MMBtu, EPA did not ultimately find that the methodology consistently used by these three facilities for calculating condensible PM emissions was appropriate for the Bonanza WCFU and instead allowed Bonanza's overestimate of ammonium sulfate to dictate the level of the total PM BACT limit. (draft Statement of Basis at 55-56.)

Response #4.d.(1): Disagree. EPA's calculated estimate is consistent with other projects cited in the draft Statement of Basis, not with Deseret's original calculation. As explained in the draft Statement of Basis, EPA found that Deseret's calculations of condensible emissions were not consistent with other permit applicants. "Consequently, EPA did a mass balance calculation that assumed all of the ammonia slip coming out of the CFB combustor unit (i.e., immediately downstream of SNCR controls) would react with sulfuric acid to form ammonium sulfate. This would occur upstream of the dry scrubber and baghouse. EPA also assumed 85% control of ammonium sulfate by the dry scrubber and baghouse. *These assumptions were consistent with analyses in permit applications reviewed by EPA for other CFB boiler projects.* EPA's calculation yielded an emission estimate of 0.0036 lb/MMBtu for ammonium sulfate. This was about one-fifth of Deseret Power's estimated emission range of 0.014 to 0.0209 lb/MMBtu." (emphasis added) (draft Statement of Basis at 56)

Federal PSD rules at 40 CFR 52.21 pertaining to BACT determination do not require EPA to review the specific details of how emission limits were calculated at other facilities. The other facilities (listed on pages 57-58 of the draft Statement of Basis) are only somewhat similar to the proposed WCFU. As explained in the Statement of Basis, in selecting an initial limit of 0.03 lb/MMBtu for total PM/PM_{10} at the WCFU (including condensible PM), EPA relied to a large extent on its own emission calculations specific to the WCFU, for individual components of condensible PM.

Comment #4.d.(1) has not resulted in any change to the permit or Statement of Basis.

Comment #4.d.(2): Commenters further alleged that the actual stack test data for similar sources are lower than EPA's proposed total PM BACT limit, with results ranging from 0.004 lb/MMBtu to 0.023 lb/MMBtu using EPA Method 202. (draft Statement of Basis at 59.) Thus, commenters argued, the majority of the data provided by EPA in its draft Statement of Basis indicate that its proposed total PM/PM_{10} BACT limit does not reflect the maximum degree of emission reduction that can be achieved as required by the definition of BACT. While EPA's proposed 0.03 lb/MMBtu emission limit incorporates a "margin of safety," the margin of safety is too lenient.

Response #4.d.(2): Disagree. The draft Statement of Basis (at page 59) actually refers to 0.03 lb/MMBtu as "an initial emission limit that EPA believes can reasonably be achieved (with appropriate margin of compliance)…" EPA does not consider the margin in this case to be too lenient. EPA's rationale for proposing an initial permit limit of 0.03 lb/MMBtu is explained in great detail on pages 54-64 of the draft Statement of Basis.

As explained in the draft Statement of Basis, EPA did evaluate stack testing data from other facilities, but there are minimal data on condensible PM emissions from CFB units, and no data for CFBs burning bituminous waste coal. The proposed permit limit of 0.03 lb/MMBtu is largely based on emission calculations that are specific to Deseret Power's proposed WCFU, as described on page 56 of the draft Statement of Basis. No numerical margin of compliance was incorporated into the calculation of 0.03 lb/MMBtu as an initial limit. Rather, EPA considers the "margin of compliance" to be the ability to revise the limit upward, to no more than 0.045 lb/MMBtu, if stack testing results show that a limit of 0.03 lb/MMBtu is not achievable.

Although EPA is establishing a total PM/PM_{10} emissions limit in the final WCFU permit that includes condensible PM consistent with the draft permit, we must note that EPA has recently acknowledged the concerns regarding the availability and implementation of test methods for condensible PM. As a result of these concerns, EPA's recent $PM_{2.5}$ implementation rule for State Implementation Plans has adopted a transition period during which EPA will assess possible revisions to available test methods and allow time for States to update emissions inventories. 72 Fed. Reg. 20586, 20650 (Apr. 25, 2007). EPA is currently considering whether it should also establish a similar transition period in its forthcoming $PM_{2.5}$ implementation rule for the New Source Review permitting program. Notwithstanding this ongoing assessment, EPA has decided to retain the proposed total PM/PM_{10} emissions limit to accommodate the request of the permit applicant that we not allow that rulemaking action to delay the completion of this permit.

Comment #4.d.(2) has not resulted in any change to the permit or Statement of Basis.

Comment #4.d.(3): In addition, commenters asserted, due to the deficiencies in EPA's 0.03 lb/MMBtu BACT determination for total PM/PM_{10}, the permit must not allow for an even further relaxation of this limit up to 0.045 lb/MMBtu. Commenters said this upper bound limit is unjustified as BACT.

Response #4.d.(3): Disagree. As explained on pages 58-64 of the draft Statement of Basis, due to the inherent uncertainty with setting a limit for total PM/PM_{10} that includes condensible PM at a CFB unit burning bituminous waste coal, EPA believes that 0.045 lb/MMBtu is an appropriate upper bound for possible adjustment of the limit. In describing its engineering calculation to estimate the amount of condensible PM that would be emitted from the WCFU, EPA pointed out the uncertainties in the calculation, due to the complexities in the chemical reactions taking place from fuel combustion. (draft Statement of Basis at 57.) As also explained in the draft Statement of Basis, there are only minimal stack test data for somewhat similar projects.

EPA believes the provision to adjust the limit upward later, to no more than 0.045 lb/MMBtu, pending EPA review of stack test results, is warranted and consistent with EPA's approach in other similar situations (e.g., AES Puerto Rico, cited on page 63 of the draft Statement of Basis and cited again below).

Comment #4.d.(3) has not resulted in any change to the permit or Statement of Basis.

Comment #4.d.(4): Commenters asserted that if Deseret Power obtains stack test data indicating that the total PM/PM_{10} BACT limit cannot reasonably be complied with, EPA can propose a revised total PM_{10} limit at a later time. Such a revised limit must be subject to public review and opportunity for comment.

Response #4.d.(4): Disagree. As explained on pages 54-64 of the draft Statement of Basis, EPA proposed an initial emission limit for total PM/PM_{10} of 0.03 lb/MMBtu, including condensible PM, based on EPA emission calculations specific to the proposed WCFU, as well as based on limited stack testing data for somewhat similar facilities. As mentioned in response #4.d.(3) above, EPA explained in the draft Statement of Basis the inherent uncertainty with setting a limit for total PM/PM_{10} that includes condensible PM at a CFB unit burning bituminous waste coal, due to the complexities in the chemical reactions taking place from fuel combustion. (draft Statement of Basis at 57.)

The Environmental Appeals Board (EAB) has recognized that "use of an adjustable limit, constrained by certain parameters, and backed by a worst case air quality analysis, is a reasonable approach." *In re AES Puerto Rico,* 8 EAD 324, 349 (1999).

As explained on page 63 of the draft Statement of Basis, EPA proposed to allow in the permit that the limit could be adjusted upward, to no more than 0.045 lb/MMBtu, pending EPA review of stack test results at the WCFU. This sets an upper bound on the possible adjustment. EPA stated in the draft permit itself that "[b]ecause condensible

particulate matter emissions from CFB boilers have not been widely quantified, there is a possibility that the actual condensible portion of particulate matter would cause the emission limit of total PM/PM_{10} to be exceeded. In the event the Permittee cannot meet that limit because of condensible particulate matter, EPA may adjust the emission limit to a level not to exceed 0.045 lb/MMBtu, pending EPA's review of stack test results at the CFB boiler." (draft WCFU permit at page 7.) The range of possible emission limits is therefore constrained between 0.03 lb/MMBtu and 0.045 lb/MMBtu, and was subject to public review and comment.

As listed on page 128 of the draft Statement of Basis, the WCFU emission rate of total PM/PM_{10} used for modeling of ambient air quality impact was 9.47 grams per second. This rate is equivalent to 0.052 lb/MMBtu multiplied by the WCFU maximum design heat input capacity of 1,445 MMBtu/hr. Even if the BACT limit of 0.03 lb/MMBtu is adjusted to the upper bound in the draft permit of 0.045 lb/MMBtu, the limit is still lower than the emission rate used for modeling. Therefore, the range of 0.03 to 0.045 lb/MMBtu is backed by a worst case air quality analysis, and at the upper bound of 0.045 lb/MMBtu, the NAAQS and PSD increment will still be protected.

In summary, EPA believes that the possible future adjustment of the total PM/PM_{10} emission limit, within the parameters specified in the permit, is a "reasonable approach" as recognized by the EAB. The range of possible adjustment was subject to public review and opportunity for comment, therefore does not require additional public review and opportunity for comment later. Comment #4.d.(4) has not resulted in any change to the permit or Statement of Basis.

Comment #4.d.(5): Commenters concluded that until such time as the limit is revised, the evidence provided by EPA indicates that the proposed total PM/PM_{10} BACT limit is too high.

Response #4.d.(5): Disagree, as explained on pages 54-64 of the draft Statement of Basis, and as explained in responses #4.d.(1) through #4.d.(3) above. Comment #4.d.(5) has not resulted in any change to the permit or Statement of Basis.

Comment #4.d.(6): Commenters further stated that the draft WCFU permit also fails to address BACT requirements when Deseret Power is using"run-of-mine" coal either in lieu of waste coal, or as a blend with waste coal, from the Deserado mine (as allowed by Condition III.E.2.c. of the draft permit). As indicated by EPA in correspondence to Deseret Power, BACT needs to be met "for the entire range of operating conditions." (Ref: April 7, 2006 e-mail from Mike Owens, EPA Region 8, to Ed Thatcher, Deseret Power.) Yet, commenters said, EPA did not provide any review of BACT or propose any emission limits to address BACT when the Bonanza WCFU is burning the much higher quality coal either wholly or in part. Commenters argued that, as discussed above, such a BACT limit must be imposed on a 24-hour average basis to ensure the maximum degree of PM emission reduction is required when 100% "run-of-mine" coal is being burned.

Response #4.d.(6): Disagree. See draft Statement of Basis at page 64: "As explained earlier in this Statement of Basis, for the proposed WCFU, Deseret Power will be permitted to use coal from their Deserado mine, consisting of either waste coal alone, or else a blend of waste coal and ROM coal yielding heat content of up to 6,500 Btu/lb. For reasons explained above, EPA believes the proposed BACT emission limit of 0.03 lb/MMBtu on a rolling 30-day average, for total PM/PM_{10}, will represent BACT for coal from the Deserado mine with heat content of up to at least 6,500 Btu/lb, and will ensure a continued high degree of PM emission control efficiency." Comment #4.d.(6) has not resulted in any change to the permit or Statement of Basis.

In summary, comments #4.d.(1) through (6) have not resulted in any changes to the permit or Statement of Basis.

4.e -- Visible Emissions:

Comment #4.e: A group of commenters asserted that EPA failed to evaluate and impose a BACT limit for visible emissions (VE), and that the BACT analysis for the Bonanza WCFU must include a visible emission limit reflective of BACT for the source.

Commenters argued that the definition of BACT at 40 CFR 52.21(b)(12) specifically indicates that BACT includes a "visible emission limitation." Commenters noted that in the draft Statement of Basis, EPA indicated that, because EPA is proposing use of a PM continuous emission monitoring system (CEMS), "EPA does not consider it necessary to also propose an opacity limit as part of BACT for total filterable particulate." (draft Statement of Basis at 47.) Commenters argued that EPA's reasoning is flawed for several reasons, described in comments #4.e.(1) through (5) below:

Comment #4.e.(1): Commenters argued that the definition of BACT in the Clean Air Act and associated federal regulations specifically mandate that BACT include a visible emission (or opacity) limitation. There are no exemptions provided for in the statutory or regulatory definition. Thus, commenters concluded, EPA is without legal authority to decide not to impose an opacity limit because it is requiring PM CEMS for the PM limit.

Response #4.e.(1): Disagree. EPA does not view the phrase "visible emission standard," in the BACT definition at 40 CFR 52.21(b)(12), as an emission limit that all PSD permittees must meet, nor as implying that a visible emission (VE) and/or opacity limit must be included in all PSD permits. However, while these limits are not required under BACT, permitting authorities have the discretion to include them in PSD permits in order to ensure compliance with BACT emission limitations.

In fact, the Circuit Court of Appeals for the District of Columbia (DC Circuit) found that visible emission limitations were properly included in PSD permits as "one such means of measuring and limiting emissions" under BACT, but stated that "EPA's inclusion of visible emission standards (among others) to be used to determine compliance with BACT sets no single standard that all PSD permittees must meet." *Alabama Power v. Costle*, 636 F.2d 323, 408 (D.C. Cir. 1979) (emphasis added). Instead, the DC Circuit found that permitting authorities "may exercise reasonable discretion" to include opacity/VE limits in BACT for a particular facility. Id. at 409. The Environmental Appeals Board has also found that such limits are not a requirement of PSD program permitting. *See Knauf Fiber Glass*, 8 E.A.D. 121, 172 (1999) (finding that opacity limits are "not a requirement of the federal PSD program").

Accordingly, in order to avoid confusion regarding the opacity limit contained in the "PSD BACT Emission Limits" section of the permit, EPA notes that the opacity limit is included for demonstrating continued proper operation and maintenance of the materials handling baghouses, not because it is required under BACT. The permit title section III.D has been amended to indicate the section includes limits in addition to BACT.

Comment #4.e.(1) has not resulted in any substantive change to the permit or Statement of Basis.

Comment #4.e.(2): Commenters argued that the Particulate Matter Continuous Emission Monitoring System (PM CEMS), required by the draft permit, will only measure filterable particulate matter, while opacity measures all particulate matter that may block the transmission of light exiting the stack including condensible particulate matter. While compliance with the total particulate matter limit must be demonstrated on a rolling 30-day average basis at the Bonanza WCFU (Condition III.D.1.a. of the draft permit), this compliance determination will be based on a once-per-year stack test of the total PM emission rate (Condition III.I.4.b of the draft permit). Commenters concluded that an opacity limit that can be continuously monitored will provide a much needed additional assurance that the total particulate matter emission limits are being complied with continuously.

Response #4.e.(2): Disagree. While it is true that PM CEMS only measures filterable particulate matter, EPA believes that opacity monitoring at Deseret Power's WCFU, as an addition to requiring a PM CEMS calibrated according to 40 CFR 60, Appendix B, Performance Specification 11, would be ineffective for assuring compliance with emission limits for either filterable PM or total PM.

Opacity monitoring can be useful as a surrogate for direct measurement of particulate emissions. However, EPA does not consider it useful for assuring compliance with PM emission limits where those limits are extremely low. The proposed emission limit for Deseret Power's WCFU for total PM/PM_{10} is 0.03 lb/MMBtu. This limit is based on a filterable PM/PM_{10} emission limit of 0.012 lb/MMBtu, added to projected emissions of no more than 0.018 lb/MMBtu for condensable PM. These emission limits are so low that EPA believes it highly improbable, if not impossible, that any form of existing opacity monitor could reliably detect opacity at levels that would correspond to these limits. Moreover, given the sensitivity of the PM CEMS, elevated emissions would be detected by PM CEMS well in advance of detection via a Continuous Opacity Monitoring System (COMS), or via a Method 9 or Method 22 visible emissions observation. Further, opacity only provides data from a subset of all particles, namely those particles whose size is roughly the same wavelength as visible light.

A status report prepared for EPA on PM CEMS, dated February 12, 1997 (available at http://www.epa.gov/ttn/emc/cem.html, labeled on website as "PM CEMS Demo. Test, Status Report 4 (Adobe format)," with website posting date of 7/31/97) in support of proposed revised regulations for hazardous waste combustors (HWC), states (in the Introduction, as quoted below) that opacity monitors are insensitive at filterable PM concentrations below 45 mg/dscm. For coal combustion, this is equivalent to about 0.04 lb/MMBtu. (The proposed total PM/PM_{10} emission limit for Deseret Power's WCFU, cited above, is lower than this.) The Introduction states:

EPA in the past has relied on opacity monitors as a form of surrogate-PM monitoring to indicate compliance with a PM standard. This approach involved a continuous opacity monitor to demonstrate compliance with a separately-enforceable opacity limit approximately aligned with, or near, the PM emission limit. However, this approach has a serious limitation relative to the proposed HWC rule, because of poor correlation between opacity and PM at low PM concentrations near the proposed PM emission limit of 69 mg/dscm (at 7 % O2).

EPA recognizes that there are two inherent problems with the opacity/PM approach: 1) the general concern about the stability of any opacity/PM correlation, which is strongly dependent on particle size distribution and composition, and 2) the specific concern about the insensitivity of opacity monitors typically below PM levels of about 45 mg/dscm (at 7 % O2).

Consequently, opacity monitors would not be sufficient because to maintain compliance with 69 mg/dscm, facilities would generally need to operate near 35 mg/dscm. Thus, emissions would typically be below the detection limits of opacity monitors most of the time. While normal emission levels below the detection limits of CEMS are acceptable, facilities often desire the detection limit to be one-tenth of the emission limit. This gives sufficient warning of how emissions are changing before the emission limit is approached, and allows the facility, based on CEMS readings, to change operations as necessary to be in compliance.

If possible, EPA desires a quantitative, continuous measure of PM mass concentrations rather than opacity. Based on surveys and preliminary testing, EPA has recently determined that CEMS do exist that do this: beta gauges and light scattering based CEMS. These CEMS rely on calibration/certification of the device by manual gravmetric measurements. Therefore, EPA is proposing use of CEMS based on the availability of these newer technologies and a related Draft CEMS Performance Specification for monitoring PM mass concentration. EPA believes that such monitoring is feasible and that opacity monitoring has borderline sensitivity relative to the proposed PM emission limit. The newer technology PM CEMS can give a real-time quantitative measure of low PM concentrations while opacity monitors cannot.

(Ref: Status Report No. IV, Particulate Matter CEMS Demonstration, prepared by Energy and Environmental Research Corporation, EPA Contract 68-D2-0164, Work Assignment 4-02, February 12, 1997.)

This same reasoning is reflected in the recent revisions to Subpart Da of New Source Performance standards (40 CFR 60). The revised Subpart Da exempts facilities from ongoing opacity monitoring where PM CEMS is installed and used.

Further, the proposed emission control technique for condensable PM at Deseret Power's WCFU is a combination of alkali injection, dry SO_2 scrubbing and a fabric filter baghouse. (Draft Statement of Basis at 50.) Each of these control techniques will be

installed and used to comply with other emission limits in the permit (alkali injection for NO_x control, dry SO_2 scrubbing for SO_2 control, and a fabric filter baghouse for filterable PM control). The permit requires compliance with these three other emission limits to be tracked continuously via CEMS. (Draft WCFU permit at pages 16-17, conditions III.I.4.a and III.I.4.c.) This continuous monitoring, in addition to annual stack tests required in the permit for condensable PM, is considered by EPA to be sufficient for ensuring good control of the both the condensable PM portion and the filterable PM portion of total PM. EPA does not agree with commenters that opacity monitoring "will provide a much needed additional assurance that the total particulate matter emission limits are being complied with continuously."

In summary, comment #4.e.(2) has not resulted in any change to the permit. However, the Statement of Basis has been revised to included the expanded explanation above on why EPA does not consider an opacity limit or opacity monitoring to be necessary at the WCFU.

Comment #4.e.(3): Commenters argued that a limitation on visible emissions serves as an indicator of proper operation and maintenance (O&M) of all pollution control equipment.

Response #4.e.(3): Disagree. EPA recognizes that opacity monitoring can be useful as an indicator of proper baghouse O&M; however, as explained in response #4.e.(2), the proposed emission control technique for condensable PM at Deseret Power's WCFU is a combination of alkali injection, dry SO_2 scrubbing and a fabric filter baghouse, not just a baghouse. EPA considers CEMS for NO_x, SO_2 and PM to be far more useful than opacity monitoring for ensuring good control of condensable PM at Deseret Power's WCFU. Further, the fact that opacity monitoring can be useful as an indicator of proper baghouse O&M does not mean it is necessarily useful for ensuring compliance with PM emission limits. As explained in response #4.e.(2), in this case EPA does not believe it is useful. Comment #4.e.(3) has not resulted in any change to the permit or Statement of Basis.

Comment #4.e.(4): Commenters argued that compliance with both the filterable and total PM/PM_{10} limits is based on a rolling 30-day average basis, whereas compliance with opacity BACT limits are based on a six-minute averaging time. Thus, commenters concluded, the 30-day rolling average filterable PM limit measured with PM CEMS is not an adequate replacement for a six-minute average opacity BACT limit.

Response #4.e.(4): Disagree. See responses #4.e.(2) and #4.e.(3).

Comment #4.e.(5): Commenters argued that with a fabric filter baghouse for PM_{10} control, an opacity BACT limit should be "at least 10%." Commenters noted that the recently permitted Sevier CFB power plant in Utah is subject to a 10% visible emissions limit. The River Hill Power Company proposed CFB power plant in Pennsylvania is also subject to a 10% opacity limit. Similarly, the Gascoyne CFB facility in North Dakota will also be subject to a 10% opacity BACT limit. Commenters also

noted that the permit for the Longview power plant in West Virginia, which will utilize a pulverized coal boiler, requires PM CEMS and imposes a 10% opacity BACT limit.

Response #4.e.(5): Disagree. Commenters have not presented any evidence that a 10% opacity limit would have any correlation with the proposed total PM/M_{10} emission limit of 0.03 lb/MMBtu for Deseret Power's WCFU. Further, as explained in response #4.e.(2), EPA believes it highly improbable, if not impossible, that any form of existing opacity monitor could reliably detect opacity at levels that would correspond to PM emission rates as low as 0.03 lb/MMBtu. EPA has also found there is poor correlation between opacity and PM at such low PM concentrations. Comment #4.e.(5) has not resulted in any change to the permit or Statement of Basis.

Comment #4.e.(6): Commenters concluded that EPA must include an evaluation of opacity BACT in its Statement of Basis and must impose a visible emission limit on the Bonanza WCFU that reflects the maximum degree of reduction achievable. Further, to ensure compliance on a continuous basis, commenters concluded that a continuous opacity monitoring system (COMS) must be required.

Response #4.e.(6): Disagree. See responses #4.e.(1) through (5) above.

In summary, comments #4.e.(1) through (6) have not resulted in any change to the permit. However, the Statement of Basis has been revised, to included the expanded explanation provided in response #4.e.(2), on why EPA does not consider it necessary to impose an opacity limit or opacity monitoring at the WCFU.

5. MEETING BACT LIMITS ON A CONTINUOUS BASIS AND MEETING ENFORCEABILITY CRITERIA

Comment #5:

One group of commenters stated that all BACT limits must be met on a continuous basis and must meet enforceability criteria, but that the draft Bonanza WCFU permit does not adequately address EPA requirements for including such provisions.

5.a – Meeting BACT Limits on a Continuous Basis:

Comment #5.a.(1): Commenters cited from the draft 1990 NSR Workshop Manual that "BACT emission limits or conditions must be met on a continual basis at all levels of operation (e.g., limits written in lb/MMBtu or percent reduction achieved), demonstrate protection of short term ambient standards (limits written in pounds per hour) and be enforceable as a practical matter (contain appropriate averaging times, compliance verification procedures and recordkeeping requirements)." (NSR Workshop Manual at B.56). Commenters argued that EPA did not propose BACT limits consistent with these criteria.

Specifically, commenters argued, with respect to all of the emission limits, there must be pound per hour emission caps established, in addition to lb/MMBtu limits, that must be reflective of BACT and consistent with what is modeled to show compliance with the NAAQS, PSD increments, and air quality related values. Commenters stated the NSR Workshop Manual indicates that it is best to express emission limits in two different ways, "with one value serving as an emissions cap (e.g., lb/hr) and the other ensuring continuous compliance at any operating capacity (e.g., lb/MMBtu)." (NSR Workshop Manual at H.5. See also *In re Steel Dynamics, Inc.*, PSD Appeal Nos. 99-4 & 99-5, Decided June 22, 2000, at 220-225.)

Commenters noted that EPA only proposed BACT limits in terms of lb/MMBtu, and EPA did not evaluate or propose BACT limits in terms of lb/hr. While EPA did propose lb/hr "modeling limits" for SO_2 and total PM_{10} (Section G. of the draft permit), commenters stated that these modeling limits are not reflective of BACT for the Bonanza WCFU. Commenters argued that at full heat input capacity, the 3-hour average 872 lb/hr SO_2 modeling limit is equivalent to 0.6 lb/MMBtu, which would be only 87% SO_2 removal from worst case uncontrolled SO_2 emissions. The 24-hour total PM_{10} modeling limit of 75.4 lb/hr is equivalent to 0.052 lb/MMBtu at full heat input capacity – which, commenters noted, is greater than the maximum level EPA has proposed the total PM_{10} limit could be raised to. Commenters concluded that these modeling limits clearly do not reflect BACT for these pollutants. Commenters also asserted that EPA failed to propose BACT limits in terms of lb/hr for NO_x, CO, or H_2SO_4.

Further, commenters argued, the averaging time of the BACT emission limits must be "of a short-term nature" and must be consistent with the averaging time of the short term NAAQS and PSD increments, including a 24-hour averaging time for PM_{10}

limits, an 8-hour averaging time for CO limits, and an 8-hour averaging time for VOC limits, as well as the 24-hour averaging time for the pollutants modeled in the visibility modeling. (NSR Workshop Manual at H.5.) Yet, commenters stated, EPA's proposed lb/MMBtu BACT limits for SO_2, NO_x, CO, and PM_{10} for the Bonanza WCFU are all based on rolling 30-day averages. Commenters concluded that, while EPA has proposed short term average emission limits for SO_2 and PM_{10} as modeling limits, these limits are not reflective of BACT for these pollutants.

Response #5.a.(1): Partially disagree. EPA agrees that a shorter-term limit than rolling 30-day should be specified for filterable PM. As discussed below, EPA has changed the averaging time to daily in the final permit.

EPA does not agree, however, that emission limitations for purposes of PSD increment protection and NAAQS protection (such as the lb/hr limits that EPA included in the draft permit, labeled "modeling limits") have to reflect BACT, nor that BACT limits must always include lb/hr limits and correspond to all averaging times of the PSD increments and NAAQS. As explained below in response #5.a.(2), EPA included Federally enforceable "modeling limits" in the draft permit specifically for ensuring compliance with the NAAQS and increments under 40 CFR 52.21(k). The rolling 30-day emission limits in lb/MMBtu in the draft permit reflect BACT, pursuant to 40 CFR 52.21(j)(3). As explained in detail on pages 30 through 90 of the draft Statement of Basis, for each pollutant for which a BACT determination is required by PSD rules, the BACT emission limit in lb/MMBtu for the WCFU was set at a level sufficiently stringent to reflect optimal emission control performance on a continual basis.

The "modeling limits" for the WCFU were established under different provisions of PSD rules than the BACT limits. As explained on page 138 of the draft Statement of Basis, "EPA interprets the PSD rules at 40 CFR 52.21(k) to require that emission limits be included in PSD permits ('modeling limits') consistent with emission rates used in dispersion modeling for ambient impacts, unless it would be physically impossible for the proposed source or modification to emit at a greater rate (i.e., maximum potential uncontrolled emissions). This requirement is *in addition to* the requirement under §52.21(j)(2) to establish BACT emission limits." [emphasis added; citation in the draft Statement of Basis should have been to §52.21(j)(3); this typographical error was corrected in the final Statement of Basis.]

EPA has proposed modeling limits in lb/hr corresponding to the emission rates assumed in dispersion modeling for cumulative ambient impacts of the proposed WCFU and all existing emitting units in the vicinity of the project. (The only exception is NO_x, for which, as explained in the draft Statement of Basis, EPA determined that the BACT limit could also serve as a modeling limit, without need to propose a separate modeling limit for NO_x in lb/hr.)

These modeling limits are enforceable emission limits in the permit, and were established based on worst-case operating scenarios used for dispersion modeling. For example, the modeling limits for 3-hour and 24-hour SO_2 are based on a cold startup,

which is the worst-case SO_2 emission scenario that might be expected, not optimal emission control performance on a continual basis. This worst-case scenario is described on page 138 of the draft Statement of Basis and was the scenario used for dispersion modeling for demonstrating PSD increment protection and NAAQS protection.

Although the permit does not list any lb/hr emission limits as BACT limits, the lb/MMBtu BACT limits in the permit, when multiplied by the maximum heat input capacity of the CFB boiler, are mathematically equivalent to lb/hr values. The draft WCFU permit says, on page 5, that the "Approved Installation" includes "One circulating fluidized bed boiler, *maximum heat input capacity not to exceed 1,445 MMBtu/hr,* designed for firing on waste coal." [emphasis added] As stated above, the lb/MMBtu limits in the permit were set at a level sufficiently stringent to reflect optimal emission control performance on a continual basis.

Furthermore, during periods of low boiler load, lb/hr emission caps would not necessarily reflect BACT. In a letter to Deseret Power dated June 22, 2005 (included in the Administrative Record for issuance of the WCFU permit), EPA questioned whether the lb/hr emission caps proposed by Deseret Power for startup/shutdown (i.e., during periods of low boiler load) could be justified as BACT, in terms of optimal use of the emission control equipment. The emission cap approach was ultimately not used in the permit for startup/shutdown periods. Instead, the permit states, at condition III.I.1, that the BACT emission limits (all expressed in lb/MMBtu) apply at all times, including periods of startup/shutdown.

Contrary to the conclusion implied by commenters, the Steel Dynamics EAB decision does <u>not</u> say PSD permits must include both emission caps (e.g., lb/hr limits) and production-based limits (e.g., lb/MMBtu). Instead, the EAB decision says, on page 225, that the Indiana Department of Environmental Management (IDEM) "…is ordered to explain why the limits it imposed are in lbs/hr (rather than in lbs/hr *and* lbs/ton, or lbs/ton alone), in particular explaining in detail the specific differences (if any) between SDI's proposed mill and the fifteen polled mills that would justify exclusive lbs/hr limits for CO and NO_x." The EAB decision does not preclude the possibility that production-based limits alone, absent lb/hr limits, could constitute BACT.

The Steel Dynamics decision involves considerations specific to the batch-type nature of the steelmaking process, which, as explained in the decision, could warrant lb/hr limits as BACT in some cases. Deseret Power's proposed WCFU is an electric utility generating unit designed to run continuously, not a batch-type operation. PSD rules define BACT as a case-by-case determination. The considerations in the BACT determination for the WCFU are different than for a batch-type steelmaking process, and affect whether or not lb/hr limits are warranted as part of BACT.

EPA Region 8's comments on state permit actions are consistent with the statements above that modeling limits in lb/hr don't necessarily have to reflect BACT, nor that BACT limits must always include lb/hr limits and correspond to all averaging

times of the PSD increments and NAAQS. In commenting to Montana on the draft PSD permit for the Roundup coal-fired electric utility project, EPA Region 8 wrote:

> *Currently the draft permit only contains SO_2 emission limitations on a 30-day rolling average. This approach may be acceptable* <u>*only*</u> *if modeling for protection of the short-term NAAQS and PSD increments was based on worst-case hourly SO_2 emissions, rather than on the 30-day emission limitations in the draft permit. At a minimum, we believe the permit action should either establish short-term emission limits in the permit itself, or justify that worst-case hourly SO2 emission limits have been modeled for protection of short-term NAAQS and PSD increments.*
>
> (Ref: Letter from Richard R. Long, EPA Region 8, to Steve Welch, Montana Department of Environmental Quality, December 18, 2002, page 6; emphasis added.)

The letter on Roundup does not say or imply that short-term emission limitations must reflect BACT, but only that short-term limits must be imposed as necessary in the permit to validate the assumptions used in dispersion modeling.

Similarly, in an EPA guideline document, on SO_2 emission limitations in State Implementation Plans (SIPs), the stated purpose of short-term emission limitations is the protection of ambient standards. The document does not say or imply that short-term emission limitations must also reflect BACT:

> *The EPA policy regarding averaging periods for SO_2 SIP emission limitations is to require enforceable limits that protect the short-term (3-hour and 24-hour) NAAQS as well as the annual NAAQS. These emission limitations must be protective with maximum emission scenarios and worst-case meteorological conditions. ... The EPA will not approve an SO_2 SIP with emission limitations based on 30-day averaging, unless the SIP also contains short-term limits established by an approved dispersion modeling analysis. This point is especially important for SO_2 sources that are complying with an NSPS (e.g., subpart Da). Although subpart Da allows 30-day averaging, parameters for evaluating the control system on a short-term basis must also be established for compliance with the NAAQS and PSD increments.*
>
> (Ref: "SO_2 Guideline Document," EPA-452/R-94-008, February 1994, page 6-14, available online at: http://nepis.epa.gov/pubtitleOAR.htm.)

Similarly, a 1986 letter signed by EPA's Assistant Administrator for Air and Radiation, regarding SO_2 SIPs, states the same purpose for short-term emission limitations:

> *EPA has had a long standing policy to require emission limitations to be enforceable on a short-term basis to protect the short-term standard.*

(Ref: Letter from J. Craig Potter, Assistant Administrator for Air and Radiation, US EPA, to Nancy Maloley, Commissioner, Indiana Department of Environmental Management, May 23, 1986, page 1, included in the Administrative Record for issuance of the WCFU permit.)

Again, there is no statement that such limitations must also reflect BACT.

Similarly, a 1986 EPA memorandum states the following:

> *The PSD regulations clearly require that the application of BACT conform with any applicable standard of performance under 40 CFR Part 60 at a minimum. However, this should not be taken to supercede any additional limitations as needed to enable the source to demonstrate compliance with the NAAQS and PSD increments. In the case of sulfur dioxide (SO2), source compliance with the 30-day rolling average emission limit under subpart D(a) does not adequately demonstrate compliance with the short-term NAAQS and PSD increments.*
>
> Ref: Memorandum from Gerald A. Emison, Director, Office of Air Quality Planning and Standards, US EPA, to David Kee, Director, Air Management Division, EPA Region 5, November 24, 1986, page 1, available online at:
>
> http://www.epa.gov/region7/programs/artd/air/nsr/nsrmemos/shrtterm.pdf

Again, the stated purpose of the short-term emission limitations is the protection of short-term ambient standards. There is no statement that such limitations must also reflect BACT.

With regard to the statement in the 1986 EPA memorandum that "the application of BACT conform with any applicable standard of performance under 40 CFR part 60 at a minimum," EPA notes that the averaging times of the proposed BACT emission limits for the WCFU conform with those in 40 CFR 60, subpart Da, with the exception of filterable PM/PM_{10}, for which the draft WCFU permit specifies a 30-day rolling average, whereas Subpart Da specifies a daily average. To conform with 40 CFR part 60, subpart Da, EPA believes it is necessary to change the averaging time of the BACT limit to daily.

In summary, EPA believes the draft permit for the WCFU includes all short-term (lb/hr) emission limitations that are necessary to protect ambient standards, to the extent required by Federal rules at 40 CFR 52.21(k). EPA also believes the draft permit contains all emission limits necessary to satisfy BACT, as required by Federal rules at 40 CFR 52.21(j)(3), with the exception that EPA believes the averaging time of the BACT limit for filterable PM/PM_{10} should be changed from 30-day rolling to daily, to conform with 40 CFR 60 subpart Da. Therefore, to the extent that commenters suggest shorter-term limits are needed than those in the draft permit, EPA agrees with commenters with regard to PM/PM_{10}.

The final permit reflects this change. Instead of 0.012 lb/MMBtu on a 30-day rolling average, the limit in the final permit is 0.012 lb/MMBtu on a daily average. Monitoring, reporting and recordkeeping requirements in the permit have been changed accordingly. Since this limit is no more stringent than filterable PM/PM_{10} limits at some other new coal-fired projects (e.g., Utah permit ("Approval Order") dated October 15, 2004, for construction of Intermountain Power Unit 3, included in the Administrative Record for issuance of the WCFU permit), EPA believes it is achievable by Deseret Power. The Statement of Basis has been revised accordingly, to reflect a daily average (i.e., a 24-hour block average from midnight to midnight) rather than a 30-day rolling average. Comment #5.a.(1) has resulted in these changes to the permit and Statement of Basis.

Comment #5.a.(2): Commenters noted that EPA's Statement of Basis explains that the lb/hr emission rates used in the modeling analyses reflect short term emission peaks from startups. (draft Statement of Basis at 135.) Commenters asserted that EPA "admitted" that the proposed BACT limits for SO_2 and PM_{10} do not adequately limit short term emissions for compliance with the NAAQS and PSD increments because the BACT limits are based on 30-day rolling averages. (draft Statement of Basis at 136.) Yet, commenters stated, "as acknowledged by EPA in the Statement of Basis," BACT emission limits must be met on a continuous basis, and there are to be no exemptions for startup and shutdown. (draft Statement of Basis at 23.)

In particular, commenters stated, EPA noted in its draft Statement of Basis that the NSR Workshop Manual states (at page B.56) "BACT emission limits or conditions must be met on a continual basis *at all levels of operation*." [emphasis added by commeneters] Yet, commenters argued, EPA's proposed BACT limits violate these principles and essentially provide for startup and shutdown exemptions from BACT by providing such long averaging times for the BACT emission limits.

Response #5.a.(2): Disagree. First, EPA included "modeling limits" in the permit, as a separate set of limits from the BACT emission limits, specifically for ensuring compliance with the NAAQS and PSD increments. (draft Statement of Basis at 138-139). Second, the draft permit does not allow any exemptions from either the BACT limits or the modeling limits for startup/shutdown periods. Section III.I.1 of the draft permit states that "The PSD BACT emission limits in this permit, as well as the modeling limits, *apply at all times, including periods of startup, shutdown and malfunction*." [emphasis added] Moreover, the requirements in the permit for continuous monitoring of emissions of particulate matter, SO_2, NO_x and CO, and accompanying averaging times, are consistent with the concept of continuous compliance identified by the commenters.

As demonstrated by the detailed discussion in the draft Statement of Basis of the BACT determination for each pollutant, the 30-day limits have been set at levels sufficiently stringent as to not allow under-utilization of control equipment. Setting a stringent 30-day limit that applies at all times creates an incentive for the source to limit its duration of startup, shutdown, and malfunction periods in order to preserve any margin of compliance the source might be operating under.

In summary, EPA believes the proposed 30-day average BACT limits (and the limit for filterable PM/PM_{10}, on a daily average in the final permit), in conjunction with continuous emission monitoring, are consistent with the EPA policy that BACT applies at all times and must be met at all levels of operation. EPA also believes that inclusion of modeling limits in the draft permit adequately addresses the commenters' concern about NAAQS and PSD increment protection. Comment #5.a.(2) has not resulted in any change to the permit or Statement of Basis.

Comment #5.a.(3): Commenters further argued that EPA's failure to propose shorter averaging time emission limits reflective of BACT is inconsistent with recently issued permits for coal-fired power plants. Commenters cited the Roundup power plant permit issued by the state of Montana as requiring 24-hour average BACT limits for NO_x and SO_2, and also a 1-hour BACT limit for SO_2. Commenters also cited the Sevier power plant permit issued by the state of Utah as including rolling 24-hour average BACT limits for SO_2, NO_x, PM_{10}, and H_2SO_4. Commenters also cited the Longview power plant permit issued by the state of West Virginia as including a 3-hour average SO_2 BACT limit, 24-hour average NO_x and SO_2 BACT limits, a 6-hour average PM_{10} BACT limit and a 3-hour average H_2SO_4 BACT limit.

Commenters concluded that for all of the above reasons, EPA must revise its proposed BACT limits for the Bonanza WCFU to require shorter averaging times consistent with the NAAQS, PSD increments, and air quality related values standards and to also set lb/hr emission limits reflective of BACT, with compliance being monitored by continuous emission monitoring systems as proposed by EPA for SO_2, NO_x, and PM.

Response #5.a.(3): Disagree. Commenters appear to be suggesting that EPA is bound by state permitting decisions; however, as EPA stated in response #2.b above, state PSD permit actions are not binding on EPA and do not establish irrefutable precedence for EPA PSD permit actions. Further, by rule, BACT is a case-by-case determination. There may be case-specific reasons why certain permits contain different averaging times for their BACT limits. Although EPA has changed the filterable PM/PM10 limit from 30-day rolling average to daily average in the final permit, EPA has done so to conform with 40 CFR 60, subpart Da, not on the basis that EPA is bound by the averaging times in state permits.

As explained in response #5.a.(1), EPA's December 18, 2002 comment letter on the draft Roundup permit did not say the permit must include BACT limits corresponding to all averaging times of the PSD increments and NAAQS. Instead, the letter said only that there should either be short-term emission limits in the permit itself, or else the State should justify that worst-case hourly SO2 emission limits have been modeled for protection of short-term NAAQS and PSD increments. In the case of the WCFU permit, EPA has chosen to establish modeling limits to reflect the worst-case short-term emission rates assumed in dispersion modeling, for demonstration of short-term NAAQS and PSD increment protection.

EPA also disagrees with the comment that there should be a short-term emission limitation for VOC. Estimated potential emissions of VOC from the proposed project are 32 tons per year, below the PSD significance threshold. (draft Statement of Basis at 14.) A demonstration of NAAQS protection is not required where the estimated potential emissions of a pollutant (or precursor, in this case, VOC as a precursor of ozone) are below significance threshold. (draft Statement of Basis at 119) A BACT analysis is also not required. (draft Statement of Basis at 24)

EPA also disagrees with the comment that there should be a short-term emission limitation for CO. Since short-term emission limitations are generally used to protect PSD increments and NAAQS, and since there are no Class I or II increments for CO in PSD rules, the only applicable ambient standard that must be considered in response to comment #5.a.(3) is the CO NAAQS. While estimated potential emissions of CO from the proposed project are above the PSD significance threshold, the modeling results for ambient impacts from the proposed project were only 3.3% of the one-hour NAAQS for CO, and only 11.9% of the eight-hour NAAQS for CO. Because of these very low results in comparison to the NAAQS, and because modeling was based on "worst-case" startup emissions, EPA did not consider it necessary to include a lb/hr emission limit for CO in the permit. Since there is no Class I or II increment for CO in PSD rules, the only applicable ambient standard is the NAAQS.

Comment #5.a.(3) has not resulted in any change in the permit or Statement of Basis.

5.b – Meeting Enforceability Criteria:

Comment #5.b.(1): Commenters asserted that the permit must also specify appropriate compliance methods and recordkeeping requirements to show compliance with the short-term emission limits in comment 5.a. above. As discussed in the NSR Workshop Manual, "the construction permit should state how compliance with each limitation will be determined." (NSR Workshop Manual at H.6.). Commenters stated that the test methods must provide for continuous compliance where feasible. Commenters argued that when compliance with BACT emission limits is determined over a 30-day averaging period – even if monitored with continuous emission monitoring systems (CEMS), this does not ensure continuous compliance.

Response #5.b.(1): Disagree. Commenters have presented no basis for the implied claim that the permit fails to specify continuous compliance test methods where feasible. Continuous Emission Monitoring Systems (CEMSs) have been specified in the permit, at the CFB boiler stack, for every PSD pollutant where feasible. This includes CEMSs for particulate matter, SO_2, NO_x and CO. Commenters also have presented no basis for the claim that use of CEMS does not ensure continuous compliance with 30-day average limits. The draft permit contains detailed requirements for testing of the accuracy of the CEMS for each pollutant, as well as quality assurance requirements, recordkeeping requirements, and reporting requirements. This includes quarterly reporting on the performance of the CEMSs. (Draft permit at pages 21-27.)

Comment #5.b.(1) has not resulted in any change to the permit or Statement of Basis.

Comment #5.b.(2): Commenters also alleged that the draft permit for the Bonanza WCFU lacks proper recordkeeping for some of the conditions of the permit:

First, EPA must require Deseret to maintain records of all weekly Method 22 visible emissions (VE) evaluations of the unenclosed coal and limestone stockpiles required by Condition III.F.3. of the draft permit, in addition to maintaining records of all Method 9 opacity observations (per Condition III.I.8.c. of the draft permit).

Second, regarding the monitoring of coal quality and sulfur content, EPA must require that heat content and sulfur content be tested and recorded on a daily basis for all coal used (i.e., washed or "run-of-mine" coal used during "emergencies" or in whole or blended in part during other times). This is necessary for comparison to a percent SO_2 removal requirement, which commenters contend is necessary to ensure BACT is met over the wide variety of coal quality and sulfur content that will be used in the Bonanza WCFU.

Response #5.b.(2): Partially agree**.** Commenters appear to be suggesting that the permit fails to require the above-mentioned recordkeeping. EPA finds that commenters are correct on the first point (Method 22 VE evaluations), but not on the second point (heat content and sulfur content of coal).

Regarding the first point, EPA agrees with commenters that language should be added to permit condition III.I.8.c, to require that records be kept of the weekly Method 22 visible emission evaluations required by condition III.F.3. EPA acknowledges that without recordkeeping, the requirement to conduct weekly Method 22 evaluations would not be enforceable as a practical matter. EPA added this recordkeeping requirement to condition III.I.8.c. in the final permit.

Regarding the second point, EPA disagrees with commenters' apparent assertion that the draft permit fails to require daily records of heat content and sulfur content of coal, for all coal used. To the contrary, permit condition III.K.6 requires that records be kept of "all measurements of coal sulfur content and heat content required by this permit." Permit condition III.J.2 states that "The as-fired coal shall be tested each boiler operating day for sulfur content and heat content." The term "boiler operating day" is defined in permit condition III.D to mean "a 24-hour period between 12 midnight and the following midnight period during which any fuel is combusted at any time in the steam generating unit" (i.e., the CFB boiler). Therefore, contrary to commenters' apparent assertion, the permit does, in fact, require that heat content and sulfur content of the coal be tested and recorded on a daily basis for all coal used.

Comment #5.b.(2) has resulted in the addition of a requirement to permit condition III.I.8.c, that records be kept of the weekly Method 22 visible emission evaluations required by condition III.F.3.

6. EPA ADJUSTMENTS TO DESERET'S MODELING ANALYSIS

Comment #6:

One group of commenters argued that EPA must present its adjustments to Deseret's modeling analysis and provide opportunity to comment on the results. Commenters noted that in its draft Statement of Basis, EPA indicated that Deseret Power improperly determined the maximum short term SO_2 emission rates expected from the Bonanza WCFU that were used in the modeling analyses. (draft Statement of Basis at 135.) EPA re-calculated worst case short term SO_2 emission rates based on data provided by Deseret, and found "[w]hen the higher emissions values are used as input for dispersion models, it still appears to EPA that the NAAQS and PSD Class I and II increments would not be exceeded." However, commenters asserted, EPA did not provide the results of its dispersion modeling analysis with the higher worst case short term SO_2 emission limits to the public for review and comment.

Commenters further stated that EPA's revised 3-hour average SO_2 emission rate is almost six times greater than the 3-hour SO_2 emission rate modeled in Deseret's analyses, and the 24-hour average SO_2 emissions rate is close to 40% higher than what Deseret modeled. Commenters noted that Deseret accepted EPA's revised short term SO_2 emission rates as an amendment to its PSD permit application. Commenters argued that these increased emission rates should have been taken into account in estimating the significant impact area of the Bonanza WCFU (which, in turn, would be used to determine which sources should have been included in cumulative NAAQS and increment analyses), and also in determining whether preconstruction monitoring and/or cumulative PSD increment analyses should have been done.

Commenters further alleged that it is not clear whether EPA determined that, cumulatively with other sources in the region, the NAAQS and PSD Class I and II increments would not be exceeded with EPA's recalculated worst case SO_2 emission rates. Thus, commenters argued, EPA must present its revised modeling so the public can understand the true scope of short term average SO_2 impacts from the Bonanza WCFU and so that the public can ensure all CAA requirements will be complied with.

Response #6:

Partially agree. EPA does not agree that revised modeling must be done. No new modeling runs are necessary to account for EPA's adjustments to the modeling inputs for 3-hour and 24-hour SO_2. Emissions and concentrations are directly proportional in this type of model, so EPA simply scaled up the modeling results generated by Deseret's consultants to estimate local-scale impacts if worst case short term emission limits (i.e., the "Modeling Limits" in the permit) are ever reached.

As explained on pages 137-138 of the draft Statement of Basis ("EPA adjustments to permit applicant's modeling analysis"), EPA found that Deseret Power's assumed worst-case 3-hour average emission rate for SO_2, for modeling purposes, should have

been 872 lb/hr rather than 147 lb/hr, to account for a cold startup. EPA similarly found that Deseret Power's assumed worst-case 24-hour average emission rate for SO_2, for modeling purposes, should have been 202 lb/hr rather than 147 lb/hr. In evaluating the effect of these worst case emissions for modeling, EPA multiplied the WCFU's contribution to the modeling results shown on pages 130-134 of the draft Statement of Basis by a factor of [872/147 = 5.93] for 3 hour SO_2, and by a factor of [202/147 = 1.37] for 24 hour average SO_2. The corrected worst-case SO_2 emission rates for the WCFU (872 lb/hr on a 3-hour average and 202 lb/hr on a 24-hour average) are included as "Modeling Limits" in the draft WCFU permit. Modeling results still show compliance with the NAAQS and PSD increments for SO_2.

EPA does agree, however, that it should be made clear in the Statement of Basis how the corrected 3-hour and 24-hour WCFU emission rates are reflected in the modeling results. EPA has therefore scaled up the WCFU's contribution to the 3-hour and 24-hour SO_2 modeling results, in the following tables, on pages 161 through 164 of the Statement of Basis:

"NAAQS Compliance Demonstration for WCFU Project Sources"
"NAAQS Compliance Demonstration for Full Impact Area Sources"
"PSD Class II Increment Compliance for WCFU Sources (Near-field Analysis)"
"PSD Class II Increment Compliance for Full Impact Area Sources"

By "scale up," EPA means the modeling results in micrograms per cubic meter (ug/m^3) for the WCFU are multiplied by the factors mentioned above, which are 5.93 for 3-hour SO_2 and 1.37 for 24-hour SO_2, to reflect the worst-case short-term SO_2 emission scenario at the WCFU. Explanations have also been included in the Statement of Basis as to why these changes to the modeling results tables have been made.

EPA has also corrected the WCFU emission rates for 3-hour and 24-hour SO_2 in the Statement of Basis table titled, "ISC3 WCFU Stack Input Parameters Used for Modeling." EPA has also included the corrected WCFU emission rates in a PSD Class I increment compliance screening analysis, described in Response #9 below and added to the Statement of Basis.

EPA also agrees that its revision to Deseret's SO_2 emissions estimate for the WCFU should be taken into account in estimating the significant impact area of the WCFU. In Deseret's original analysis, the Class II significant impact area for SO_2 was a 16-kilometer radius from the proposed WCFU. Deseret added 50 kilometers to the impact area radius and looked for other increment affecting sources within 66 kilometers of the proposed WCFU. Other than Bonanza Unit 1, there were no other sources in the 66-kilometer radius impact area. The revised emission estimate for the WCFU would expand the impact area somewhat, but there are no additional large SO_2 sources near the edge of the 66-kilometer impact area. (This area is very remote.) At distances exceeding 66 kilometers, it would take a huge source to materially affect increment concentrations and there are none that large within at least 100 kilometers of the proposed WCFU. This additional explanation will be added to the final Statement-of-Basis.

In summary, Comment #6 has not resulted in any change to the permit; however, the changes described above have been made to the WCFU stack parameter table and to the modeling results tables in the Statement of Basis. Additional changes to the modeling results tables, to account for higher 3-hour and 24-hour SO_2 emission rates at Bonanza Unit 1 (140 g/sec and 106 g/sec, respectively), and to account for a 29% increase in SO_2 emissions at Unit 1 since 1991-93, are described in Responses #7 and #8.b. below, respectively.

7. CUMULATIVE NAAQS/INCREMENT ANALYSIS FOR SULFUR DIOXIDE

Comment #7:

One group of commenters asserted that Deseret's cumulative SO_2 NAAQS and Class II PSD increment analysis is flawed because the 2002 SO_2 emission rate modeled for Bonanza Unit 1 is much lower than the peak short term SO_2 emission rate for this unit in 2002. Specifically, commenters stated, Deseret assumed an SO_2 emission rate, purportedly based on 2002 actual emissions, of 56.30 grams per second (g/s). However, commenters stated, a review of the 2002 SO_2 emission data for Bonanza Unit 1 on EPA's Clean Air Markets Database indicates that the maximum three-hour average SO_2 emission rate was 126 g/s (1000 lb/hr) and the maximum 24-hour average SO_2 emission rate was 115.9 g/s (920 lb/hr).

Thus, commenters argued, Deseret Power underestimated Bonanza Unit 1's impacts on the short term average SO_2 NAAQS and increment. Commenters concluded that the NAAQS and increment analyses must be revised to model the highest 3-hour and 24-hour average emission rate of Bonanza Unit 1, as well as to model the EPA adjusted worst case 3-hour and 24-hour average SO_2 emission rates expected from the Bonanza WCFU. Commenters also asserted that the peak 3-hour and 24-hour SO_2 emission rates of Bonanza Unit 1 must be used in the cumulative Class I SO_2 increment modeling that is required. (See related comment #9 below.)

Response #7:

Partially agree. EPA does not agree that the modeling is flawed. Deseret Power conducted additional PSD increment analysis, in response to EPA comments that Bonanza Unit 1 SO_2 emission rates appeared to be too low for use in modeling PSD short term increments. (Ref: EPA letters to Deseret dated November 22 and 29, 2004, and Deseret's response letter to EPA dated March 23, 2005, all included in the Administrative Record for issuance of the WCFU permit.)

Specifically, Deseret Power re-modeled for PSD Class I SO_2 increment consumption at the nearest state-classified Class I area (Dinosaur National Monument in Colorado) using the maximum actual emission rates for Bonanza Unit 1 from the 2001-2002 period (140 g/sec for 3-hour increment and 106 g/sec for 24-hour increment). (Note: Dinosaur is not a mandatory Federal Class I area, but is classified as Class I by the State of Colorado.) The results are summarized in Deseret Power's March 23, 2005 letter to EPA. As explained in Response #9 below, EPA has conducted a separate screening analysis for impact on mandatory Federal Class I areas, which are somewhat more distant (Arches, Canyonlands and Capitol Reef National Parks).

The results of these analyses show that cumulative impacts of the proposed WCFU and existing Bonanza Unit 1 do not threaten the PSD Class I increment at Dinosaur and the impacts are expected to be even smaller at other more distant Class I areas. Deseret Power stated that no continuous emission monitoring system (CEMS) data

for startup/shutdown/ malfunction were excluded in determining the corrected maximum 3-hour and 24-hour emission rates from Bonanza Unit 1 used as inputs for the revised modeling. (Ref: E-mail dated November 13, 2006, from Ed Thatcher of Deseret Power to Mike Owens of EPA Region 8.)

EPA does agree, however, that the corrected 3-hour and 24-hour peak emission rates for Bonanza Unit 1 and the revised modeling analysis, as reported by Deseret Power to EPA on March 23, 2005, as well as revised modeling results to account for worst-case 3-hour and 24-hour SO_2 emission rates at the WCFU, should be reflected in the modeling results tables in the Statement of Basis. Revisions to those tables to account for the worst-case 3-hour and 24-hour emission rates at the WCFU are described in Response #6 above. Additional revisions, to account for the higher 3-hour and 24-hour emission rates at Unit 1, have been made to the following modeling results tables, on pages 162 and 164 of the Statement of Basis:

"NAAQS Compliance Demonstration for Full Impact Area Sources"
"PSD Class II Increment Compliance for Full Impact Area Sources"

In addition, the 3-hour and 24-hour SO_2 emission rates for Bonanza Unit 1 have been revised in the table titled, "Bonanza Unit 1 Stack Parameters Used for Modeling." Further, a reference to Deseret Power's March 23, 2005 Class I increment analysis for Dinosaur National Monument has been added to the Statement of Basis.

In summary, Comment #7 has not resulted in any change to the permit; however, the changes described above have been made to the Statement of Basis.

Additional changes to the modeling results tables in the Statement of Basis, to account for worst-case 3-hour and 24-hour SO_2 emission rates at the WCFU, and to account for a 29% increase in SO_2 emissions at Unit 1 since 1991-93, are described in Responses #6 and #8.b, respectively.

8. PRE-APPLICATION AMBIENT MONITORING FOR SULFUR DIOXIDE

Comment #8:

Comment #8.a: One group of commenters stated that it appears that Deseret should not have been exempted from one year of pre-construction ambient monitoring for SO_2. Commenters asserted that although the PSD permit application shows that the SO_2 impacts from the Bonanza WCFU would be less than the monitoring significance levels, this modeling was based on Deseret's flawed approach of estimating worst case short term emission rates.

Commenters noted that Deseret Power's worst case SO_2 emission rate modeled was 146.99 lb/hr. (draft Statement of Basis at 135.) EPA's recalculated worst case 24-hour average SO_2 emission rate was 201.9 lb/hr. Multiplying Deseret Power's original 24-hour maximum near field modeling result of 10.8 ug/m3 for SO_2 (as provided in the Statement of Basis at 128) by the ratio of the revised worst case short term emission rate to the originally modeled worst case SO_2 emission rate, results in a maximum 24-hour average SO_2 ambient concentration of 14.8 ug/m3. This exceeds the 24-hour SO_2 monitoring significance level of 13 ug/m3. Thus, commenters argue, it appears that Deseret should have conducted one year of pre-application ambient monitoring for SO_2. Consequently, commenters asserted, EPA must delay issuing the permit until this data is collected.

Response #8.a: Disagree. There is no reason to require one year of pre-construction ambient SO_2 monitoring, if representative ambient SO_2 concentration data are already available. Ambient SO_2 air quality monitoring data are available from the plant site for the period 1991-1993. These data are considered by EPA to be representative, since the data were collected on site, and there were no other major sources of SO_2 in the area then, and none have been added since that time. The data were collected in accordance with EPA's PSD monitoring guidelines

EPA's ambient monitoring guidelines for PSD list the following three criteria that must be met, for pre-construction monitoring data to be considered representative of pre-construction ambient air quality for PSD purposes

- Section 2.4.1 - Monitor location;

- Section 2.4.2 - Data Quality [the guideline says the data should be of similar quality as would be obtained if the applicant monitored according to the PSD requirements]; and

- Section 2.4.3 - Currentness of the data [the guideline says the data should be current, which generally means, for the pre-construction phase, the data must have been collected in the 3-year period preceding the permit application, provided the data are still representative of current conditions].

(Ref: Pages 6 through 9 of EPA's Ambient Monitoring Guidelines for Prevention of Significant Deterioration (PSD), EPA-450/4-87- 007 (May 1987), available through website at: http://www.epa.gov/ttnamti1/files/ambient/criteria/reldocs/4-87-007.pdf)

The Environmental Appeals Board (EAB) has also recognized that the permitting authority may allow for representative data gathered from other time periods. (*See In re Encogen Cogeneration Facility,* 8 EAD 244, 255-257 (1999); *In re Hawaii Electric Light Co*., 8 EAD 66, 97 (1998); *In re Kawaihae Cogeneration Project*, 7 EAD 107, 128 (1997).)

The first criterion above was met because the 1991-93 ambient data were collected on-site (Ref: November 1, 2004 PSD permit application, volume titled "Dispersion Modeling, Deposition and Visibility Analyses," page 3-3, included in the Administrative Record for issuance of the WCFU permit.)

The second criterion above was met because all of the data were collected in compliance with the quality assurance provisions in EPA's above-mentioned ambient monitoring guidelines. (Ref: E-mail from Ed Thatcher, Deseret Power, to Mike Owens, EPA Region 8, dated November 9, 2006, included in the Administrative Record for issuance of the WCFU permit.)

The third criterion above was met because, although the data were not collected within a three-year period preceding the PSD permit application, the data are considered representative because there have been no substantive emission changes in the vicinity of the proposed project since the 1991-93 period. Emission changes at Bonanza Unit 1 since 1991-93 have been accounted for as described in the remainder of this response.

In summary, comment #8.a has not resulted in any change in the permit; however, an expanded explanation has been included in the Statement of Basis, on why Deseret Power is exempt from pre-construction ambient monitoring for SO_2.

Comment #8.b: As mentioned in comment #6 above, EPA re-calculated maximum short term SO_2 emission rates but, according to commenters, did not present the results of its revised modeling analyses. Commenters noted that, considering the emissions rate is all that would be changed in the revised modeling, one can simply adjust the results proportionately based on the EPA's revised emission rate as compared to Deseret's modeled SO_2 emission rate.

Response #8.b: Agree. EPA inadvertently failed to revise the modeling results tables in the draft Statement of Basis to account for EPA's re-calculation of maximum (worst case) short term SO_2 emission rates at the WCFU. The tables have been revised in the Statement of Basis.

When re-evaluating the modeling in response to comments, EPA found, however, that Deseret used ambient air quality data from the Bonanza site for 1991-1993 for determining ambient background concentrations, but failed to consider the effect of

potential growth in Bonanza Unit 1 emissions since that time. (Ref: November 13, 2006 e-mail from Deseret Power to EPA, included in the Administrative Record for issuance of the WCFU permit.)

To consider the effect of Unit 1 emission changes, EPA's AirData base was reviewed by Deseret Power and emissions, in tons per year for Uintah County, for SO_2, NO_x, CO, PM_{10}, and VOC were obtained for 1990 and 2001. These data are presented below. Based on Table 1, emissions of CO, NO_x, and VOC have decreased; SO_2 and PM_{10} emissions have increased by 27.3 and 7.6%, respectively.

Table 1. Criteria Pollutant Emissions for Uintah County for 1990 and 2001

Pollutant	1990 (tons)	2001 (tons)	Change (tons)
CO	33,530	28,597	- 4,933
NO_x	10,110	8,991	- 1,119
SO_2	1,029	1,416	+ 389
VOC	5,818	2,952	- 2,866
PM_{10}	8,958	9,690	+ 732

Ref: November 1, 2006 e-mail from Deseret Power to EPA.

As noted above, the background values used did not account for the relatively small increase in Bonanza Unit I SO_2 emissions since 1993. The background concentrations used to determine National Ambient Air Quality Standards (NAAQS) compliance in Deseret Power's PSD permit application are listed in Table 2.

Table 2. Background Concentration Values

Pollutant	Averaging Period	Concentration ($\mu g/m^3$)
SO_2	3-hour	20
SO_2	24-hour	10
SO_2	Annual	5
NO_2	Annual	5
PM_{10}	24-hour	10
PM_{10}	Annual	28
CO	1-hour	1 ppm
CO	8-hour	1 ppm

SO_2 emissions from Bonanza Unit 1 from 1991 through 1993, and 1994 through 2005, were reviewed to determine the percent increase over the period. The average SO_2 emissions from Unit 1, in tons, from 1991 – 1993 was 774; the average SO_2 emissions from Unit 1 from 1994 – 2005 was 1000.7. This represents a 29% emission increase from 1991 to 2005. (Ref: November 1, 2006 e-mail from Deseret Power to EPA.)

If we assume a 29% increase in the measured ambient SO_2 data collected at the Bonanza Power Plant from 1991 – 1993 (to account for the 29% emissions increase) and scale up the background concentrations used for the NAAQS compliance demonstration

by 29%, the resultant three-hour, 24-hour, and annual SO_2 background concentrations would be 25.8 $\mu g/m^3$, 12.9 $\mu g/m^3$, and 6.5 $\mu g/m^3$, respectively. Adding to this the highest modeled maximum SO_2 concentrations, from both the proposed WCFU and Bonanza Unit 1, based on 1993 meteorological data, the predicted ambient concentrations for the 3-hour, 24-hour, and annual averaging periods are 27.3%, 7.6% and 9.6% of the NAAQS, respectively.

In summary, comment #8 has not resulted in any change to the permit; however, the following modeling results tables, on pages 161 and 163 of the Statement of Basis, have been changed, to reflect the revised results for background concentrations, accounting for a 29% increase in Bonanza Unit 1 SO_2 emissions since 1991-93:

"NAAQS Compliance Demonstration for WCFU Project Sources"
"NAAQS Compliance Demonstration for Full Impact Area Sources"

Additional changes to the modeling results tables, to account for account for worst-case 3-hour and 24-hour SO_2 emission rates at the WCFU, and to account for higher 3-hour and 24-hour emission rates at Bonanza Unit 1, are described in Responses #6 and #7 above, respectively.

9. CUMULATIVE PSD INCREMENT ANALYSIS FOR CLASS I AREAS (AND FOR COLORADO CLASS I AREAS)

Comment #9:

One group of commenters asserted that Deseret Power failed to provide any cumulative PSD increment analysis for any affected Class I area in its permit application for the Bonanza WCFU, and that neither Deseret Power's PSD permit application, nor EPA's draft Statement of Basis, explains why cumulative increment analyses were not completed for Class I areas. Commenters asserted that PSD permitting regulations indicate that no PSD permit can be issued unless the source demonstrates that it will not cause *or contribute to* a violation of any PSD increment. 40 CFR 52.21(k)(2). Commenters argued that since Deseret has not made that demonstration, EPA cannot issue the permit.

Commenters postulated that one possible reason Deseret did not perform any cumulative Class I PSD increment analyses might be because Deseret considers the impacts of the Bonanza WCFU to be less than significance levels. (Commenters cited the Class I area impact tables on pages 4-21 through 4-28 of the dispersion modeling portion of Deseret's November 2004 PSD permit application, which identify the Bonanza WCFU's impact at each Class I area in terms of "Percent of EPA Class I Significance Levels.") However, commenters stated, there are no Class I area significance levels authorized in any federal regulation. While EPA proposed use of such Class I significant impact levels in July of 1996, EPA never finalized promulgation of those significant impact levels. Thus, commenters concluded, until EPA adopts significant impact levels for Class I increments, *any* impact must warrant a cumulative analysis.

Moreover, commenters argued, even if use of proposed but never finalized significant impact levels were appropriate to exempt the Bonanza WCFU from a cumulative increment analysis in affected Class I areas, cumulative SO_2 increment analyses would be required because the SO_2 impacts of the Bonanza WCFU would be greater than the proposed Class I significant impact levels for SO_2 in several Class I areas as follows:

Commenters argued that Deseret Power's modeling showed that its impact on the Colorado portion of Dinosaur National Monument would be greater than the SO_2 3-hour and 24-hour average proposed significant impact levels and greater than the 24-hour average Class I proposed significant impact level in Colorado National Monument. (Commenters cited pages 4-23, 4-24 and 4-30 of the dispersion modeling portion of the PSD permit application.) Colorado's regulations mandate that Dinosaur National Monument and Colorado National Monument, although Class II areas, will be subject to the more stringent Class I increments for SO_2. (Colorado Regulation 3, Part B, Section VIII.B.1.b.). Thus, commenters concluded, Deseret Power should have been required to perform a cumulative increment analysis for Dinosaur National Monument and Colorado National Monument.

Further, commenters asserted, Deseret Power's analysis of the Bonanza WCFU's impacts on short term average SO_2 concentrations in Class I areas was flawed because, as noted by EPA, Deseret underestimated worst case short term SO_2 emission rates from the Bonanza WCFU. (draft Statement of Basis at 135.) Commenters noted that, as discussed in the above comment regarding the monitoring significance threshold, the predicted SO_2 impacts on the Class I areas can be proportionately adjusted based on the EPA's revised SO_2 emission rates as compared to Deseret's modeled SO_2 emission rate.

Commenters further noted that EPA re-calculated Bonanza's WCFU worst case 3-hour average SO_2 emission rate to be 872 lb/hr, which is almost six times as high as the 146.99 lb/hr SO_2 emission rate modeled by Deseret. Commenters concluded that proportionately adjusting the 3- hour average SO_2 impacts of the Bonanza WCFU using EPA's revised worst case 3-hour average emission rate shows that the Bonanza WCFU would have an impact greater than the 3-hour average proposed significant impact level for SO_2 for most of the Class I areas in the region.

Commenters created and submitted a table to EPA, showing the revised Class I area 3-hour average SO_2 impacts based on EPA's revised worst case emission rates for those Class I areas where the Bonanza WCFU would exceed the proposed Class I significant impact levels. Based on that table, commenters argued that even if it were appropriate to exempt a facility from a cumulative Class I increment analysis based on its impacts being less than the proposed significant impact levels, the Bonanza WCFU would not be exempt from performing cumulative analyses of impacts on the 3-hour average SO_2 increment at Arches National Park, Canyonlands National Park, Capitol Reef National Park, Colorado National Monument, the Colorado portion of Dinosaur National Monument, the Flat Tops Wilderness area, and the Mt. Zirkel Wilderness Area.

Thus, commenters argued, Deseret Power must be required to conduct cumulative Class I increment analyses for the nearby Class I areas. EPA must not issue a PSD permit for the Bonanza WCFU without ensuring that the facility will not cause *or contribute* to a violation of any PSD increment.

Further, commenters argued, the cumulative Class I increment analyses must include the PSD increment consuming emissions of all other sources that could be affecting air quality in those Class I areas. This would include all large sources of air pollution within 200 kilometers of each Class I area, such as nearby coal-fired power plants (e.g., the Bonanza Unit 1, Hunter, Huntington, and Intermountain power plants in Utah, and the Craig, Hayden and Nucla power plants in Colorado).

In addition, commenters argued, Deseret Power must be required to model those facilities that have submitted complete PSD permit applications, and/or that have received air quality permits, but that have not yet constructed. This would include NEVCO's Sevier Power plant, Unit 3 of the Intermountain Power Plant, and Unit 4 of the Hunter Power plant, all to be located in Utah. Commenters further argued that Deseret Power must also include the existing and proposed oil and gas development occurring near the Class I areas that Bonanza will affect. Commenters concluded that until

complete and thorough Class I increment modeling analyses are completed, EPA cannot issue the permit because EPA will not know whether the facility will cause or contribute to a Class I increment violation.

Although commenters did not say their comment pertains only to SO_2, EPA interprets this to be the case, since the only pollutant mentioned in the comment was SO_2. Therefore, EPA's response below pertains only to SO_2.

Response #9:

Disagree. Given the modeling results from Deseret Power's PSD permit application of November 1, 2004 that indicate very small or no impacts on any Class I areas, EPA concluded that a cumulative PSD increment analysis for nearby Class I areas would not be necessary or required for the WCFU project. Further, an e-mail from the National Park Service to EPA on June 16, 2005, regarding Deseret's November 1, 2004 PSD permit application for the WCFU project, stated that "the modeling analyses for Class I and II PSD increments and impacts to Air Quality Related Values has been performed correctly and all issues regarding impacts to the NPS Class I and Class II units have been addressed."

The commenters' suggested use of worst case short term SO_2 emission rates ("modeling limits" in the permit), in determining impacts to Air Quality Related Values (AQRVs) or PSD increments, greater than 50 kilometers from the source, is not an approach EPA would require, since the worst case emission rate is not intended to represent a routine or frequent operating condition. The low frequency of occurrence of the WCFU facility operating at the worst case emission rate (reflecting a cold startup), combining with simultaneous meteorology to transport emissions a considerable distance to the nearest Class I area, makes the likelihood of impacts on the nearest Class I areas extremely unlikely.

Nevertheless, to be responsive to commenters, EPA conducted a screening analysis for cumulative impact on nearby mandatory Federal Class I areas (except Dinosaur National Monument, which is not mandatory Federal Class I and is addressed separately in response #7 above), using worst-case emission rates cited by public commenters. This was done by scaling Deseret Power's PSD Class I modeling analysis to the level of the worst case short term emission rates (as noted in response #6 above), even though this is not an approach EPA would require (as explained above).

Specifically, the 3-hour PSD increment concentrations were multiplied by 5.93 and the 24-hour PSD increment concentrations by 1.37. The adjusted modeled concentrations from the WCFU were then added to the cumulative PSD increment concentrations calculated by Intermountain Power Agency (IPA) for their Unit 3 PSD permit application in May of 2003. The modeling analysis in the IPA Unit 3 application has been reviewed and approved by the Utah Division of Air Quality. The State of Utah has a SIP-approved PSD permitting program and implements the PSD program in Utah.

That analysis showed that the PSD Class1 increment is not threatened in these areas. (See Table 3 below.)

Table 3
Cumulative PSD Increments Consumption for Selected Utah Class 1 Areas Based on Combined Modeled Impacts from Deseret WCFU and Reported PSD Increment Modeling Results from Intermountain Power Project, Unit 3

Location	3-hour SO2	24-Hour SO2	Annual SO2	24-Hour PM10	Annual PM10
Arches NP	3.8	0.8	0.1	0.2	.02
Canyonlands NP	9.6	2.2	0.1	0.2	.02
Capitol Reef NP	4.0	0.9	0.1	0.2	0.2
PSD Class 1 Increment	25	5	2	8	4

Ref: Addendum to Final Permit Application ("Notice-Of-Intent"), Intermountain Power Project, Proposed Unit 3, June 16, 2003.

In summary, Comment #9 has not resulted in any change to the permit; however, the Class I increment screening analysis described above has been added to the Statement of Basis. A separate analysis by Deseret Power, for impact on PSD Class I SO_2 increment at Dinosaur National Monument (a Colorado Class I area, not mandatory Federal Class I) is described in Response #7 above.

10. PSD INCREMENT CONCERNS AT CAPITOL REEF NATIONAL PARK

Comment #10:

One group of commenters asserted that EPA must not issue the PSD permit for the Bonanza WCFU in light of the PSD SO_2 increment violations that commenters asserted are occurring at Capitol Reef National Park. Commenters cited a March 25, 2004 letter from the National Park Service to the Utah Division of Air Quality, submitted during the permit review and proceedings for the proposed Unit 3 of the Intermountain Power Plant located in Delta, Utah. Commenters asserted that the letter expressed the concern that there are increment violations in Capitol Reef National Park.

On the basis of the NPS letter, commenters asserted that the Bonanza WCFU will contribute to existing SO_2 increment violations at Capitol Reef National Park, at a level greater than the proposed Class I significance level for 3-hour SO_2, and that EPA should not issue the PSD permit for the Bonanza WCFU until the increment violations are addressed.

Response #10:

Disagree. Neither EPA nor the State of Utah, the two agencies with authority to do so under the Federal Clean Air Act, has determined there is an increment violation at Capitol Reef National Park. The State of Utah has an EPA-approved PSD permitting program. In issuing the PSD permit for construction of Unit 3 at Intermountain Power Plant, the State of Utah concluded that the PSD increments are not threatened. In addition, in response to comment #9 above, EPA added the impact of Deseret Power's WCFU to the Intermountain Power Unit 3 PSD cumulative increment analysis results for Capitol Reef National Park and found no PSD increment violations.

Moreover, any concern for the potential of increment violations at Capitol Reef National Park should be further minimized by the fact that Pacificorp recently applied for permits from the State of Utah for installation of additional controls at the Hunter and Huntington plants in Delta, Utah, both of which are closer to Capitol Reef National Park than Deseret Power's proposed WCFU at Bonanza, Utah. These additional controls are projected by Pacificorp and the State of Utah to yield a total of 21,560 tons per year of SO_2 emission reductions.

The permit ("Approval Order" in Utah's terminology) for additional emission controls at the Huntington plant was issued by the State of Utah on April 6, 2005, and cites expected emission reductions of 17,479 tons per year of SO_2, 2,781 tons per year of NO_x, and 1.432 tons per year of PM_{10}. (Ref: Approval Order dated April 6, 2005, included in the Administrative Record for issuance of the WCFU permit.)

The permit for additional emission controls at the Hunter plant was proposed by the State of Utah on March 2, 2007 ("Intent-To-Approve" in Utah's terminology), but is not yet finalized, as of early August 2007. The ITA cites expected emission reductions of

4,081 tons per year of SO_2, 8,754 tons per year of NO_x, and 1,441 tons per year of PM_{10}. (Ref: Intent-To-Approve dated March 2, 2007, included in the Administrative Record for issuance of the WCFU permit.)

These additional controls at the Huntington and Hunter plants are expected by EPA to further reduce the level of PSD increment consumption from those shown in Table 3 above. The potential controlled SO_2 emissions from the WCFU project are only 348 tons per year based on the proposed upper-tier emission allowable of 0.055 lb/MMBtu, or only 253 tons per year based on the proposed lower-tier emission allowable of 0.040 lb/MMBtu.

In summary Comment #10 has not resulted in any change to the permit or Statement of Basis.

11. VISIBILITY MODELING

Comment #11:

One group of commenters asserted that Deseret Power's visibility modeling analysis of the Bonanza WCFU is flawed because Deseret Power failed to model maximum 24-hour average emissions of SO_2 and because Deseret Power failed to properly document why it was necessary or appropriate to rollback the relative humidity in the regional haze modeling to 95%. Consequently, commenters argued, the modeling likely underestimated the impacts of the Bonanza WCFU on visibility in nearby Class I areas.

Comment #11.a: Commenters noted that, as discussed above, EPA adjusted the worst case 24-hour SO_2 emission rate based on data from Deseret Power because Deseret Power's estimate of worst case SO_2 emissions did not properly include emissions from start-ups. (draft Statement of Basis at 135.) With EPA's adjustment, the worst case 24-hour average SO_2 emission rate is 37% higher than the emission rate that was modeled in Deseret's visibility analysis. Thus, commenters argued, Deseret's visibility analysis underestimated visibility impacts in all affected Class I areas. Commenters asserted that Deseret must be required to re-model visibility impacts using the adjusted worst case 24-hour average SO_2 emission rate of 201.9 lb/hr and such modeling must be provided to the Federal Land Managers for review.

Response #11.a: Disagree. As noted in response #9 above, the National Park Service stated on June 16, 2005, in regard to the PSD permit application for the WCFU, that "the modeling analyses for Class I and II PSD increments and impacts to Air Quality Related Values has been performed correctly and all issues regarding impacts to the NPS Class I and Class II units have been addressed."

Also, it appears that commenters may have used a scaling technique to determine that the Bonanza WCFU would have an adverse visibility impact at some nearby Class 1 areas. Scaling the Calpuff model is not appropriate when reviewing visibility and deposition results. Calpuff converts a portion of the SO_2 emissions to sulfate particulate and a portion of the NO_X emissions to nitrate particulate as the plume is transported. Thus, visibility and deposition values are not linear to the emission rate. Comment #11.a has not resulted in any change to the permit or Statement of Basis.

Comment #11.b: Commenters also argued that Deseret estimated visibility impacts using both a maximum relative humidity of 98%, consistent with the Federal Land Managers' guidance, and rolling back relative humidity to 95%. (Commenters cited page 4-49 of the modeling portion of Deseret Power's PSD permit application.) However, commenters argued, the National Park Service has indicated that any analysis rolling back relative humidity to 95% would have to be "well documented as to why it is appropriate to. . .roll back relative humidity to 95% . . ." (Commenters cited an August 6, 2004 e-mail from John Notar, National Park Service, to Ed Thatcher of Deseret Power and Kevin Golden of EPA Region 8.) Commenters asserted that Deseret Power did not

provide any such documentation, therefore the results of its visibility analysis capping relative humidity at 95% cannot be relied upon.

Response #11.b: Disagree. With regard to the maximum relative humidity assumption, the Federal Land Managers (FLMs) have reviewed the draft permit package for this project and had no comment. It is EPA's understanding that in recent permit applications, the FLMs have broadly accepted the use of the 95 percent humidity threshold. The Draft Calpuff Reviewer's Guide, dated September 2005, prepared for the USDA Forest Service and the National Park Service (and included in the Administrative Record for issuance of the WCFU permit), indicates the general consensus among FLMs is that use of a maximum relative humidity value of 95% is appropriate for visibility modeling. (Ref: Page 6-1 of the Guide: "The CALPOST default value for RHMAX is 98, but the general consensus among FLMs is that RHMAX = 95 is appropriate.") Comment #11.b has not resulted in any change to the permit or Statement of Basis.

Comment #11.c: Commenters further argued that, based on the visibility modeling done by Deseret that is consistent with current guidance of the Federal Land Managers (i.e., capping relative humidity at 98%), the Bonanza WCFU will have an adverse impact on visibility (greater than a 5% change) at Arches and Capitol Reef National Parks. (Commenters cited page 4-51 of the modeling portion of Deseret's PSD permit application.) This analysis, commenters stated, must be redone with the EPA's worst case 24-hour average SO_2 emission rate and the results transmitted to the appropriate Federal Land Managers.

Response #11.c: Disagree. EPA does not agree that 98% relative humidity should have been used to model visibility impacts. See response #11.b above. Comment #11.c has not resulted in any change to the permit or Statement of Basis.

Comment #11.d: Commenters also asserted that, because the impacts on visibility will be greater using the higher SO_2 worst case 24-hour average emission rate, it appears the Bonanza WCFU will have an adverse visibility impact at some nearby Class I areas. Commenters concluded that EPA Region 8 must ensure that, in issuing a permit for the Bonanza WCFU, its actions are consistent with the intent of the PSD requirements of the Clean Air Act – specifically, whether its actions will preserve, protect, and enhance the air quality in nearby national parks and wilderness areas (i.e., pursuant to §160(1) of the Clean Air Act), and whether its actions will ensure that emissions from the Bonanza WCFU will not interfere with portions of State Implementation Plans aimed at preventing significant deterioration of air quality including preventing future visibility impairment (i.e., pursuant to §160(4) and 169(a)(1) of the Clean Air Act).

Response #11.d: Disagree. As explained in responses #11.a, 11.b and 11.c above, EPA's analysis and determinations have followed applicable rules and guidance, and the FLMs have no issue with the analysis and determinations. Comment #11.d has not resulted in any change to the permit or Statement of Basis.

In summary, Comments #11.a, b c and d have not resulted in any change to the permit or Statement of Basis.

12. MERCURY SIGNIFICANCE THRESHOLD

Comment #12:

One commenter asserted that an estimate of potential-to-emit for mercury should have been presented in the draft permit, and that a determination of whether the expected mercury emissions from the proposed WCFU will exceed PSD significance threshold should have been presented in the Statement of Basis. The commenter asserted the significance threshold of 0.1 tons per year (tpy) appears in the Federal PSD rules at 40 CFR 52.21(b)(23)(i). The commenter also remarked that coal-fired electric generating units are known to represent one of the largest sectors for mercury emissions, and that the issue of mercury significance should be explicitly discussed.

Response #12:

Disagree, for two reasons. First, the commenter's assertion is incorrect. The current Federal PSD rules at 40 CFR 52.21(b)(23)(i) have no significance threshold for mercury. On December 31, 2002, EPA revised the PSD rules to remove the significance threshold for mercury from §52.21(b)(23)(i). (Ref: 67 Fed. Reg. 80186, 80239-80240 (December 31, 2002)) Second, as discussed in detail in the final preamble to the December 31, 2002 rulemaking, EPA took final action to promulgate the proposed revisions and indicated that the "1990 Amendments to the CAA at section 112(b)(6) exempted HAP listed under section 112(b)(1) from the PSD requirements in part C." *Id.* EPA went on to indicate that the HAPs listed in section 112(b)(1) of the Clean Air Act, including mercury, are excluded from the PSD provisions of part C.

Comment #12 has not resulted in any change to the permit or Statement of Basis.

13. COMPLIANCE WITH NEW SOURCE PERFORMANCE STANDARD FOR MERCURY

Comment #13:

One commenter asserted that section III.I of the proposed permit for the WCFU should contain a provision to demonstrate compliance with 40 CFR 60.45Da, Standard for mercury. The commenter also asserted that the Statement of Basis should contain a discussion of the rank(s) of coal the CFB will burn and which standard within §60.45Da is applicable.

Response #13:

Disagree. Federal PSD rules at 40 CFR 52.21 do not require PSD permits to include emission standards from 40 CFR part 60, for mercury or any other pollutant. Also, the commenter's assertion that the draft Statement of Basis fails to cite the applicable mercury standard and coal category is incorrect. The applicable mercury standard from 40 CFR 60.45Da(a)(4) was shown in a table on page 115 of the draft Statement of Basis. The table may be found on page 143 of the final Statement of Basis, and is titled "Emission Limits in Amended NSPS Subpart Da as of July 1, 2007, Applicable to Units Commencing Construction after February 28, 2005."

Comment #13 has not resulted in any change to the permit or Statement of Basis.

14. COMPLIANCE DEMONSTRATIONS FOR OPACITY AT MATERIALS HANDLING VENT FILTERS AND BAGHOUSES

Comment #14:

One commenter noted that draft permit condition III.I.6, "Compliance demonstrations for opacity" at the materials handling vent filters and baghouses, states that "If no visible emissions are observed in three consecutive monthly observations, frequency of observations at that baghouse or vent filter may be reduced to quarterly." The commenter asked EPA, "If the opacity observations have been reduced to quarterly, and an observation finds visible emissions, do the observations remain at quarterly, or return to monthly?"

Response #14:

Agree. The commenter appears to be suggesting that the permit should clarify whether the required frequency of visible emission observations reverts back to monthly from quarterly, in the event that visible emissions are observed. EPA agrees that the permit should be clear on this. Since conditions at a materials handling vent filter or bagbouse can vary over time, it is EPA's intent that frequency of observations revert back to the original frequency (i.e., monthly), if visible emissions are observed. Relaxation of observation frequency should serve as an ongoing incentive (not just a one-time-only incentive) to maintain good particulate control.

Comment #14 has resulted in the above-mentioned clarification in the final permit and Statement of Basis.

15. REFERENCES IN STATEMENT-OF-BASIS TO INTERMOUNTAIN POWER UNIT 3 PROJECT

Comment #15:

One commenter noted that the draft Statement of Basis, at page 51, references the Utah Division of Air Quality's "Modified Source Plan Review" for the Intermountain Power Unit 3 (IPP3) project. The commenter stated that the IPP3 permit action is under challenge by the Sierra Club and Grand Canyon Trust, therefore "possibly it is unwise to add into the record an analysis that may be overturned."

Response #15:

Disagree, for two reasons. First, since the IPP3 permit has not been overturned, EPA sees no reason to delete references to Utah's "Modified Source Plan Review" for that permit action. Second, EPA does not believe its reference to Utah's "Modified Source Plan Review" is connected to the "challenge" cited by the commenter. EPA's draft Statement of Basis referenced the "Modified Source Plan Review" only in regard to a statement by the Utah Division of Air Quality (DAQ) that no more than 80 percent removal efficiency might be expected for sulfuric acid, for a wet electrostatic precipitator (ESP) at a pulverized coal-fired boiler, under optimum conditions. EPA's draft Statement of Basis also cited other sources of information on sulfuric acid removal efficiency where a wet ESP is used, and concluded that an estimate of 86% should be used for EPA's analysis, not 80%. EPA presented the Utah DAQ's estimate only to show that estimates vary.

While EPA is not certain what the commenter means by a "challenge" to the IPP3 permit, EPA presumes that the "challenge" arose from a comment letter dated May 20, 2004, on the draft PSD permit for the Intermountain Power Unit 3 project. The letter was submitted to the Utah DAQ by the Sierra Club, Grand Canyon Trust, and several other environmental organizations. Page 31 of the comment letter notes the Utah DAQ's estimate of 80% removal efficiency for sulfuric acid. The comment letter does not question that estimate. Rather, the comment letter states that Utah DAQ and Intermountain Power did not properly follow EPA's cost effectiveness formulas, and thus these calculations cannot be relied on to eliminate a wet ESP from review. (A copy of the May 20, 2004 comment letter is included in the Administrative Record for issuance of the WCFU permit.)

In summary, EPA does not consider the challenge to the Intermountain Power Unit 3 permit to be a reason to remove from its own Statement of Basis the reference to the Utah DAQ's "Modified Source Plan Review." Comment #15 has not resulted in any change to the permit or Statement of Basis.

16. TYPOGRAPHICAL ERROR IN PERMIT

Comment #16: One commenter stated that permit condition III.H.1.a has a typographical error. It should cross-reference permit condition III.E.3 rather than III.E.4.

Response #16: Agree. The correction will be made in the final permit.

C. CHANGES TO THE PERMIT AND STATEMENT OF BASIS IN RESPONSE TO PUBLIC COMMENT

Permit:

From response #4.b.(1): Revised the "cutpoint" in permit conditions III.D.1.b.(ii)*(a)* and *(b)* from 1.9 lb/MMBtu to 2.2 lb/MMBtu.

From response #5.a.(1):

- Changed the averaging time of the emission limits at the CFB boiler stack in permit condition III.D.1.a, for total particulate matter and for filterable particulate matter, from 30-day rolling average to 24-hour block average.

- Changed permit condition III.I.4.d, to delete "total particulate matter including condensibles" and "total filterable particulate matter" from the first sentence on 30-day rolling averages.

- Added a paragraph to permit condition III.I.4.d, to say that emissions of "total particulate matter" and "total filterable particulate matter" shall be calculated on a 24-hour block average basis (midnight to midnight).

- Changed permit condition III.J.1.f, to add the phrase "and 24-hour block average emission rates" to the second sentence.

- Changed permit condition III.L.2.a, to add "24-hour block averages" to the title line. Made corresponding change in the Table of Contents. Changed the language in the condition to say that for SO_2, NO_x and CO, reports of 30-day rolling average emissions are required, but that for total particulate and total filterable particulate matter, reports of 24-hour block average emissions are required.

- Changed permit condition III.L.2.a.(iii), to say that the language about 30 successive boiler operating days pertains only to SO_2, NO_x and CO, not to total particulate matter or total filterable particulate matter.

- Added a new permit condition III.L.2.a.(iv), to say that for total particulate matter and for total filterable particulate matter, the average emission rate in lb/MMBtu for each boiler operating day shall be reported. ("Boiler operating day" is defined at the beginning of permit condition III.D and means a period from midnight to midnight, which corresponds to a 24-hour block average.)

- Moved the language about reporting for periods of non-compliance, startups, shutdowns and malfunctions from permit condition III.L.2.a.(iii) to a new permit condition III.L.2.a.(v).

- Re-numbered the remaining provisions of permit condition III.L.2.a., from III.L.2.a.(iv) through.(viii), to III.L.2.a.(vi) through (x).

From response #5.a.(1): Changed title of permit section III.D from "PSD BACT Emission Limits" to "PSD BACT and Other Emission Limits." Added a footnote to permit condition III.D.3 regarding opacity limit.

From response #5.b.(2): Added requirement in permit condition III.I.8.c to keep records of the weekly Method 22 observations required by condition III.F.3.

From response #14: Added a clarifying statement to permit condition III.I.6 that if any visible emissions are observed in a quarterly observation at a baghouse or vent filter, the frequency of observation at that baghouse or vent filter shall return to monthly.

From response #16: Corrected typographical error in permit condition III.H.1.a, to cross-reference permit condition III.E.3 rather than III.E.4.

Other minor administrative changes made to the draft permit:

In permit condition III.H.3, at the end of the condition, added the phrase "and any changes required by EPA."

In permit condition III.I.2.a, corrected the references to NSPS Subpart Da, regarding exemptions from emission standards. The meaning of the permit condition was not changed.

Added a permit condition III.L.9, to require written notification to EPA of the date that construction commences on the WCFU project, within 15 days after commencement. Re-numbered existing condition III.L.9 to III.L.10.

Updated the EPA street address in permit condition III.L.9, now condition III.L.10.

Reworded permit condition IV.B, "Permit Effective Date," to say "This PSD Permit becomes effective 30 days after the service of notice of the final permit decision, unless review of the permit decision is requested pursuant to 40 CFR 124.19."

Statement of Basis:

From response #3:

Added a new SOB subsection VI.E ("Supercritical Boiler Technology for BACT"), to explain why supercritical CFB boiler technology was eliminated as a BACT control option. Remaining subsections of section VI have been re-numbered accordingly.

From response #4.a:

In SOB subsection VI.D.1 ("Alternative from Deserado mine" as a BACT option), adjusted the estimate of potential emission reductions of condensible PM that might be achieved by switching from waste coal to ROM coal at the Deserado mine. Adjusted the $/ton annualized cost of BACT accordingly. EPA's conclusion that cost of ROM coal is excessive for BACT remains unchanged.

In SOB section VI.D.2 ("Alternative coal from other mines" as a BACT option), expanded the explanation of why alternative coal from other mines has been eliminated as a BACT option. The explanation now includes $/ton calculations on cost of this BACT option, along with a comparison of this cost versus the cost that other similar sources have to bear for BACT. EPA's conclusion that cost of alternative coal from other mines is excessive for BACT remains unchanged.

From response #4.b.(1):

In SOB subsection VI.K.5 (Step 5 of the SO_2 BACT analysis), added an explanation titled "Revision of proposed cutpoint" on why EPA has revised the "cutpoint" from 1.9 to 2.2 lb/MMBtu in permit condition III.D.1.b.(ii). Also added AES-Puerto Rico and Nevco Energy to the table titled, "Coal Scenarios and Sulfur Dioxide Control Efficiency, Comparisons for CFB Projects: EPA Compilation."

Also corrected the calculations in subsection VI.K.5, for "worst-case" coal at AES-Puerto Rico, from 1.7 lb/MMBtu and 98.7% control efficiency to 1.3 lb/MMBtu and 98.3% control efficiency.

The numerical value of the SO_2 BACT emission limit itself remains unchanged.

From response #4.c.(1):

In SOB subsection VI.G.1 (Step 1 of the NO_x BACT analysis), added a discussion of potential NO_x control options from the Nov. 1999 EPA Technical Bulletin that were not already addressed in the draft SOB, along with an explanation of why all but two of the options were eliminated at Step 1 of the top-down BACT analysis.

In SOB subsection VI.G.2, added an explanation of why the remaining two options from the Nov. 1999 Bulletin were eliminated at Step 2. EPA's conclusion that SCR and SNCR are the only technically feasible NO_x control options remains unchanged.

From responses #4.c.(4) and (5):

In SOB subsection VI.G.3 (Step 3 of the NO_x BACT analysis), added a statement that the potential NO_x control effectiveness of SCR has been revised from 0.04 to 0.015 lb/MMBtu at Step 3 of the BACT analysis, to reflect the possibility that a rate as low as 0.015 lb/MMBtu could be achieved, rather than 0.04 lb/MMBtu. Explained that this revision is based on information from Babcock & Wilcox, cited in public comments on the draft SOB. The cost effectiveness ranking at Step 3 remains unchanged.

In SOB subsection VI.G.4 (Step 4 of the NO_x BACT analysis), revised the reheat cost analysis for SCR in VI.G.4.a, to account for the aforementioned change in potential control effectiveness. EPA's conclusion that SCR should be eliminated at Step 4 remains unchanged.

From response #4.c.(8):

In SOB subsection VI.G.5 (Step 5 of the NO_x BACT analysis), added the Kentucky Mountain Power Project to the table titled "Summary of Recent CFB Projects Permitted or Proposed: NO_x Emission Rates Using SNCR." Also added an explanation titled "Note 1" on why EPA has discounted to some degree the significance of KMPP's initial NO_x emission limit of 0.07 lb/MMBtu. EPA's conclusion on what emission limit to impose as NO_x BACT remains unchanged.

From response #4.e.(2):

In SOB subsecton VI.H.7 ("Proposed compliance monitoring approach" for PM/PM_{10} filterable emissions), expanded the explanation of why no opacity limit or opacity monitoring for the CFB boiler exhaust stack are considered necessary in the permit. EPA's conclusion that no opacity limit or opacity monitoring is necessary remains unchanged.

From response #5.a.(1):

In SOB subsection VI.H.5 (Step 5 of the BACT analysis for PM/PM_{10} filterable emissions), added an explanation of why EPA has revised the averaging time of the PM/PM_{10} filterable emission limit in permit condition III.D.1.a from 30-day rolling to daily.

In SOB subsection VI.I.5 (Step 5 of the BACT analysis for PM/PM_{10} condensible emissions), revised the averaging time of the total PM/PM_{10} emission limit (including condensibles) from 30-day rolling to daily, to be consistent with the revised averaging time of the emission limit for the filterable portion.

From response #6:

In SOB subsection VIII.C.7, corrected the WCFU emission rates for 3-hour and 24-hour SO_2 in the table retitled, "ISC3 WCFU Stack Input Parameters Used for Modeling, As Corrected by EPA for 3-Hour and 24-Hour SO_2." The 3-hour rate was scaled up by a factor of 5.93 and the 24-hour rate was scaled up by a factor of 1.37.

In SOB subsection VIII.E.1.a, scaled up the WCFU's 3-hour and 24-hour SO_2 modeling results, in the table retitled "NAAQS Compliance Demonstration for WCFU Project Sources (Results as Corrected by EPA for SO_2)." Added an explanation why the results were scaled up.

In SOB subsection VIII.E.1.b, scaled up the WCFU's contribution to the 3-hour and 24-hour SO_2 full impact modeling results, in the table retitled "NAAQS Compliance Demonstration for Full Impact Area Sources (Results as Corrected by EPA for SO_2)." Added an explanation why the results were scaled up.

In SOB subsection VIII.E.2.a, scaled up the WCFU's 3-hour and 24-hour SO_2 modeling results, in the table retitled "PSD Class II Increment Compliance for WCFU Sources (Near-field Analysis) (Results as Corrected by EPA for SO_2)." Added an explanation why the results were scaled up.

In SOB subsection VIII.E.2.b, scaled up the WCFU's contribution to the 3-hour and 24-hour SO_2 full impact modeling results, in the table retitled "PSD Class II Increment Compliance for Full Impact Area Sources (Results as Corrected by EPA for SO_2)." Added an explanation why the results were scaled up.

(By "scale up" for the WCFU, EPA means the modeling results in micrograms per cubic meter (ug/m^3) attributable to the WCFU are multiplied by 5.93 for 3-hour SO_2 and 1.37 for 24-hour SO_2, to reflect the worst-case short-term SO_2 emission scenario at the WCFU.)

In SOB subsection VIII.E.3, added a reference to Deseret Power's March 23, 2005 Class I increment analysis for Dinosaur National Monument, which revised the modeling results to account for the higher short-term SO_2 emission rates at Bonanza Unit 1, as well as to account for the worst-case short-term SO_2 emission scenario at the WCFU.

In SOB subsection VIII.E.3, included the corrected WCFU emission rates for 3-hour and 24-hour SO_2 in a PSD Class I increment compliance screening analysis (described by EPA in response #9).

EPA's conclusion that the NAAQS and the PSD Class II increments will not be exceeded remains unchanged.

From response #7:

In SOB subsection VIII.C.7, corrected the Bonanza Unit 1 emission rates for 3-hour and 24-hour SO_2 in the table retitled, "Bonanza Unit 1 Stack Parameters Used for Modeling, As Corrected by EPA for 3-Hour and 24-Hour SO_2." The 3-hour rate was changed from 56.3 g/sec to 140 g/sec. The 24-hour rate was changed from 56.3 g/sec to 106 g/sec. These higher rates reflect maximum actual emission rates from the 2001-2002 period.

In SOB subsection VIII.E.1.b, scaled up Unit 1's contribution to the 3-hour and 24-hour SO_2 full impact modeling results, in the table retitled "NAAQS Compliance Demonstration for Full Impact Area Sources (Results as Corrected by EPA for SO_2)." Added an explanation why the results were scaled up.

In SOB subsection VIII.E.2.b, scaled up Unit 1's contribution to the 3-hour and 24-hour SO_2 full impact modeling results, in the table retitled "PSD Class II Increment Compliance for Full Impact Area Sources (Results as Corrected by EPA for SO_2)." Added an explanation why the results were scaled up.

(By "scale up" for Unit 1, EPA means the modeling results in micrograms per cubic meter (ug/m^3) attributable to Unit 1 are multiplied by a factor of 140/56.3 for 3-hour SO_2 and by a factor of 106/56.3 for 24-hour SO_2, to reflect higher short-term SO_2 emission rates at Unit 1.)

In SOB subsection VIII.E.3, added a reference to Deseret Power's March 23, 2005 Class I increment analysis for Dinosaur National Monument (which revised the modeling results to account for the higher short-term SO_2 emission rates at Bonanza Unit 1, as well as to account for the worst-case short-term SO_2 emission scenario at the WCFU).

EPA's conclusion that the NAAQS and the PSD Class II increments will not be exceeded remains unchanged.

From response #8.a:

In SOB subsection VIII.D, revised the 3-hour and 24-hour SO_2 "modeled maximums" in the table retitled "Near-Field WCFU Modeling Results and Comparison to Monitoring Exemption Levels (Modeled Maximums As Corrected by EPA for 3-Hour and 24-Hour SO2)," to reflect a 29% increase in Bonanza Unit 1's SO_2 emissions since the 1991-1993 period.

Also expanded the explanation of why Deseret Power should still qualify for the exemption from pre-construction ambient monitoring, even though the corrected 24-hour SO_2 modeled maximum is above the exemption threshold in 40 CFR 52.21(i)(5)(i). EPA's conclusion that Deseret Power should qualify for exemption remains unchanged.

From response #8.b:

In SOB subsection VIII.C.5, revised the SO_2 values in the table retitled "Background Pollutant Concentration Values (As Corrected by EPA for SO_2)," to reflect a 29% increase in Bonanza Unit 1's SO_2 emissions since the 1991-1993 period.

In SOB subsection VIII.E.1.a, revised the "Background Concentration" in the table retitled "NAAQS Compliance Demonstration for WCFU Project Sources (Results as Corrected by EPA for SO_2)," to reflect a 29% increase in Bonanza Unit 1's SO_2 emissions since the 1991-1993 period.

In SOB subsection VIII.E.1.b, revised the "Background Concentration" in the table retitled "NAAQS Compliance Demonstration for Full Impact Area Sources (Results as Corrected by EPA for SO_2)." to reflect a 29% increase in Bonanza Unit 1's SO_2 emissions since the 1991-1993 period.

EPA's conclusion that the NAAQS and the PSD Class II increments will not be exceeded remains unchanged.

From response #9:

In SOB subsection VIII.E.3, incorporated EPA's cumulative PSD Class I increment consumption screening analysis on nearby mandatory Federal Class I areas, as described in response #9. EPA's conclusion that Class I increment will not be exceeded or threatened remains unchanged.

From response #14:

In SOB subsection VI.Q.6, added a clarifying statement that if any visible emissions are observed in a quarterly observation at a baghouse or vent filter, the frequency of observation at that baghouse or vent filter shall return to monthly.

Other minor changes made to the draft SOB:

In SOB section III, "Public Notice, Comments, Hearings and Appeals," added a citation to public comments that were received. Also added a statement that the final WCFU permit, responses to public comments, final Statement of Basis, and Administrative Record of permit-related correspondence, will be available on EPA website. Also added a statement that since commenters requested changes in the draft permit, the effective date of the final permit is thirty days after permit issuance, unless the permit is appealed.

In SOB subsection V.C, "Application Submittals and Addendums," added citation of two e-mails dated April 5, 2007 from Deseret Power, which constituted additional amendments to their PSD permit application of November 1, 2004.

In SOB subsection VI.D.1, corrected an error in the calculated annual cost of using waste coal. (The corrected cost is $6 million/year.) Made corresponding corrections in the table titled, "Annualized Cost of Potential Emission Reductions if Run-of-Mine Coal is Used Rather Than Waste Coal for Deseret Power's Proposed WCFU."

Added document listings to Appendix A, to cover the period from June 13, 2006 until issuance of the final permit, and retitled the appendix, "List of Documents in the Administrative Record for Issuance of Federal PSD Permit #PSD-OU-0002-04.00."

U.S. Department of Health and Human Services
and
U.S. Environmental Protection Agency

May 2001; Updated February 2006

Mercury Levels in Commercial Fish and Shellfish

Return to Advisory on Mercury in Seafood

See also Mercury in Fish: FDA Monitoring Program

Table 1. Fish and Shellfish With Highest Levels of Mercury

SPECIES	MERCURY CONCENTRATION (PPM)					NO. OF SAMPLES	SOURCE OF DATA
	MEAN	MEDIAN	STDEV	MIN	MAX		
MACKEREL KING	0.730	N/A	N/A	0.230	1.670	213	GULF OF MEXICO REPORT 2000
SHARK	0.988	0.830	0.631	ND	4.540	351	FDA 1990-02
SWORDFISH	0.976	0.860	0.510	ND	3.220	618	FDA 1990-04
TILEFISH (Gulf of Mexico)	1.450	N/A	N/A	0.650	3.730	60	NMFS REPORT 1978

Table 2. Fish and Shellfish With Lower Levels of Mercury†

SPECIES	MERCURY CONCENTRATION (PPM)					NO. OF SAMPLES	SOURCE OF DATA
	MEAN	MEDIAN	STDEV	MIN	MAX		
ANCHOVIES	0.043	N/A	N/A	ND	0.340	40	NMFS REPORT 1978
BUTTERFISH	0.058	N/A	N/A	ND	0.360	89	NMFS REPORT 1978
CATFISH	0.049	ND	0.084	ND	0.314	23	FDA 1990-04

CLAM *	ND	ND	ND	ND	ND	6	FDA 1990-02
COD	0.095	0.087	0.080	ND	0.420	39	FDA 1990-04
CRAB [1]	0.060	0.030	0.112	ND	0.610	63	FDA 1990-04
CRAWFISH	0.033	0.035	0.012	ND	0.051	44	FDA 2002-04
CROAKER ATLANTIC (Atlantic)	0.072	0.073	0.036	0.013	0.148	35	FDA 1990-03
FLATFISH [2]*	0.045	0.035	0.049	ND	0.180	23	FDA 1990-04
HADDOCK (Atlantic)	0.031	0.041	0.021	ND	0.041	4	FDA 1990-02
HAKE	0.014	ND	0.021	ND	0.048	9	FDA 1990-02
HERRING	0.044	N/A	N/A	ND	0.135	38	NMFS REPORT 1978
JACKSMELT	0.108	0.060	0.115	0.040	0.500	16	FDA 1990-02
LOBSTER (Spiny)	0.09	0.14	‡	ND	0.27	9	FDA SURVEY 1990-02
MACKEREL ATLANTIC (N.Atlantic)	0.050	N/A	N/A	0.020	0.160	80	NMFS REPORT 1978
MACKEREL CHUB (Pacific)	0.088	N/A	N/A	0.030	0.190	30	NMFS REPORT 1978
MULLET	0.046	N/A	N/A	ND	0.130	191	NMFS REPORT 1978
OYSTER	0.013	ND	0.042	ND	0.250	38	FDA 1990-04
PERCH OCEAN *	ND	ND	ND	ND	0.030	6	FDA 1990-02
POLLOCK	0.041	ND	0.106	ND	0.780	62	FDA 1990-04
SALMON (CANNED)	ND	ND	ND	ND	ND	23	FDA 1990-

*							02
SALMON (FRESH/FROZEN) *	0.014	ND	0.041	ND	0.190	34	FDA 1990-02
SARDINE	0.016	0.013	0.007	0.004	0.035	29	FDA 2002-04
SCALLOP	0.050	N/A	N/A	ND	0.220	66	NMFS REPORT 1978
SHAD AMERICAN	0.065	N/A	N/A	ND	0.220	59	NMFS REPORT 1978
SHRIMP *	ND	ND	ND	ND	0.050	24	FDA 1990-02
SQUID	0.070	N/A	N/A	ND	0.400	200	NMFS REPORT 1978
TILAPIA *	0.010	ND	0.023	ND	0.070	9	FDA 1990-02
TROUT (FRESHWATER)	0.072	0.025	0.143	ND	0.678	34	FDA 2002-04
TUNA (CANNED, LIGHT)	0.118	0.075	0.119	ND	0.852	347	FDA 2002-04
WHITEFISH	0.069	0.054	0.067	ND	0.310	28	FDA 2002-04
WHITING	ND	ND	‡	ND	ND	2	FDA SURVEY 1990-02

Table 3. Mercury Levels of Other Fish and Shellfish†

SPECIES	MERCURY CONCENTRATION (PPM)					NO. OF SAMPLES	SOURCE OF DATA
	MEAN	MEDIAN	STDEV	MIN	MAX		
BASS (SALTWATER, BLACK, STRIPED)[3]	0.219	0.130	0.227	ND	0.960	47	FDA 1990-04
BASS CHILEAN	0.386	0.303	0.364	0.085	2.180	40	FDA 1990-04
BLUEFISH	0.337	0.303	0.127	0.139	0.634	52	FDA 2002-04
BUFFALOFISH	0.19	0.14	‡	0.05	0.43	4	FDA SURVEY

							1990-02
CARP	0.14	0.14	‡	0.01	0.27	2	FDA SURVEY 1990-02
CROAKER WHITE (Pacific)	0.287	0.280	0.069	0.180	0.410	15	FDA 1990-03
GROUPER (ALL SPECIES)	0.465	0.410	0.293	0.053	1.205	43	FDA 2002-04
HALIBUT	0.252	0.200	0.233	ND	1.520	46	FDA 1990-04
LOBSTER (NORTHERN/AMERICAN)	0.310	N/A	N/A	0.050	1.310	88	NMFS REPORT 1978
LOBSTER (Species Unknown)	0.169	0.182	0.089	ND	0.309	16	FDA 1991-2004
MACKEREL SPANISH (Gulf of Mexico)	0.454	N/A	N/A	0.070	1.560	66	NMFS REPORT 1978
MACKEREL SPANISH (S. Atlantic)	0.182	N/A	N/A	0.050	0.730	43	NMFS REPORT 1978
MARLIN *	0.485	0.390	0.237	0.100	0.920	16	FDA 1990-02
MONKFISH	0.180	N/A	N/A	0.020	1.020	81	NMFS REPORT 1978
ORANGE ROUGHY	0.554	0.563	0.148	0.296	0.855	49	FDA 1990-04
PERCH (Freshwater)	0.14	0.15	‡	ND	0.31	5	FDA SURVEY 1990-02
SABLEFISH	0.220	N/A	N/A	ND	0.700	102	NMFS REPORT 1978
SCORPIONFISH	0.286	N/A	N/A	0.020	1.345	78	NMFS REPORT 1978
SHEEPSHEAD	0.128	N/A	N/A	0.020	0.625	59	NMFS REPORT 1978

SKATE	0.137	N/A	N/A	0.040	0.360	56	NMFS REPORT 1978
SNAPPER	0.189	0.114	0.274	ND	1.366	43	FDA 2002-04
TILEFISH (Atlantic)	0.144	0.099	0.122	0.042	0.533	32	FDA 2002-04
TUNA (CANNED, ALBACORE)	0.353	0.339	0.126	ND	0.853	399	FDA 2002-04
TUNA(FRESH/FROZEN, ALL)	0.383	0.322	0.269	ND	1.300	228	FDA 2002-04
TUNA (FRESH/FROZEN, ALBACORE)	0.357	0.355	0.152	ND	0.820	26	FDA 2002-04
TUNA (FRESH/FROZEN, BIGEYE)	0.639	0.560	0.184	0.410	1.040	13	FDA 2002-04
TUNA (FRESH/FROZEN, SKIPJACK)	0.205	N/A	0.078	0.205	0.260	2	FDA 1993
TUNA (FRESH/FROZEN, YELLOWFIN)	0.325	0.270	0.220	ND	1.079	87	FDA 2002-04
TUNA (FRESH/FROZEN, Species Unknown)	0.414	0.339	0.316	ND	1.300	100	FDA 1991-2004
WEAKFISH (SEA TROUT)	0.256	0.168	0.226	ND	0.744	39	FDA 2002-04

Source of data: FDA 1990-2004, "National Marine Fisheries Service Survey of Trace Elements in the Fishery Resource" Report 1978,
"The Occurrence of Mercury in the Fishery Resources of the Gulf of Mexico" Report 2000

Mercury was measured as Total Mercury except for species (*) when only Methylmercury was analyzed.

ND - mercury concentration below detection level (Level of Detection (LOD)=0.01ppm)
N/A - data not available

†The following species have been removed from the tables:

Bass (freshwater) – not commercial
Pickerel – not commercial

‡ Standard deviation data generated for new data 2004 or later only.

[1]Includes: Blue, King, Snow

[2]Includes: Flounder, Plaice, Sole
[3]Includes: Sea bass/ Striped Bass/ Rockfish

NOTE: On February 8, 2006, technical changes were made to the data that was posted on January 19, 2006. The changes corrected data or more properly characterized the species of fish or shellfish sampled.

Advisory on Mercury in Seafood

Mercury in Fish: FDA Monitoring Program

Historical data: March 2004 Mercury Levels in Commercial Fish and Shellfish.

FDA/Center for Food Safety & Applied Nutrition
Hypertext updated by cjm March 2, 2006

TOXICOLOGICAL PROFILE FOR MERCURY

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

March 1999

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UPDATE STATEMENT

A Toxicological Profile for Mercury–Draft for Public Comment was released in September 1997. This edition supersedes any previously released draft or final profile.

Toxicological profiles are revised and republished as necessary, but no less than once every three years. For information regarding the update status of previously released profiles, contact ATSDR at:

Agency for Toxic Substances and Disease Registry
Division of Toxicology/Toxicology Information Branch
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

QUICK REFERENCE FOR HEALTH CARE PROVIDERS

Toxicological Profiles are a unique compilation of toxicological information on a given hazardous substance. Each profile reflects a comprehensive and extensive evaluation, summary, and interpretation of available toxicologic and epidemiologic information on a substance. Health care providers treating patients potentially exposed to hazardous substances will find the following information helpful for fast answers to often-asked questions.

Primary Chapters/Sections of Interest

Chapter 1: Public Health Statement: The Public Health Statement can be a useful tool for educating patients about possible exposure to a hazardous substance. It explains a substance's relevant toxicologic properties in a nontechnical, question-and-answer format, and it includes a review of the general health effects observed following exposure.

Chapter 2: Health Effects: Specific health effects of a given hazardous compound are reported by *route of exposure*, by *type of health effect* (death, systemic, immunologic, reproductive), and by *length of exposure* (acute, intermediate, and chronic). In addition, both human and animal studies are reported in this section.

NOTE: Not all health effects reported in this section are necessarily observed in the clinical setting. Please refer to the Public Health Statement to identify general health effects observed following exposure.

Pediatrics: Four new sections have been added to each Toxicological Profile to address child health issues:

Section 1.6 How Can (Chemical X) Affect Children?
Section 1.7 How Can Families Reduce the Risk of Exposure to (Chemical X)?
Section 2.6 Children's Susceptibility
Section 5.6 Exposures of Children

Other Sections of Interest:

Section 2.7 Biomarkers of Exposure and Effect
Section 2.10 Methods for Reducing Toxic Effects

ATSDR Information Center

Phone: 1-800-447-1544 (to be replaced by 1-888-42-ATSDR in 1999) or 404-639-6357 ***Fax:*** 404-639-6359
E-mail: atsdric@cdc.gov ***Internet:*** http://atsdr1.atsdr.cdc.gov:8080

The following additional material can be ordered through the ATSDR Information Center:

Case Studies in Environmental Medicine: Taking an Exposure History—The importance of taking an exposure history and how to conduct one are described, and an example of a thorough exposure history is provided. Other case studies of interest include *Reproductive and Developmental Hazards; Skin Lesions and Environmental Exposures; Cholinesterase-Inhibiting Pesticide Toxicity;* and numerous chemical-specific case studies.

Managing Hazardous Materials Incidents is a three-volume set of recommendations for on-scene (prehospital) and hospital medical management of patients exposed during a hazardous materials incident. Volumes I and II are planning guides to assist first responders and hospital emergency department personnel in planning for incidents that involve hazardous materials. Volume III—*Medical Management Guidelines for Acute Chemical Exposures*—is a guide for health care professionals treating patients exposed to hazardous materials.

Fact Sheets (ToxFAQs) provide answers to frequently asked questions about toxic substances.

Other Agencies and Organizations

The National Center for Environmental Health (NCEH) focuses on preventing or controlling disease, injury, and disability related to the interactions between people and their environment outside the workplace. *Contact:* NCEH, Mailstop F-29, 4770 Buford Highway, NE, Atlanta, GA 30341-3724 • Phone: 770-488-7000 • FAX: 770-488-7015.

The National Institute for Occupational Safety and Health (NIOSH) conducts research on occupational diseases and injuries, responds to requests for assistance by investigating problems of health and safety in the workplace, recommends standards to the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA), and trains professionals in occupational safety and health. *Contact:* NIOSH, 200 Independence Avenue, SW, Washington, DC 20201 • Phone: 800-356-4674 or NIOSH Technical Information Branch, Robert A. Taft Laboratory, Mailstop C-19, 4676 Columbia Parkway, Cincinnati, OH 45226-1998 • Phone: 800-35-NIOSH.

*The National Institute of Environmental Health Sciences (*NIEHS) is the principal federal agency for biomedical research on the effects of chemical, physical, and biologic environmental agents on human health and well-being. *Contact:* NIEHS, PO Box 12233, 104 T.W. Alexander Drive, Research Triangle Park, NC 27709 • Phone: 919-541-3212.

Referrals

The Association of Occupational and Environmental Clinics (AOEC) has developed a network of clinics in the United States to provide expertise in occupational and environmental issues. *Contact:* AOEC, 1010 Vermont Avenue, NW, #513, Washington, DC 20005 • Phone: 202-347-4976 • FAX: 202-347-4950 • e-mail: aoec@dgs.dgsys.com • AOEC Clinic Director: http://occ-env-med.mc.duke.edu/oem/aoec.htm.

The American College of Occupational and Environmental Medicine (ACOEM) is an association of physicians and other health care providers specializing in the field of occupational and environmental medicine. *Contact:* ACOEM, 55 West Seegers Road, Arlington Heights, IL 60005 • Phone: 847-228-6850 • FAX: 847-228-1856.

CONTRIBUTORS

CHEMICAL MANAGER(S)/AUTHORS(S):

John Risher, Ph.D.
ATSDR, Division of Toxicology, Atlanta, GA

Rob DeWoskin, Ph.D.
Research Triangle Institute, Research Triangle Park, NC

THE PROFILE HAS UNDERGONE THE FOLLOWING ATSDR INTERNAL REVIEWS:

1. Health Effects Review. The Health Effects Review Committee examines the health effects chapter of each profile for consistency and accuracy in interpreting health effects and classifying end points.

2. Minimal Risk Level Review. The Minimal Risk Level Workgroup considers issues relevant to substance-specific minimal risk levels (MRLs), reviews the health effects database of each profile, and makes recommendations for derivation of MRLs.

3. Data Needs Review. The Research Implementation Branch reviews data needs sections to assure consistency across profiles and adherence to instructions in the Guidance.

PEER REVIEW

A peer review panel was assembled for mercury. The panel consisted of the following members:

1. Mr. Harvey Clewell, K.S. Crump Group, ICF Kaiser International, Inc., Ruston, LA

2. Dr. Ingeborg Harding-Barlow, Private Consultant, Environmental and Occupational Toxicology, 3717 Laguna Ave., Palo Alto, California;

3. Dr. Thomas Hinesly, Professor (Emeritus), Department of Natural Resources and Environmental Sciences, University of Illinois, Champaign-Urbana, Illinois;

4. Dr. Loren D. Koller, Professor, College of Veterinary Medicine, Oregon State University, Corvallis, Oregon; and

5. Dr. Kenneth Reuhl, Professor, Neurotoxicology Laboratory, Rutgers University, Piscataway, New York.

These experts collectively have knowledge of mercury's physical and chemical properties, toxicokinetics, key health end points, mechanisms of action, human and animal exposure, and quantification of risk to humans. All reviewers were selected in conformity with the conditions for peer review specified in Section 104(i)(13) of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended.

Scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) have reviewed the peer reviewer's comments and determined which comments will be included in the profile. A listing of the profile. A listing of the peer reviewers' comments not incorporated in the profile, with brief explanation of the rationale for their exclusion, exists as part of the administrative record for this compound. A list of databases reviewed and a list of unpublished documents cited are also included in the administrative record.

The citation of the peer review panel should not be understood to imply its approval of the profile's final content. The responsibility for the content of this profile lies with the ATSDR.

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1. PUBLIC HEALTH STATEMENT

This public health statement tells you about mercury and the effects of exposure.

The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup activities. Mercury has been found in at least 714 of the 1,467 current or former NPL sites. However, the total number of NPL sites evaluated for this substance is not known. As more sites are evaluated, the sites at which mercury is found may increase. This information is important because exposure to this substance may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance or by skin contact.

If you are exposed to mercury, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals to which you're exposed, as well as your age, sex, diet, family traits, lifestyle, and state of health.

1.1 WHAT IS MERCURY?

Mercury occurs naturally in the environment and exists in several forms. These forms can be organized under three headings: metallic mercury (also known as elemental mercury), inorganic mercury, and organic mercury. Metallic mercury is a shiny, silver-white metal that is a liquid at room temperature. Metallic mercury is the elemental or pure form of mercury (i.e., it is not combined with other elements). Metallic mercury metal is the familiar liquid metal used in thermometers and some electrical switches. At room temperature, some of the metallic mercury

will evaporate and form mercury vapors. Mercury vapors are colorless and odorless. The higher the temperature, the more vapors will be released from liquid metallic mercury. Some people who have breathed mercury vapors report a metallic taste in their mouths. Metallic mercury has been found at 714 hazardous waste sites nationwide.

Inorganic mercury compounds occur when mercury combines with elements such as chlorine, sulfur, or oxygen. These mercury compounds are also called mercury salts. Most inorganic mercury compounds are white powders or crystals, except for mercuric sulfide (also known as cinnabar) which is red and turns black after exposure to light.

When mercury combines with carbon, the compounds formed are called "organic" mercury compounds or organomercurials. There is a potentially large number of organic mercury compounds; however, by far the most common organic mercury compound in the environment is methylmercury (also known as monomethylmercury). In the past, an organic mercury compound called phenylmercury was used in some commercial products. Another organic mercury compound called dimethylmercury is also used in small amounts as a reference standard for some chemical tests. Dimethylmercury is the only organic mercury compound that has been identified at hazardous waste sites. It was only found in extremely small amounts at two hazardous waste sites nationwide, but it is very harmful to people and animals. Like the inorganic mercury compounds, both methylmercury and phenylmercury exist as "salts" (for example, methylmercuric chloride or phenylmercuric acetate). When pure, most forms of methylmercury and phenylmercury are white crystalline solids. Dimethylmercury, however, is a colorless liquid.

Several forms of mercury occur naturally in the environment. The most common natural forms of mercury found in the environment are metallic mercury, mercuric sulfide (cinnabar ore), mercuric chloride, and methylmercury. Some microorganisms (bacteria and fungi) and natural processes can change the mercury in the environment from one form to another. The most common organic mercury compound that microorganisms and natural processes generate from other forms is methylmercury. Methylmercury is of particular concern because it can build up in certain edible

freshwater and saltwater fish and marine mammals to levels that are many times greater than levels in the surrounding water (see Section 1.2).

Mercury is mined as cinnabar ore, which contains mercuric sulfide. The metallic form is refined from mercuric sulfide ore by heating the ore to temperatures above 1,000 degrees Fahrenheit. This vaporizes the mercury in the ore, and the vapors are then captured and cooled to form the liquid metal mercury. There are many different uses for liquid metallic mercury. It is used in producing of chlorine gas and caustic soda, and in extracting gold from ore or articles that contain gold. It is also used in thermometers, barometers, batteries, and electrical switches. Silver-colored dental fillings typically contain about 50% metallic mercury. Metallic mercury is still used in some herbal or religious remedies in Latin America and Asia, and in rituals or spiritual practices in some Latin American and Caribbean religions such as Voodoo, Santeria, and Espiritismo. These uses may pose a health risk from exposure to mercury both for the user and for others who may be exposed to mercury vapors in contaminated air.

Some inorganic mercury compounds are used as fungicides. Inorganic salts of mercury, including ammoniated mercuric chloride and mercuric iodide, have been used in skin-lightening creams. Mercuric chloride is a topical antiseptic or disinfectant agent. In the past, mercurous chloride was widely used in medicinal products including laxatives, worming medications, and teething powders. It has since been replaced by safer and more effective agents. Other chemicals containing mercury are still used as antibacterials. These products include mercurochrome (contains a small amount of mercury, 2%), and thimerosal and phenylmercuric nitrate, which are used in small amounts as preservatives in some prescription and over-the-counter medicines. Mercuric sulfide and mercuric oxide may be used to color paints, and mercuric sulfide is one of the red coloring agents used in tattoo dyes.

Methylmercury is produced primarily by microorganisms (bacteria and fungi) in the environment, rather than by human activity. Until the 1970s, methylmercury and ethylmercury compounds were used to protect seed grains from fungal infections. Once the adverse health effects of methylmercury were known, the use of methymercury- and ethylmercury as fungicides was

banned. Up until 1991, phenylmercuric compounds were used as antifungal agents in both interior and exterior paints, but this use was also banned because mercury vapors were released from these paints.

Chapter 3 contains more information on the physical and chemical properties of mercury. Chapter 4 contains more information on the production and use of mercury.

1.2 WHAT HAPPENS TO MERCURY WHEN IT ENTERS THE ENVIRONMENT?

Mercury is a naturally occurring metal found throughout the environment. Mercury enters the environment as the result of the normal breakdown of minerals in rocks and soil from exposure to wind and water, and from volcanic activity. Mercury releases from natural sources have remained relatively constant in recent history, resulting in a steady rise in environmental mercury. Human activities since the start of the industrial age (e.g., mining, burning of fossil fuels) have resulted in additional release of mercury to the environment. Estimates of the total annual mercury releases that result from human activities range from one-third to two-thirds of the total mercury releases. A major uncertainty in these estimates is the amount of mercury that is released from water and soils that were previously contaminated by human activities as opposed to new natural releases. The levels of mercury in the atmosphere (i.e., the air you breathe in the general environment) are very, very low and do not pose a health risk; however, the steady release of mercury has resulted in current levels that are three to six times higher than the estimated levels in the preindustrial era atmosphere.

Approximately 80% of the mercury released from human activities is elemental mercury released to the air, primarily from fossil fuel combustion, mining, and smelting, and from solid waste incineration. About 15% of the total is released to the soil from fertilizers, fungicides, and municipal solid waste (for example, from waste that contains discarded batteries, electrical switches, or thermometers). An additional 5% is released from industrial wastewater to water in the environment.

With the exception of mercury ore deposits, the amount of mercury that naturally exists in any one place is usually very low. In contrast, the amount of mercury that may be found in soil at a particular hazardous waste site because of human activity can be high (over 200,000 times natural levels). The mercury in air, water, and soil at hazardous waste sites may come from both natural sources and human activity.

Most of the mercury found in the environment is in the form of metallic mercury and inorganic mercury compounds. Metallic and inorganic mercury enters the air from mining deposits of ores that contain mercury, from the emissions of coal-fired power plants, from burning municipal and medical waste, from the production of cement, and from uncontrolled releases in factories that use mercury. Metallic mercury is a liquid at room temperature, but some of the metal will evaporate into the air and can be carried long distances. In air, the mercury vapor can be changed into other forms of mercury, and can be further transported to water or soil in rain or snow. Inorganic mercury may also enter water or soil from the weathering of rocks that contain mercury, from factories or water treatment facilities that release water contaminated with mercury, and from incineration of municipal garbage that contains mercury (for example, in thermometers, electrical switches, or batteries that have been thrown away). Inorganic or organic compounds of mercury may be released to the water or soil if mercury-containing fungicides are used.

Microorganisms (bacteria, phytoplankton in the ocean, and fungi) convert inorganic mercury to methylmercury. Methylmercury released from microorganisms can enter the water or soil and remain there for a long time, particularly if the methylmercury becomes attached to small particles in the soil or water. Mercury usually stays on the surface of sediments or soil and does not move through the soil to underground water. If mercury enters the water in any form, it is likely to settle to the bottom where it can remain for a long time.

Mercury can enter and accumulate in the food chain. The form of mercury that accumulates in the food chain is methylmercury. Inorganic mercury does not accumulate up the food chain to any extent. When small fish eat the methylmercury in food, it goes into their tissues. When larger fish eat smaller fish or other organisms that contain methylmercury, most of the methylmercury

originally present in the small fish will then be stored in the bodies of the larger fish. As a result, the larger and older fish living in contaminated waters build up the highest amounts of methyl-mercury in their bodies. Saltwater fish (especially sharks and swordfish) that live a long time and can grow to a very large size tend to have the highest levels of mercury in their bodies. Plants (such as corn, wheat, and peas) have very low levels of mercury, even if grown in soils containing mercury at significantly higher than background levels. Mushrooms, however, can accumulate high levels if grown in contaminated soils. For further information on what happens to mercury in the environment, see Chapters 4 and 5.

1.3 HOW MIGHT I BE EXPOSED TO MERCURY?

Because mercury occurs naturally in the environment, everyone is exposed to very low levels of mercury in air, water, and food. Between 10 and 20 nanograms of mercury per cubic meter (ng/m^3) of air have been measured in urban outdoor air. These levels are hundreds of times lower than levels still considered to be “safe” to breathe. Background levels in nonurban settings are even lower, generally about 6 ng/m^3 or less. Mercury levels in surface water are generally less than 5 parts of mercury per trillion parts of water (5 ppt, or 5 ng per liter of water), about a thousand times lower than “safe” drinking water standards. Normal soil levels range from 20 to 625 parts of mercury per billion parts of soil (20–625 ppb; or 20,000–625,000 ng per kilogram of soil). A part per billion is one thousand times bigger than a part per trillion.

A potential source of exposure to metallic mercury for the general population is mercury released from dental amalgam fillings. An amalgam is a mixture of metals. The amalgam used in silver-colored dental fillings contains approximately 50% metallic mercury, 35% silver, 9% tin, 6% copper, and trace amounts of zinc. When the amalgam is first mixed, it is a soft paste which is inserted into the tooth surface. It hardens within 30 minutes. Once the amalgam is hard, the mercury is bound within the amalgam, but very small amounts are slowly released from the surface of the filling due to corrosion or chewing or grinding motions. Part of the mercury at the surface of the filling may enter the air as mercury vapor or be dissolved in the saliva. The total amount of mercury released from dental amalgam depends upon the total number of fillings and

surface areas of each filling, the chewing and eating habits of the person, and other chemical conditions in the mouth. Estimates of the amount of mercury released from dental amalgams range from 3 to 17 micrograms per day (µg/day). The mercury from dental amalgam may contribute from 0 to more than 75% of your total daily mercury exposure, depending on the number of amalgam fillings you have, the amount of fish consumed, the levels of mercury (mostly as methylmercury) in those fish, and exposure from other less common sources such as mercury spills, religious practices, or herbal remedies that contain mercury. However, it should be kept in mind that exposure to very small amounts of mercury, such as that from dental amalgam fillings, does not necessarily pose a health risk.

Whether the levels of exposure to mercury vapor from dental amalgam are sufficiently high to cause adverse health effects, and exactly what those effects are, continues to be researched and debated by scientists and health officials. U.S. government summaries on the effects of dental amalgam conclude that there is no apparent health hazard to the general population, but that further study is needed to determine the possibility of more subtle behavioral or immune system effects, and to determine the levels of exposure that may lead to adverse effects in sensitive populations. Sensitive populations may include pregnant women, children under the age of 6 (especially up to the age of 3), people with impaired kidney function, and people with hypersensitive immune responses to metals. If you belong to this group, you should discuss your medical condition with your dentist prior to any dental restoration work. Removal of dental amalgams in people who have no indication of adverse effects is not recommended and can put the person at greater risk, if performed improperly. Chelation therapy (used to remove metals from the body tissues) itself presents some health risks, and should be considered only when a licensed occupational or environmental health physician determines it necessary to reduce immediate and significant health risks due to high levels of mercury in the body. For additional information on health risks associated with mercury dental amalgam, see Section 2.5, "More on the Health Effects of Dental Amalgam."

Some religions have practices that may include the use of metallic mercury. Examples of these religions include Santeria (a Cuban-based religion whose followers worship both African deities

and Catholic saints), Voodoo (a Haitian-based set of beliefs and rituals), Palo Mayombe (a secret form of ancestor worship practiced mainly in the Caribbean), and Espiritismo (a spiritual belief system native to Puerto Rico). Not all people who observe these religions use mercury, but when mercury is used in religious, ethnic, or ritualistic practices, exposure to mercury may occur both at the time of the practice and afterwards from contaminated indoor air. Metallic mercury is sold under the name "azogue" (pronounced ah-SEW-gay) in stores called “botanicas.” Botanicas are common in Hispanic and Haitian communities, where azogue may be sold as an herbal remedy or for spiritual practices. The metallic mercury is often sold in capsules or in glass containers. It may be placed in a sealed pouch to be worn on a necklace or in a pocket, or it may be sprinkled in the home or car. Some people may mix azogue in bath water or perfume, or place azogue in devotional candles. Because metallic mercury evaporates into the air, these practices may put anyone breathing the air in the room at risk of exposure to mercury. The longer people breathe the contaminated air, the greater their risk will be. The use of metallic mercury in a home or an apartment not only threatens the health of the people who live there now, but also threatens the health of future residents who may unknowingly be exposed to further release of mercury vapors from contaminated floors or walls.

Metallic mercury is used in a variety of household products and industrial items, including thermostats, fluorescent light bulbs, barometers, glass thermometers, and some blood pressure devices. The mercury in these devices is contained in glass or metal, and generally does not pose a risk unless the item is damaged or broken, and mercury vapors are released. Spills of metallic mercury from broken thermometers or damaged electrical switches in the home may result in exposure to mercury vapors in indoor air. You must be careful when you handle and dispose of all items in the home that contain metallic mercury.

Very small amounts of metallic mercury (for example, a few drops) can raise air concentrations of mercury to levels that may be harmful to health. The longer people breathe the contaminated air, the greater the risk to their health. Metallic mercury and its vapors are extremely difficult to remove from clothes, furniture, carpet, floors, walls, and other such items. If these items are not

properly cleaned, the mercury can remain for months or years, and continue to be a source of exposure.

It is possible for you to be exposed to metallic mercury vapors from breathing contaminated air around hazardous waste sites, waste incinerators, or power plants that burn mercury-containing fuels (such as coal or other fossil fuels), but most outdoor air is not likely to contain levels that would be harmful. Exposure to mercury compounds at hazardous waste sites is much more likely to occur from handling contaminated soil (i.e., children playing in or eating contaminated surface soil), drinking well-water, or eating fish from contaminated waters near those sites. Not all hazardous sites contain mercury, and not all waste sites that do contain mercury have releases of mercury to the air, water, or surface soils.

You can be exposed to mercury vapors from the use of fungicides that contain mercury. Excess use of these products may result in higher-than-average exposures. You may also be exposed to mercury from swallowing or applying to your skin outdated medicinal products (laxatives, worming medications, and teething powders) that contain mercurous chloride. Exposure may also occur from the improper or excessive use of other chemicals containing mercury, such as skin-lightening creams and some topical antiseptic or disinfectant agents (mercurochrome and thimerosal).

Workers are mostly exposed from breathing air that contains mercury vapors, but may also be exposed to other inorganic mercury compounds in the workplace. Occupations that have a greater potential for mercury exposure include manufacturers of electrical equipment or automotive parts that contain mercury, chemical processing plants that use mercury, metal processing, construction where building parts contain mercury (e.g., electrical switches, thermometers), and the medical professions (medical, dental, or other health services) where equipment may contain mercury (e.g., some devices that measure blood pressure contain liquid mercury). Dentists and their assistants may be exposed to metallic mercury from breathing in mercury vapor released from amalgam fillings and to a much lesser extent from skin contact with

amalgam restorations. Family members of workers who have been exposed to mercury may also be exposed to mercury if the worker's clothes are contaminated with mercury particles or liquid.

Some people may be exposed to higher levels of mercury in the form of methylmercury if they have a diet high in fish, shellfish, or marine mammals (whales, seals, dolphins, and walruses) that come from mercury-contaminated waters. Methylmercury accumulates up the food chain, so that fish at the top of the food chain will have the most mercury in their flesh. Of these fish, the largest (i.e., the oldest) fish will have the highest levels. The Food and Drug Administration (FDA) estimates that most people are exposed, on average, to about 50 ng of mercury per kilogram of body weight per day (50 ng/kg/day) in the food they eat. This is about 3.5 micrograms (μg) of mercury per day for an adult of average weight. This level is not thought to result in any harmful effects. A large part of this mercury is in the form of methylmercury and probably comes from eating fish. Commercial fish sold through interstate commerce that are found to have levels of methylmercury above an "action level" of 1 ppm (established by the FDA) cannot be sold to the public. This level itself is below a level associated with adverse effects. However, if you fish in contaminated waters and eat the fish you catch, you may be exposed to higher levels of mercury. Public health advisories are issued by state and federal authorities for local waters that are thought to be contaminated with mercury. These advisories can help noncommercial (sport and subsistence) fishermen and their families to avoid eating fish contaminated with mercury. Foods other than fish that may contain higher than average levels of mercury include wild game, such as wild birds and mammals (bear) that eat large amounts of contaminated fish. People in the most northern climates may be exposed to high levels of mercury from eating meat or fat from marine mammals including whales, dolphins, walruses, and seals. These marine mammals are at or near the top of their marine food chain. Plants contain very little methylmercury or other forms of mercury. Mushrooms grown in mercury-contaminated soil may contain levels of mercury that could pose some risk to health, if large amounts were eaten.

See Chapter 5 for more information on how you might be exposed to mercury.

1.4 HOW CAN MERCURY ENTER AND LEAVE MY BODY?

A person can be exposed to mercury from breathing in contaminated air, from swallowing or eating contaminated water or food, or from having skin contact with mercury. Not all forms of mercury easily enter your body, even if they come in contact with it; so it is important to know which form of mercury you have been exposed to, and by which route (air, food, or skin).

When you swallow small amounts of metallic mercury, for example, from a broken oral thermometer, virtually none (less than 0.01%) of the mercury will enter your body through the stomach or intestines, unless they are diseased. Even when a larger amount of metal mercury (a half of a tablespoon, about 204 grams) was swallowed by one person, very little entered the body. When you breathe in mercury vapors, however, most (about 80%) of the mercury enters your bloodstream directly from your lungs, and then rapidly goes to other parts of your body, including the brain and kidneys. Once in your body, metallic mercury can stay for weeks or months. When metallic mercury enters the brain, it is readily converted to an inorganic form and is “trapped” in the brain for a long time. Metallic mercury in the blood of a pregnant woman can enter her developing child. Most of the metallic mercury will accumulate in your kidneys, but some metallic mercury can also accumulate in the brain. Most of the metallic mercury absorbed into the body eventually leaves in the urine and feces, while smaller amounts leave the body in the exhaled breath.

Inorganic mercury compounds like mercurous chloride and mercuric chloride are white powders and do not generally vaporize at room temperatures like elemental mercury will. If they are inhaled, they are not expected to enter your body as easily as inhaled metallic mercury vapor. When inorganic mercury compounds are swallowed, generally less than 10% is absorbed through the intestinal tract; however, up to 40% may enter the body through the stomach and intestines in some instances. Some inorganic mercury can enter your body through the skin, but only a small amount will pass through your skin compared to the amount that gets into your body from swallowing inorganic mercury.

Once inorganic mercury enters the body and gets into the bloodstream, it moves to many different tissues. Inorganic mercury leaves your body in the urine or feces over a period of several weeks or months. A small amount of the inorganic mercury can be changed in your body to metallic mercury and leave in the breath as a mercury vapor. Inorganic mercury accumulates mostly in the kidneys and does not enter the brain as easily as metallic mercury. Inorganic mercury compounds also do not move as easily from the blood of a pregnant woman to her developing child. In a nursing woman, some of the inorganic mercury in her body will pass into her breast milk.

Methylmercury is the form of mercury most easily absorbed through the gastrointestinal tract (about 95% absorbed). After you eat fish or other foods that are contaminated with methylmercury, the methylmercury enters your bloodstream easily and goes rapidly to other parts of your body. Only small amounts of methylmercury enter the bloodstream directly through the skin, but other forms of organic mercury (in particular dimethylmercury) can rapidly enter the body through the skin. Organic mercury compounds may evaporate slowly at room temperature and may enter your body easily if you breathe in the vapors. Once organic mercury is in the bloodstream, it moves easily to most tissues and readily enters the brain. Methylmercury that is in the blood of a pregnant woman will easily move into the blood of the developing child and then into the child's brain and other tissues. Like metallic mercury, methylmercury can be changed by your body to inorganic mercury. When this happens in the brain, the mercury can remain there for a long time. When methylmercury does leave your body after you have been exposed, it leaves slowly over a period of several months, mostly as inorganic mercury in the feces. As with inorganic mercury, some of the methylmercury in a nursing woman's body will pass into her breast milk.

For more information on how mercury can enter and leave your body, please see Chapter 2.

1.5 HOW CAN MERCURY AFFECT MY HEALTH?

The nervous system is very sensitive to mercury. In poisoning incidents that occurred in other countries, some people who ate fish contaminated with large amounts of methylmercury or seed grains treated with methylmercury or other organic mercury compounds developed permanent

damage to the brain and kidneys. Permanent damage to the brain has also been shown to occur from exposure to sufficiently high levels of metallic mercury. Whether exposure to inorganic mercury results in brain or nerve damage is not as certain, since it does not easily pass from the blood into the brain.

Metallic mercury vapors or organic mercury may affect many different areas of the brain and their associated functions, resulting in a variety of symptoms. These include personality changes (irritability, shyness, nervousness), tremors, changes in vision (constriction (or narrowing) of the visual field), deafness, muscle incoordination, loss of sensation, and difficulties with memory.

Different forms of mercury have different effects on the nervous system, because they do not all move through the body in the same way. When metallic mercury vapors are inhaled, they readily enter the bloodstream and are carried throughout the body and can move into the brain. Breathing in or swallowing large amounts of methylmercury also results in some of the mercury moving into the brain and affecting the nervous system. Inorganic mercury salts, such as mercuric chloride, do not enter the brain as readily as methylmercury or metallic mercury vapor.

The kidneys are also sensitive to the effects of mercury, because mercury accumulates in the kidneys and causes higher exposures to these tissues, and thus more damage. All forms of mercury can cause kidney damage if large enough amounts enter the body. If the damage caused by the mercury is not too great, the kidneys are likely to recover once the body clears itself of the contamination.

Short-term exposure (hours) to high levels of metallic mercury vapor in the air can damage the lining of the mouth and irritate the lungs and airways, causing tightness of the breath, a burning sensation in the lungs, and coughing. Other effects from exposure to mercury vapor include nausea, vomiting, diarrhea, increases in blood pressure or heart rate, skin rashes, and eye irritation. Damage to the lining of the mouth and lungs can also occur from exposure to lower levels of mercury vapor over longer periods (for example, in some occupations where workers were exposed to mercury for many years). Levels of metallic mercury in workplace air are generally much greater than the levels normally encountered by the general population. Current

levels of mercury in workplace air are low, due to increased awareness of mercury's toxic effects. Because of the reduction in the allowable amount of mercury in workplace air, fewer workers are expected to have symptoms of mercury toxicity. Most studies of humans who breathed metallic mercury for a long time indicate that mercury from this type of exposure does not affect the ability to have children. Studies in workers exposed to metallic mercury vapors have also not shown any mercury-related increase in cancer. Skin contact with metallic mercury has been shown to cause an allergic reaction (skin rashes) in some people.

In addition to effects on the kidneys, inorganic mercury can damage the stomach and intestines, producing symptoms of nausea, diarrhea, or severe ulcers if swallowed in large amounts. Effects on the heart have also been observed in children after they accidentally swallowed mercuric chloride. Symptoms included rapid heart rate and increased blood pressure. There is little information on the effects in humans from long-term, low-level exposure to inorganic mercury.

To protect the public from the harmful effects of toxic chemicals and to find ways to treat people who have been harmed, scientists use many tests.

One way to see if a chemical will hurt people is to learn how the chemical is absorbed, used, and released by the body; for some chemicals, animal testing may be necessary. Animal testing may also be used to identify health effects such as cancer or birth defects. Without laboratory animals, scientists would lose a basic method to get information needed to make wise decisions to protect public health. Scientists have the responsibility to treat research animals with care and compassion. Laws today protect the welfare of research animals, and scientists must comply with strict animal care guidelines.

Studies using animals indicate that long-term oral exposure to inorganic mercury salts causes kidney damage, effects on blood pressure and heart rate, and effects on the stomach. Study results also suggest that reactions involving the immune system may occur in sensitive populations after swallowing inorganic mercury salts. Some animal studies report that nervous system damage occurs after long-term exposure to high levels of inorganic mercury. Short-term, high-

level exposure of laboratory animals to inorganic mercury has been shown to affect the developing fetus and may cause termination of the pregnancy.

Animals exposed orally to long-term, high levels of methylmercury or phenylmercury in laboratory studies experienced damage to the kidneys, stomach, and large intestine; changes in blood pressure and heart rate; adverse effects on the developing fetus, sperm, and male reproductive organs; and increases in the number of spontaneous abortions and stillbirths. Adverse effects on the nervous system of animals occur at lower doses than do harmful effects to most other systems of the body. This difference indicates that the nervous system is more sensitive to methylmercury toxicity than are other organs in the body. Animal studies also provide evidence of damage to the nervous system from exposure to methylmercury during development, and evidence suggests that the effects worsen with age, even after the exposure stops.

Some rat and mice strains that are susceptible to autoimmune responses develop kidney damage as a result of an immune response when exposed to relatively low levels of mercury vapor or mercury chloride.

Animals given inorganic mercury salts by mouth for most of their lifetime had increases in some kinds of tumors at the highest dose tested. Rats and mice that received organic mercury (methyl-mercury or phenylmercury) in their drinking water or feed for most of their lives had an increased incidence of cancer of the kidney, but this affected only the males that received the highest amount of mercury given (not the females). Since the high doses caused severe damage to the kidneys prior to the cancer, these animal studies provide only limited information about whether mercury causes cancer in humans. As a result, the Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have not classified mercury as to its human carcinogenicity. The Environmental Protection Agency has determined that mercury chloride and methylmercury are possible human carcinogens. Chapter 2 contains more information on the health effects of mercury in humans and animals.

1.6 HOW CAN MERCURY AFFECT CHILDREN?

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans. Potential effects on children resulting from exposures of the parents are also considered.

Children are at risk of being exposed to metallic mercury that is not safely contained, to mercury that may be brought home on work clothes or tools, or to methylmercury-contaminated foods. Methylmercury eaten or swallowed by a pregnant woman or metallic mercury that enters her body from breathing contaminated air can also pass into the developing child. Inorganic mercury and methylmercury can also pass from a mother's body into breast milk and into a nursing infant. The amount of mercury in the milk will vary, depending on the degree of exposure and the amount of mercury that enter the nursing woman's body. There are significant benefits to breast feeding, so any concern that a nursing woman may have about mercury levels in her breast milk should be discussed with her doctor. Methylmercury can also accumulate in an unborn baby's blood to a concentration higher than the concentration in the mother.

For similar exposure routes and forms of mercury, the harmful health effects seen in children are similar to the effects seen in adults. High exposure to mercury vapor causes lung, stomach, and intestinal damage and death due to respiratory failure in severe cases. These effects are similar to those seen in adult groups exposed to inhaled metallic mercury vapors at work.

Children who had been exposed to excessive amounts of mercurous chloride tablets for worms or mercurous chloride-containing powders for teething discomfort had increased heart rates and elevated blood pressure. Abnormal heart rhythms were also seen in children who had eaten grains contaminated with very high levels of methylmercury.

Other symptoms of poisonings in children who were treated with mercurous chloride for constipation, worms, or teething discomfort included swollen red gums, excessive salivation, weight loss, diarrhea and/or abdominal pain, and muscle twitching or cramping in the legs and/or arms. Kidney damage is very common after exposure to toxic levels of inorganic mercury.

Metallic mercury or methylmercury that enters the body can also be converted to inorganic mercury and result in kidney damage.

Children who breathe metallic/elemental mercury vapors, eat foods or other substances containing phenylmercury or inorganic mercury salts, or use mercury-containing skin ointments for an extended period may develop a disorder known as acrodynia, or pink disease. Acrodynia can result in severe leg cramps; irritability; and abnormal redness of the skin, followed by peeling of the hands, nose, and soles of the feet. Itching, swelling, fever, fast heart rate, elevated blood pressure, excessive salivation or sweating, rashes, fretfulness, sleeplessness, and/or weakness may also be present. It was once believed that this syndrome occurred only in children, but recent reported cases in teenagers and adults have shown that they can also develop acrodynia.

In critical periods of development before they are born, and in the early months after birth, children and fetuses are particularly sensitive to the harmful effects of metallic mercury and methylmercury on the nervous system. Harmful developmental effects may occur when a pregnant woman is exposed to metallic mercury and some of the mercury is transferred into her developing child. Thus, women who are normally exposed to mercury vapors in the workplace (such as those working in thermometer/barometer or fluorescent light manufacturing or the chlor-alkali industry) should take measures to avoid mercury vapor exposures during pregnancy. Exposures to mercury vapors are relatively rare outside of the workplace, unless metallic mercury is present in the home.

As with mercury vapors, exposure to methylmercury is more dangerous for young children than for adults, because more methylmercury easily passes into the developing brain of young children and may interfere with the development process.

Methylmercury is the form of mercury most commonly associated with a risk for developmental effects. Exposure can come from foods contaminated with mercury on the surface (for example, from seed grain treated with methylmercury to kill fungus) or from foods that contain toxic levels of methylmercury (as in some fish, wild game, and marine mammals). Mothers who are exposed to methylmercury and breast-feed their infant may also expose the child through the milk. The

effects on the infant may be subtle or more pronounced, depending on the amount to which the fetus or young child was exposed. In cases in which the exposure was very small, some effects might not be apparent, such as small decreases in IQ or effects on the brain that may only be determined by the use of very sensitive neuropsychological testing. In instances in which the exposure is great, the effects may be more serious. In some such cases of mercury exposure involving serious exposure to the developing fetus, the effects are delayed. In such cases, the infant may be born apparently normal, but later show effects that may range from the infant being slower to reach developmental milestones, such as the age of first walking and talking, to more severe effects including brain damage with mental retardation, incoordination, and inability to move. Other severe effects observed in children whose mothers were exposed to very toxic levels of mercury during pregnancy include eventual blindness, involuntary muscle contractions and seizures, muscle weakness, and inability to speak. It is important to remember, however, that the severity of these effects depends upon the level of mercury exposure and the time of exposure. The very severe effects just mentioned were reported in large-scale poisoning instances in which pregnant and nursing women were exposed to extremely high levels of methylmercury in contaminated grain used to make bread (in Iraq) or seafood (in Japan) sold to the general population.

Researchers are currently studying the potential for less serious developmental effects, including effects on a child's behavior and ability to learn, think, and solve problems that may result from eating lower levels of methylmercury in foods. A main source of exposure to methylmercury for the pregnant woman and the young child is from eating fish. Most fish purchased in the market in the United States do not have mercury levels that pose a risk to anyone, including pregnant women. Since mercury accumulates in the muscles of fish, larger fish that feed on smaller fish and live for long periods usually have larger concentrations of methylmercury than fish that feed on plants. For example, shark and swordfish normally contain the highest levels of mercury out of all ocean fish. Scientists have an ongoing debate about the value of fish in the diet versus any risk from increased exposure of pregnant women to methylmercury that may be in the fish. The safety of most fish sold commercially in the United States is regulated by the FDA. These fish pose no health risk to those who purchase and eat them. Only fish or wildlife containing relatively high

levels of methylmercury are of concern, and these are discussed in Section 1.7 of this toxicological profile.

1.7 HOW CAN FAMILIES REDUCE THE RISK OF EXPOSURE TO MERCURY?

If your doctor finds that you have been exposed to significant amounts of mercury, ask whether your children might also be exposed. Your doctor might need to ask your state health department to investigate.

Children may be exposed to metallic mercury if they play with it. Metallic mercury is a heavy, shiny, silver liquid. When metallic mercury is spilled, it forms little balls or beads. Children are sometimes exposed to metallic mercury when they find it in abandoned warehouses or closed factories, and then play with it or pass it around to friends. Children have also taken metallic mercury from school chemistry and physics labs. Broken thermometers and some electrical switches are other sources of metallic mercury. Sometimes children find containers of metallic mercury that were improperly disposed of, or adults may bring home metallic mercury from work, not knowing that it is dangerous.

To protect your children from metallic mercury, teach them not to play with shiny, silver liquids. Schoolteachers (particularly science teachers) and school staff need to know about students' fascination with metallic mercury. Teachers and school staff should teach children about the dangers of getting sick from playing with mercury, and they should keep metallic mercury in a safe and secured area (such as a closed container in a locked storage room) so that children do not have access to it without the supervision of a teacher. Metallic mercury evaporates slowly, and if it is not stored in a closed container, children may breathe toxic mercury vapors.

In the past, mercurous chloride was widely used in medicinal products such as laxatives, worming medications, and teething powders. These older medicines should be properly disposed of and replaced with safer and more effective medicines. Other chemicals containing mercury, such as mercurochrome and thimerosal (sold as Merthiolate and other brands), are still used as antiseptics or as preservatives in eye drops, eye ointments, nasal sprays, and vaccines. Some skin-lightening

creams contain ammoniated mercuric chloride and mercuric iodide. These and all other mercury-containing medicines should be kept safely out of the reach of children to prevent an accidental poisoning. Nonmedicinal products, including some fungicides that contain mercury compounds and paints that contain mercuric sulfide or mercuric oxide, should also be safely stored out of the reach of children.

You should check to see if any medicines or herbal remedies that you or your child use contain mercury. Some traditional Chinese and Indian remedies for stomach disorders (for example, herbal balls) contain mercury, and if you give these remedies to your children, you may harm them. If you are pregnant or nursing a baby and you use mercury-containing ethnic or herbal remedies, you could pass some of the mercury to your unborn child or nursing infant.

If you use metallic mercury or azogue in religious practices, you may expose your children or unborn child to mercury or contaminate your home. Such practices in which mercury containing substances have traditionally been used include Santeria (a Cuban-based religion whose followers worship both African deities and Catholic saints), Voodoo (a Haitian-based set of beliefs and rituals), Palo Mayombe (a secret form of ancestor worship practiced mainly in the Caribbean), or Espiritismo (a spiritual belief system native to Puerto Rico).

Metallic mercury is used in a variety of household products and industrial items, including thermostats, fluorescent light bulbs, barometers, glass thermometers, and some blood pressure measuring devices. You must be careful when you handle and dispose of all items in the home that contain metallic mercury.

If small amounts of mercury are spilled, be very careful cleaning it up. Do not try to vacuum spilled metallic mercury. Using a vacuum cleaner to clean up the mercury causes the mercury to evaporate into the air, creating greater health risks. Trying to vacuum spilled metallic mercury also contaminates the vacuum cleaner. Also, take care not to step on the mercury and track it into other areas of the home. Metallic mercury vapors are very toxic and have no odor. Do not remain unnecessarily in that room, and try not to let metallic mercury contact your eyes, skin, or clothing. If you think you have been exposed directly to metallic mercury, wash yourself

thoroughly and discard contaminated clothing by placing them in a sealed plastic bag. Perhaps the most important thing to remember if you break a household thermometer is do not panic. The amount of mercury contained in an oral thermometer is small and does not present an immediate threat to human health. However, if it is not properly cleaned up and disposed of, it may present a health risk over time, particularly to infants, toddlers, and pregnant women.

If a thermometer breaks on a counter top or uncarpeted floor, remove children from the area. Mercury is not absorbent, so do not try to wipe or blot it up with a cloth or paper towel; that will only spread the mercury and break it up into smaller beads, making it more difficult to find and remove. Instead, clean up the beads of metallic mercury by using one sheet of paper to carefully roll them onto a second sheet of paper, or by sucking very small beads of mercury into an eye dropper. After picking up the metallic mercury in this manner, put it into a plastic bag or airtight container. The paper and eye dropper should also be bagged in a zip-lock plastic container. All plastic bags used in the cleanup should then be taken outside of the house or apartment and disposed of properly, according to instructions provided by your local health department or environmental officials. Try to ventilate the room with outside air, and close the room off from the rest of the home. Use fans (that direct the air to the outside and away from the inside of the house) for a minimum of one hour to speed the ventilation.

If a thermometer breaks and the liquid/metallic mercury spills onto a carpeted floor, try to collect the mercury beads in the manner described in the above paragraph. Depending on the cut or pile of the carpeting, however, it may not be possible to collect all of the spilled mercury. Regardless, do not vacuum. Instead, call your local (county, city, or state) health department and tell them of your situation. (You may also call the Agency for Toxic Substances and Disease Registry [ATSDR] toll-free at 1-888-42-ATSDR [1-888-422-8737] to obtain additional guidance, if local assistance cannot be obtained.)

If larger amounts of metallic mercury are found (for example, a jar of liquid mercury), it should be contained in an airtight container, and you should call your local health department for instructions on how to safely dispose of it. If the mercury is in an open container or the container does not have a lid, place a piece of plastic wrap around the top of the container to prevent

vapors from escaping; then wash your hands thoroughly. If a larger amount is spilled, leave the area and contact your local health department and fire department. Do not simply throw metallic mercury away, but instead seek professional help.

ATSDR and EPA strongly recommend against the use of metallic (liquid) mercury that is not properly enclosed in glass, as it is in thermometers. This form of mercury should not be used or stored in homes, automobiles, day-care centers, schools, offices, or other public buildings. If you notice a child with metallic mercury on his or her clothing, skin, or hair, call the fire department and let them know that the child needs to be decontaminated.

Metallic or inorganic mercury can be carried into the home from a workers' contaminated clothing and shoes. Increased exposure to mercury has been reported in children of workers who are exposed to mercury at work, and increased levels of mercury were measured in places where work clothes were stored and in some washing machines. The children most likely to be exposed to risky levels of mercury are those whose parents work in facilities that use mercury (for example, a scientific glassware manufacturing plant or a chlor-alkali chemical plant), but where no protective uniforms or footgear are used. In some reported cases in which children were exposed in this way, protective clothing was used in the workplace by the parent, but work gloves, clothes, and boots, which were contaminated with mercury, were taken home, thus exposing family members. If you have questions or concerns about exposure to mercury at work, you have a right to obtain information from your employer about your safety and health on the job without fear of punishment. The Occupational Safety and Health Administration (OSHA) requires employers to provide Material Safety Data Sheets (MSDSs) for many of the chemicals used at the workplace. Information on these sheets should include chemical names and hazardous ingredients, important properties (such as fire and explosion data), potential health effects, how you get the chemical(s) in your body, how to properly handle the materials, and what to do in an emergency. Your occupational health and safety officer at work can and should tell you whether chemicals you work with are dangerous and likely to be carried home on your clothes, body, or tools, and whether you should be showering and changing clothes before you leave work, storing your street clothes in a separate area of the workplace, or laundering your work clothes at home separately from other clothes.

Your employer is legally responsible for providing a safe workplace and should freely answer your questions about hazardous chemicals. Your OSHA-approved state occupational safety and health program or OSHA can also answer any further questions you might have, and help your employer identify and correct problems with hazardous substances. If you would like to make a formal complaint about health hazards in your workplace, your OSHA-approved state occupational safety and health program or OSHA office will listen to your complaint and inspect your workplace when necessary.

One way in which people are routinely exposed to extremely small amounts of mercury is through the gradual (but extremely slow) wearing-away process of dental amalgam fillings, which contain approximately 50% mercury. The amount of mercury to which a person might be exposed from dental amalgams would depend on the number of amalgams present and other factors. The Centers for Disease Control and Prevention (CDC) has determined that dental amalgam fillings do not pose a health risk, although they do account for some mercury exposure to those having such fillings. People who frequently grind their teeth or often chew gum can add to the small amount of mercury normally released from those fillings over time. If you are pregnant, the decision of whether to have dental amalgam or a nonmercury material used for fillings, or whether existing amalgam fillings should be repaired or replaced during pregnancy, should be made in consultation with your dentist. The practice of having all your dental amalgam fillings replaced with non-mercury filling materials just to remove the possibility of mercury exposure is not recommended by ATSDR. In fact, the removal of the mercury amalgam fillings would actually expose the patient to a greater amount of mercury for a while. Other sources of mercury may increase your overall exposure, such as the amount of fish consumed per week, especially if caught in local waters contaminated with mercury or of certain species known to be higher in mercury content (shark and swordfish), or an exposure to mercury from a nearby hazardous waste site or incinerator.

You or your children may be exposed to methylmercury when eating certain types of fish caught from contaminated waters, or when eating certain types of wildlife from mercury contaminated areas. Most states, Native American tribes, and U.S. Territories have issued fish and/or wildlife advisories to warn people about methylmercury contaminated fish and/or wildlife. Most of the

methylmercury advisories relate to specific types of freshwater or saltwater fish or shellfish, or freshwater turtles. Each state, Native American tribe, or U.S. Territory sets its own criteria for issuing fish and wildlife advisories. A fish or wildlife advisory will specify which bodies of water or hunting areas have restrictions. The advisory will tell you what types and sizes of fish or game are of concern. The advisory may completely ban eating fish or tell you to limit your meals of a certain type of fish. For example, an advisory may tell you to eat a certain type of fish no more than once a month; or an advisory may tell you only to eat certain parts of fish or game, or how to prepare it to decrease your exposure to methylmercury. The fish or wildlife advisory may be stricter to protect pregnant women, nursing women, and young children. To reduce your children's exposure to methylmercury, you should follow the instructions recommended in the fish or wildlife advisories. Information on Fish and Wildlife Advisories in your state is available from your state public health or natural resources department. Signs may also be posted in certain fishing and hunting areas with information about contaminated fish or wildlife.

FDA currently advises that pregnant women and women of childbearing age who may become pregnant limit their consumption of shark and swordfish to no more that one meal per month. This advice is given because methylmercury levels are relatively high in these fish species. Women of childbearing age are included in this advice because dietary practices immediately before the pregnancy could have a direct bearing on fetal exposure during pregnancy, particularly during the earlier months of pregnancy.

FDA further advises that persons other than pregnant women and women of childbearing age in the general population limit their regular consumption of shark and swordfish (which typically contains methylmercury around 1 ppm) to about 7 ounces per week (about one serving) to stay below the acceptable daily intake for methylmercury. For fish species with methylmercury levels averaging 0.5 ppm, regular consumption should be limited to 14 ounces per week. Recreational and subsistence fishers who eat larger amounts of fish than the general population and routinely fish the same waterbodies may have a higher exposure to methylmercury if these waters are contaminated. People who consume greater than 100 grams of fish (approximately 3.5 ounces) every day are considered high-end consumers. This is over 10 times more than the amount of fish consumed by members of the general population (6.5 g/day). No consumption advice is necessary

for the top ten seafood species that make up about 80% of the seafood sold in the United States: canned tuna, shrimp, pollock, salmon, cod, catfish, clams, flatfish, crabs, and scallops. The methylmercury in these species is generally less than 0.2 ppm, and few people eat more than the suggested weekly limit of fish (i.e., 2.2 pounds).

If you are concerned about a mercury exposure or think that you or your child are experiencing the adverse effects of mercury, you should consult with a doctor or public health official who is familiar with the health effects of mercury.

1.8 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO MERCURY?

There are reliable and accurate ways to measure mercury levels in the body. These tests all involve taking blood, urine, or hair samples, and must be performed in a doctor's office or in a health clinic. Nursing women may have their breast milk tested for mercury levels, if any of the other samples tested are found to contain significant amounts of mercury. Most of these tests, however, do not determine the form of mercury to which you were exposed. Mercury levels found in blood, urine, breast milk, or hair may be used to determine if adverse health effects are likely to occur (see Section 2.5). Mercury in urine is used to test for exposure to metallic mercury vapor and to inorganic forms of mercury. Measurement of mercury in whole blood or scalp hair is used to monitor exposure to methylmercury. Urine is not useful for determining whether exposure has occurred to methylmercury. Levels found in blood, urine, and hair may be used together to predict possible health effects that may be caused by the different forms of mercury.

Blood and urine levels are used as markers to determine whether someone has been exposed to mercury. They are used to determine whether exposure to mercury has occurred and to give a rough idea of the extent of exposure, but they do not tell exactly how much exposure has occurred. Except for methylmercury exposures, blood is considered useful if samples are taken within a few days of exposure. This is because most forms of mercury in the blood decrease by one-half every three days if exposure has been stopped. Thus, mercury levels in the blood provide

more useful information after recent exposures than after long-term exposures. Several months after an exposure, mercury levels in the blood and urine are much lower. Hair, which is considered useful only for exposures to methylmercury, can be used to show exposures that occurred many months ago, or even more than a year ago if the hair is long enough and careful testing methods are used. After short-term exposures to metallic mercury, mercury vapor can be detected in the breath, but this occurs to a significant extent only within a few days after exposure, and is not a method normally used to determine if mercury exposure has occurred. For more information on testing for mercury levels in the body, see Chapters 2 and 6.

1.9 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA). Recommendations, on the other hand, provide valuable guidelines to protect public health, but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Institute for Occupational Safety and Health (NIOSH).

Regulations and recommendations can be expressed in not-to-exceed levels in air, water, soil, or food that are usually based on levels that affect animals; then they are adjusted to help protect people. Sometimes these not-to-exceed levels differ among federal organizations because of different exposure times (an 8-hour workday or a 24-hour day), the use of different animal studies, or other factors.

Recommendations and regulations are also periodically updated as more information becomes available. For the most current information, check with the federal agency or organization that provides it for the substance in which you are interested. Some regulations and recommendations for mercury include the following:

EPA and FDA have set a limit of 2 parts inorganic mercury per billion (ppb) parts of water in drinking water. EPA is in the process of revising the Water Quality Criteria for mercury. EPA currently recommends that the level of inorganic mercury in rivers, lakes, and streams be no more than 144 parts mercury per trillion (ppt) parts of water to protect human health (1 ppt is a thousand times less than 1 part per billion, or ppb). EPA has determined that a daily exposure (for an adult of average weight) to inorganic mercury in drinking water at a level up to 2 ppb is not likely to cause any significant adverse health effects. FDA has set a maximum permissible level of 1 part of methylmercury in a million parts (ppm) of seafood products sold through interstate commerce (1 ppm is a thousand times more than 1 ppb). FDA may seize shipments of fish and shellfish containing more than 1 ppm of methylmercury, and may seize treated seed grain containing more than 1 ppm of mercury.

OSHA regulates levels of mercury in the workplace. It has set limits of 0.1 milligrams of mercury per cubic meter of air (mg/m^3)for organic mercury and 0.05 mg/m^3 for metallic mercury vapor in workplace air to protect workers during an 8-hour shift and a 40-hour work week. NIOSH recommends that the amount of metallic mercury vapor in workplace air be limited to an average level of 0.05 mg/m^3 during a 10-hour work shift.

1.10 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop E-29
Atlanta, GA 30333

* Information line and technical assistance

Phone: 1-888-42-ATSDR (1-888-422-8737)
Fax: (404) 639- 6315 or -6324

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

* To order toxicological profiles, contact

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22161
Phone: (800) 553-6847 or (703) 605-6000

2. HEALTH EFFECTS

2.1 INTRODUCTION

The primary purpose of this chapter is to provide public health officials, physicians, toxicologists, and other interested individuals and groups with an overall perspective of the toxicology of mercury. It contains descriptions and evaluations of toxicological studies and epidemiological investigations and provides conclusions, where possible, on the relevance of toxicity and toxicokinetic data to public health.

A glossary and list of acronyms, abbreviations, and symbols can be found at the end of this profile.

Mercury is a metal element that occurs naturally in the environment. Metallic or elemental mercury (Hg^0) is the main form of mercury released into the air by natural processes. Mercury bound to other chemicals may have valence states of either +1 (Hg^{+1}) or +2 (Hg^{+2}). Mercury with a valence state of +1 is referred to as mercurous mercury, and mercury with a valence state of +2 is referred to as mercuric mercury. Many inorganic and organic compounds of mercury can be formed from the mercuric (divalent) cation (Hg^{+2}). For information on the physical and chemical properties of mercury, refer to Chapter 3.

There are many similarities in the toxic effects of the various forms of mercury, but there are also significant differences. In the text, tables, and figures of this profile, the metallic mercury and the inorganic salts, including mercurous chloride, mercuric chloride, mercuric acetate, and mercuric sulfide, are organized under the general heading of inorganic mercury. The organic mercury compounds including methyl-mercuric chloride, dimethylmercury, and phenylmercuric acetate are addressed in this document under the heading of organic mercury. In most discussion in the text, the specific effects are attributable to a particular form, and the form is specified.

The general population is most commonly exposed to mercury primarily from two sources: (1) eating fish and marine mammals (e.g., whales, seals) that may contain some methylmercury in their tissues or (2) from the release of elemental mercury from the dental amalgam used in fillings. It is not known how much of the elemental mercury released from dental amalgam is inhaled as a mercury vapor, how much is breathed out, how much is swallowed in a liquid form, or how much is converted into a mercuric salt that is either swallowed of directly absorbed into the oral mucosa. Exposure to mercury, however, does not necessarily mean that adverse health effects will result. Health effects depend upon the amount of exposure, the form

of mercury, and the route of exposure. Each form and route leads to different effects, and these are discussed in detail in this chapter. The levels of mercury that the general population are exposed to from either fish or dental amalgam are discussed in Chapter 5. Hazard assessments combine the information in Chapter 5 on exposure levels with the dose-response information in this chapter to develop an estimate of the potential for adverse health effects from any given exposure.

In the environment, inorganic mercury can be methylated by microorganisms to methylmercury. Methylmercury will accumulate in the tissues of organisms. The animals at the top of the food chain tend to accumulate the most methylmercury in their bodies. Any source of mercury release to the environment may, therefore, lead to increased levels of methylmercury in tissues of large fish and mammals. Occupational exposures are primarily to metallic mercury vapor. Accidental exposures to mercury are more common than accidental exposures to many hazardous substances, because liquid mercury is shiny and interesting, and because liquid mercury has been used in many electrical and mechanical devices. Accidental exposures, even to small amounts of mercury, may be harmful. Liquid mercury is poorly absorbed by the skin and from the intestines, but vapors that are released from liquid mercury are readily absorbed through the lungs and are very harmful when inhaled. The text in this chapter provides considerable detail on a number of accidental exposures to all forms of mercury. This information is intended to inform the reader and help prevent accidental exposures in the future.

The literature on the health effects of mercury is extensive. However, the human and animal data are generally limited to inhalation exposure to metallic mercury vapors and oral exposure to inorganic and organic mercury compounds. There is limited dermal exposure information on adverse effects from ointments and creams that contain inorganic mercury compounds.

Once absorbed, metallic and inorganic mercury enter an oxidation-reduction cycle. Metallic mercury is oxidized to the divalent inorganic cation in the red blood cells and lungs of humans and animals. Evidence from animal studies suggests that the liver is an additional site of oxidation. Absorbed divalent cation from exposure to mercuric compounds can, in turn, be reduced to the metallic or monovalent form and released as exhaled metallic mercury vapor. In the presence of protein sulfhydryl groups, mercurous mercury (Hg^{+}) disproportionates to one divalent cation (Hg^{+2}) and one molecule at the zero oxidation state (Hg^{0}). The conversion of methylmercury or phenylmercury into divalent inorganic mercury can probably occur soon after absorption, also feeding into the oxidation-reduction pathway.

This profile contains a discussion of acrodynia under Relevance to Public Health (Section 2.5). Acrodynia is an idiosyncratic hypersensitivity response from exposure to mercury and is characterized by certain cardiovascular, dermal, and neurological effects, among others. In the section on health effects by route of exposure, the relevant symptoms are discussed under the appropriate headings without reference to the syndrome. This occurs, in part, because there is some overlap between symptoms characteristic of acrodynia and those seen in persons who are not hypersensitive and, in part, because not every report of a study in which the symptoms were observed states whether the authors considered the affected person to have suffered from acrodynia.

This profile also contains a general discussion of the human exposures to mercury associated with dental amalgam. This discussion is at the end of the Relevance to Public Health Section 2.5, under the heading More on Health Effects and Dental Amalgam.

2.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

To help public health professionals and others address the needs of persons living or working near hazardous waste sites, the information in this section is organized first by route of exposure — inhalation, oral, and dermal; and then by health effect — death, systemic, immunological, neurological, reproductive, developmental, genotoxic, and carcinogenic effects. These data are discussed in terms of three exposure periods — acute (14 days or less), intermediate (15–364 days), and chronic (365 days or more).

Levels of significant exposure (LSE) for each route and duration are presented in Tables 2-1, 2-2, and 2-3 and illustrated in Figures 2-1, 2-2, and 2-3. The points in the figures showing no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELs) reflect the actual doses (levels of exposure) used in the studies. LOAELs have been classified into "less serious" or "serious" effects. "Serious" effects are those that evoke failure in a biological system and can lead to morbidity or mortality (e.g., acute respiratory distress or death). "Less serious" effects are those that are not expected to cause significant dysfunction or death, or those whose significance to the organism is not entirely clear. ATSDR acknowledges that a considerable amount of judgment may be required in establishing whether an end point should be classified as a NOAEL, "less serious" LOAEL, or "serious" LOAEL, and that in some cases, there will be insufficient data to decide whether the effect is indicative of significant dysfunction. However, the Agency has established guidelines and policies that are used to classify these end-points. ATSDR believes that there is sufficient merit in this approach to warrant an attempt at distinguishing between "less

serious" and "serious" effects. The distinction between "less serious" effects and "serious" effects is considered to be important because it helps the users of the profiles to identify levels of exposure at which major health effects start to appear. LOAELs or NOAELs should also help in determining whether or not the effects vary with dose and/or duration, and place into perspective the possible significance of these effects to human health.

The significance of the exposure levels shown in the Level of Significant Exposure (LSE) tables and figures may differ depending on the user's perspective. Public health officials and others concerned with appropriate actions to take at hazardous waste sites may want information on levels of exposure associated with more subtle effects in humans or animals (LOAELs) or exposure levels below which no adverse effects (NOAELs) have been observed. Estimates of levels posing minimal risk to humans (Minimal Risk Levels or MRLs) may be of interest to health professionals and citizens alike.

Levels of exposure associated with carcinogenic effects (Cancer Effect Levels, CELs) of mercury are indicated in Tables 2-2 and 2-3 and Figures 2-2 and 2-3. Cancer effects could occur at lower exposure levels; however, a range for the upper bound of estimated excess risks (ranging from a risk of 1 in 10,000 to 1 in 10,000,000 [10^{-4} to 10^{-7}]) has not been developed by EPA.

Estimates of human Minimal Risk Levels (or MRLs) have been made for mercury. An MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (noncarcinogenic) over a specified duration of exposure. Although the term, MRL, may seem to imply a slight level of risk, MRLs are, in fact, considered to represent safe levels of exposure for all populations, including sensitive subgroups. MRLs are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration within a given route of exposure. MRLs are based on noncancerous health effects only and do not consider carcinogenic effects. MRLs can be derived for acute, intermediate, and chronic duration exposures for inhalation and oral routes. Appropriate methodology does not exist to develop MRLs for dermal exposure.

Although methods have been established to derive these levels (Barnes and Dourson 1988; EPA 1990a), uncertainties are associated with these techniques. Furthermore, ATSDR acknowledges additional uncertainties inherent in the application of the procedures to derive less than lifetime MRLs. As an example, acute inhalation MRLs may not be protective for health effects that are delayed in development or are acquired following repeated acute insults, such as hypersensitivity reactions, asthma, or chronic

bronchitis. As these kinds of health effects data become available and methods to assess levels of significant human exposure improve, these MRLs may be revised.

2.2.1 Inhalation Exposure

Most of the studies on inhalation exposure concern exposure to metallic mercury vapor. For this reason, the term “metallic mercury” will be used in this section instead of “inorganic mercury.” Other forms of inorganic mercury do not pose a risk by the inhalation pathway. Inhalation of sufficient levels of metallic mercury vapor has been associated with systemic toxicity in both humans and animals. The major target organs of metallic mercury-induced toxicity are the kidneys and the central nervous system. At high-exposure levels, respiratory, cardiovascular, and gastrointestinal effects also occur. Some metallic mercury vapor may condense (Milne et al. 1970), or in the case of vapors from dental amalgam, may dissolve in saliva and be ingested (WHO 1991). Condensed droplets are more likely to be ingested than inhaled (resulting in a lower absorbed dose than would be expected for a given concentration in air). Mercury vapor concentrations in the general work environment may also be lower than those in the micro-environment immediately surrounding workers (Bell et al. 1973; Stopford et al. 1978); therefore, estimates of air mercury values in occupational studies should be carefully evaluated for bias towards a level that may be lower than actual exposure levels.

No studies were located concerning effect levels following inhalation exposure to inorganic salts of mercury (e.g., mercuric or mercurous salts, oxides). Also, much of the information located regarding effects of metallic mercury vapors or volatile organic compounds (VOCs) comes from studies with significant limitations. Information on inhalation exposure to organic mercury compounds (e.g., alkyl mercury compounds) in humans is limited to case reports and includes only qualitative data on gastro-intestinal, renal, muscular, and neurological effects. In many cases, it is difficult to determine whether effects observed in exposed persons were directly attributable to mercury exposure. In addition, a great deal of the information on effects associated with inhalation exposure to metallic mercury vapor comes from studies conducted several decades ago, when methods for determining exposure levels were less precise than current methods.

2.2.1.1 Death

Metallic Mercury. Several studies have reported death in humans following accidental acute-duration exposure to high, but unspecified, concentrations of metallic mercury vapor (Campbell 1948; Kanluen and Gottlieb 1991; Matthes et al. 1958; Rowens et al. 1991; Soni et al. 1992; Taueg et al. 1992; Teng and Brennan 1959; Tennant et al. 1961). Death in all cases was attributed to respiratory failure. In all of these cases, high levels of mercury vapors were generated by volatilizing metallic mercury by heating.

Available animal data on death from exposure to metallic mercury vapors were also limited to acute-duration exposures (Ashe et al. 1953; Christensen et al. 1937; Livardjani et al. 1991b). Rats, guinea pigs, and mice died from severe pulmonary edema following a 24–48-hour exposure to an unspecified concentration of metallic mercury vapor resulting from spillage of mercury droplets on the floor of a static exposure chamber (Christensen et al. 1937). Exposure of rats to 27 mg/m^3 of elemental mercury vapors for 2 hours, followed by observation for 15 days, resulted in substantial mortality (20 of 32 rats died prior to their scheduled sacrifice) (Livardjani et al. 1991b). Rabbits appeared to be less sensitive, with death occurring in 1 of 2 rabbits exposed to 28.8 mg/m^3 metallic mercury for 30 hours and no deaths in rabbits exposed to the same concentration for 20 hours or less (Ashe et al. 1953).

All reliable LOAEL values for death following exposure to inorganic mercury in each species and duration category are recorded in Table 2-1 and plotted in Figure 2-1.

Organic Mercury. Case studies of occupational exposure to alkyl mercury compounds have reported deaths in humans following inhalation exposure to organic mercury vapors. The cause of death was not reported, but most subjects died after developing profound neurotoxicity (Hill 1943; Hook et al. 1954). Exposure to diethylmercury vapor (estimated exposure level = 1–1.1 mg/m^3) for 4–5 months resulted in the death of 2 women (Hill 1943). The cause of death was not reported; however, the symptoms experienced by the women were consistent with mercury toxicity, and autopsies revealed pronounced gastrointestinal disorder. It is unclear whether the gastrointestinal effects were directly attributable to the mercury exposure. A 41-year-old man with 3–4 years of exposure to alkyl mercury compounds used in seed dressing died within approximately 3 months after cleaning up a spill of liquid containing alkyl mercury (Hook et al. 1954). A 57-year-old male employed for 5 years treating lumber with an alkyl mercury preparation (unspecified) died soon after developing neurological toxicity (Lundgren and Swensson 1949).

A 39-year-old farmer who had treated seeds with phenylmercuric acetate for 6–7 seasons died within several months of developing severe neurological toxicity (Brown 1954).

Four rats died soon after developing severe ataxia following inhalation of unspecified concentrations of methylmercury iodide vapor for 22 days (Hunter et al. 1940).

2.2.1.2 Systemic Effects

The highest NOAEL values and all reliable LOAEL values for systemic effects in each species and duration category are recorded in Table 2-1 and plotted in Figure 2-1.

Respiratory Effects

Metallic Mercury. In humans, respiratory symptoms are a prominent effect of acute-duration high-level exposure to metallic mercury vapors. The most commonly reported symptoms include cough, dyspnea, and tightness or burning pains in the chest (Bluhm et al. 1992a; Gore and Harding 1987; Haddad and Sternberg 1963; Hallee 1969; Kanluen and Gottlieb 1991; King 1954; Lilis et al. 1985; Matthes et al. 1958; McFarland and Reigel 1978; Milne et al. 1970; Rowens et al. 1991; Snodgrass et al. 1981; Soni et al. 1992; Taueg et al. 1992; Teng and Brennan 1959; Tennant et al. 1961). X-ray analyses of the lungs have primarily shown diffuse infiltrates or pneumonitis (Bluhm et al. 1992a; Garnier et al. 1981; Gore and Harding 1987; Hallee 1969; King 1954; Soni et al. 1992; Tennant et al. 1961). Pulmonary function may also be impaired. Airway obstruction, restriction, hyperinflation (Snodgrass et al. 1981), and decreased vital capacity (Lilis et al. 1985; McFarland and Reigel 1978) have been reported. The decreased vital capacity observed by Lilis et al. (1985) persisted for 11 months after exposure. In the more severe cases, respiratory distress, pulmonary edema (alveolar and interstitial), lobar pneumonia, fibrosis, and desquamation of the bronchiolar epithelium have been observed. The ensuing bronchiolar obstruction by mucus and fluid results in alveolar dilation, emphysema, pneumothorax, and possibly death (Campbell 1948; Gore and Harding 1987; Jaffe et al. 1983; Kanluen and Gottlieb 1991; Matthes et al. 1958; Taueg et al. 1992; Teng and Brennan 1959; Tennant et al. 1961).

Little information is available regarding exposure levels resulting in the above symptoms. However, workers accidentally exposed to mercury vapors at an estimated concentration of up to 44.3 mg/m^3 for 4–8 hours exhibited chest pains, dyspnea, cough, hemoptysis, impairment of pulmonary function (i.e.,

reduced vital capacity), diffuse pulmonary infiltrates, and evidence of interstitial pneumonitis (McFarland and Reigel 1978).

Very little information was located regarding respiratory effects associated with intermediate-duration exposures. However, two studies noted chronic coughs in subjects exposed to metallic mercury vapor for several weeks (Schwartz et al. 1992; Sexton et al. 1976). No respiratory symptoms and no abnormalities were noted upon examining chest X-rays or the results of pulmonary function tests in a group of chloralkali workers exposed for an average of >6 years to levels of mercury ranging from near 0 to 0.27 mg/m^3 (85% of the group was exposed at or below 0.1 mg/m^3) (Smith et al. 1970).

Respiratory effects in animals have been observed following acute inhalation exposure of metallic mercury vapors. Rats exposed to 27 mg/m^3 of elemental mercury vapors for 2 hours then observed for 15 days displayed dyspnea and death due to asphyxiation (Livardjani et al. 1991b). Respiratory tract lesions included lung edema, necrosis of the alveolar epithelium and hyaline membranes, and occasional lung fibrosis.

Exposure to 28.8 mg/m^3 of mercury vapor lasting from 1 to 20 hours produced effects ranging from mild to moderate pathological changes (unspecified) (Ashe et al. 1953). For exposures lasting 30 hours, marked cellular degeneration and some necrosis were observed in the lungs of 1 rabbit. Less severe respiratory changes (unspecified mild-to-moderate pathological changes) were reported in rabbits following exposure to metallic mercury vapor at 6 mg/m^3 for 7 hours a day, 5 days a week for 1–11 weeks (Ashe et al. 1953). The usefulness of these results is limited because the study did not specify the pathological changes nor distinguish between primary and secondary effects (i.e., pathological changes secondary to induced shock).

Congested lungs were observed in rats exposed to 1 mg/m^3 metallic mercury vapors for 100 hours continuously per week for 6 weeks (Gage 1961). In rats exposed to 3 mg/m^3 mercury vapor for only 3 hours a day, 5 days a week for 12–42 weeks, pathological examination revealed no significant changes in the respiratory system (Kishi et al. 1978). The potential for oral exposure was not quantified in these studies; however, it is likely that most of the exposure was via inhalation.

Organic Mercury. Dyspnea, respiratory depression, and respirations frequently obstructed by mucus were observed in a farmer who had treated grain with phenylmercuric acetate for several seasons (Brown 1954). An autopsy revealed purulent bronchopneumonia. It is unclear whether the respiratory effects were direct

effects of the phenylmercuric acetate or secondary to the severe neurotoxicity also seen in this subject. A case study reported that no respiratory effects were observed in four men inhaling unspecified concentrations of methylmercury for several months (Hunter et al. 1940). Both of these studies are limited because exposure levels were unknown.

No studies were located regarding respiratory effects in animals after inhalation exposure to organic mercury.

Cardiovascular Effects

Metallic Mercury. Increases in heart rate and blood pressure have been reported following inhalation exposure to metallic mercury in humans. Acute inhalation exposure to high concentrations of metallic mercury vapor generated by heating metallic mercury resulted in increased blood pressure (Haddad and Sternberg 1963; Hallee 1969; Snodgrass et al. 1981) and heart rate/palpitations (Bluhm et al. 1992a; Haddad and Sternberg 1963; Hallee 1969; Jaffe et al. 1983; Snodgrass et al. 1981; Soni et al. 1992; Teng and Brennan 1959). In one of these cases, the increase in heart rate was characterized as a sinus tachycardia (Soni et al. 1992). Exposures of longer durations due to spills or occupational exposures have also been reported to result in increased blood pressure (Fagala and Wigg 1992; Foulds et al. 1987; Friberg et al. 1953; Karpathios et al. 1991; Taueg et al. 1992) and increased heart rate (Fagala and Wigg 1992; Foulds et al. 1987). A single case report was located regarding cardiovascular effects resulting from inhalation of mercury vapors released from a paint that contained a high level of phenylmercuric acetate (Aronow et al. 1990). The affected child was diagnosed with acrodynia and exhibited a rapid heart beat and hypertension.

Chronic-duration occupational exposures, however, have given mixed results regarding effects on blood pressure and heart rate. Two studies of workers exposed to relatively low levels of mercury (near 0–0.27 mg/m^3 in one study and an average of 0.075 mg/m^3 in the other) for an average of greater than 6 or 7 years showed no effects on blood pressure or electrocardiography (Schuckmann 1979; Smith et al. 1970). In contrast, workers exposed to an estimated 0.03 mg/m^3 of mercury vapor (estimate based on blood levels) for at least 5 years reported an increased incidence of palpitations, and cardiovascular reflex responses were slightly reduced compared to unexposed matched controls (Piikivi 1989). Also, workers in a thermometer plant had a high incidence of hypertension (5 of 9 workers) (Vroom and Greer 1972). A morbidity and mortality study of chloralkali workers showed an increased likelihood of death due to

ischemic heart and cerebrovascular disease (Barregard et al. 1990). These studies are limited, however, because exposure to other chemicals may have contributed to the effects observed, exposure levels may have been estimated from only a few actual determinations, and other risk factors were not consistently considered.

Significant increases in systolic blood pressure and diastolic blood pressure were found in volunteers with dental amalgam containing mercury when compared to a control group (matched for age and sex) that had no amalgam fillings (Siblerud 1990). However, the length of time that the individuals had the dental amalgams was not reported. Furthermore, the blood pressure levels of the amalgam group were closer than those of the nonamalgam group to "normal" blood pressure levels reported for the general population. The authors suggested that the populations from which such normal values are drawn are likely to include many people with amalgam dental fillings, but without additional data to determine which control group would best represent “normal,” these results have limited use.

In animals, cardiovascular effects were noted following inhalation exposure to mercury vapor. Marked cellular degeneration with some necrosis of heart tissue was observed in rabbits following acute intermittent exposure to 28.8 mg/m^3 metallic mercury vapor for periods ranging from 4 to 30 hours (Ashe et al. 1953). Mild-to-moderate pathological changes (unspecified) were seen for 1–4-hour exposures. Exposures to lower concentrations (0.86–6 mg/m^3) of mercury vapor for periods ranging from 2 to 12 weeks also resulted in mild-to-moderate pathological changes (unspecified) in the hearts of rabbits. The usefulness of these results is limited because the study did not specify the pathological changes nor distinguish between primary and secondary effects (i.e., pathological changes secondary to induced shock).

Organic Mercury. Only two case histories were located regarding cardiovascular effects in persons exposed by inhalation to organic mercury compounds. No cardiovascular effects were reported in four men hospitalized for neurological symptoms after inhaling an unspecified concentration of methylmercury dust for at least several months (Hunter et al. 1940). Elevated blood pressure was reported in two men exposed occupationally to methylmercury compounds (dose not known) (Hook et al. 1954).

No studies were located regarding cardiovascular effects in animals after inhalation exposure to organic mercury.

Gastrointestinal Effects

Metallic Mercury. Many instances of gastrointestinal effects have been reported in humans following acute inhalation exposure to metallic mercury vapor. A classical sign of mercury intoxication is stomatitis (inflammation of the oral mucosa). Accordingly, a number of case studies have reported stomatitis after acute-duration exposure to high concentrations of metallic mercury vapors (Bluhm et al. 1992a; Garnier et al. 1981; Haddad and Sternberg 1963; Snodgrass et al. 1981; Tennant et al. 1961). Occasionally, the stomatitis was accompanied by excessive salivation (Hallee 1969; Karpathios et al. 1991) or difficulty swallowing (Campbell 1948). Other gastrointestinal effects observed after acute-duration exposure to high levels of mercury include abdominal pains (Bluhm et al. 1992a; Campbell 1948; Haddad and Sternberg 1963; Milne et al. 1970; Teng and Brennan 1959), nausea and/or vomiting (Haddad and Sternberg 1963; Hallee 1969; Kanluen and Gottlieb 1991; Lilis et al. 1985; Milne et al. 1970; Rowens et al. 1991; Snodgrass et al. 1981; Soni et al. 1992; Taueg et al. 1992), and diarrhea (Bluhm et al. 1992a; Kanluen and Gottlieb 1991; Rowens et al. 1991; Taueg et al. 1992; Teng and Brennan 1959). The autopsy of a young child who was intoxicated with mercury vapor and died of pulmonary edema revealed a grayish, necrotic mucosa of the stomach and duodenum (Campbell 1948).

Intermediate-duration exposures to mercury spills have also resulted in similar gastrointestinal effects. A case study reported that teenage girls exhibited anorexia, intermittent abdominal cramps, mild diarrhea, painful mouth, and bleeding gingiva 2 weeks after a spill of metallic mercury in their home (on carpet) resulted in the release of metallic mercury vapor (Sexton et al. 1976). Air levels in the home were measured 6 months after the initial spill and ranged from 0.02 to 1 mg Hg/m^3, depending upon the degree of ventilation and proximity to the spill. Fagala and Wigg (1992) reported a case of colicky abdominal pain and diarrhea in a 12-year-old girl exposed to mercury vapors for approximately 6 months after a spill in her home.

Limited information was located regarding gastrointestinal effects in persons who are chronically exposed to elemental mercury vapors. Stomatitis was observed in 22 of 72 workers exposed to mercury vapors in the manufacture of thermometers in the 1940s (Bucknell et al. 1993). Drooling, sore gums, ulcerations of the oral mucosa, and/or diarrhea were observed in 5 of 9 workers in a thermometer-manufacturing plant (Vroom and Greer 1972). A correlation was also observed between mercury exposure levels and unspecified oropharyngeal symptoms in workers from a chloralkali plant (Smith et al. 1970).

Two animal studies assessed the gastrointestinal effects from mercury vapor exposure. In rabbits, effects ranging from mild pathological changes to marked cellular degeneration and some necrosis of the colon were observed following exposure to 28.8 mg/m^3 mercury vapor for 4–30 hours (Ashe et al. 1953). A single exposure to 28.8 mg/m^3 for 1–2 hours or multiple exposures of 6 mg/m^3 for 7 hours a day, 5 days a week for up to 11 weeks resulted in either no changes or mild pathological changes. The usefulness of these results is limited because the study did not specify the pathological changes nor distinguish between primary and secondary effects (i.e., pathological changes secondary to induced shock).

Organic Mercury. Gastrointestinal effects were reported in several case studies of humans exposed to organomercurial compounds. A 39-year-old farmer who had dressed his seeds for several seasons with phenylmercuric acetate exhibited a swollen mouth, reddened and tender gums, carious teeth, a thin blue line at the gums, and an infected and swollen posterior pharyngeal wall (Brown 1954). Similarly, two women who died following 3–5 months of occupational exposure to diethylmercury vapors exhibited inflammation of the mouth and gums, excessive salivation, and unspecified gastrointestinal disorders (Hill 1943). Marked salivation was observed in one man and nausea was observed in another occupationally exposed to alkyl-mercury compounds used for dressing seeds (Hook et al. 1954). Gastrointestinal effects were not, however, observed in four men after inhalation of dust containing methylmercury for several months (Hunter et al. 1940).

No studies were located regarding gastrointestinal effects in animals after inhalation exposure to organic mercury.

Hematological Effects

Metallic Mercury. Initial exposure to high concentrations of elemental mercury vapors produces a syndrome similar to "metal fume fever," which is characterized by fatigue, fever, chills, and elevated leukocyte count. Evidence of moderate-to-high leukocytosis with neutrophilia was reported following acute inhalation exposure to metallic mercury vapor (Campbell 1948; Haddad and Sternberg 1963; Hallee 1969; Jaffe et al. 1983; Lilis et al. 1985; Matthes et al. 1958; Rowens et al. 1991).

Similarly, an elevated white cell count was observed in a 12-year-old girl with a 6-month exposure to mercury vapors from a spill of metallic mercury in her home (Fagala and Wigg 1992). Thrombocytopenia and frequent nosebleeds were reported in two of four family members exposed to mercury vapors in their

home as a result of a spill of metallic mercury (Schwartz et al. 1992). The authors considered this to be a unique reaction to the mercury exposure.

In volunteers with dental amalgam, significantly decreased hemoglobin and hematocrit and increased mean corpuscular hemoglobin concentrations were found compared to controls without dental amalgams (Siblerud 1990). δ-Aminolevulinic acid dehydratase activity in erythrocytes was decreased in workers exposed to elemental mercury in the manufacture of tungsten rods (Wada et al. 1969). The decreases correlated with increases in urinary mercury. The estimated exposure level to mercury in the plant was slightly less than 0.1 mg/m^3. In workers exposed to 0.106–0.783 mg/m^3 mercury vapor, there was a significant increase in α2-macroglobulin and ceruloplasmin (an α-globulin protein active in the storage and transport of copper) compared to unexposed workers (Bencko et al. 1990).

No studies were located regarding hematological effects in animals after inhalation exposure to inorganic mercury.

Organic Mercury. No studies were located regarding hematological effects in humans or animals after inhalation exposure to organic mercury.

Musculoskeletal Effects

Metallic Mercury. A number of studies have reported increases in tremors, muscle fasciculations, myoclonus, or muscle pains after acute (Adams et al. 1983; Bluhm et al. 1992a; Karpathios et al. 1991; McFarland and Reigel 1978), intermediate (Aronow et al. 1990; Barber 1978; Sexton et al. 1976; Taueg et al. 1992), or chronic (Albers et al. 1982, 1988; Bidstrup et al. 1951; Chaffin et al. 1973; Chapman et al. 1990; Fawer et al. 1983; Smith et al. 1970; Verberk et al. 1986; Vroom and Greer 1972; Williamson et al. 1982) exposure to metallic mercury vapor. These effects are probably neurally mediated and are discussed more fully in Section 2.2.1.4.

No studies were located regarding musculoskeletal effects in animals after inhalation exposure to metallic mercury.

Organic Mercury. Exposure to unspecified alkyl mercury compounds has caused muscular effects (e.g., muscle fasciculations, absence of deep reflexes in arms, Babinski reflex) (Brown 1954; Hook et al. 1954;

Hunter et al. 1940). These effects may have been secondary to neurological changes and are discussed more fully in Section 2.2.1.4.

No studies were located regarding musculoskeletal effects in animals after inhalation exposure to organic mercury.

Hepatic Effects

Metallic Mercury. A case study described the acute poisoning of a young child who was exposed to mercury vapors that were produced from heating an unknown quantity of mercury (Jaffe et al. 1983). Hepatocellular effects were characterized by biochemical changes (e.g., elevated serum alanine aminotransferase [ALT]), ornithine carbamyl transferase, and serum bilirubin levels) and evidence of a decrease in the synthesis of hepatic coagulation factors. Similarly, hepatomegaly and central lobular vacuolation were observed in a man who died following acute-duration exposure to high levels of elemental mercury vapors (Kanluen and Gottlieb 1991; Rowens et al. 1991).

Serious liver effects have been noted in animals at high exposure concentrations. Acute inhalation exposure of rabbits to metallic mercury vapor concentrations of 28.8 mg/m^3 for 6–30 hours resulted in effects ranging from moderate pathological changes (unspecified) to severe liver necrosis (Ashe et al. 1953). These effects were less severe (mild effects to degeneration) at shorter exposure durations and following exposure to 6 mg/m^3 mercury vapors for 7 hours a day, 5 days a week for 1–5 weeks (Ashe et al. 1953). Effects ranging from moderate pathological changes to marked cellular degeneration and some necrosis were seen at mercury concentrations of 6 mg/m^3 for 7 hours a day, 5 days a week for 6–11 weeks (Ashe et al. 1953). No hepatic changes were present in a pathological examination of the livers of rats intermittently exposed to 3 mg/m^3 mercury vapor for only 3 hours a day, 5 days a week for 12–42 weeks (Kishi et al. 1978). The studies by Ashe et al. (1953) and Kishi et al. (1978) were deficient in quantitative data, and used a small number of animals. However, available human and animal data suggest that metallic mercury vapors can cause liver effects following acute exposures.

Organic Mercury. Midzonal necrosis in the liver was observed during the autopsy of a farmer who died after treating grain with phenylmercuric acetate for several seasons (Brown 1954). No conclusions can be drawn from this study, however, because other factors may have contributed to the hepatic effects in this subject.

No studies were located regarding hepatic effects in animals after inhalation exposure to organic mercury.

Renal Effects

Metallic Mercury. The kidney is a sensitive target organ of toxicity following inhalation exposure to metallic mercury. This sensitivity may be, in part, because of the relatively high accumulation of mercury in the kidneys. Acute high-concentration inhalation exposure in humans has resulted in effects ranging from mild transient proteinuria or s syndrome has been reported light changes in urinary acid excretion (Bluhm et al. 1992b; Soni et al. 1992); to frank proteinuria, hematuria, and/oliguria (Campbell 1948; Hallee 1969; Snodgrass et al. 1981); to acute renal failure with degeneration or necrosis of the proximal convoluted tubules (Campbell 1948; Jaffe et al. 1983; Kanluen and Gottlieb 1991; Rowens et al. 1991). Actual exposure concentrations are unknown in these cases, but urinary mercury excretion as high as 59–193 μg/hour has been reported (Bluhm et al. 1992b).

A nephrotic in two case studies of intermediate-duration exposure (Agner and Jans 1978; Friberg et al. 1953). In one report, the exposure was to a spill in the home (Agner and Jans 1978); in the other, the exposure was occupational (Friberg et al. 1953). The nephrotic syndrome was characterized by edema and proteinuria with albumin and hyaline casts in the urine. These changes usually abated within a few months following termination of exposure. Among a group of 10 patients who reported adverse effects associated with dental amalgams (the route of exposure in dental amalgams is probably a mixture of inhalation exposure to mercury vapor released from the amalgams, absorption of the vapor through the oral mucosa, and ingestion), a decrease in the ability to concentrate the urine and elevated urinary albumin were observed (Anneroth et al. 1992). Removal of one amalgam resulted in a significant decrease in urinary albumin (it is unknown whether other amalgams remained). In a study of renal function in 10 healthy volunteers having an average of 18 amalgam-filled tooth surfaces both before and after amalgam removal (Sandborgh-Englund and Nygren 1996), no signs of renal toxicity were found in conjunction with mercury released from the amalgam fillings. Although plasma mercury levels increased significantly one day after removal of the fillings (all removals were accomplished in one dental session), glomerular filtration rates were similar both before and after mercury exposure (amalgam removal). Blood, plasma, and urine mercury concentrations were significantly lower 60 days after amalgam removal.

The results from a number of studies show renal toxicity in workers chronically exposed to mercury vapor (Barregard et al. 1988; Bernard et al. 1987; Buchet et al. 1980; Cardenas et al. 1993; Danziger and

Possick 1973; Ehrenberg et al. 1991; Kazantzis et al. 1962; Langworth et al. 1992b; Piikivi and Ruokonen 1989; Roels et al. 1982; Stewart et al. 1977; Stonard et al. 1983; Sunderman 1978; Tubbs et al. 1982). Several of these reports have focused on workers with proteinuria (Danziger and Possick 1973; Kazantzis et al. 1962; Tubbs et al. 1982), while others have examined a variety of urinary parameters in exposed populations. Biopsies in the studies of workers with proteinuria have shown both proximal tubular and glomerular changes. In the report by Kazantzis et al. (1962), heavy albuminuria was reported to be accompanied by both proximal tubular damage and glomerulosclerosis. Examination of tissue samples from two other workers with proteinuria showed changes in the foot processes of cells associated with the glomerular basement membrane and deposition of IgG and C3 (Tubbs et al. 1982).

Comparisons of exposed populations to controls have shown a variety of changes in exposed workers, ranging from no effects (Bernard et al. 1987; Piikivi and Ruokonen 1989) to increases in urinary protein (Stewart et al. 1977), the specific gravity of the urine (Ehrenberg et al. 1991), and urinary *N*-acetyl-β-glucosaminidase (NAG) (Barregard et al. 1988; Boogaard et al. 1996; Langworth et al. 1992b). A detailed examination of markers for urinary dysfunction showed increases in urinary excretion of Tamm-Horsfall glycoprotein and tubular antigens and decreases in urinary pH and excretion of glycoamino-glycans, prostaglandin E2 and F2α, and thromboxane B2 (Cardenas et al. 1993). Several studies have also shown correlations with some of these parameters and urinary mercury content (Buchet et al. 1980; Cardenas et al. 1993; Ehrenberg et al. 1991; Langworth et al. 1992b; Roels et al. 1982; Stonard et al. 1983). Attempts to define threshold levels for effects have produced mixed results. A no-effect level of 72 μg Hg/g creatinine was determined for urinary excretion of albumin, β_2-microglobulin, or retinol binding protein (Bernard et al. 1987). However, other studies have shown increases in urinary albumin at urinary mercury levels >50 μg Hg/g creatinine (Buchet et al. 1980) and increases in urinary *N*-acetyl-β-glucosaminidase at urinary mercury levels of >50 or >100 μg Hg/g creatinine. Boogaard et al. (1996) reported that after exposure to mercury with urinary levels below the biological exposure index of 35 μg/g creatinine, a transient increase in NAG was observed, but there was no correlation with duration of exposure and that this increase was not an early indicator of developing renal dysfunction. More information on correlation between urinary mercury levels and renal toxicity can be found in Section 2.5.

Serious degenerative effects have been observed in the kidneys of animals exposed to moderate-to-high levels of metallic mercury vapors following acute- and intermediate-duration exposures (Ashe et al. 1953). Effects ranging from marked cellular degeneration to tissue destruction and widespread necrosis were observed in rabbits exposed to mercury vapor at a concentration of 28.8 mg/m^3 for 2–30 hours. Moderate

pathological changes (unspecified) were also seen for 1-hour exposures. As the duration of exposure increased to 30 hours, extensive cell necrosis in the kidneys became evident. These results and the following results are limited as to their usefulness because the pathological changes are not described.

In an intermediate-duration study, rabbits exposed to mercury vapor concentrations of 0.86 mg/m^3 for 7 hours a day, 5 days a week for 12 weeks exhibited moderate pathological kidney changes that were reversible with cessation of exposure (Ashe et al. 1953). Larger doses (6 mg/m^3) administered for 7 hours a day, 5 days a week for up to 11 weeks, produced effects that ranged from mild, unspecified, pathological changes to marked cellular degeneration and widespread necrosis (Ashe et al. 1953).

In rats, slight degenerative changes (i.e., dense deposits in tubule cells and lysosomal inclusions) in the renal tubular epithelium were evident following exposure to 3 mg/m^3 mercury vapor for 3 hours a day, 5 days a week for 12–42 weeks (Kishi et al. 1978).

Low-level chronic-duration inhalation exposures to 0.1 mg/m^3 metallic mercury vapor for 7 hours a day, 5 days a week for 72–83 weeks in rats, rabbits, and dogs produced no microscopic evidence of kidney damage (Ashe et al. 1953). Only two dogs were tested in the study.

Organic Mercury. An autopsy of a man who died after acute high-level exposure to alkyl mercury vapor revealed necrosis of the tubule epithelium, swollen granular protoplasm, and nonstainable nuclei in the kidneys (Hook et al. 1954). No studies were available on renal effects following intermediate or chronic-duration exposure to organic mercury vapors in humans.

No studies were located regarding renal effects in animals after inhalation exposure to organic mercury.

Endocrine Effects

Metallic Mercury. A 13-year-old boy exposed to mercury vapors for 2 weeks developed a thyroid enlargement with elevated triiodothyronine, and thyroxine; and low thyroid-stimulating hormone levels (Karpathios et al. 1991). Serum-free thyroxine (T4) and the ratio of free thyroxine to free 3,5,3'-triiodo-thyronine (T3) were found to be slightly, but significantly, higher in workers with the highest exposure concentrations in a study of chloralkali workers exposed an average of 10 years to metallic mercury vapor (Barregard et al. 1994a, 1994b). Further, serum-free T3 was inversely associated with cumulative mercury exposure, suggesting a possible inhibitory effect of mercury on 5'-deiodinases, which is responsible for the

conversion of T4 to the active hormone T3. In this study, serum total testosterone (but not free testosterone) was positively correlated with cumulative mercury exposure, while prolactin, thyrotrophin, and urinary cortisol concentrations were not associated with exposure. However, two other occupational studies found no relationship between mercury exposure (unspecified concentration) and endocrine function (i.e., testicular, thyroid, and pituitary) (Erfurth et al. 1990; McGregor and Mason 1991). Biochemical indices that were measured in the occupational study by McGregor and Mason (1991) to assess endocrine effects included serum testosterone, sex-hormone binding globulin, thyroid-stimulating hormone, and prolactin. Erfurth et al. (1990) measured both basal serum concentrations of thyrotropin, thyroxine, triiodothyronine, and cortisol, as well as the response to a thyrotropin challenge.

No studies were located regarding endocrine effects in animals after inhalation exposure to metallic mercury.

Organic Mercury. No studies were located regarding endocrine effects in humans or animals after inhalation exposure to organic mercury.

Dermal Effects

Metallic Mercury. Inhalation exposure of individuals to elemental mercury vapors for acute and intermediate durations has resulted in erythematous and pruritic skin rashes (Aronow et al. 1990; Bluhm et al. 1992a; Foulds et al. 1987; Karpathios et al. 1991; Schwartz et al. 1992; Sexton et al. 1976). Other dermal reactions to mercury exposure include heavy perspiration (Aronow et al. 1990; Fagala and Wigg 1992; Karpathios et al. 1991; Sexton et al. 1976) and reddened and/or peeling skin on the palms of the hands and soles of the feet (Aronow et al. 1990; Fagala and Wigg 1992; Karpathios et al. 1991).

No studies were located regarding dermal effects in animals after inhalation exposure to metallic mercury.

Organic Mercury. No studies were located regarding dermal effects in humans or animals after inhalation exposure to organic mercury.

Ocular Effects

Metallic Mercury. Ocular effects observed following acute exposure included red, burning eyes and conjunctivitis (Bluhm et al. 1992a; Sexton et al. 1976). Workers chronically exposed to mercury have also exhibited a peculiar grayish-brown or yellow haze on the outer surface of their lenses (Atkinson 1943; Bidstrup et al. 1951; Locket and Nazroo 1952). These case studies contained insufficient quantitative data for dose-response assessment.

No studies were located regarding ocular effects in animals after inhalation exposure to metallic mercury.

Organic Mercury. No studies were located regarding ocular effects in humans or animals after inhalation exposure to organic mercury.

Other Systemic Effects

Metallic Mercury. Initial exposure to high concentrations of elemental mercury vapors produces a syndrome similar to "metal fume fever," which is characterized by fatigue, fever, chills, and an elevated leukocyte count. Accordingly, several studies have reported fever and/or chills in humans after exposure to high concentrations of elemental mercury vapors (Aronow et al. 1990; Bluhm et al. 1992a; Garnier et al. 1981; Lilis et al. 1985; McFarland and Reigel 1978; Milne et al. 1970; Schwartz et al. 1992; Snodgrass et al. 1981).

Organic Mercury. No studies were located regarding other systemic effects in humans or animals after inhalation exposure to organic mercury.

2.2.1.3 Immunological and Lymphoreticular Effects

Metallic Mercury. The immune reaction in humans to mercury exposure appears to be idiosyncratic, with either increases or decreases in immune activity depending on individual genetic predisposition (see Section 2.4). Therefore, it is not surprising that several studies of workers exposed to elemental mercury vapor have failed to show consistent or marked changes in immune function parameters in large populations. For example, no effect on serum immunoglobulins (IgA, IgG, or IgM) and no increase in autoantibody titres were observed in a group of chloralkali workers exposed for an average of 13.5 years

(Langworth et al. 1992b). Similarly, no increases in antilaminin antibodies were observed in workers exposed for an average of 7.9 years (Bernard et al. 1987), and no increase in antiglomerular basement membrane antibodies or IgE was seen in workers exposed for between 1.5 and 25 years (Cardenas et al. 1993). Slight decreases in IgA and IgG were observed in workers after more than 20 years of exposure to metallic mercury vapors when compared to unexposed controls (Moszczynski et al. 1990b). No significant differences in the concentrations of immunoglobulins or complement components were found in a study of 76 chloralkali workers previously exposed to mercury vapor for an average of 7.9 years (range, 1.1–36.2 years) (Ellingsen et al. 1994). No increase in the prevalence of autoantibodies was observed between the formerly exposed worker group and a control group of 53 age-matched referents. The average time elapsed since the cessation of occupational exposure was 12.3 years (range, 1–35 years).

Evidence of a human autoimmune response has been obtained in a few studies. Examination of the kidneys of two workers with proteinuria revealed granular deposition of IgG and the complement C3 in the glomeruli (Tubbs et al. 1982). Among a group of 10 patients who reported adverse effects associated with dental amalgams (the route of exposure is probably a mixture of inhalation exposure to mercury vapor released from the amalgams and dermal exposure to the amalgams), 3 had increased antiglomerular basement membrane antibodies and 2 had elevated antinuclear antibodies (Anneroth et al. 1992). After removal of one amalgam, there was a significant decrease in IgE (it is unknown whether other amalgams remained). Also, 1 of 89 workers examined by Langworth et al. (1992b) showed a weak reaction to antiglomerular basement membrane, and 8 of 44 workers examined by Cardenas et al. (1993) showed an abnormally high anti-DNA antibody titre. Only two studies have shown increases in immune parameters in exposed populations. Increases in IgA and IgM were observed in workers in a mercury producing plant (Bencko et al. 1990). The study is limited by a lack of information on daily dose levels, duration of employment and potential confounding factors (smoking, alcohol). An increase in anti-DNA antibodies was observed in workers from a chloralkali plant (Cardenas et al. 1993).

Other experimental evidence suggests that mercury can alter a number of parameters of the host's immune system and lead to increased susceptibility to infections, autoimmune diseases, and allergic manifestations. In workers exposed to mercury vapor concentrations of 0.024–0.09 mg/m^3 for less than 10 and up to 31 years (Moszczynski et al. 1995), the stimulation of T-lymphocytes (as manifested by an increased number of T-cells [CD3+], T-helpers [CD4+], and T-suppressors [CD8+]) was observed in peripheral blood; however, no significant effect was seen on NK-cell (CD16+) count. A positive correlation was found between the T-helper cell count and the duration of exposure ($p<0.05$). The combined stimulation of

the T-cell line and an observed decrease in the helper/suppressor ratio were suggestive of an autoimmune response.

In a mercury-producing plant, neutrophil function was found to be significantly reduced in workers with a mean exposure duration of 8 months (range, 0.5–46 months) (Perlingeiro and Queiroz 1995). In this study, both chemotactic and chemical-specific reducing activities of the neutrophils of exposed workers were found to be affected. While improved industrial hygiene practices over a 6-month period resulted in a decrease in urine mercury concentration in the workers, it did not result in the return of neutrophil migration activity to within the normal range. There was, however, no observed increase in the incidence of infections in the mercury-exposed group compared to controls. Based on their observations, Perlingeiro and Queiroz (1995) suggested that even exposures to levels of mercury considered "safe" in some industrial settings may lead to impairment of neutrophil function.

Exposure of genetically susceptible mice to mercury vapor for a period of 10 weeks resulted in an autoimmune response similar to that seen in similar mice after treatment with mercuric chloride by subcutaneous injections and in drinking water (Warfvinge et al. 1995). This response was manifested as a syndrome, which included a general stimulation of the immune system, with hyperimmunoglobulinemia, anti-nucleolar-fibrillarin autoantibodies, and glomerular disease accompanied by vascular immune complex deposits. Actual inhalation exposure times for the 0.3–1 mg Hg/m^3 exposure concentrations varied from 0.5 to 19 hours a day (5 days a week), but doses for individual exposure groups were also expressed in µg/kg/week units. The LOAEL for serum antinucleolar antibodies was determined to be an absorbed dose of 0.170 mg Hg/kg/week (from a 1.5-hour daily exposure to 0.5 mg/m^3) and the corresponding NOAEL was a calculated absorbed dose of 0.075 mg/kg/day (from a 0.5-hour daily exposure to 0.0005 mg/m^3). Higher doses were required for B-cell stimulation and for the development of immune complex deposits.

The highest NOAEL values and all reliable LOAEL values for immunological effects in each species and duration category are recorded in Table 2-1 and plotted in Figure 2-1.

Organic Mercury. No studies were located regarding immunological and lymphoreticular effects in humans or animals after inhalation exposure to organic mercury.

2.2.1.4 Neurological Effects

Metallic Mercury. The central nervous system is probably the most sensitive target organ for metallic mercury vapor exposure. Nervous system disorders following exposure to metallic mercury vapors are both consistent and pronounced. Acute-, intermediate-, and chronic-duration exposures elicit similar neurological effects. Symptoms intensify and may become irreversible as exposure duration and/or concentration increases. Most occupational studies discuss chronic-duration exposure to a time-weighted average (TWA) concentration or to a concentration range, thereby preventing the assessment of dose-response relationships within the populations studied. However, the average exposure levels for affected groups are similar in many of these studies.

In humans, several case studies have reported adverse neurological effects following acute inhalation of high concentrations of mercury vapor. A wide variety of cognitive, personality, sensory, and motor disturbances have been reported. The most prominent symptoms include tremors (initially affecting the hands and sometimes spreading to other parts of the body), emotional lability (characterized by irritability, excessive shyness, confidence loss, and nervousness), insomnia, memory loss, neuromuscular changes (weakness, muscle atrophy, muscle twitching), headaches, polyneuropathy (paresthesia, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory and motor nerve conduction velocities), and performance deficits in tests of cognitive function (Adams et al. 1983; Bluhm et al. 1992a; Hallee 1969; Jaffe et al. 1983; Karpathios et al. 1991; Lilis et al. 1985; McFarland and Reigel 1978; Snodgrass et al. 1981). A few individuals have also noted hearing loss, visual disturbances (visual field defects), and/or hallucinations (Bluhm et al. 1992a; McFarland and Reigel 1978). In a case study of exposure to a calculated metallic mercury vapor level of 44 mg/m^3 for <8 hours, workers experienced long-lasting feelings of irritability, lack of ambition, and lack of sexual desire (McFarland and Reigel 1978). Three and one-half months after exposure to high levels of mercury vapor during 2 days of an industrial liquid mercury salvaging operation, a 54-year-old man exhibited a syndrome resembling amyotrophic lateral sclerosis, characterized by slowed conduction velocities (suggestive of peripheral nerve damage). Urinary mercury levels were 100 µg/g creatinine at the time of the exam; after an additional 2 months (no treatment administered), levels dropped to less than 30 µg/g creatinine and symptoms disappeared (Adams et al. 1983). In contrast, chelation therapy (2.3 dimercaptosuccinic acid [DMSA] or – acetyl-D,L-penicillamine [NAP]) and lowering of urinary mercury levels did not result in improvement in depression, anxiety, phobias, psychotic-like behavior, interpersonal sensitivity, and hostility observed in another group of workers exposed to high concentrations of mercury vapor for up to 16 hours (Bluhm et al. 1992a).

In case reports of individuals exposed to inorganic mercury vapor for an intermediate duration, similar effects were reported (Barber 1978; Fagala and Wigg 1992; Foulds et al. 1987; Friberg et al. 1953; Sexton et al. 1976; Taueg et al. 1992). After 6 months of exposure to a spill of metallic mercury in the place where she slept, a 12-year-old girl experienced dizziness, joint pains, weakness, insomnia, numbness and tingling in her palms, decreased pinprick and vibration sensations in the lower extremities, intentional tremors, a slowing of the background rhythms on electroencephalograms, irritability, outbursts of temper, shyness, sensitivity, auditory hallucinations, and photophobia (Fagala and Wigg 1992). Similarly, a 4-year-old boy exposed for approximately 1 month to mercury vapors released from paint containing phenylmercuric acetate exhibited irritability, personality change, insomnia, headaches, weakness, and nerve dysfunction in the lower extremities (Aronow et al. 1990). This study is not discussed under organic mercury because the exposure was to metallic mercury vapors released from the paint.

Two adolescents (ages 13 and 15) who were unintentionally exposed to concentrated mercury vapors for 3 months developed a variety of more immediate- and long-term effects (Yeates and Mortensen 1994). In the 15-year-old male, the earliest symptoms included declining school performance, irritability, depression, neurobehavioral complaints, tremor, rash, hypertension, cold intolerance, diaphoresis, headaches, sleep disturbance, paresthesias, and anorexia. He was referred to a pediatric teaching hospital, where he was diagnosed with acrodynia and mercury poisoning. Before undergoing two courses of chelation therapy with 2,3-dimercaptosuccoinic acid (DMSA), his average 24-hour urine mercury and blood mercury levels were 1,314 µg/L and 23 µg/L, respectively. His 13-year-old half-sister, who was also exposed, had pretreatment average 24-hour urine mercury and blood mercury levels of 624 µg/L and 69 µg/L, respectively; her pre-treatment medical symptoms included tremor, rash, anorexia, paresthesias, and neuropsychiatric complaints (e.g., irritability, social withdrawal, and emotional lability). On hospital admission, she was diagnosed with acrodynia and underwent three courses of DMSA treatment, which were complicated by severe peripheral neuropathy, accompanied by a significant weight loss. Although the neuropathy was relatively mild at the time of initial neurological evaluation, it became progressively worse, and eventually the patient required a wheelchair and assistance eating. The neuropathy had resolved by the 1-year follow-up neuropsychological evaluation; however, despite removal from exposure, return of blood and urinary mercury to acceptable levels, and resolution of clinical signs of mercury poisoning and associated neuropsychiatric symptoms, both patients continued to show major deficits in visuoperceptual and constructional skills, nonverbal memory, and abstract reasoning.

A worker (age, mid-40s) exposed to mercury in a thermometer factory for approximately 3.5 years experienced acute, intermediate, chronic, and delayed neurological effects (White et al. 1993). During his employment, he performed a variety of functions, including sweeping mercury off floors with a vacuum cleaner or hose blower, repairing and cleaning machines, disassembling machines containing mercury, and operating a machine that crushed instruments so that he could then separate the mercury from other materials for reuse. From approximately the beginning of his employment at the factory, he experienced a number of symptoms, including blurred vision, ocular pain, rash, a strange taste in the mouth, weakness, memory loss, rage, and irrational behavior. The month following his release by the factory, his urine mercury concentration was measured at 690 µg/L, which confirmed a diagnosis of mercury poisoning. He was treated by chelation with penicillamine over a 2-month period; approximately 2 months after the completion of treatment, his urine mercury level was only 17 µg/L. Approximately 21months after termination of his employment, neurological examination revealed nystagmus on upward gaze, bilateral manual tremor, diminished sensation to pain, peripheral neuropathy, and abnormalities in nerve conduction. An magnetic resonance imaging (MRI) examination revealed mild central and cortical atrophy, with punctiform foci of T2 in both frontal regions, especially underlying the precentral gyri and in the white matter (both subcortical and gyri). The MRI data were interpreted as consistent with diffuse and focal white matter disease. Neuropsychological testing conducted during the same time period revealed problems with cognitive function, fine manual motor coordination, visuospatial analysis and organization, memory for visuospatial information, affect, and personality almost 2 years after cessation of employment at the factory.

In contrast with the long-term (perhaps permanent) effects noted in the previous study, Yang et al. (1994) reported that recovery from chronic elemental mercury intoxication may be complete when patients are removed early from the exposure environment. A 29-year-old worker in a Taiwanese lampsocket-manufacturing facility, with an initial urinary mercury concentration of 610 µg/L (in a 24-hour sample) and a blood mercury concentration of 237 µg/L (reference range, <10 µg/L), exhibited a variety of symptoms, including blurred vision, dysarthria, prominent gingivitis, tremors (usually postural and intentional), unsteady gait, and slow mental response. The TWA concentration of mercury in the air in the room where he spent most of his working time during his 5 years on the job was 0.945 mg/m^3. The worker also had a higher blood lead concentration of 450 µg/L (reference range, <20 µg/L), and lead toxicity or interactions with mercury could have occurred. The man underwent an 8-week course of chelation with D-penicillamine, which resulted in a rapid improvement in gait; a complete recovery from all symptoms occurred over a 4-month period.

A 27-year-old female, who worked primarily in a room with a TWA mercury air concentration of 0.709 g/m^3 and who had been on the job for 1.5 years, showed a variety of symptoms, including gum pain, dizziness, poor attention, bad temper, some numbness, hypersalivation, hyperhidrosis, dizziness, and fatigue. She had initial urine and blood mercury levels of 408 µg/L and 105 µg/L, respectively, but did not require chelation; the symptoms abated fully approximately 2 months following discontinuation of exposure (Yang et al. 1994).

Other chronic-duration exposures to metallic mercury vapor have resulted in tremors (which may be mild or severe depending on the degree of exposure), unsteady walking, irritability, poor concentration, short-term memory deficits, tremulous speech, blurred vision, performance decrements in psychomotor skills (e.g., finger tapping, reduced hand-eye coordination), paresthesias, decreased nerve conduction, and other signs of neurotoxicity (Albers et al. 1988; Bidstrup et al. 1951; Chaffin et al. 1973; Chang et al. 1995; Chapman et al. 1990; Fawer et al. 1983; Langolf et al. 1978; Piikivi et al. 1984; Smith et al. 1970; Sunderman 1978; Uzzell and Oler 1986; Vroom and Greer 1972; Williamson et al. 1982). The majority of studies suggest that motor system disturbances are reversible upon exposure cessation, while cognitive impairments, primarily memory deficits, may be permanent (Chaffin et al. 1973; Hanninen 1982; Miller et al. 1975).

Several studies have noted correlations between exposure level or duration and effects (e.g., memory deficits, psychomotor coordination, motor and sensory nerve conduction velocities, electromyographic abnormalities, evidence of polyneuropathy, tremor, emotional changes, reflex abnormalities, and electroencephalographic changes) (Albers et al. 1982; Iyer et al. 1976; Levine et al. 1982; Smith et al. 1983; Vroom and Greer 1972; Williamson et al. 1982). Early studies suggested that frank neurotoxicity (pronounced tremors, erethism, restriction of visual fields, difficulty seeing) was generally observed at >300 µg mercury in a 24-hour urine (Bidstrup et al. 1951) or at >0.1 mg/m^3 (Smith et al. 1970). More recent studies using sensitive tests for psychomotor skills, tremor, and peripheral nerve function suggest that adverse effects may be associated with very low exposures (see below). However, conflicting information exists regarding thresholds for neurotoxic effects.

Several reports have presented essentially negative findings at low exposure levels (0.025–0.076 mg/m^3). Chloralkali workers exposed to low air levels of mercury vapors for at least 5 years (group average, 14 years) reported an increase in memory disturbances, sleep disorders, anger, fatigue, confusion, and hand tremors compared to the controls (Piikivi and Hanninen 1989). However, tests of psychomotor

coordination and memory showed no significant deficits in the exposed group. The exposed and control groups were matched for age, sex, vocational status, education, and mean number of amalgam fillings. A group-average exposure concentration of 0.025 mg/m^3 mercury vapors was estimated from repeated analyses of blood mercury concentration (mean, 51.3 nmol/L . 10 µg/L) (see the discussion regarding these estimated exposure levels in Section 2.5). Also, no effects on tremors, bimanual coordination, color determination, or reaction time were observed in chloralkali workers with more than 7 years of exposure to low levels of mercury; ambient air levels measured for 2 years prior to testing averaged 0.076 mg/m^3 and the average blood level in the workers was 19.9 µg/L (Schuckmann 1979). Negative findings were also noted when the results of tremor frequency spectra and psychometric tests of a group of chloralkali workers exposed for an average of 13.5 years were compared to unexposed controls (Langworth et al. 1992a). The TWA exposure level was estimated to be 0.025 mg/m^3, based on measurements taken at the time of the study, and blood levels in the workers averaged 55 nmol/L (. 11 µg/L). Despite the negative objective findings, subjective reports of fatigue, memory disturbances, and confusion were significantly higher in the exposed workers.

Boogaard et al. (1996) evaluated the effects of exposure to elemental mercury on the nervous system and the kidneys of workers producing natural gas in the Netherlands. Early signs of alterations in renal and neurological functions were studied in three groups of workers who were exposed to different levels of mercury that were below the current ACGIH biological exposure index of 35 µg/g creatinine. Air concentrations ranged from 10 to 1,500 µg/m^3 (median, 67) at locations where mercury exposure was anticipated; the potential 8-hour TWA exposure ranged from 33 to 781 µg/m^3 (median, 88). Air concentrations ranged from 0 to 6 µg/m^3 at locations where little mercury exposure was expected. Current mercury concentrations in urine were 23.7, 4.1, and 2.4 µg/g in high, low, and control exposure groups, respectively; mercury concentrations in blood were 3.5, 1.5, and 2.2 µg/L, respectively. There were no differences among the three study groups with respect to either motor nerve conduction velocity or tremor frequency spectra of physiological tremors. Also, no significant correlations were found between the results of the neurological tests and any of the present or historical biological monitoring data.

In contrast to the negative findings above, several studies have shown significant effects on tremor or on cognitive skills at comparable or lower group-average exposure levels (0.014–0.076 mg/m^3). Using the same paradigm as Langworth et al. (1992a), a significant difference was seen in the tremor frequency spectra in mercury-exposed workers from three industries who were exposed to low levels of mercury for an average of 15.3 years (range, 1–41 years) when compared to unexposed controls (Fawer et al. 1983).

The TWA mercury concentration measured in the work area at the time of the study was 0.026 mg Hg/m^3 (range not reported). It was assumed that the workers were exposed to the same concentration of mercury for the duration of their employment. However, the group size was small, and the results may have been influenced by a small number of more severely affected individuals. It is also possible that the tremors may have resulted from intermittent exposure to concentrations higher than the TWA. Urinary mercury levels in these workers averaged 11.3 µmol/mol creatinine (. 20 µg/g creatinine). Tremors have also been associated with occupational exposures that produced urinary concentrations of 50–100 µg/g creatinine and blood levels of 10–20 µg/L (Roels et al. 1982). Difficulty with heel-to-toe gait was observed in thermometer-plant workers subjected to mean personal-breathing-zone air concentrations of 0.076 mg/m^3 (range, 0.026–0.27 mg/m^3) (Ehrenberg et al. 1991).

Decreases in performance on tests that measured intelligence (a similarities test) and memory (digit span and visual reproduction tests) were observed in chloralkali workers exposed for an average of 16.9 years to low levels of mercury when compared to an age-matched control group (Piikivi et al. 1984). Significant differences from controls were observed among workers with blood levels >75 nmol/L (. 15 µg/L) and urine levels >280 nmol/L (. 56 µg/L).

Dentists (n=98, mean age 32, range 24–49) with an average of 5.5 years of exposure to low levels of mercury showed impaired performance on several neurobehavioral tests (Ngim et al. 1992). Exposure levels measured at the time of the study ranged from 0.0007 to 0.042 mg/m^3 (average, 0.014 mg/m^3) and blood levels ranged from 0.6 to 57 µg/L (average, 9.8 µg/L). Controls were matched for age, fish consumption, and number of amalgam fillings. Differences in education, sex distribution, and reported use of Chinese traditional medicines that might contain mercury were adjusted for in the statistical analysis. The dentists showed significantly poorer performance on finger tapping (measures motor speed), trail making (measures visual scanning), digit symbol (measures visuomotor coordination and concentration), digit span, logical memory delayed recall (measures visual memory), and Bender-Gestalt time (measures visuomotor coordination). The dentists had a higher aggression score than the controls. Correlations were observed for exposure levels and duration. This study is limited, however, by lack of blinding and failure to report control mercury levels; the statistical procedures used for confounders (use of traditional Chinese medicines) were not reported.

In a study of the relation between cumulative exposure to mercury and chronic health impairment, 298 dentists had their mercury levels measured by an X-ray fluorescence technique. Electrodiagnostic and neuropsychological findings in the dentists with more than 20 µg/g tissue (head and wrist) mercury levels

were compared with those of a control group consisting of dentists with no detectable mercury levels. Twenty-three out of 298 dentists with the highest mercury levels were administered neurological tests and compared to controls. The high mercury group had slowed conduction velocities in motor (median nerve) and sensory (suralnerve) nerves, mild neuropsychological impairment (increased errors in the Bender-Gestalt test), mild visuographic dysfunction, and higher distress levels (self-reported) than the control group. Seven of the high exposure dentists showed manifestations of polyneuropathy. Exposure concentrations were not specified. No polyneuropathies were detected in the control group (Shapiro et al. 1982). Abnormal nerve conduction velocities have also been observed at a mean urine concentration of 450 μg/L in workers from a chloralkali plant (Levine et al. 1982). These workers also experienced weakness, paresthesias, and muscle cramps. Prolongation of brainstem auditory-evoked potentials was observed in workers with urinary mercury levels of 325 μg/g creatinine (Discalzi et al. 1993). Prolonged somatosensory-evoked potentials were found in 28 subjects exposed to 20–96 mg/m^3 of mercury (Langauer-Lewowicka and Kazibutowska 1989).

In animals, as in humans, adverse neurological and behavioral effects are prominent following inhalation exposure to high concentrations of metallic mercury vapor. However, animals appear to be less sensitive than humans. Marked cellular degeneration and widespread necrosis were observed in the brains of rabbits following exposures to metallic mercury vapor at 28.8 mg/m^3 for durations ranging from 2 to 30 hours (Ashe et al. 1953). Exposures of 1 hour produced moderate (unspecified) pathological changes.

Intermediate-duration exposure of rabbits to 6 mg/m^3 mercury vapor for periods of 1–11 weeks produced effects ranging from mild, unspecified, pathological changes to marked cellular degeneration and some necrosis in the brain (Ashe et al. 1953). The more serious degenerative changes were observed at longer exposure durations (i.e., 8 and 11 weeks). Mild-to-moderate pathological changes were revealed in the brains of rabbits exposed to a metallic mercury vapor concentration of 0.86 mg/m^3 for 12 weeks (Ashe et al. 1953). The usefulness of these results is limited because the pathological changes are not specified and no distinction is made between primary and secondary effects (i.e., pathological changes secondary to induced shock).

Two of 6 rabbits exposed to 4 mg/m^3 metallic mercury vapor for 13 weeks exhibited slight tremors and clonus and had mercury concentrations of 0.8–3.7 μg/g wet tissue in the brain (Fukuda 1971). Following intermittent exposure to 3 mg/m^3 for 12–39 weeks, rats exhibited a decline in conditioned avoidance response; however, no histopathological changes were evident (Kishi et al. 1978). The change was

reversible within 12 weeks after exposure cessation and was associated with a decrease in the mercury concentration in brain tissue to below 10 μg/g wet weight (w/w). Mice exposed to an unspecified concentration of metallic mercury vapor intermittently for more than 3 weeks exhibited progressive neurological dysfunction (i.e., wobbling and unresponsiveness to light), beginning 22 days after initial exposure, and subsequently died 4 days later (Ganser and Kirschner 1985).

No studies were located regarding neurological effects in animals following chronic inhalation exposure to inorganic mercury.

Organic Mercury. Exposure to organic mercury via inhalation is extremely rare. The only reports of even its potential occurrence come from a few case histories. Case reports have described neurological effects in humans after inhalation exposure to organic mercury; however, no quantitative data were provided. Following acute inhalation exposure of dust containing methylmercury, four men had initial symptoms including numbness and tingling of limbs, unsteadiness in gait, difficulty in performing fine movements (e.g., buttoning a shirt), irritability, and constricted vision (Hunter et al. 1940). At least 2 years after these occupational exposures, the subjects had not fully recovered from their symptoms. Acute high-level exposure to an unspecified alkyl mercury compound has reportedly caused neurological symptoms (e.g., ataxia, unsteady gait, slurred speech, memory difficulties, tremors) in exposed workers (Hook et al. 1954; Lundgren and Swensson 1949).

A case study reporting neurological effects in a boy after exposure to mercury vapor released from paint containing phenylmercuric acetate (Aronow et al. 1990) was discussed under metallic mercury because the exposure was to metallic mercury vapors released from the paint.

Dimethylmercury is extremely volatile, and extremely toxic (in the 5 mg/kg body weight range). The following case history describes an accidental death due to an occupational spill of only a few drops of dimethylmercury. The primary exposure route is thought to have been dermal, but dimethylmercury is so volatile that inhalation exposure might also have occurred. Blayney et al. (1997) provided the first account of this tragic event. The case history was subsequently detailed by Nierenberg et al. (1998). The exposure occurred to a 48-year-old female chemistry professor who was admitted to the hospital 5 months (154 days) after, as best as can be determined, she inadvertently spilled several drops (estimated at 0.4–0.5 mL, about 1,500 mg) of dimethylmercury from the tip of her pipette onto the back of her disposable latex gloves. The spill was cleaned and the gloves disposed of. Hair analysis on a long strand

of hair revealed that after a brief lag time, mercury content rose rapidly to almost 1,100 ppm (normal level, <0.26 ppm; toxic level, >50 ppm), and then slowly declined with a half-life of 74.6 days. These results support the occurrence of one or several episodes of exposure, and are consistent with laboratory notebook accounts of a single accidental exposure. Testing of family members, laboratory coworkers, and laboratory surfaces failed to reveal any unsuspected mercury spills or other cases of toxic blood or urinary mercury levels. Permeation tests subsequently performed on disposable latex gloves similar to those the patient had worn at the time of the lone exposure revealed that dimethylmercury penetrates such gloves rapidly and completely, with penetration occurring in 15 seconds or less and perhaps instantly. Polyvinyl chloride gloves were equally permeable to dimethylmercury. Five days prior to hospital admission, the patient developed a progressive deterioration in balance, gait, and speech. During the previous 2 months, she had experienced brief episodes (spaced weeks apart) of nausea, diarrhea, and abdominal discomfort, and had lost 6.8 kg (15 lb). Medical examination revealed moderate upper-extremity dysmetria, dystaxic handwriting, a widely based gait, and "mild scanning speech." Routine laboratory test results were normal. Computed tomography (CT) and magnetic resonance imaging (MRI) of the head were normal except for the incidental finding of a probable meningioma, 1 cm in diameter. The cerebrospinal fluid was clear, with a protein concentration of 42 mg/dL and no cells. A preliminary laboratory report indicated that the whole-blood mercury concentration was more than 1,000 μg/L (normal range, 1–8 μg/L; toxic level, >200 μg/L). Chelation therapy with oral succimer (10 mg/kg orally every 8 hours) was begun on day 168 after exposure. Whole blood concentrations rose to 4,000 μg/L after one day of chelation, and urinary mercury levels were 234 μg/L (normal range, 1–5 μg/L; toxic level, >50 μg/L). Despite the initial success of chelation therapy, administration of vitamin E, and a blood exchange transfusion, at 176 days postexposure, the patient became comatose. Further aggressive general support and chelation therapy failed, life support ws removed (following the patient's advance directive), and the patient died 298 days postexposure. Autopsy results revealed diffusely thin cortex of the cerebral hemispheres (to 3 mm), and extensive gliosis of the visual cortex around the calcarine fissure and the superior surface of the superior temporal gyri. The cerebellum showed diffuse atrophy of both vermal and hemispheric folia. Microscope evaluation revealed extensive neuronal loss and gliosis bilaterally within the primary visual and auditory cortices, with milder loss of neurons and gliosis in the motor and sensory cortices. There was widespread loss of cerebellar granular-cell neurons, Purkinje cells, and basket-cell neurons, with evidence of loss of parallel fibers in the molecular layer. Borgmann's gliosis was well developed and widespread.

The highest NOAEL values and all reliable LOAEL values for neurological effects in each species and duration category are recorded in Table 2-1 and plotted in Figure 2-1.

2.2.1.5 Reproductive Effects

Metallic Mercury. No acute-duration exposure data were located regarding reproductive effects in humans after inhalation exposure to metallic mercury. However, several studies found no effect on fertility following intermediate or chronic inhalation exposure to metallic mercury in humans (Alcser et al. 1989; Cordier et al. 1991; Lauwerys et al. 1985). A retrospective cohort study reported that male workers in a U.S. Department of Energy (DOE) plant exposed for at least 4 months had urinary mercury concentrations of 2,144–8,572 µg/L (Alcser et al. 1989). This sample population showed no significant difference in fertility compared to controls (unexposed workers); however, they were never monitored for elemental mercury exposure. In a questionnaire study assessing the fertility of male workers exposed to mercury vapor from various industries (i.e., zinc-mercury amalgam, chloralkali, or electrical equipment product plants), there was no statistically significant difference in the number of children of the exposed group compared to a matched control group (Lauwerys et al. 1985). The concentration of mercury in the urine of these exposed workers ranged from 5.1 to 272.1 µg/g creatinine. No correlation was observed between prolactin, testosterone, luteinizing hormone, and follicle stimulating hormone levels and blood or urine mercury levels in male workers exposed to mercury vapors (Erfurth et al. 1990; McGregor and Mason 1991). Also, no effect on the response of these hormones to challenge with gonadotropin releasing hormone was observed (Erfurth et al. 1990).

Although no effect on fertility was observed in exposed workers, an increase in the rate of spontaneous abortions was reported in association with increased mercury concentrations in the urine of the fathers exposed to metallic mercury in chloralkali plants before the pregnancy (Cordier et al. 1991). There was a significantly increased risk of spontaneous abortion, at a rate of 18.4%, when fathers had more than 50 µg/L mercury in the urine, compared to a rate of 8.6% when fathers were unexposed. Sikorski et al. 1987) reported that women occupationally exposed to metallic mercury vapors (dentists and dental assistants) had more reproductive failures (spontaneous abortions, stillbirths, congenital malformations) and irregular, painful, or hemorrhagic menstrual disorders than a control (unexposed) group of women. The reproductive difficulties and menstrual disorders were correlated with mercury levels identified in scalp and pubic hair collected from the women. It should be noted that this study has been recently severely criticized for what Larsson (1995) calls "erroneous interpretation of results and distortion of conclusions." The Sikorski et al. (1987) paper is nonetheless presented in this toxicological profile as part of the available published data on reported human mercury exposure. Its presence here is based upon its publication in a

credible peer-reviewed international journal and is intended neither as endorsement nor condemnation of the data or conclusions in the 1987 paper.

Rowland et al. (1994) report that 418 women with high exposure to mercury (i.e., female dental assistants) were less fertile than unexposed controls. In this study, the probability of conception with each menstrual cycle (called "fecundability" by the authors) in women who prepared 30 or more amalgams per week and who were evaluated as having 4 or more poor mercury-hygiene practices was 63% of the fecundity of the unexposed controls. Rowland et al. (1994) noted that occupational groups with roughly the same potential for exposure often contain subjects whose actual exposures are quite different, depending on their particular work environment and their work practices within that environment. For example, 20% of the women in the final sample in this study reported preparing more than 30 amalgams per week with 4 or more poor hygiene factors. Among the women preparing the same number of amalgams, this study found differences in "fecundability," based upon each dental assistant's reported number of poor mercury-hygiene factors. One peculiar observation, however, was that women determined to have had low exposure to mercury in their dental occupation were found to be more fertile than unexposed controls. The reason(s) for the observed U-shaped dose-response curve were not known.

In animals, exposure to metallic mercury vapors causes prolongation of the estrous cycle. In a study by Baranski and Szymczyk (1973), female rats exposed via inhalation to metallic mercury (at an average of 2.5 mg/m^3, 6 hours a day, 5 days a week for 21 days) experienced longer estrous cycles than unexposed animals. In addition, estrous cycles during mercury exposure were longer than normal estrous cycles in the same animals prior to exposure. Although the initial phase of the cycle was protracted, complete inhibition of the cycle did not occur. During the second and third weeks of exposure, these rats developed signs of mercury poisoning including restlessness, seizures, and trembling of the entire body. The authors speculated that the effects on the estrous cycle were caused by the action of mercury on the central nervous system (i.e., damage to the hypothalamic regions involved in the control of estrous cycling).

Organic Mercury. No studies were located regarding reproductive effects in humans or animals after inhalation exposure to organic mercury.

The highest NOAELs and all reliable LOAELs for reproductive effects in each species and duration category are recorded in Table 2-1 and plotted in Figure 2-1.

2.2.1.6 Developmental Effects

Metallic Mercury. No association was demonstrated between inhalation exposure of the father and increased rates of major fetal malformations or serious childhood illnesses in a retrospective cohort study of workers at a U.S. DOE plant (Alcser et al. 1989).

A case study of a woman chronically exposed to an undetermined concentration of mercury vapor reported that her first pregnancy resulted in spontaneous abortion, and her second resulted in the death of the newborn soon after birth (Derobert and Tara 1950). It is unclear whether the reproductive toxicity experienced by the woman was due to the mercury exposure. However, after recovery from overt mercury poisoning, she gave birth to a healthy child. A woman occupationally exposed to mercury vapors for 2 years prior to pregnancy and throughout pregnancy was reported to have delivered a viable infant at term (Melkonian and Baker 1988). Urinary mercury in the woman at 15 weeks of pregnancy was 0.875 mg/L (normal levels are approximately 0.004 mg/L). Also, a case report of a woman exposed to mercury vapors in her home during the first 17 weeks of pregnancy reported that the woman delivered a normal child who met all developmental milestones (although the child was not formally tested for psychological development) (Thorpe et al. 1992). Although mercury exposure was not measured, the child was born with hair levels of 3 mg/kg (3 ppm) of mercury. This hair level is comparable to that observed in populations consuming fish once a week (WHO 1990) and suggests that exposure in this case may have been relatively low.

Exposure of neonatal rats to metallic mercury vapor at 0.05 mg/m^3 for 1 or 4 hours a day for 1 week during a period of rapid brain growth (postpartum days 11–17) resulted in subtle behavioral changes when the rats were tested at 4 and 6 months of age (Fredriksson et al. 1992). Offspring of rats exposed for 1 hour/day showed increases in the time necessary to finish a task in the radial arm maze (spatial learning). Offspring of rats exposed for 4 hours a day showed increases in both the time to finish the task and in the number of errors committed. When tested for locomotor activity at 2 months, an increase in rearing was observed in the 4 hour/day group, but repeat testing at 4 months showed lower locomotor, rearing, and total activity than controls. The 1-hour/day exposure group showed no difference from controls at 2 months, and increased activity and decreased rearing at 4 months when compared to controls.

Three groups of 12 pregnant Sprague-Dawley rats were exposed by inhalation to 1.8 mg/m^3 metallic mercury vapor on gestation days (Gd) 11–14 and 17–20 for 1 hour ("low dose") or 3 hours ("high dose").

Hg/kg/day ("high dose"). At postpartum day 3, each litter was reduced to 4 male and 4 female offspring. No significant differences between the mercury-treated offspring and the controls were observed for surface righting, negative geotaxis, pinna unfolding, and tooth eruption. Tests of spontaneous motor activity (locomotion, rearing, rearing time, and total activity) showed that the mercury-treated offspring were hypoactive at 3 months of age; at 14 months, only total activity differed between exposed and control groups. In spatial learning tasks, exposed offspring showed retarded acquisition in the radial-arm maze but no differences in the circular-swim maze. A simple test of learning, habituation to a novel environment (activity chambers), indicated a reduced ability to adapt. The authors conclude that prenatal exposure to mercury vapor results in behavior changes in the offspring similar to those reported for methylmercury. On postpartum days 3–4, the mercury contents in the brain, liver, and kidneys were 0.001, 0.004, and 0.002 mg Hg/kg, respectively, for control offspring; 0.005, 0.053, and 0.033 mg Hg/kg, respectively, for animals exposed for 1 hour a day; and 0.012, 0.112, and 0.068 mg Hg/kg, respectively, for animals exposed for 3 hours a day (Danielsson et al. 1993).

Four groups of 12 pregnant Sprague-Dawley rats were exposed to methylmercury or elemental mercury alone or in combination as follows: (1) administered 2 mg/kg/day methylmercury via gavage during Gd 6–9; (2) exposed by inhalation to 1.8 mg/m^3 metallic mercury (elemental mercury) vapor for 1.5 hours per day during Gd 14–19; (3) exposed to both methylmercury by gavage (2 mg/kg/day, Gd 6–9) and elemental Hg vapor by inhalation (1.8 mg/m^3, Gd 14–19) (methylmercury + elemental mercury); or (4) given combined vehicle administration for each of the 2 treatments (control). The inhalation regimen corresponded to an approximate dose of 0.1 mg Hg/kg/day. At postpartum day 3, each litter was reduced to 4 male offspring. There were no differences between any of the groups in maternal body weight gain before parturition. No differences in body weight, pinna unfolding, tooth eruption, surface righting reflex, and negative geotaxis were observed in the offspring. Offspring of dams exposed to elemental Hg showed hyperactivity in the spontaneous motor activity test chambers over all three parameters: locomotion, rearing, and total activity; this effect was potentiated in the animals of the methylmercury + elemental Hg group. In the swim maze test, the methylmercury + elemental mercury and elemental mercury groups evidenced longer latencies to reach a submerged platform, which they had learned to mount the day before, compared to either the control group or the methylmercury group. In the modified enclosed radial-arm maze, both the methylmercury + elemental Hg and elemental Hg groups showed more ambulations and rearings in the activity test prior to the learning test. During the learning trial, the same groups (i.e., methylmercury + elemental Hg and elemental Hg) showed longer latencies and made more errors in

acquiring all eight pellets. Generally, the results indicate that prenatal exposure to elemental mercury causes alterations to both spontaneous and learned behaviors, suggesting some deficit in the adaptive functions of the rats. Co-exposure to methylmercury, which by itself did not alter these functions at the dose given in this study, served to aggravate the changes significantly. Brain mercury concentrations in offspring were 1 ng/g w/w in the controls, 4 ng/g in the methylmercury group, 5 ng/g in the elemental Hg group, and 12 ng/g in the methylmercury + elemental Hg group (Fredriksson et al. 1996).

Adult female rats were exposed to metallic mercury vapor at 2.5 mg/m^3 for 3 weeks prior to fertilization and during Gd 7–20 (Baranski and Szymczyk 1973). A decrease in the number of living fetuses was observed in these dams compared to unexposed controls, and all pups born to the exposed dams died by the sixth day after birth. However, no difference in the occurrence of developmental abnormalities was observed between exposed and control groups. The cause of death of the pups in the mercury-exposed group was unknown, although an unspecified percentage of the deaths was attributed by the authors to a failure of lactation in the dams. Death of pups was also observed in another experiment in which dams were only exposed to the same dose level prior to fertilization, supporting the conclusion that high mortality in the first experiment was due, at least in part, to the poor health of the mothers. Without further information, this study must be considered inconclusive regarding developmental effects.

Newland et al. (1996) studied the offspring of pregnant squirrel monkeys exposed to 0.5 or 1 mg/m^3 of mercury vapor for 4 or 7 hours per day, 5 days per week during the last two-thirds or more of the gestation period. One female and 2 male offspring came from mothers exposed to 0.5 mg/m^3 mercury vapor during gestation weeks 5–19, 5–21, or 6–22 for a total of 247–510 hours, resulting in total doses of 1,304–2,900 μg (20–38 μg/day); and 3 male offspring came from mothers exposed to 1 mg/m^3 mercury vapor during gestation weeks 7–21, 3–18, or 8–21 for a total of 283–402 hours, resulting in total doses of 2,901–4,305 μg (42–62 μg/day). Five male offspring born about the same time as the exposed monkeys served as controls. Lever pressing was maintained under a Concurrent Random-Interval 30 schedule of reinforcement. Time allocation on each lever was examined during behavioral transitions and in a steady state. Median maternal blood levels ranged from 0.025 to 0.09 μg/g in animals exposed to 0.5 mg/m^3 and from 0.12 to 0.18 μg/g in animals exposed to 1 mg/m^3. No differences in birth weight, weight gain, or body weight at time of behavioral testing were observed between exposed and control offspring. No difference in sensitivity to reinforcer ratios was identified in the steady state, but there was much more variability in the steady-state performance of exposed monkeys, as indicated by the standard deviation of the regression, than in controls. Logistic regression was used to examine the transition to new schedule

parameters. Exposed monkeys were found to produce smaller or slower transitions than controls. The magnitude and stability of lever-press durations for controls and exposed monkeys were indistinguishable early in the experiment, but at the end, the exposed monkeys had longer lever-press durations and the session-to-session variability was much greater. One monkey's exposure began during the third week of gestation (earlier than any of the others) and its behavior was so erratic that some of the analyses could not be accomplished. Long-term effects of prenatal mercury vapor exposure included instability in lever-press durations and steady-state performance under concurrent schedules of reinforcement as well as aberrant transitions (Newland et al. 1996).

Organic Mercury. No studies were located regarding developmental effects in humans or animals after inhalation exposure to organic mercury.

The highest NOAELs and all reliable LOAELs for developmental effects in each species and duration category are recorded in Table 2-1 and plotted in Figure 2-1.

2.2.1.7 Genotoxic Effects

There is inconclusive evidence that occupational exposure to metallic mercury and to organic and inorganic mercury compounds, primarily through inhalation, causes structural and numerical chromosome aberrations in human lymphocytes. In one study, significant increases in the frequency of acentric fragments (chromosome breaks) occurred in 4 workers exposed to high concentrations of metallic mercury and in 18 workers exposed to a mixture of mercuric chloride, methylmercuric chloride, and ethylmercuric chloride (Popescu et al. 1979). Mercury concentrations in the workplace ranged from 0.15 to 0.44 mg/m^3; the urinary excretion level of mercury for both exposed groups was . 890 µg/L. The findings of this study are suspect because the control group was not matched for sex, smoking habits, or sample size. Additionally, one of the four individuals in the metallic mercury group had a history of benzene poisoning, which was reflected in the unusually high frequency of abnormal chromosome morphology seen in this individual. No difference in the incidence of aneuploidy was found between the exposed workers and the controls. In an earlier study, an apparent association between increased chromosome aberrations and workplace exposure to mercury (as measured by urinary mercury levels) was reported (Verschaeve et al. 1976). However, the study was not well controlled (i.e., not matched for sex, smoking habits, or sample size), and the only significant increase in structural aberrations occurred in the three workers exposed to ethylmercury. Significant increases in aneuploid were also noted for the exposure groups compared to the

control subjects. However, these data should also be interpreted with caution since age has an influence on aneuploidy, and in this study, there was a general trend toward a higher incidence of aneuploidy in the older exposed workers (ages 36–63 years). It is noteworthy that in a subsequent study performed by these investigators (Verschaeve et al. 1979), no adverse effects on the structure or number of chromosomes were demonstrated in 28 subjects exposed to moderate levels of metallic mercury (urinary levels of 50 μg/L). The authors concluded that the results from their 1976 study, showing an association between increased chromosomal aberrations and occupational exposure to mercury, may have been affected by factors other than exposure to mercury compounds.

No increased frequency of structural aberrations was found in 22 workers exposed to mercury vapors; no information was provided on numerical aberrations (Mabille et al. 1984). The mean duration of exposure was 4 years, and the mean urinary and blood mercury levels in the exposed group were 117 μg/g creatinine and 0.031 μg/mL, respectively. More recently, peripheral lymphocytes from 26 male chloralkali workers exposed to mercury vapors (25–50 $\mu g/m^3$), for a mean exposure time of 10 years, were analyzed for micronucleus induction. The results were compared to results obtained from 26 unexposed subjects (Barregard et al. 1991). Groups were matched for age (±7 years) and smoking habits; plasma, erythrocyte, and urine mercury levels were determined. Parallel lymphocyte cultures from each donor group were incubated in the presence of pokeweed mitogen, which stimulates both B- and T-lymphocytes, and phytohemagglutinin, which primarily activates T-cells. The analysis showed no significant increase in the frequency or the size of micronuclei in the exposed versus the control group. Nor was there a correlation between micronuclei induction and plasma, erythrocyte, or urinary levels of mercury. Within the exposed group, however, there was a significant correlation between micronuclei induction in phytohemagglutinin-stimulated lymphocytes and cumulative exposure (whole-blood mercury level over employment time); the response was independent of age or smoking habits. These results, suggesting a genotoxic effect on T-lymphocytes, are unusual since there is evidence that B-lymphocytes may be more sensitive indicators of chemically induced clastogenesis than T-lymphocytes (Högstedt et al. 1988). The authors stated that the evidence of a genotoxic response confined to T-lymphocytes could have been a random finding but hypothesized that long-term exposure to mercury may cause an accumulation of cytogenetic effects.

Similarly, there was no correlation between urinary mercury levels (60–245 μg/L) or the duration of exposure (11–34 years) and increased frequency of structural aberrations and micronuclei in the lymphocytes of 29 male workers exposed to mercury fulminate (Anwar and Gabal 1991). From the overall

results, the authors concluded that mercury in the manufacturing process may not have been the clastogen. Other genotoxicity studies are discussed in Section 2.5.

2.2.1.8 Cancer

Metallic Mercury. There is no evidence from epidemiological studies that indicates inhalation of metallic mercury produces cancer in humans (Cragle et al. 1984; Kazantzis 1981). No evidence of an association between metallic mercury exposure and cancer mortality was found in a group of workers employed in a facility utilizing the metal in a lithium isotope separation process (Cragle et al. 1984). Overall mortality in the mercury-exposed group was less than that of the standard white male population and that of a control group of men who were not exposed to mercury. Similarly, no excess of cancer of the kidneys or nervous system was found among a cohort of 674 Norwegian men exposed to mercury vapors for more than 1 year at 2 chloralkali plants (Ellingsen et al. 1993). An excess in lung cancer (type not specified) was found in Swedish chloralkali workers 10 years after the end of long-term, high-level exposure to metallic mercury (Barregard et al. 1990). However, these workers had also been exposed to asbestos. Furthermore, no data on smoking status was provided, although the study implied that the workers did not smoke much.

No studies were located regarding cancer in animals after inhalation exposure to metallic mercury.

Organic Mercury. Associations were reported between the use of mercury-containing fungicides (i.e., mercury levels in hair) and leukemia in farmers and between the use of mercury-containing seed dressings and leukemia in cattle (Janicki et al. 1987). However, the study was limited in reporting methodology used to conduct this study. Furthermore, the study did not adequately address exposure to other chemicals, or adjust for other leukemia risk factors.

No studies were located regarding cancer in animals after inhalation exposure to organic mercury.

2.2.2 Oral Exposure

The bulk of the information regarding toxicity resulting from oral exposure to inorganic mercury comes from studies of mercuric chloride. However, a few studies are also available on the effects of oral exposure to mercuric acetate, mercurous chloride (calomel), and mercuric sulfide (cinnabar). Discussion of these

compounds has not been separated in this section, but the specific inorganic compound responsible for any effect is noted both in the text and in Table 2-2 and Figure 2-2.

Health effects following oral exposure to organic mercury were observed in humans and animals. The majority of the studies used to derive the NOAELs and LOAELs shown in Table 2-3 and Figure 2-3 concern exposure to methylmercuric chloride; however, in several studies, exposure was to methylmercuric acetate, methylmercuric hydroxide, methylmercuric dicyanidiamide, or phenylmercuric acetate. These chemicals are discussed together in Table 2-3 and Figure 2-3. In order to facilitate a comparison of studies using different compounds of mercury (either organic or inorganic), all doses are expressed in terms of the mercury exposure (mg Hg/kg/day) rather than to the mercury compound (HgX or RHgX/kg/day) to which one is exposed. For example, a dose of 1 mg/kg (when the compound is methylmercuric chloride) refers to 1 mg/kg mercury rather than 1 mg/kg methylmercuric chloride.

2.2.2.1 Death

Inorganic Mercury. A lethal dose of mercuric chloride was estimated to be 10–42 mg Hg/kg for a 70-kg adult (Gleason et al. 1957). Death from oral exposure to inorganic mercury is usually caused by shock, cardiovascular collapse, acute renal failure, and severe gastrointestinal damage (Gleason et al. 1957; Murphy et al. 1979; Troen et al. 1951). Eighteen cases of human poisoning (suicide attempts in some cases) were reported by Troen et al. (1951); 9 patients died following oral ingestion of single doses of mercuric chloride (range, 29–>50 mg Hg/kg). The most common findings in these cases were gastro-intestinal lesions (e.g., mild gastritis to severe necrotizing ulceration of the mucosa) and renal involvement (e.g., albuminuria, anuria, and uremia). Death of a 50-year-old woman due to ingestion of an unspecified amount of mercurous chloride in Chinese medicine has also been reported (Kang-Yum and Oransky 1992). The death was attributed to renal failure.

In rats, the oral LD_{50} values (lethal dose, 50% kill) ranged from 25.9 to 77.7 mg Hg/kg as mercuric chloride (Kostial et al. 1978). The signs of acute mercury toxicity in animals were similar to those described above for humans. Male rats appeared to be slightly more sensitive to the lethal effects of mercuric chloride; 2 of 5 male rats and no female rats died when given gavage doses of 14.8 mg Hg/kg, 5 days a week for 2 weeks (Dieter et al. 1992; NTP 1993). Mice showed slightly less toxicity, with no deaths at 14.8 mg Hg/kg, death in 1 male at 29 mg Hg/kg, and deaths in 5 of 5 males and 4 of 5 females at 59 mg Hg/kg when administered by gavage over the same period (NTP 1993).

Chronic exposure to mercuric chloride resulted in increased mortality in male rats at 1.9 mg Hg/kg/day but no increase in mortality in female rats at up to 3.7 mg Hg/kg/day or in either male or female mice at up to 7.4 mg Hg/kg/day (NTP 1993). Renal lesions in the male rats were thought to contribute to the early deaths in these animals.

The highest NOAEL values and all reliable LOAEL values for death for each species and duration category are recorded in Table 2-2 and plotted in Figure 2-2 for inorganic mercury.

Organic Mercury. The acute lethal dose of organic mercury compounds for humans is difficult to assess from the available literature. Death resulting from organic mercury ingestion has been amply documented following outbreaks of poisoning (Minamata disease) after consumption of methylmercury-contaminated fish in Minamata, Japan (Tsubaki and Takahashi 1986) and after consumption of grains contaminated with methyl- and ethylmercury in Iraq (Al-Saleem and the Clinical Committee on Mercury Poisoning 1976; Bakir et al. 1973). Death occurred in two boys who ate meat from a butchered hog that had been fed seed treated with ethylmercuric chloride (Cinca et al. 1979). However, primarily because of the delay between mercury consumption and the onset of symptoms, the amount of organic mercury ingested in these cases is difficult to determine. Fatal doses estimated from tissue concentrations range from 10 to 60 mg/kg (EPA 1985b). A case-control study examining the cause of death for patients with Minamata disease compared to the cause of death in unexposed persons showed that those patients who died prior to 1970 had significantly increased noninflammatory diseases of the nervous system; Minamata disease was reported as the underlying cause of death (Tamashiro et al. 1984). For this group, pneumonia and nonischemic heart disease were reported as prominent secondary cause of death. For those patients who died between 1970 and 1980, significant increases in Minamata disease were reported as the primary cause of death. Nonischemic heart disease correlated with the incidence of Minamata disease, and noninflammatory central nervous system disease was a prominent secondary cause of death in this group.

Methylmercury toxicity is very strain- and sex-specific in mice. A single oral dose of methylmercuric chloride at 16 mg Hg/kg resulted in the death of 4 of 6 male mice (C57BL/6N Jcl strain) but no deaths in females (Yasutake et al. 1991b). No increase in mortality was observed in female mice until 40 mg Hg/kg was administered, at which dosage 4 of 6 females died. Twenty-six weeks of dietary exposure to methyl-mercuric chloride resulted in increased mortality in both male and female mice (ICR strain) at 3.1 mg Hg/kg/day (Mitsumori et al. 1981). Chronic (104 weeks) dietary exposure to methylmercuric

chloride resulted in increased deaths in male mice (B6C3F$_1$ strain) at 0.69 mg Hg/kg/day but no increased mortality in females at up to 0.60 mg Hg/kg/day (Mitsumori et al. 1990).

The highest NOAEL values and all reliable LOAEL values for death for each species and duration category are recorded in Table 2-3 and plotted in Figure 2-3 for organic mercury.

2.2.2.2 Systemic Effects

Ingestion of mercury compounds has been associated with systemic toxicity in both humans and animals. As with inhalation exposure to metallic mercury vapor, the major target organs of toxicity following oral exposure to inorganic and organic mercury are the kidneys and the central nervous system, respectively. Available information is limited mainly to that concerning exposure to mercuric chloride and methyl-mercuric chloride. Oral exposure to mercury, especially the organic mercury form, has also been observed to result in adverse developmental effects in humans and experimental animals. A discussion of the differences in the toxicities of metallic mercury, inorganic compounds, and organic compounds of mercury is presented in Section 2.5. The systemic effects observed after oral exposure are discussed below.

The highest NOAEL values and all reliable LOAEL values for systemic effects for each species and duration category are recorded in Table 2-2 and plotted in Figure 2-2 for inorganic mercury, and recorded in Table 2-3 and plotted in Figure 2-3 for organic mercury.

Respiratory Effects

Inorganic Mercury. Extremely limited information was located regarding respiratory effects in humans after oral exposure to inorganic forms of mercury. A 35-year-old man who swallowed an unknown amount of mercuric chloride had severe pulmonary edema and required artificial ventilation (Murphy et al. 1979). Fine rales were detected in a 19-month-old boy who swallowed powdered mercuric chloride (Samuels et al. 1982). A 50-year-old female who ingested 5 tablets of a Chinese medicine that contained an unspecified amount of mercurous chloride (Kang-Yum and Oransky 1992) experienced shortness of breath.

The only study located regarding respiratory effects in animals after oral exposure to inorganic mercury described forceful and labored breathing, bleeding from the nose, and other unspecified respiratory

difficulties in Long-Evans rats after dietary exposure to 2.2 mg Hg/kg/day as mercuric chloride for 3 months (Goldman and Blackburn 1979).

Organic Mercury. Limited information was located regarding respiratory effects in humans after oral exposure to organic mercury. Two boys who died after eating meat from a hog that had eaten seed treated with ethylmercuric chloride developed bronchopneumonia and edematous alveolitis, and required artificial ventilation (Cinca et al. 1979). Bronchopneumonia was also identified as the cause of death in four adults and one infant who died as the result of methylmercury poisoning in Iraq during 1972 (Al-Saleem and the Clinical Committee on Mercury Poisoning 1976). It is unclear whether these respiratory effects were the result of direct effects on the respiratory system or were secondary to other effects.

The only information located regarding respiratory effects in animals after oral exposure to organic mercury comes from a study in which rats were exposed to methylmercuric chloride in the diet for 2 years (Verschuuren et al. 1976). This study showed no treatment-related histopathological lesions in the lungs of exposed rats at 0.1 mg Hg/kg/day.

Cardiovascular Effects

Inorganic Mercury. Cardiovascular toxicity has been observed following ingestion of mercuric chloride and mercurous chloride in humans. The majority of the information regarding cardiovascular effects comes from reports of children who were treated with mercurous chloride tablets for worms or mercurous chloride-containing powders for teething discomfort (Warkany and Hubbard 1953). These authors described multiple cases in which tachycardia and elevated blood pressure were observed in the affected children. The only information located regarding cardiovascular effects in humans after ingestion of mercuric chloride comes from a case study of a 22-year-old who attempted suicide by ingesting approximately 20 mg Hg/kg as mercuric chloride (Chugh et al. 1978). An electrocardiogram showed no P wave, prolongation of the QRS segment, and a high T wave. The authors suggested that these cardiovascular effects were secondary to severe hyperkalemia.

Exposure of rats to 28 mg Hg/kg/day as mercuric chloride for 180 days in drinking water resulted in an increase in blood pressure, a decrease in cardiac contractility, and no effect on heart rate (Carmignani et al. 1992). The increase in blood pressure was attributed to a vasoconstrictor effect, and the decrease in contractility was attributed to the direct toxic effect of the mercury on the cardiac muscle. Slightly

different results were obtained following 350-day exposure of a different strain of rats to 7 mg Hg/kg/day as mercuric chloride in drinking water (Boscolo et al. 1989; Carmignani et al. 1989). In the chronic study, positive inotropic response, increased blood pressure and cardiac contractility, and decreased baroreceptor reflex sensitivity were observed. The investigators suggested that the mechanism for the cardiac effects in the chronic study involved the release of norepinephrine from presynaptic nerve terminals. Evidence of this release was provided by the fact that mercury administration reduced the cardiovascular response to bretylium (which blocks presynaptic release of the neurotransmitter norepinephrine) but not tyramine (which releases neurotransmitter from nerve terminals).

Organic Mercury. Electrocardiography in four family members who ate meat from a hog that had consumed seed treated with ethylmercuric chloride had abnormal heart rhythms (ST segment depression and T wave inversion) (Cinca et al. 1979). Death of the two children in the family was attributed to cardiac arrest, and autopsy of these boys showed myocarditis. Cardiovascular abnormalities were also observed in severe cases of poisoning in the Iraqi epidemic of 1956, when widespread poisoning resulted from eating flour made from seed grains treated with ethylmercury *p*-toluene sulfonanilide (Jalili and Abbasi 1961). These abnormalities included irregular pulse, occasionally with bradycardia, and electrocardiograms showing ventricular ectopic beats, prolongation of the Q-T interval, depression of the S-T segment, and T inversion.

A decrease in heart rate was observed in male rats given 2 gavage doses of 12 mg Hg/kg as methylmercuric chloride (Arito and Takahashi 1991). An increase in systolic blood pressure was observed in male rats after daily oral gavage doses of 0.4 mg Hg/kg/day as methylmercuric chloride for 3–4 weeks (Wakita 1987). This effect began approximately 60 days after initiation of exposure and persisted for at least 9 months. No treatment-related histopathological changes were observed in the hearts of rats exposed to 0.1 mg Hg/kg/day as methylmercuric chloride in the diet for up to 2 years (Verschuuren et al. 1976).

Gastrointestinal Effects

Inorganic Mercury. Ingestion of metallic mercury results in negligible absorption and little effect on the gastrointestinal tract. The two case histories identified are unusual in that the dose levels could be reasonably well quantified. The first case history reported ingestion of 15 mL (204 g) of metallic mercury by a 17-year-old male storekeeper who swallowed mercury from the pendulum of a clock (apparently out of curiosity rather than as a suicide attempt). On admission, and 24 hours later, he was symptom free, and

physical examination was normal. The patient complained of no gastrointestinal symptoms, and was treated with a mild laxative and bedrest (Wright et al. 1980).

In a second and massive incidence of ingestion, a 42-year-old man who had spent much of his life (since the age of 13) repairing instruments that contained mercury, intentionally ingested an estimated 220 mL (about 3,000 g) while repairing a sphygmomanometer (Lin and Lim 1993). Upon admission, the patient presented with significantly elevated mercury blood levels (103 μg/L, normal <10 μg/L) and urine levels (73μg/L, normal <20μg/L). In the previous 2 years he had developed mild hand tremors, forgetfulness, irritability, and fatigue. Only a mild abdominal discomfort and no hepatic complications were observed at admission. The neurological symptoms were attributed to the long occupational exposure to mercury and not to the recent acute exposure. The initial radiological examination showed a conglomeration of mercury globules in the fundus of the stomach and ascending colon, with fine metallic spots dispersed throughout the small intestine. Abdominal ultrasonography was normal. He was treated with immediate gastric lavage and cathartics. He also received D-penicillamine 1 g/day orally for 7 days. Seven days later, there were only spots of metallic mercury in the ascending colon. By 2 weeks, most of the mercury had been excreted in the feces and was measured at a total volume of 220 mL (this number was used to estimate the amount initially ingested). The authors reported that systemic absorption appeared low, based on the return to low levels of mercury in the urine and blood over the 10 days of monitoring following the exposure. A subsequent evaluation 6 months later revealed no further gastrointestinal involvement.

Ingestion of mercuric chloride is highly irritating to the tissues of the gastrointestinal tract. Blisters and ulcers on the lips and tongue and vomiting were observed in a 19-month-old boy who ingested an unknown amount of mercuric chloride powder (Samuels et al. 1982). Similarly ingestion of a lethal dose of mercuric chloride by a 35-year-old man resulted in vomiting, diarrhea, colicky abdominal pain, oropharyngeal pain, and ulceration and hemorrhages throughout the length of the gastrointestinal tract (Murphy et al. 1979). Ingestion by a woman of 30 mg Hg/kg as mercuric chloride resulted in severe abdominal pain, diarrhea, nausea, and vomiting (Afonso and deAlvarez 1960). Another report of an attempted suicide by a 22-year-old reported ulceration of the mouth and throat and bloody vomit after ingestion of approximately 20 mg Hg/kg (Chugh et al. 1978). Because of vomiting, the actual effective dose was unknown.

Reports of ingestion of mercurous chloride have not found similar caustic effects; however, a 50-year-old woman who ingested an unspecified amount of mercurous chloride in a Chinese medicine experienced nausea and vomiting (Kang-Yum and Oransky 1992). Several children who were treated with mercurous

chloride for constipation, worms, or teething discomfort had swollen red gums, excessive salivation, anorexia, diarrhea, and/or abdominal pain (Warkany and Hubbard 1953).

Inflammation and necrosis of the glandular stomach were observed in mice that were given oral doses of 59 mg Hg/kg as mercuric chloride 5 days a week for 2 weeks (NTP 1993). In a 2-year gavage study, an increased incidence of forestomach hyperplasia was observed in male rats exposed to 1.9 or 3.7 mg Hg/kg/day as mercuric chloride compared to the control group.

Organic Mercury. Case studies of individuals who were orally exposed to alkyl mercury compounds (unspecified form) reported diarrhea, tenesmus, irritation, and blisters in the upper gastrointestinal tract (Lundgren and Swensson 1949). Ingestion of meat from a hog that was fed seed treated with ethylmercuric chloride resulted in vomiting in two of the family members (Cinca et al. 1979). No quantitative data were available. Ingestion of flour made from seed grains that had been treated with ethylmercury *p*-toluene sulfonanilide also commonly resulted in abdominal pain and vomiting, diarrhea, or constipation (Jalili and Abbasi 1961).

Pfab et al. (1996) reported a case of a 44-year-old man who ingested 83 mg/kg Thiomersal in a suicide attempt (5 g/60 kg). Thiomersal is a widely used alkyl-aryl-organomercurial bactericide. The man developed gastritis, renal tubular failure, dermatitis, gingivitis, delirium, coma, polyneuropathy, and respiratory failure. Treatment was symptomatic plus gastric lavage and the oral chelation with dimercaptopropane sulfonate and dimercaptosuccinic acid. The patient's condition was at its worst on day 17; however, the patient recovered completely (after several months). Maximum mercury concentrations were: blood, 14 mg/L; serum, 1.7 mg/L; urine, 10.7 mg/L; and cerebrospinal fluid, 0.025 mg/L. Mercury concentration in blood declined with two velocities: first with a half-time of 2.2 days, then with a half-time of 40.5 days. The decline of mercury concentration in blood, urinary mercury excretion, and renal mercury clearance were not substantially influenced by chelation therapy.

Exposure of rats to phenylmercuric acetate for 2 years resulted in necrosis and ulceration of the cecum at doses as low as 4.2 mg Hg/kg/day in drinking water; no effect was observed at 1.7 mg Hg/kg/day in the feed (Fitzhugh et al. 1950; Solecki et al. 1991). Mice showed ulceration of the glandular stomach after 2 years of dietary exposure to methylmercuric chloride at 0.69 mg Hg/kg/day (Mitsumori et al. 1990). In contrast, no treatment-related histopathological lesions in the stomach or jejunum were observed in rats exposed via the diet to 0.1 mg Hg/kg/day as methylmercuric chloride (Verschuuren et al. 1976).

Hematological Effects

Inorganic Mercury. Information is limited regarding hematological effects in humans after ingestion of inorganic mercury. The only information located regarding hematological effects in humans was the report of anemia that developed (probably secondary to massive gastrointestinal hemorrhaging) in a 35-year-old man who ingested a lethal amount of mercuric chloride (Murphy et al. 1979). Bone marrow activity in the afflicted man was normal, but thrombocytopenia was also observed.

Groups of 10 female Sprague-Dawley rats were administered a single gavage dose of mercuric chloride at 7.4 or 9.2 mg Hg/kg in water and necropsied at 14 days postexposure. Blood samples were analyzed for hemoglobin concentration, hematocrit value, erythrocyte counts, total and differential leukocyte counts, and platelet counts. Serum was analyzed for sodium, potassium, inorganic phosphorus, total bilirubin, alkaline phosphatase, aspartate aminotransferase (AST), total protein, calcium, cholesterol, glucose, uric acid, and lactate dehydrogenase (LDH). There were no effects on body weight, and weights of other organs were not affected. Significant decreases in hemoglobin, erythrocytes, and hematocrit were also reported. There was a significant decrease in serum protein and calcium in the low-dose mercury group only. Mercury was found mainly in the kidneys (12.6 and 18.9 ppm at the low and high dose, respectively), but trace amounts were also detected in the liver, brain, and serum (Lecavalier et al. 1994).

No other studies were located regarding hematological effects in animals after oral exposure to inorganic mercury.

Organic Mercury. No studies were located regarding hematological effects in humans after oral exposure to organic mercury.

Rats that received phenylmercuric acetate in their drinking water for 2 years showed decreases in hemoglobin, hematocrit, and red blood cell counts at a dose of 4.2 mg Hg/kg/day (Solecki et al. 1991). The anemia observed in this study may have been secondary to blood loss associated with the ulcerative lesions in the large intestine seen at this dose (see Gastrointestinal Effects above). No treatment-related changes were observed in hematological parameters measured in rats (strain not specified) exposed via the diet for 2 years to 0.1 mg Hg/kg/day as methylmercuric chloride (Verschuuren et al. 1976).

Musculoskeletal Effects

Inorganic Mercury. A single case report was identified that found evidence of skeletal muscle degeneration (markedly elevated serum aldolase, LDH, and creatinine phosphokinase; and the presence of pigment granular casts and myoglobin in the urine) in a 22-year-old man who ingested 2 g of mercuric chloride in an attempt to commit suicide (Chugh et al. 1978). Several children who were treated with mercurous chloride for constipation, worms, or teething discomfort experienced muscle twitching or cramping in the legs and/or arms (Warkany and Hubbard 1953). The muscular effects were probably secondary to changes in electrolyte balance (i.e., potassium imbalance due to fluid loss or renal wasting).

No studies were located regarding musculoskeletal effects in animals after oral exposure to inorganic mercury.

Organic Mercury. Autopsy of one of two boys who died after eating meat from a hog that had consumed seed treated with ethylmercuric chloride showed muscle wasting (Cinca et al. 1979). This effect was probably secondary to neurotoxicity. Electromyography in the two surviving members of the family showed no abnormalities. Musculoskeletal effects observed in Iraqis poisoned by consuming flour made from grains treated with ethylmercury *p*-toluene sulfonanilide included deep skeletal pain and muscle twitching or fasciculations (Jalili and Abbasi 1961). It is likely that these effects were secondary to effects on the nervous system.

No treatment-related histopathological changes in skeletal muscle were observed in rats exposed via the diet for 2 years to 0.1 mg Hg/kg/day as methylmercuric chloride (Verschuuren et al. 1976).

Hepatic Effects

Inorganic Mercury. Limited information was located regarding hepatic effects in humans who ingested inorganic mercury. A 35-year-old man who ingested a lethal dose of mercuric chloride became jaundiced and exhibited elevated AST, alkaline phosphatase, LDH, and bilirubin (Murphy et al. 1979). An autopsy revealed an enlarged and softened liver. Hepatic enlargement was also observed in a 19-month-old boy who ingested an unknown amount of powdered mercuric chloride (Samuels et al. 1982).

Limited information was located regarding the hepatic effects of inorganic mercury in animals.

Groups of 10 female Sprague-Dawley rats were administered a single gavage dose of mercuric chloride at 7.4 or 9.2 mg Hg/kg in water and necropsied at 14 days postexposure. There were no effects on body or relative liver weights from mercuric chloride exposure. LDH activity was significantly decreased in animals exposed to $HgCl_2$ at both dose levels. Mercury was found mainly in the kidneys (12.6 and 18.9 ppm at the low and high dose, respectively), but trace amounts were also detected in the liver, brain, and serum (Lecavalier et al. 1994).

Two intermediate-duration studies in rats showed biochemical changes following ingestion of mercuric chloride (Jonker et al. 1993b; Rana and Boora 1992). Increases in hepatic lipid peroxidation and decreases in glutathione peroxidase were observed in rats orally exposed to an unspecified dose of mercuric chloride for 30 days (Rana and Boora 1992). In a 4-week range-finding study, groups of 5 rats per sex (10 per sex for controls) received diets containing mercuric chloride at 5, 10, or 20 mg Hg/kg/day in males and 5.5, 11.1, and 22.2 mg Hg/kg/day in females. Absolute liver weight decreased starting at the mid-dose group in males and in the high-dose group in females (Jonker et al. 1993b). The liver weight significantly increased in mice given 2.9 mg Hg/kg/day as mercuric chloride in the drinking water for 7 weeks; however, no histopathological changes were observed (Dieter et al. 1983). Male rats administered mercuric chloride by gavage for 2 years showed a slight increase in acute hepatic necrosis (11 of 50 versus 4 of 50 in controls); however, it is unclear whether this increase was statistically significant (NTP 1993).

Organic Mercury. Extremely limited information was also obtained regarding the hepatic effects of organic mercury exposure. An autopsy of four adults and four infants who died as the result of methyl-mercury poisoning in Iraq in 1972 reported fatty changes in the liver occurred in most cases (Al-Saleem and the Clinical Committee on Mercury Poisoning 1976). It is unclear whether these changes were the direct result of methylmercury on the liver or whether they were due to other causes. The prevalence of liver disease in a population from the Minamata area was not significantly increased when compared to unexposed controls (Futatsuka et al. 1992).

No treatment-related changes were observed in hepatic parameters measured in rats exposed via the diet to 0.1 mg Hg/kg/day as methylmercuric chloride (Verschuuren et al. 1976).

Renal Effects

Inorganic Mercury. The kidney appears to be the critical organ of toxicity for the ingestion of mercuric salts. Renal effects in humans have been observed following acute oral exposure to inorganic mercury. Acute renal failure has been observed in a number of case studies of mercuric chloride ingestion (Afonso and deAlvarez 1960; Murphy et al. 1979; Samuels et al. 1982). An autopsy of a 35-year-old man who ingested a lethal dose of mercuric chloride and exhibited acute renal failure showed pale and swollen kidneys (Murphy et al. 1979). A case study reported acute renal failure characterized by oliguria, proteinuria, hematuria, and granular casts in a woman who ingested 30 mg Hg/kg as mercuric chloride (Afonso and deAlvarez 1960). Another case study reported a dramatic increase in urinary protein secretion by a patient who ingested a single dose of 15.8 mg Hg/kg as mercuric chloride (assuming a body weight of 70 kg) (Pesce et al. 1977). The authors of the report surmised that the increased excretion of both albumin and β_2-microglobulin was indicative of mercury-induced tubular and glomerular pathology. Acute renal failure that persisted for 10 days was also observed in a 19-month-old child who ingested an unknown amount of powdered mercuric chloride (Samuels et al. 1982). Decreased urine was observed in a 22-year-old who attempted suicide by ingesting approximately 20 mg Hg/kg (Chugh et al. 1978). Myoglobin and pigmented casts were observed in the urine, and the authors suggested that these observations, in combination with a highly elevated level of serum creatine phosphokinase, indicated that rhabdomyolysis may have contributed to the renal failure.

Ingestion of mercurous chloride has also resulted in renal toxicity in humans. Decreased urinary output and edema were observed in a 60-year-old woman who ingested an unspecified amount of mercurous chloride in a Chinese medicine (Kang-Yum and Oransky 1992). Renal failure was a contributing factor in the death of this woman. Renal failure also developed in two female patients who chronically ingested a mercurous chloride-containing laxative (Davis et al. 1974).

Renal toxicity has been observed in Fischer 344 rats and B6C3F$_1$ mice following acute-, intermediate-, and chronic-duration exposures to mercuric chloride (Dieter et al. 1992; NTP 1993). In the 14-day study, male and female rats were exposed by gavage to 0.93–14.8 mg Hg/kg/day as mercuric chloride for 5 days a week. There was a significant increase in the absolute and relative kidney weights of males beginning at the 1.9-mg/kg/day dose level. An increased incidence of tubular necrosis was observed in rats exposed to at least 3.7 mg/kg/day; severity progressed with increasing dose levels. Increases in urinary levels of alkaline phosphatase, AST, and LDH were also observed at 3.7 mg Hg/kg/day; at 7.4 mg Hg/kg/day,

increased urinary γ-glutamyltransferase activity was also observed. Mice given a single gavage dose of 10 mg/Hg/kg as mercuric chloride showed minor renal tubular damage and rapid regeneration of the tubular epithelium (Nielsen et al. 1991). At 20 mg Hg/kg/day, the mice showed necrosis of the proximal tubules. Mice given gavage doses of mercuric chloride 5 days a week for 2 weeks showed an increase in absolute and relative kidney weights at 3.7 mg Hg/kg/day and acute renal necrosis at 59 mg Hg/kg/day (NTP 1993).

Groups of 10 female Sprague-Dawley rats were administered a single gavage dose of mercuric chloride at 7.4 or 9.2 mg Hg/kg in water and necropsied at 14 days postexposure. No effects on body weight or weights of other organs were found. Mercury was found mainly in the kidneys (12.6 and 18.9 ppm at the low and high doses, respectively), but trace amounts were also detected in the liver, brain, and serum. Mild-to-moderate morphological changes, consisting of protein casts, cellular casts, and interstitial sclerosis, were noted in the kidneys of $HgCl_2$-treated animals in both groups (Lecavalier et al. 1994).

In a 4-week range-finding study, groups of 5 rats per sex (10 per sex for controls) received diets containing mercuric chloride at 5, 10, or 20 mg Hg/kg/day for males and 5.5, 11.1, and 22.2 mg Hg/kg/day for females. Nephrosis and proteinaceous casts in the kidneys were observed in all groups (males and females) fed mercuric chloride. An increased number of epithelial cells in the urine was observed in males exposed at the low dose; however, this effect was not observed at higher dose levels and the authors noted that the effect could not be ascribed to treatment. The minimum-nephrotoxic-effect level (MNEL) and the no-nephrotoxic-effect level (NNEL) for mercuric chloride in feed were determined to be 8 mg Hg/kg/day in males and 8.9 mg Hg/kg/day in females and 1 mg Hg/kg/day in males and 1.1 mg Hg/kg/day in females, respectively (Jonker et al. 1993b). In a follow-up 4-week study, 10-week-old Wistar rats were fed mercuric chloride at the MNEL and NNEL. In males, the MNEL resulted in the presence of ketones in urine and an increase in the relative weight of kidneys. Effects observed in females in the MNEL group included decreased density of urine and increased absolute and relative kidney weights. Increased absolute and relative kidney weights were also seen in females at the NNEL. A few histopathological changes were found in the basophilic tubules in the outer cortex of the kidneys in 5 of 5 males and 1 of 5 females exposed to the MNEL (Jonker et al. 1993b).

Similarly, male mice receiving mercuric chloride in drinking water for 7 weeks showed slight degeneration of the tubular epithelial cells (nuclear swelling) at 2.9 mg Hg/kg/day and minimal renal nephropathy

(dilated tubules with either flattened eosinophilic epithelial cells or large cytomegalic cells with foamy cytoplasm) at 14.3 mg Hg/kg/day (Dieter et al. 1983).

In a 6-month exposure to 0.23–3.7 mg Hg/kg/day, a significant increase in severity of nephropathy (i.e., dilated tubules with hyaline casts, foci of tubular regeneration, and thickened tubular basement membrane) was observed in Fischer 344 rats exposed to 0.93 mg/kg/day of mercuric chloride compared to the controls (NTP 1993). The absolute and relative kidney weights were increased in males at 0.46 mg/kg/day. In $B6C3F_1$ mice, the incidence and severity of cytoplasmic vacuolation of renal tubule epithelium increased in males exposed to at least 3.7 mg Hg/kg/day as mercuric chloride for 6 months (NTP 1993). Administration of large doses of mercuric chloride (28 mg Hg/kg/day) in the drinking water for 6 months also resulted in focal degeneration of the tubular cells with decreased acid phosphatase in the lysosomes (indicative of the release of the lysosomal contents) (Carmignani et al. 1992). Notably, at this dose, renal glomerular changes were also evident. The glomeruli showed hypercellularity, and there was deposition of amorphous material in the mesangium; thickening of the basement membrane with IgM present was also observed.

When a strain of mice (SJL/N) sensitive to the immunotoxic effects of mercury was given mercuric chloride in the drinking water at 0.56 mg Hg/kg/day for 10 weeks, slight glomerular cell hyperplasia with granular IgG deposits in the renal mesangium and glomerular blood vessels were observed (Hultman and Enestrom 1992). No tubular necrosis was observed.

In a 2-year study, male rats gavaged with 1.9 or 3.7 mg Hg/kg/day as mercuric chloride 5 days a week exhibited an incidence of marked nephropathy (described as thickening of glomerular and tubular basement membranes and degeneration and atrophy of tubular epithelium) that was significantly greater in severity than in the control group (NTP 1993). In addition, the incidence of renal tubule hyperplasia was increased in the high-dose male rats. In the same study, the incidence and severity of nephropathy were significantly greater in male and female mice gavaged with 3.7 and 7.4 mg Hg/kg/day as mercuric chloride 5 days a week than in the controls. Administration of 7 mg Hg/kg/day as mercuric chloride to rats in the drinking water resulted in hydropic degeneration and desquamation of tubule cells (Carmignani et al. 1989). Electron microscopy showed lysosomal alterations in the proximal tubules and thickening of the basal membrane of the glomeruli.

Organic Mercury. Data on renal toxicity associated with ingestion of methylmercury in humans come from several case studies. An outbreak of ethylmercury fungicide-induced poisoning was reported by Jalili and Abbasi (1961). Affected individuals exhibited polyuria, polydypsia, and albuminuria. Two boys who ingested meat from a hog that had consumed seed treated with ethylmercuric chloride also had increased blood urea, urinary protein, and urinary sediment (Cinca et al. 1979); an autopsy revealed nephritis. A 13-month-old boy who ate porridge made from flour treated with an alkyl mercury compound (specific mercury compound not reported) experienced albuminuria, red and white cells, and casts in the urine (Engleson and Herner 1952). In autopsies carried out to evaluate the cause of death in 4 adults and 4 infants from the Iraqi epidemic of 1972, one case exhibited tubular degeneration in the kidneys (whether an adult or child was not specified) (Al-Saleem and the Clinical Committee on Mercury Poisoning 1976).

Organic mercury-induced nephrotoxicity has been demonstrated in rodents following acute-, intermediate-, and chronic-duration exposure. The usefulness of results from subchronic studies may be limited because the pathological changes observed were often not distinguished as primary or secondary effects (i.e., pathological changes secondary to induced shock). Nonetheless, they provide some useful indication of potential effects.

Administration of methylmercuric chloride to mice in a single gavage dose of 16 mg Hg/kg resulted in decreased renal function (decreased phenolsulfonphthalein excretion), increased plasma creatinine, and swelling of tubular epithelial cells, with exfoliation of the cells into the tubular lumen (Yasutake et al. 1991b). Although no effects were observed after a single gavage dose of 8 mg Hg/kg (Yasutake et al. 1991b), 5 daily gavage doses of 8 mg Hg/kg/day as methylmercuric chloride in rats resulted in vacuolization and tubular dilation in the proximal tubules with ongoing regeneration (Magos et al. 1985). Similar effects were observed after 5 doses of 8 mg Hg/kg/day as ethylmercuric chloride (Magos et al. 1985).

In an intermediate-duration study, histopathological changes were observed in the kidneys of female rats exposed to 0.86, 1.68, or 3.36 mg Hg/kg/day as methylmercury dicyanidiamide by gavage 5 days a week for 3–12 weeks (Magos and Butler 1972). The low-dose group exhibited large foci of basophilic tubular epithelial cells, desquamation, fibrosis, and inflammation in the renal cortex; however, no control group was used in the study (Magos and Butler 1972). A 12-week diet containing 0.08 mg Hg/kg/day as methyl-mercury caused ultrastructural changes (cytoplasmic masses containing ribosomes and bundles of smooth endoplasmic reticulum) in kidney proximal tubule cells of female rats, despite the normal appearance of the

glomeruli at the light microscopic level (Fowler 1972). The author concluded that these changes could be a result of metabolism to inorganic mercury and may account for proteinuria observed in exposed humans. Administration of methylmercuric chloride in the diet of mice for 26 weeks at a dose of 0.6 mg Hg/kg/day resulted in degeneration of the proximal tubules characterized by nuclear swelling and vacuolation of the cytoplasm (Hirano et al. 1986).

Rats fed daily doses of phenylmercuric acetate for up to 2 years exhibited slight-to-moderate renal damage (e.g., tubular dilatation, atrophy, granularity, fibrosis) (Fitzhugh et al. 1950). These effects were evident at doses (beginning at 0.02 mg Hg/kg/day) that were two orders of magnitude lower than those required to induce detectable effects in the mercuric acetate-treated rats (Fitzhugh et al. 1950). A NOAEL of 0.005 mg Hg/kg/day was determined. The authors concluded that some of the histological changes were present to some degree in the control animals, suggesting that low levels of mercury apparently hasten the normal degenerative processes of the kidneys (see Inorganic Mercury above). Problems in this study limit its usefulness in determining effect levels. Increased severity of renal nephrosis was also observed in another study in which rats were given 0.4 mg Hg/kg/day as phenylmercuric acetate in the drinking water for 2 years (Solecki et al. 1991). Lower doses in this study were not tested. Mice given methylmercuric chloride in the diet at a dose of 0.13 mg Hg/kg/day showed epithelial cell degeneration and interstitial fibrosis, with ongoing regeneration of the tubules present (Mitsumori et al. 1990); no effect was observed at 0.03 mg Hg/kg/day. Similar effects were seen in mice given methylmercuric chloride in the diet for 2 years at a dose of 0.11 mg Hg/kg/day (Hirano et al. 1986). Rats given methylmercuric chloride in the diet for 2 years at a dose of 0.1 mg Hg/kg/day had increased kidney weights and decreased enzymes (alkaline phosphatase, ATPase, NADH- and NADPH-oxidoreductase, and AMPase) in the proximal convoluted tubules (Verschuuren et al. 1976). However, histopathological examination revealed no treatment-related lesions.

A 2-year study conducted with mercuric acetate in the feed of rats showed an increased severity of renal damage at doses of mercury as low as 2 mg Hg/kg/day (Fitzhugh et al. 1950). Rats initially showed hypertrophy and dilation of the proximal convoluted tubules. At this stage, eosinophilia, rounding, and granular degeneration of the epithelial cells were observed. Occasionally basophilic cytoplasm and sloughing of the cells were observed. As the lesion progressed, tubular dilation increased, and hyaline casts appeared within the tubules; fibrosis and inflammation were observed. Finally, tubules appeared as cysts, and extensive fibrosis and glomerular changes were observed. However, this study was limited because

group sizes were small, survival data were not reported, and a considerable number of early deaths from pneumonia were noted.

Endocrine Effects

Inorganic Mercury. No studies were located regarding endocrine effects in humans after oral exposure to inorganic mercury.

Several studies have reported effects on the thyroid after acute- or intermediate-duration exposure to mercuric chloride. An increase in iodine release from the thyroid was observed following gavage administration of 7.4 mg Hg/kg/day as mercuric chloride to rats for 6 days (Goldman and Blackburn 1979). Serum levels of thyroid hormones (triiodothyronine and/or thyroxine) in mice decreased after administration of 6 mg Hg/kg/day as mercuric chloride or mercuric sulfide for 10 days by gavage (Sin et al. 1990). Similar effects were observed after 4 weeks of dosing with mercuric sulfide (Sin and The 1992). Administration by gavage of 5.3 mg Hg/kg/day as mercuric chloride to rats for 40 days resulted in increased thyroid weight, thyroidal iodine uptake, and protein-bound iodine in the serum (Goldman and Blackburn 1979). Decreased triiodothyronine and monoiodotyrosine were also observed. Dietary exposure of rats to 2.2 mg Hg/kg/day as mercuric chloride for 3 months resulted in decreased thyroidal iodine uptake, release, and turnover (Goldman and Blackburn 1979). Adrenocortical function was evaluated in male rats exposed to 0, 9, 18, or 36 mg Hg/kg/day as mercuric chloride in drinking water for 60–180 days (Agrawal and Chansouria 1989). A significant increase in adrenal and plasma corticosterone levels in all dose groups was observed after 120 days of exposure. After 180 days of exposure, corticosterone levels had returned to control values. The relative adrenal gland weight was significantly increased for all exposed groups compared to control values.

In a 4-week range-finding study, groups of 5 rats per sex (10 per sex for controls) received diets containing mercuric chloride at 5, 10, or 20 mg Hg/kg/day in males and 5.5, 11.1, and 22.2 mg Hg/kg/day in females. The high dose resulted in an increased relative adrenal weight in males and a decreased absolute adrenal weight in females (Jonker et al. 1993b)

Organic Mercury. No studies were located regarding endocrine effects in humans or animals after oral exposure to organic mercury.

Dermal Effects

Inorganic Mercury. Limited information was located regarding dermal effects of inorganic mercury in humans. Several children who were treated with medications containing mercurous chloride for constipation, worms, or teething discomfort exhibited flushing of the palms of the hands and soles of the feet (Warkany and Hubbard 1953). The flushing was frequently accompanied by itching, swelling, and desquamation of these areas. Morbilliform rashes, conjunctivitis, and excessive perspiration were also frequently observed in the affected children. Patch tests conducted in several children revealed that the rashes were not allergic reactions to the mercury. Kang-Yum and Oransky (1992) reported hives in a woman who ingested a Chinese medicine containing an unspecified amount of mercurous chloride, which suggests an allergic response to the medicine.

No studies were located regarding dermal effects in animals after oral exposure to inorganic mercury.

Organic Mercury. Only a few studies were identified regarding dermal effects of organic mercury, however, the case history concerning dimethylmercury exposure is a very important alert to the hazards of this organomercurial.

Blayney et al. (1997) originally reported the fatal case of a dimethylmercury exposure after a dermal exposure to an extremely small amount of material. The case history was subsequently detailed by Nierenberg et al. (1998). The exposure occurred to a 48-year-old female chemistry professor who was admitted to the hospital 5 months (154 days) after, as best as can be determined, she inadvertently spilled several drops (estimated at 0.4–0.5 mL; about 1,500 mg) of dimethylmercury from the tip of her pipette onto the back of her disposable latex gloves. The spill was cleaned and the gloves disposed of. Hair analysis on a long strand of hair revealed that after a brief lag time, mercury content rose rapidly to almost 1,100 ppm (normal level, <0.26 ppm; toxic level, >50 ppm), and then slowly declined with a half-life of 74.6 days. These results support the occurrence of one or several episodes of exposure, and are consistent with laboratory notebook accounts of a single accidental exposure. Testing of family members, laboratory coworkers, and laboratory surfaces failed to reveal any unsuspected mercury spills or other cases of toxic blood or urinary mercury levels. Permeation tests subsequently performed on disposable latex gloves similar to those the patient had worn at the time of the lone exposure revealed that dimethylmercury penetrates such gloves rapidly and completely, with penetration occurring in 15 seconds or less and perhaps instantly. Polyvinyl chloride gloves were equally permeable to dimethylmercury. Five days prior to

admission, the patient developed a progressive deterioration in balance, gait, and speech. During the previous 2 months, she had experienced brief episodes (spaced weeks apart) of nausea, diarrhea, and abdominal discomfort; and had lost 6.8 kg (15 lb). Medical examination revealed moderate upper-extremity dysmetria, dystaxic handwriting, a widely based gait, and "mild scanning speech." Routine laboratory test results were normal. Computed tomography (CT) and magnetic resonance imaging (MRI) of the head were normal except for the incidental finding of a probable meningioma, 1 cm in diameter. The cerebrospinal fluid was clear, with a protein concentration of 42 mg/dL and no cells. A preliminary laboratory report indicated that the whole-blood mercury concentration was more than 1,000 μg/L (normal range, 1–8 μg/L; toxic level, >200 μg/L). Chelation therapy with oral succimer (10 mg/kg orally every 8 hours) was begun on day 168 after exposure. Whole blood concentrations rose to 4,000 μg/L after one day of chelation, and urinary mercury levels were 234 μg/L (normal range, 1–5 μg/L; toxic level, >50 μg/L). Despite the initial success of chelation therapy, administration of vitamin E, and a blood exchange transfusion, at 176 days postexposure, the patient became comatose. Further aggressive general support and chelation therapy failed, life support ws removed (following the patient's advance directive), and the patient died 298 days post exposure. Autopsy results revealed diffusely thin cortex of the cerebral hemispheres (to 3 mm), and extensive gliosis of the visual cortex around the calcarine fissure and the superior surface of the superior temporal gyri. The cerebellum showed diffuse atrophy of both vermal and hemispheric folia. Microscope evaluation revealed extensive neuronal loss and gliosis bilaterally within the primary visual and auditory cortices, with milder loss of neurons and gliosis in the motor and sensory cortices. There was widespread loss of cerebellar granular-cell neurons, Purkinje cells, and basket-cell neurons, with evidence of loss of parallel fibers in the molecular layer. Bergmann's gliosis was well developed and widespread.

In the only other organic mercury studies identified for dermal exposures, a study of a large group of people who consumed methylmercury-contaminated bread over a 1- to 3-month period showed a dose-related history of rashes (Al-Mufti et al. 1976). These may also have been allergic responses. A 13-month-old child who ingested porridge made from flour that had been treated with an alkyl mercury compound (specific mercury compound not reported) developed a measles-like rash, fever, and facial flushing (Engleson and Herner 1952). Also, Iraqis who consumed flour made from grain treated with ethylmercury *p*-toluene sulfonanilide exhibited skin lesions consisting of pruritus on the palms, soles, and genitalia (Jalili and Abbasi 1961). In severe cases, exfoliative dermatitis of the hands and feet was also observed.

The only information located regarding dermal effects in animals after oral exposure to organic mercury comes from a study in which rats were exposed to methylmercuric chloride in the diet for 2 years (Verschuuren et al. 1976). No treatment-related lesions were observed upon histopathological examination of the skin of rats exposed to 0.1 mg Hg/kg/day.

Ocular Effects

Inorganic Mercury. No information was located regarding ocular effects in humans from ingestion of inorganic mercury.

No studies were located regarding ocular effects in animals after oral exposure to inorganic mercury.

Organic Mercury. No information was located regarding ocular effects in humans from ingestion of organic mercury. While visual effects result from methylmercury exposure, they are cortical in origin (see neurotoxicity below).

The only report of ocular effects in animals after oral exposure to organic mercury comes from a study in which rats were exposed to methylmercuric chloride via the diet for 2 years (Verschuuren et al. 1976). No treatment-related lesions were observed upon histopathological examination of the eyes of rats exposed to 0.1 mg Hg/kg/day. As in humans, the visual effects resulting from methylmercury exposure in primates are considered to be centrally mediated (Rice and Gilbert 1982, 1990).

Body Weight Effects

Inorganic Mercury. No information was located regarding body weight effects in humans from ingestion of inorganic mercury.

A single dose of mercuric chloride administered to female Sprague-Dawley rats (10/group) at 7.4 or 9.2 mg Hg/kg in water resulted in no effects on body weight at 14 days postexposure (Lecavalier et al. 1994). However, a number of animal studies have reported decreases in body weight or body weight gain after ingestion of mercuric chloride (Chang and Hartmann 1972a; Dieter et al. 1992; NTP 1993). After a 4-week exposure to mercuric chloride in the food, male Wistar rats had a 21% body weight decrease at 10 mg Hg/kg/day, and female Wistar rats had a 27% decrease in body weight at 22.2 mg Hg/kg/day. No

significant loss was observed at the next-lower-dose groups of 5 and 11.1 mg Hg/kg/day in males and females, respectively (Jonker et al. 1993b).

Doses of 14.8 mg Hg/kg/day administered to rats 5 days a week for 2 weeks resulted in a 10% decrease in male body weight gain (NTP 1993). Much lower doses produced decreases in body weight gain when administered over longer periods. In rats, decreases in body weight gain of approximately 10% were observed with doses of 0.93 mg Hg/kg as mercuric chloride when administered by gavage 5 days a week for 6 months (NTP 1993). Mice were less sensitive, showing no effect at 7.4 mg Hg/kg/day and a 26% decrease in body weight gain at 14.8 mg Hg/kg/day in the same study (NTP 1993).

Organic Mercury. No information was located regarding body weight effects in humans from ingestion of organic mercury.

A number of animal studies have reported decreases in body weight or body weight gain after ingestion of methyl or phenyl mercury. A 20–25% decrease in body weight gain in male and female rats was observed after 5 gavage doses of 8 mg Hg/kg/day as methylmercuric chloride or ethylmercuric chloride (Magos et al. 1985). In intermediate-duration studies with methylmercury, biologically significant decreases in body weight gain have been observed in rats after exposure to doses as low as 0.8 mg Hg/kg/day for 6 weeks (Chang and Hartmann 1972a) and in mice after exposure to 1 mg Hg/kg/day for 60 days (Berthoud et al. 1976). No effect on female body weight gain was observed after dietary exposure to 0.195 mg Hg/kg/day as methylmercuric chloride for 14 weeks (Lindstrom et al. 1991). A 2-year exposure to 0.4 mg Hg/kg/day as phenylmercuric acetate in the feed resulted in a 10% decrease in body weight gain in rats (Solecki et al. 1991). Gavage administration of methylmercuric chloride to rats for 2 days at 12 mg Hg/kg/day resulted in a persistent decrease in the body temperature of the rats (Arito and Takahashi 1991).

Other Systemic Effects

Inorganic Mercury. Several children who were treated with mercurous chloride contained in powders or tablets for constipation, worms, or teething discomfort exhibited low-grade or intermittent fevers (Warkany and Hubbard 1953).

No studies were located on other systemic effects in animals after oral exposure to inorganic mercury.

Organic Mercury. No studies were located regarding other systemic effects in humans or animals after oral exposure to organic mercury.

2.2.2.3 Immunological and Lymphoreticular Effects

Inorganic Mercury. No studies were located regarding immunological or lymphoreticular effects in humans after oral exposure to inorganic mercury.

The immune response to mercury exposure is complex, depending in part on the dose of mercury and the genetic characteristics of the exposed population (see Section 2.4). Administration of 14.8 mg Hg/kg/day as mercuric chloride to $B6C3F_1$ mice 5 days a week for 2 weeks resulted in a decrease in thymus weight (NTP 1993), suggesting immune suppression. However, a 2-week exposure to 0.7 mg Hg/kg/day as mercuric chloride in the drinking water resulted in an increase in the lymphoproliferative response after stimulation with T-cell mitogens in a strain of mice particularly sensitive to the autoimmune effects of mercury (SJL/N) (Hultman and Johansson 1991). In contrast, a similar exposure of a strain of mice (DBA/2) not predisposed to the autoimmune effects of mercury showed no increase in lymphocyte proliferation.

A significant suppression of the lymphoproliferative response to T-cell mitogens, concanavalin A, and phytohemagglutinin was observed in male $B6C3F_1$ mice administered 2.9 or 14.3 mg Hg/kg/day as mercuric chloride in drinking water for 7 weeks (Dieter et al. 1983). A significant decrease in the weight of the thymus and spleen and a decrease in antibody response were also exhibited at 14.3 mg Hg/kg/day. An increase in B-cell-mediated lymphoproliferation was, however, observed at both 2.9 and 14.3 mg Hg/kg/day. No immunological effects were observed at the lowest dose of 0.57 mg Hg/kg/day. When SJL/N mice were administered mercuric chloride in the drinking water for 10 weeks, an increase in circulating antinucleolar antibodies was observed at 0.28 mg Hg/kg/day, and deposition of granular IgG deposits was observed in the renal mesangium and glomerular blood vessels at 0.56 mg Hg/kg/day (Hultman and Enestrom 1992).

In rats, immune deposits have been observed in the basement membrane of the intestines and kidneys following gavage exposure to 2.2 mg Hg/kg/day as mercuric chloride twice weekly for 2 months, although no functional changes were evident in these tissues (Andres 1984). The observation of these deposits suggests that autoimmunity to specific components of these tissues has developed.

The highest NOAEL values and all reliable LOAEL values for immunological and lymphoreticular effects in each species and duration category are recorded in Table 2-2 and plotted in Figure 2-2 for inorganic mercury.

Organic Mercury. No studies were located regarding immunological effects in humans after oral exposure to organic mercury.

In BALB/c mice administered a diet containing 0.5 mg Hg/kg/day as methylmercury for 12 weeks, the thymus weight and cell number decreased by 22 and 50%, respectively, compared to the control group (Ilback 1991). The natural killer cell activity was reduced by 44 and 75% in the spleen and blood, respectively. However, the lymphoproliferative response in the spleen increased at this dose of mercury.

The LOAEL value for immunological and lymphoreticular effects in mice for intermediate-duration oral exposure to organic mercury is recorded in Table 2-3 and plotted in Figure 2-3.

2.2.2.4 Neurological Effects

Inorganic Mercury. The oral absorption of metallic mercury is negligible, and even massive doses have not resulted in neurological effects. The wo case histories identified are unusual in that the dose levels could be reasonably well quantified. The first case history reported ingestion of 15 mL (204 g) of metallic mercury by a 17-year-old male storekeeper who swallowed mercury from the pendulum of a clock (apparently out of curiosity rather than as a suicide attempt). On admission, and 24 hours later, he was symptom free, and physical examination was normal. The patient complained of no gastrointestinal symptoms, and was treated with a mild laxative and bed rest. The results of serial daily urine mercury estimates were normal (all less than 15 µg) and did not suggest significant absorption. The radiological investigation illustrated a characteristic pattern of finely divided globules of mercury in the gastrointestinal tract (Wright et al. 1980).

The second and massive incidence of ingestion involved a 42-year-old man who had spent much of his life (since the age of 13) repairing instruments that contained mercury. He intentionally ingested an estimated 220 mL (or about 3,000 g) while repairing a sphygmomanometer (Lin and Lim 1993). Upon admission, the patient presented with significantly elevated mercury blood levels (103 µg/L, normal <10 µg/L) and urine levels (73 µg/L, <20 µg/L). In the previous 2 years he had developed mild hand tremors,

forgetfulness, irritability, and fatigue. The occupational exposures made it difficult to determine any additional neurological effects from the acute exposure. There was no history of peripheral neuropathy, vertigo, insomnia, or muscular weakness. Neuropsychiatric and psychology evaluations indicated poor concentration and a defect in recent memory. EEG results indicated diffuse cortical dysfunction predominantly on the left hemisphere. He was treated with immediate gastric lavage and cathartics. He also received D-penicillamine 1 g/day orally for 7 days. Blood and urine mercury levels obtained 3 days after chelating therapy were 116.9 and 22.9μg/L, respectively. By 2 weeks postexposure, most of the mercury had been excreted in the feces and was measured at a total volume of 220 mL (this number was used to estimate the amount initially ingested). The patient was lost to follow-up, but returned to the hospital 6 months later (for glycemic control), at which time examination revealed a lessening of his hand tremors.

Most case studies of neurotoxicity in humans induced by oral exposure to inorganic mercury salts have reported neurotoxic effects as the result of ingestion of therapeutic agents that contain mercurous chloride (e.g., teething powders, ointments, and laxatives). Several children treated with tablets or powders containing mercurous chloride exhibited irritability, fretfulness, sleeplessness, weakness, photophobia, muscle twitching, hyperactive or hypoactive tendon reflexes, and/or confusion (Warkany and Hubbard 1953). A 4-year-old boy who had been given a Chinese medicine containing mercurous chloride for 3 months developed drooling, dysphagia, irregular arm movements, and impaired gait (Kang-Yum and Oransky 1992). Davis et al. (1974) reported that two women developed dementia and irritability due to chronic ingestion of a tablet laxative that contained 120 mg of USP-grade mercurous chloride (0.72 mg Hg/kg/day, assuming an average body weight of 70 kg). One woman had taken 2 tablets daily for 25 years, and the other woman took 2 tablets daily for 6 years. Both patients died from inorganic mercury poisoning, and at autopsy, low brain weight and volume and a reduced number of nerve cells in the cerebellum were seen. Light microscopic analysis revealed granules of mercury within neuronal cytoplasm. Electron microscopy revealed mercury deposits in some neurons.

In addition, neurotoxicity has been observed after ingestion of lethal doses of mercuric chloride. Blurred vision and diplopia were reported by a 35-year-old man who ingested a lethal dose of mercuric chloride (Murphy et al. 1979). Prior to death, the man experienced repeated seizures. An autopsy revealed abscesses on the occipital lobe and cerebellum.

Acute- and intermediate-duration studies describing neurotoxic effects in animals following exposure to inorganic mercury salts are limited. A study was conducted by Chang and Hartmann (1972b) in which mercuric chloride was administered both by gavage and subcutaneously. Evidence of disruption of the blood-brain barrier (i.e., leakage of dye into the brain tissue) was observed 12 hours after a single dose of 0.74 mg Hg/kg as mercuric chloride in rats (Chang and Hartmann 1972b). These investigators also administered 0.74 mg Hg/kg/day as mercuric chloride to rats for up to 11 weeks. Within 2 weeks, there were coagulative or lucid changes in cerebellar granule cells and fragmentation, vacuolation, and cytoplasmic lesions in the neurons of dorsal root ganglia. Neurological disturbances consisted of severe ataxia and sensory loss, with an accompanying loss in body weight. No conclusions regarding the oral neurotoxicity of mercuric chloride can be drawn from the results of this study because the discussion of the results observed in the study did not clearly differentiate whether the effects were observed as the result of oral or subcutaneous exposure. It is expected that mercuric chloride administered subcutaneously would be much more toxic than that administered orally because of the very poor absorption of inorganic forms of mercury from the gastrointestinal tract.

Dietary exposure of rats to 2.2 mg Hg/kg/day as mercuric chloride for 3 months resulted in inactivity and abnormal gait (Goldman and Blackburn 1979). However, it is unclear whether the effects observed in this study were the direct result of effects on the nervous system, or whether they may have been secondary to other toxic effects. No evidence of neurotoxicity (clinical signs of neurotoxicity and optic and peripheral nerve structure) was seen in mice administered 0.74 or 2.2 mg Hg/kg/day as mercuric chloride in the drinking water for 110 days (Ganser and Kirschner 1985). The investigators increased the dose administered to the low-dosed animals to 7.4–14.8 mg Hg/kg/day for an additional 400 days; however, still no neurotoxic effects were observed. Similarly, no histopathological evidence of brain lesions was observed in rats receiving gavage doses of mercuric chloride as high as 3.7 mg Hg/kg/day 5 days a week for up to 2 years or in mice receiving gavage doses as high as 7.4 mg Hg/kg/day 5 days a week for up to 2 years (NTP 1993).

The highest NOAEL values and all reliable LOAEL values for neurotoxic effects in each species and duration category are recorded in Table 2-2 and plotted in Figure 2-2 for inorganic mercury.

Organic Mercury. Most of the information concerning neurotoxicity in humans following oral exposure to organic mercury comes from reports describing the effects of ingesting contaminated fish or fungicide-treated grains (or meat from animals fed such grains). Information about doses at which the effects

occurred is frequently limited because of difficulties in retracing prior exposure and uncertainties in estimating dose levels based on assumed food intake and contamination levels.

Although isolated instances of alkyl mercury poisoning have been reported (Cinca et al. 1979; Engleson and Herner 1952), the epidemic poisonings in Japan and Iraq focused attention on the neurotoxicity of these compounds. The first reported widespread outbreak of neurological disorders associated with the ingestion of methylmercury-contaminated fish occurred in the Minamata area of Japan (Kutsuna 1968). The neurological syndrome was characterized by a long list of symptoms including prickling, tingling sensation in the extremities (paresthesia); impaired peripheral vision, hearing, taste, and smell; slurred speech; unsteadiness of gait and limbs; muscle weakness; irritability; memory loss; depression; and sleeping difficulties (Kutsuna 1968; Tsubaki and Takahashi 1986). Elevated concentrations of methylmercury were observed in the hair and brains of victims (see Section 2.5). Epidemics of similar neurological disorders were reported in Iraq in 1956 and 1960 (Bakir et al. 1973; Jalili and Abbasi 1961) as the result of eating flour made from seed grain treated with ethylmercury *p*-toluene sulfonanilide. Affected individuals had an inability to walk, cerebellar ataxia, speech difficulties, paraplegia, spasticity, abnormal reflexes, restriction of visual fields or blindness, tremors, paresthesia, insomnia, confusion, hallucinations, excitement, and loss of consciousness. In the winter of 1971–1972 in Iraq, more than 6,530 patients required hospitalization and 459 deaths occurred, usually due to central nervous system damage, after the ingestion of contaminated bread prepared from wheat and other cereals treated with a methylmercury fungicide (Bakir et al. 1973).

Al-Mufti et al. (1976) attempted to correlate symptoms of the poisoning incident with an estimate of methylmercury intake based on average levels found in grain and self-reported estimates of the number of loaves ingested. A number of assumptions were made in the estimates, and there were logistical constraints in surveying the widely spread rural population in Iraq. Moreover, only a total mercury intake was derived and compared with the results of a clinical evaluation and a survey for symptoms. Nonetheless, interesting and useful results were reported based on the 2,147 people surveyed. The mean period of exposure for the Iraqi population exposed to contaminated bread was 32 days, with some people consuming the bread for as long as 3 months. A mean of 121 loaves per person was eaten; the maximum was 480 loaves. Based on the mean number of loaves, the total intake of methylmercury was estimated at between 80 mg and 250 mg. However, those who had consumed the most loaves may have ingested up to 1,000 mg of methylmercury over a 3-month period. Of those with symptoms of alkylmercury poisoning at the time of the survey (October 1972–May 1973), 80% had eaten more than 100 loaves. Of the 75 people who had reported eating more than 200 loaves, 53 (71%) presented with some evidence of poisoning. The incidence rate for

poisoning was estimated at 271 per 1,000; this includes a mortality of 59 per 1,000, a severe disability rate of 32 per 1,000, a rate of mild or moderate disability of 41 per 1,000; and a rate for those with only a subjective evidence of poisoning of 138 per 1,000. Based on estimates of total intake, dose-related increases were observed in the incidence and severity of paresthesia, astereognosis (loss of the ability to judge the form of an object by touch), persistent pain in the limbs, persistent headaches, difficulty walking, difficulty using the arms, and changes in speech, sight, and hearing. The most commonly observed symptom was paresthesia, most frequently involving the extremities but also on the trunk and the circumoral region. Difficulty walking and a feeling of weakness were the next most common symptoms. The total estimated intake in total milligrams associated with the four categories (no evidence of poisoning, subjective evidence, mild to moderate evidence, and severe symptoms) is as follows for all ages combined (number of persons in parentheses): 95 mg (n=59), 141 mg (n=131), 160 mg (n=35), 173 mg (n=22). This dose range is small for such dramatically different health states, and does not widen when the data are evaluated by age group. Interestingly, the total intake associated with severity of symptoms decreases on a mg/kg body weight basis with increasing age in contrast with what would be expected if children were more susceptible. For example, intakes (mg/kg over the total exposure period) associated with severe symptoms are as follows for the age groups 5–9 years, 10–14 years, and 15 years and older, respectively: 7.8 mg/kg (n=9), 4 mg/kg (n=7), and 3.6 mg/kg (n=6). Comparable numbers are for the mild/moderate symptoms and the subjective symptoms (shown): 6 mg/kg (n=19), 3.4 mg/kg (n=20), and 2.4 mg/kg (n=92). It is possible that child sensitivity may not be as large a factor when exposures reach the levels experienced in Iraq.

Neurotoxic effects seen in the Minamata (Japan) and Iraqi poisonings have been associated with neuronal degeneration and glial proliferation in the cortical and cerebellar gray matter and basal ganglia (Al-Saleem and the Clinical Committee on Mercury Poisoning 1976), and derangement of basic developmental processes such as neuronal migration (Choi et al. 1978; Matsumoto et al. 1965) and neuronal cell division (Sager et al. 1983). In the brain, Purkinje, basket, and stellate cells were severely affected. Granule cells were variably affected. Sural nerves removed from two women with neurotoxicity associated with the Minamata incident also showed evidence of peripheral nerve degeneration and regeneration (Miyakawa et al. 1976).

Similar effects have been observed in persons ingesting meat contaminated with ethylmercuric chloride (Cinca et al. 1979). Neurotoxic signs observed in two boys who ultimately died as the result of the exposure included gait disturbance, ataxia, dysarthria, dysphagia, aphonia, hyperreactive tendon reflexes,

hypotonia, spasticity, mydriasis, horizontal nystagmus, agitation, and coma. Electroencephalography showed decreased alpha activity and increased slow-wave activity. Autopsy showed nerve cell loss and glial proliferation in the cerebral cortex (calcarine cortex, midbrain, bulbar reticular formation), demyelination, granule cell loss in the cerebellum, and motor neuron loss in the ventral horns of the spinal cord. Neurotoxic signs in the surviving family members were generally similar (ataxia, gait impairment, spasticity, drowsiness, intentional tremor, agitation, hypoesthesia in the limbs, speech difficulties, and visual disturbances); all but the narrowing of the visual fields resolved after termination of exposure.

A New Mexico family, including a pregnant woman, a 20-year-old female, and 2 children (a 13-year-old male and an 8-year-old female) ate meat from a hog inadvertently fed seed grain treated with a fungicide containing methylmercury and experienced severe, delayed neurological effects (Davis et al. 1994). Several months after the exposures, the children developed symptoms of neurological dysfunction. The newborn child of the exposed mother showed signs of central nervous system disorder from birth. Twenty-two years after the 3-month exposure period, the people who were 20 and 13 years old at time of exposure had developed cortical blindness or constricted visual fields, diminished hand proprioception, choreoathetosis, and attention deficits. MRI examination of these two revealed residual brain damage in the calcarine cortices, parietal cortices, and cerebellum. The brain of the person who was exposed at age 8 (who died of aspiration pneumonia with a superimposed *Klebsiella* bronchopneumonia and sepsis at age 29) showed cortical atrophy, neuronal loss, and gliosis, most pronounced in the paracentral and parieto-occipital regions. Regional brain mercury levels correlated with the extent of brain damage. The youngest (*in utero* at the time of exposure) developed quadriplegia, blindness, severe mental retardation, choreoathetosis, and seizures, and died at age 21. The inorganic mercury levels in different regions of the brain of the 29-year-old patient ranged from 82 to 100% of the total mercury present. Since inorganic mercury crosses the blood-brain barrier poorly, biotransformation of the methylmercury to inorganic mercury may have occurred after the methylmercury crossed the blood-brain barrier, accounting for its observed persistence in the brain and its possible contribution to the brain damage.

LeBel et al. (1996) studied early nervous system dysfunction in Amazonian populations exposed to low levels of methylmercury. A preliminary study was undertaken in two villages on the Tapajos River, an effluent of the Amazon, situated over 200 km downstream from the methylmercury extraction areas. The study population included 29 young adults $35 years (14 women and 15 men) randomly chosen from a previous survey. Hair analyses were conducted with cold vapor atomic fluorescence spectrophotometry. Total hair Hg (THg) varied between 5.6 µg/g and 38.4 µg/g, with MeHg levels from 72.2% to 93.3% of

the THg. A quantitative behavioral neurophysiological test battery, designed for use under standard conditions in an area without electricity and for persons with minimal formal education was administered to all participants. The results of visual testing showed that although all participants had good near and far visual acuity, color discrimination capacity (Lanthony D-15 desaturated panel) decreased with increasing THg ($F=4.1$; $p=0.05$); near visual contrast sensitivity profiles (Vistech 6000) and peripheral visual field profiles (Goldman Perimetry with Targets I and V) were reduced for those with the highest levels of THg. For the women, manual dexterity (Santa Ana, Helsinki version) decreased with increasing THg ($F=16.7$; $p<0.01$); this was not the case for the men. Although the women showed a tendency towards reduced grip strength, muscular fatigue did not vary with THg for either sex. The authors claim that this study demonstrates that it is possible, using a sensitive test battery, to detect alterations in nervous system functions, consistent with knowledge of Hg toxicity, at levels below the currently recognized threshold of 50 μg/g THg.

Mental retardation has not generally been reported as a neurotoxic effect of alkyl mercurial exposure in adults. However, a 9-month-old infant who received porridge made from alkyl mercury-contaminated grains for approximately 4 months lost the ability to crawl or walk and exhibited persistent mental retardation (Engleson and Herner 1952). These effects are similar to those seen in infants born to mothers who consumed methylmercury-contaminated food during pregnancy (see Section 2.2.2.6), suggesting that in addition to the prenatal period, infancy may also be a susceptible period for the development of these types of effects.

Studies in experimental animals also indicate that organic mercury is a potent neurotoxicant. Adult female monkeys *(Macaca fascicularis)* were exposed to methylmercury (0.050 mg Hg/kg/day) in apple juice by mouth for 6, 12, or 18 months, or 12 months followed by 6 months without exposure (clearance group). A fifth group of monkeys was administered mercuric chloride (0.200 mg Hg/kg/day) by constant-rate intravenous infusion through an in-dwelling catheter for 3 months. Controls were housed and handled with the exposed monkeys, but were not administered mercury. The number of neurons, astrocytes, reactive glia, oligodendrocytes, endothelia, and pericytes in the cortex of the calcarine sulcus was estimated by use of the optical volume fractionator stereology technique. Reactive glia showed a significant increase in number for every treatment group, increasing 72% in the 6-month, 152% in the 12-month, and 120% in the 18-month methylmercury-exposed groups, and the number of reactive glia in the clearance group remained elevated (89%). In the mercuric chloride group, there was a 165% increase in the number of reactive glia. Neurons, astrocytes, oligodendrocytes, endothelia, and pericytes showed no significant change in number in

any exposure group. The methylmercury-treated monkeys (all groups) appeared normal in terms of cage behavior throughout the entire exposure period, supporting the conclusion that there was no significant loss in the neuron population. Examination of tissue samples did not reveal any apparent degradation in the structure of neurons or chronic changes in the glial cells (e.g., the appearance of hypertrophic astrocytes), which are commonly observed following exposure to high levels of mercury. No apparent dilation of the perivascular spaces was observed. The average volume of the cortex of the calcarine sulcus did not differ significantly from controls for any methylmercury-treated group. The methylmercury-clearance group had low levels of methylmercury present in tissues; however, the level of inorganic mercury was also elevated. The astrocytes and microglia in the methylmercury group contained the largest deposits of inorganic mercury. Comparing the results of the methylmercury and inorganic mercury groups suggests that inorganic mercury may be responsible for the increase in reactive glia (Charleston et al. 1994).

Charleston et al. (1996) studied the effects of long-term subclinical exposure to methylmercury on the number of neurons, oligodendrocytes, astrocytes, microglia, endothelial cells and pericytes within the thalamus from the left side of the brain of the monkey *Macaca fascicularis.* These parameters were determined by use of the Optical Volume Fractionator stereological method. The accumulated burden of inorganic mercury (IHg) within these same cell types has been determined by autometallographic methods. Four groups of female monkeys (n=4-5) were exposed to 50 µg Hg/kg/day methylmercury in apple juice for 6, 12, or 18 months, or 12 months followed by 6 months without exposure (clearance group). One control animal each was sacrificed with the 6- and 12-month exposure groups, and two additional animals were sacrificed at the end of the experiment. All monkeys appeared normal—no changes in behavior or motor skills were observed. Hematological function (white blood cell count and differentiation, erythrocyte count, hemoglobin, hematocrit, and red cell indices) and blood chemistry (urea nitrogen, creatine, bilirubin, albumin, total protein, alkaline phosphatase, and electrolytes) were normal. No weight loss was observed. Neurons, oligodendrocytes, endothelia, and pericytes did not show a significant change in cell number for any exposure group. Astrocyte cell number exhibited a significant decline for both the 6-month (44.6%) and clearance exposure groups (37.2%); decreased astrocyte counts were also observed in the other exposure groups, but these were not significant. The microglia, in contrast, showed a significant increase in the 18-month (228%) and clearance exposure groups (162%). Results from mercury speciation and quantification analysis of contralateral matched samples from the thalamus of the right side of the brain from these same monkeys indicated that methylmercury concentrations plateaued at around 12 months exposure, whereas the inorganic levels, presumably derived from demethylation of methylmercury, continued to increase throughout all exposure durations. Autometallographic determination of the

distribution of IHg by cell type indicates that both the astrocytes and microglia contain substantially elevated IHg deposits relative to all other cell types. The data suggest that the inorganic mercury present in the brain, accumulating after long-term subclinical methylmercury exposure, may be a proximate toxic form of mercury responsible for the changes within the astrocyte and microglial populations.

Rice (1996a) evaluated delayed neurotoxicity produced by methylmercury in monkeys treated with methylmercury from birth to 7 years of age. When these monkeys reached 13 years of age, individuals began exhibiting clumsiness not present previously. Further exploration revealed that treated monkeys required more time to retrieve treats than did nonexposed monkeys and displayed abnormalities on a clinical assessment of sense of touch in hands and feet, despite the fact that clinical examinations performed routinely during the period of dosing had not yielded abnormal results. Another group of monkeys, dosed from *in utero* to 4 years of age, also took longer to retrieve treats when assessed years after cessation of exposure. These observations were pursued in both groups of monkeys by objective assessment of somatosensory function in the hands: both groups of monkeys exhibited impaired vibration sensitivity. The results suggest that a delayed neurotoxicity occurred when these monkeys reached middle age. The author notes persons with Minamata disease also have symptoms of delayed neurotoxicity. The results from a study of more than 1,100 Minamata patients over the age of 40 indicated a difficulty in performing daily activities that increased as a function of age compared to matched controls. Methylmercury may represent the only environmental toxicant for which there is good evidence for delayed neurotoxicity observable many years after cessation of exposure.

Rice (1996b) further compares the sensory and cognitive effects of developmental methylmercury exposure in monkeys to the effects in rodents. Developmental exposure to methylmercury in the Macaque monkey produced impairment of function in the visual, auditory, and somatosensory systems. In addition, delayed neurotoxicity was observed in monkeys years after cessation of dosing, manifested as overall clumsiness and slowness in reaching for objects. The effects of developmental methylmercury exposure on cognitive function in monkeys are more equivocal; both positive and negative results have been obtained, with no obvious pattern with regard to possible domains of impairment. Prenatal methylmercury exposure in rodents produced retarded development and impairment of motor function, while the evidence for cognitive impairment is less consistent. Derivation of reference doses based on these data from monkeys and rodents is remarkably congruent, and is virtually identical to values derived from evidence for developmental impairment in humans. Research needs include determination of neurotoxic effects at lower body burdens

in the monkey, including dose-effect data, and a more systematic exploration of the pattern of behavioral deficits in both primates and rodents.

Gilbert et al. (1996) used fixed interval/fixed ratio performance in adult monkeys to evaluate effects from exposure *in utero* to methylmercury. The fixed interval/fixed ratio (FI/FR) schedule is considered to be a sensitive indicator of neurotoxicity. In the present study, monkeys (*Macaca fascicularis*) were exposed *in utero* to methylmercury. Maternal doses of methylmercury of 0, 50, 70, or 90 µg/kg/day (in apple juice) (n=11, 9, 2, and 2, respectively) resulted in infant blood mercury levels at birth ranging from 1.04 to 2.45 ppm. Monkeys were tested on a multiple FI/FR schedule of reinforcement at 8–10 years of age. Four FI/FR cycles were run per session. Pause time and run rate were calculated for FI and FR components, as well as FI quarter-life and local FI response rates. Methylmercury treatment and sex effects were investigated by fitting a linear orthogonal polynomial regression to each monkey's profile across sessions and performing two-way ANOVAs on the resulting linear and intercept terms. Results from all treated monkeys were combined and compared to the control group. There were no treatment-related effects on either the fixed interval (FI) or fixed ratio (FR) component for pause time or run rate. Analysis of the quarter-life revealed a significant treatment by sex effect as well as a main effect for sex. Post hoc t-tests revealed a significant difference in quarter-life of treated male and female monkeys and a marginal difference between treated and control males. The FI run rate of the male monkeys was significantly greater than that of the female monkeys whereas the FR run rate of the males was marginally greater. These results indicate that there may be a differential effect of methylmercury on male and female monkeys, which could be interpreted as an effect on temporal discrimination. The authors concluded that adult monkeys exposed to *in utero* methylmercury exhibited very limited sex-related effects on the FI/FR intermittent schedule of reinforcement.

Typical neurotoxic signs observed in rats exposed to methylmercury include muscle spasms, gait disturbances, flailing, and hindlimb crossing (Fuyuta et al. 1978; Inouye and Murakami 1975; Magos et al. 1980, 1985). These effects have been observed after acute-duration gavage dosing with methylmercury concentrations at doses as low as 4 mg Hg/kg/day for 8 days (Inouye and Murakami 1975) and may not be observed until several days after cessation of dosing (Inouye and Murakami 1975; Magos et al. 1985). Histopathological examination of the nervous systems of affected rats has shown degeneration of cerebellar granule cells and dorsal root ganglia (Magos et al. 1980, 1985) and degenerative changes in peripheral nerves (Fehling et al. 1975; Miyakawa et al. 1974, 1976). Comparison of the effects of 5 doses of 8 mg Hg/kg/day as ethyl- or methylmercury showed dorsal root damage as well as flailing and hindlimb

crossing after exposure to both chemicals, but only methylmercury caused substantial cerebellar damage (Magos et al. 1985). Additional changes in rats exposed to methylmercury have also been observed. Rats exposed to a single gavage dose of 19.9 mg Hg/kg as methylmercuric chloride were found to have statistically significant differences in open-field tests, such as decreases in standing upright, area traversed, and activity, compared to controls. However, no accompanying histopathological changes were observed (Post et al. 1973). The exposed animals were also lethargic and ataxic initially, but symptoms disappeared within 2–3 hours. Changes in the phases of sleep were also observed in rats given 2 doses of 4 mg Hg/kg/day as methylmercuric chloride (Arito and Takahashi 1991). Paradoxical sleep was decreased and slow-wave sleep was increased. At a higher dose (12 mg Hg/kg/day for 2 days), circadian sleep patterns were also disrupted. Administration of a single dose of methylmercuric chloride (0.8 mg Hg/kg) produced blood-brain barrier dysfunction in rats (Chang and Hartmann 1972b) similar to that reported for inorganic mercury as discussed previously. In rabbits given 5.5 mg Hg/kg as methylmercuric acetate for 1–4 days, widespread neuronal degenerative changes (in cervical ganglion cells, cerebellum, and cerebral cortex) have been observed without accompanying behavioral changes (Jacobs et al. 1977).

Longer-duration studies in animals have shown qualitatively similar effects, but generally at lower daily doses with increasing durations of exposure. Rats given a dose of 10 mg Hg/kg as methylmercuric chloride once every 3 days for 15 days showed degeneration in the cerebellum with flailing and hind leg crossing (Leyshon and Morgan 1991). Rats given a TWA dose of 2.1 mg Hg/kg/day as methylmercury iodide or 2.4 mg Hg/kg/day as methylmercury nitrate by oral gavage for 29 days became weak and severely ataxic and developed paralysis of the hind legs (Hunter et al. 1940). Severe degeneration of peripheral nerves, posterior spinal roots, and trigeminal nerves were reported. Severe degenerative changes were also observed in the dorsal root fibers of rats given 1.6 mg Hg/kg/day as methylmercuric chloride for 8 weeks (Yip and Chang 1981). Similarly, ataxia (beginning the second week of exposure) and cerebellar edema and necrosis occurred in rats after 7 weeks of exposure by gavage to 1.68 mg Hg/kg as methylmercury dicyanidiamide for 5 days a week (Magos and Butler 1972). When rats were administered 0.8 mg Hg/kg/day as methylmercuric chloride by gavage for up to 11 weeks, effects similar to those reported for mercuric chloride (e.g., neuronal degeneration of the cerebellum and dorsal root ganglia and neurotoxic clinical signs) were seen but with increased severity (Chang and Hartmann 1972a).

Mice have shown comparable effects at similar doses. Mice exposed to 1.9 or 9.5 mg Hg/kg/day as methylmercury in the drinking water for 28 weeks exhibited degeneration of Purkinje cells and loss of granular cells in the cerebellum (MacDonald and Harbison 1977). At the higher of these doses, hind limb

paralysis was observed as early as 8 days, whereas at 1.9 mg Hg/kg/day, decreases in motor activity and hind limb paralysis did not develop until 24 weeks of exposure. Interestingly, cerebellar lesions were observed at 1.9 mg Hg/kg/day as early as 8 days after the start of dosing. Neuronal degeneration and microgliosis were observed in the corpus striatum, cerebral cortex, thalamus, and hypothalamus, accompanied by hind leg weakness, in mice administered 1 or 4 mg Hg/kg/day as methylmercuric chloride by gavage for 60 days (Berthoud et al. 1976). Similarly, a marked neurotoxic disturbance (not further identified) was reported in mice that received 3.1 mg Hg/kg/day as methylmercuric chloride in the diet for 26 weeks (Mitsumori et al. 1981). No effects of this type were observed in this study at 1.6 mg Hg/kg/day, but it is unknown whether more subtle neurological effects may have been missed, as the intent of this study was not to identify neurotoxic effects of methylmercury.

Some studies suggest that cats and monkeys are more sensitive to the neurotoxic effects of organic mercury than rodents. Cats fed tuna contaminated with methylmercury at doses equivalent to 0.015 mg Hg/kg/day for 11 months, starting when the cats were kittens, displayed degenerative changes in the cerebellum and the cerebral cortex (Chang et al. 1974). However, only 3 of 16 animals exhibited incoordination and weakness. Similarly, cats given gavage doses of methylmercuric chloride as low as 0.25 mg Hg/kg/day for 44–243 days displayed degenerative lesions in the granule and Purkinje cells of the cerebral cortex and/or cerebellum and degenerative changes in the white matter, but no manifestations of neurotoxicity (ataxia, loss of righting reflex, visual and sensory impairments) were observed until 0.5 mg Hg/kg/day was given (Khera et al. 1974). Neonatal monkeys given 0.5 mg Hg/kg/day as methylmercuric chloride in infant formula for 28–29 days exhibited stumbling and falling prior to termination of exposure (Willes et al. 1978). Despite the termination of exposure, abnormalities in the several reflexes; blindness; abnormal behavior consisting of shrieking, crying, and temper tantrums; and coma developed. Histopathological analyses showed diffuse degeneration in the cerebral cortex (especially the calcarine, insular, pre-, and postcentral gyri, and occipital lobe), cerebellum, basal ganglia, thalamus, amygdala, and lateral geniculate nuclei. Macaque monkeys exposed to methylmercuric chloride in biscuits exhibited tremors and visual field impairment (Evans et al. 1977). These effects were observed in animals that were first administered 4–5 priming doses of 1 mg Hg/kg at 5-day intervals (no toxicity observed), followed by "maintenance" doses of 0.5–0.6 mg Hg/kg once a week for 87–256 days. Squirrel monkeys developed similar symptoms after receiving a single priming dose of 1 or 2 mg Hg/kg as methylmercuric chloride by gavage, followed 77 days later by maintenance doses of 0.2 mg Hg/kg once a week for 90–270 days (Evans et al. 1977). The doses were adjusted to maintain steady-state blood mercury levels in the range of 1–4 ppm. No tremors or convulsions were observed in female monkeys (*Macaca fascicularis*) during a 150-day exposure

to methylmercury chloride at 0.04 mg Hg/kg/day (Petruccioli and Turillazzi 1991). However, beginning at 177–395 days after exposure to methylmercuric hydroxide at 0.08 mg Hg/kg/day, 6 of 7 female monkeys (*Macaca fascicularis*) exhibited slight tremors and decreased sucking responses, followed by claw-like grasp, gross motor incoordination, and apparent blindness (Burbacher et al. 1984, 1988). These effects were also observed in one animal from each of the lower-dose groups (0.04 and 0.06 mg Hg/kg/day) (Burbacher et al. 1988).

Miyama et al. (1983) attempted to correlate electrophysiological changes with "early" neurological signs in rats during dietary exposure to methylmercuric chloride for an unspecified period of time. They observed the following sequence in the onset of electrophysiological-somatic signs: fall in compound action potential > decrease in sensory nerve conduction velocity > tail rotation > weight loss. However, varying doses of selenium were co-administered with the methylmercury, complicating the interpretation of these results.

Evidence for a neurochemical component of methylmercury-induced toxicity following intermediate-duration exposures has been reported (Concas et al. 1983; Sharma et al. 1982; Tsuzuki 1981). A depression in the synthesis of the neurotransmitter, dopamine (whole-brain levels), was observed in the absence of clinical signs of neurotoxicity in rats fed doses as low as 0.8 mg Hg/kg/day as methylmercuric chloride once every 3 days for 15 days (Sharma et al. 1982). An increased number (but not an affinity) of benzodiazepine receptor binding sites and a decreased content of cyclic guanosine monophosphate (cGMP) were observed in the cerebellar cortex of rats administered 3.92 mg Hg/kg/day as methylmercuric chloride in the drinking water for 20 days (Concas et al. 1983). Activities of several enzymes associated with central neurotransmitter metabolism in the cerebellum (e.g., acetylcholinesterase, tryptophan hydroxylase, monoamine oxidase, catechol-*o*-methyltransferase) were depressed in rats administered 3.2 mg Hg/kg/day as methylmercury by gavage for 50 days (Tsuzuki 1981). These findings suggest that an alteration in neurotransmission may be one mechanism of action for mercury-induced neurotoxicity. However, the observed effects on the neurotransmitters may be secondary to other effects on the nervous system.

The chronic neurotoxic effects of methylmercury in animals are similar to those seen after intermediate exposure. Mice administered methylmercuric chloride in the diet for 2 years at approximately 0.6 mg Hg/kg/day showed posterior paralysis and sensory neuropathy, characterized by cerebral and cerebellar necrosis, as well as degeneration of spinal dorsal nerve roots and the sciatic nerve (Hirano et al. 1986; Mitsumori et al. 1990). Cats fed contaminated fish or contaminated fish and methylmercury at doses

as low as 0.046 mg Hg/kg/day for 2 years exhibited neurobehavioral toxic signs, including mild impairment of motor activity and diminished sensitivity to pain (Charbonneau et al. 1976). These effects began after 60 weeks of exposure but did not progress during the remainder of the 2 years of exposure. At higher doses of 0.074 and 0.18 mg Hg/kg/day, ataxia, alterations in gait, motor incoordination, muscle weakness, changes in temperament, and convulsions were also observed. Histopathological analyses showed neuronal degeneration in the motor, sensory, auditory, and occipital cortices and cerebellar granule cell degeneration. Five monkeys fed 0.05 mg Hg/kg/day as methylmercuric chloride from birth until the age of 3–4 years displayed impaired spatial vision at that time (Rice and Gilbert 1982). Continued dosing until 6.5–7 years of age resulted in clumsiness, decreased fine motor performance, and insensitivity to touch when tested at 13 years of age (Rice 1989c). Impaired high-frequency hearing was also displayed by these monkeys when tested at 14 years of age (Rice and Gilbert 1992). It is noteworthy that a wide range of neurotoxic symptoms (motor, visual, and auditory) were observed in a species similar to humans several years after dosing had ceased. No clinical signs or histopathological evidence of neurotoxicity was observed in rats that received 0.1 mg Hg/kg/day as methylmercuric chloride in the diet for 2 years (Verschuuren et al. 1976).

Deficiencies in many of the studies make it difficult to fully evaluate the quality of the data reported. General problems include the following: (1) many details of experimental protocols were omitted, thereby prohibiting an evaluation of the study's adequacy; (2) very often, only one dose was used, so an analysis of any possible dose-response relationships was not possible, and the possibility that certain observed effects were not compound-related cannot be excluded; (3) control data often were not presented; and (4) the results were frequently described in subjective terms, and no attempt was made to quantitate the data. Despite these limitations, animal studies do provide irrefutable evidence that the central and peripheral nervous systems are target organs for organic mercury-induced toxicity.

In summary, methylmercury is neurotoxic to humans and to several species of experimental animals following acute, intermediate, and chronic oral exposure. The major effects that are seen across the studies include motor disturbances, such as ataxia and tremors, as well as signs of sensory dysfunction, such as impaired vision. The predominant neuropathological feature is degenerative changes in the cerebellum, which is likely to be the mechanism involved in many of the motor dysfunctions. In humans, disruptions of higher functions have also been noted, as evidenced by depression and irritability.

The highest NOAEL values and all reliable LOAEL values for neurotoxic effects in each species and duration category are listed in Table 2-3 and plotted in Figure 2-3 for organic mercury.

2.2.2.5 Reproductive Effects

Inorganic Mercury. In an attempt to terminate her pregnancy, a 31-year-old woman ingested 30 mg Hg/kg as mercuric chloride in week 10 of her pregnancy (Afonso and deAlvarez 1960). Despite gastric lavage and treatment with dicapmerol, 13 days after exposure vaginal bleeding and uterine cramps occurred, followed by spontaneous abortion of the fetus and placenta. It was inconclusive whether the abortion was directly due to the mercury exposure.

Organic Mercury. No studies were located regarding reproductive effects in humans following oral exposure to organic mercury.

Abortions and decreased mean litter size are the predominant reproductive effects in different species of animals following oral exposure to organic mercury. Groups of 30 pregnant Fischer 344 rats were orally administered 10, 20, or 30 mg/kg as methylmercuric chloride dissolved in saline on Gd 7. Controls were given saline only (n=30). Maternal body weight gain and deaths were monitored. On Gd 20, the dams were laparotomized under ether anesthesia, and the fetuses were removed. Surviving fetuses were examined for gross toxic effects, sex, and weight; half were stained for skeletal examination. Mercury levels in maternal and fetal organs were measured. The LD_{50} of methylmercuric chloride for fetuses was calculated. Maternal body weights were decreased for 2 days in rats given 10 mg/kg, for 6 days in rats given 20 mg/kg, and were continuously decreased for rats given 30 mg/kg methylmercuric chloride. Survival rates of fetuses were 19.2, 41.4, and 91.1% less than controls for the 10, 20, and 30 mg/kg methylmercuric chloride groups, respectively. Implantation sites in the 3 groups decreased by 5.9, 13.7, and 22.5%, respectively, compared with controls. Preimplantation losses in the 3 groups were 17.2, 24.8, and 30.1%, respectively; postimplantation losses were 16.7, 34.1, and 88.9%, respectively. The reduction of litter weight was greatly enhanced with increasing methylmercuric chloride doses (32.3, 67.0, and 89.2%, respectively), presumably due to postimplantation loss, which already increased at high treatment levels. The LD_{50} of methylmercuric chloride for fetuses was determined to be 16.5 mg/kg. Mercury content in maternal organs was highest in the kidneys, followed by blood, spleen, liver, and brain, while in fetal organs it was highest in the liver (Lee and Han 1995).

Pregnant hamsters that received a single oral gavage dose of mercuric acetate on Gd 8 showed an increase in the incidence of resorptions at doses as low as 22 mg Hg/kg (Gale 1974). The incidence of resorptions was 35% at 22 mg Hg/kg, and increases were observed in a dose-related manner (53% at 32 mg Hg/kg, 68% at 47 mg Hg/kg, and 99% at 63 mg Hg/kg).

In a study by Khera (1973), after 5–7 days of oral gavage doses of 1, 2.5, or 5 mg Hg/kg/day as methylmercuric chloride, male rats were mated to unexposed female rats. A dose-related reduction of mean litter size was attributed to preimplantation losses from incompatibility of sperm-to-implantation events after mercury treatment of the parent male rat. At doses of 2 mg Hg/kg/day as methylmercury by gavage during Gd 6–9, pregnant Sprague-Dawley rats showed no differences in maternal body weight gain before parturition or in the body weights of the offspring (Fredriksson et al. 1996).

In male mice, no reduction in the incidence of fertile matings was observed after administration of 5–7 oral doses of up to 5 mg Hg/kg/day as methylmercuric chloride (Khera 1973). There was a significant dose-related decrease in the number of pups born per litter in mice receiving oral doses of 3, 5, or 10 mg Hg/kg administered on Gd 8 as methylmercuric hydroxide (Hughes and Annau 1976). Effects were not observed at 2 mg Hg/kg/day. Similarly, female mice administered 20 mg Hg/kg/day as methylmercuric chloride by gavage on Gd 10 had increased resorptions, decreased live fetuses per litter, and decreased numbers of fetuses per litter (Fuyuta et al. 1978). After guinea pigs were exposed to 11.5 mg Hg/kg as methylmercuric chloride by gavage on Gd 21, 28, 35, 42, or 49, half of the litters were aborted 4–6 days after treatment (Inouye and Kajiwara 1988). An increased rate of reproductive failure due to decreased conceptions and increased early abortions and stillbirths occurred in female monkeys exposed to 0.06 or 0.08 mg Hg/kg/day as methylmercury for 4 months (Burbacher et al. 1988). The menstrual cycle length was not affected at these dose levels. Reproductive effects were not observed in monkeys exposed to 0.04 mg/kg/day for the same duration.

Testicular functions were studied in monkeys *(M. fascicularis)* exposed to 0.025 or 0.035 mg Hg/kg/day as methylmercury by gavage for 20 weeks (Mohamed et al. 1987). The mean percentage of motile spermatozoa and the mean sperm speed were significantly decreased for both treatment groups compared to controls. Morphological examination of semen smears indicated an increased incidence of tail defects (primarily bent and kinked tails) in both exposed groups. No histopathological effects were evident on the testes. The study was limited because there were only three animals in each exposure group.

Testicular effects were also observed after chronic-duration exposure to methylmercuric chloride. Tubular atrophy of the testes was observed in mice ingesting 0.69 mg Hg/kg/day in their feed for up to 2 years (Mitsumori et al. 1990). Decreased spermatogenesis was observed in mice receiving 0.73 mg Hg/kg/day in the diet (Hirano et al. 1986). No adverse effects on the testes were observed in these studies at 0.14–0.15 mg Hg/kg/day. Similarly, no adverse effects were observed in the testes, prostate, ovaries, or uterus of rats exposed through the diet to 0.1 mg Hg/kg/day as methylmercuric chloride for 2 years (Verschuuren et al. 1976).

The highest NOAEL values and all reliable LOAEL values for reproductive effects in each species and duration category are recorded in Table 2-3 and plotted in Figure 2-3 for organic mercury.

2.2.2.6 Developmental Effects

Inorganic Mercury. No studies were located regarding developmental effects in humans or animals following oral exposure to inorganic mercury.

Organic Mercury. When grains treated with fungicides containing mercury have been accidentally consumed or when fish with high levels of methylmercury have been eaten, epidemics of human mercury poisonings have occurred with high incidences of developmental toxicity. These episodes, as well as case reports from isolated incidences of maternal consumption of organic forms of mercury during pregnancy, have provided evidence that the developing nervous system of the fetus is highly sensitive to mercury toxicity. The first such incident was reported in Sweden in 1952 when flour from grain treated with an unspecified alkyl mercury compound ingested by a pregnant woman was associated with developmental toxicity. An apparently normal infant was born, but the infant later displayed brain damage manifested by mental retardation, incoordination, and inability to move (Engleson and Herner 1952). A 40-year-old woman, 3 months pregnant, consumed methylmercury-contaminated meat for an unspecified duration and subsequently delivered a male infant with elevated urinary mercury levels (Snyder and Seelinger 1976). At 3 months, the infant was hypotonic, irritable, and exhibited myoclonic seizures. At 6 years of age, the child displayed severe neurological impairment (e.g., blindness, myoclonic seizures, neuromuscular weakness, inability to speak) (Snyder and Seelinger 1976). In the 1955 mercury poisoning outbreak in Minamata, Japan, severe brain damage was described in 22 infants whose mothers had ingested fish contaminated with methylmercury during pregnancy (Harada 1978). The types of nervous system effects described in the Minamata outbreak included mental retardation; retention of primitive reflexes; cerebellar symptoms;

dysarthria; hyperkinesia; hypersalivation; atrophy and hypoplasia of the cerebral cortex, corpus callosum, and granule cell layer of the cerebellum; dysmyelination of the pyramidal tracts; and an abnormal neuronal cytoarchitecture. It has been suggested that the widespread damage involved derangement of basic developmental processes, such as neuronal migration (Choi et al. 1978; Matsumoto et al. 1965) and neuronal cell division (Sager et al. 1983).

Large-scale poisonings also occurred in Iraq in 1956 and 1960 (Bakir et al. 1973). Thirty-one pregnant women were victims of poisoning; 14 women died from ingesting wheat flour from seeds treated with ethylmercury *p*-toluene sulfonanilide (Bakir et al. 1973). Infants were born with blood mercury concentrations of 250 μg/100 mL and suffered severe brain damage. Similar cases of severe brain damage resulting from prenatal exposure to methylmercury were reported in an outbreak of methylmercury poisoning in Iraq occurring in 1971–1972 (Amin-Zaki et al. 1974). Attempts to correlate symptoms with exposure levels have shown that a dose-response relationship exists between the severity of the neurological symptoms in offspring and the maternal intake of methylmercury (as determined using analysis of hair for mercury content) (Cox et al. 1989; Marsh et al. 1980, 1981, 1987). Delays in walking and talking were more often associated with lower peak hair levels during pregnancy than were mental retardation and seizures (Marsh et al. 1981, 1987). These studies showed that the most severely affected children had been exposed to methylmercury during the second trimester of pregnancy. Male offspring were more severely affected than female offspring. Neurological abnormalities have also been observed among offspring of Cree Indians in Quebec, Canada, exposed to methylmercury in fish (McKeown-Eyssen et al. 1983).

A significant correlation was observed between male offspring with abnormal muscle tone or reflexes and maternal prenatal exposure (as determined using hair levels). An analysis of peak hair mercury levels during pregnancy in mothers exposed during the 1971–1972 outbreak in Iraq has led to an estimated population threshold of 10 ppm (highest value during pregnancy, for total mercury in hair) associated with delays in the onset of walking in infants (Cox et al. 1989). However, this estimated threshold for the Iraqi population depends heavily on the assumed background frequency for abnormal onset of walking time, as well as the threshold chosen to define onset of walking as abnormal. Furthermore, most of the positive responses (i.e., reported delays in onset of walking or talking) were observed for maternal hair levels above about 60 ppm. Only 3 of 24 children with positive responses were born to mothers with hair levels below 59 ppm. The peak total mercury hair levels during pregnancy for the mothers of those 3 children were 14, 18, and 38 ppm (WHO 1990). A maternal exposure level of 0.0012 mg/kg/day, corresponding to a hair

level of 14 ppm, was estimated for the Iraqi women using a simple, one-compartment pharmacokinetic model (see Section 2.4).

Davidson et al. (1995b) studied the effects of prenatal methylmercury exposure from a diet high in fish on developmental milestones in children living in the Republic of Seychelles (i.e., the Seychelles Child Development Study (SCDS). In this double blind study, children were evaluated with the Bayley Scales of Infant Development (BSID) at 19 months of age (n=738). The 19-month cohort represented 94% of the initially enrolled pairs. The cohort was evaluated again at 29 months (n=736) with the BSID and the Bayley Infant Behavior Record. Mercury exposure was determined by cold vapor atomic absorption analysis of maternal hair segments during pregnancy. The 29-month cohort represented approximately 50% of all live births in the year 1989. This particular study population was carefully selected based on the following reasons: (1) they regularly consume a high quantity and variety of ocean fish; (2) pre-study mercury concentration in maternal hair was in the appropriate range (<5 to >45 ppm) to study low-level exposure; (3) there is no local industry for pollution, and the Seychelles location is 1,000 miles from any continent or large population center; (4) the Seychellois population is highly literate, cooperative, and has minimal immigration; and (5) the Seychellois constitute a generally healthy population, with low maternal alcohol and tobacco use. The association between maternal hair mercury concentrations and neurodevelopmental outcomes at 19 and 29 months of age was examined by multiple regression analysis with adjustment for confounding variables. Testing was performed by a team of Seychellois nurses extensively trained in administration of the BSID.

Maternal hair concentrations measured in hair segments that corresponded to pregnancy ranged from 0.5 to 26.7 ppm, with a median exposure of 5.9 ppm for the entire study group. The mean BSID Mental Scale Indexes at both 19 and 29 months were comparable to the mean performance of U.S. children. The mean BSID Psychomotor Scale Indexes at 19 and 29 months were 2 standard deviation units above U.S. norms, but consistent with previous findings of motoric precocity in children reared in African countries. No effect of mercury was detected on BSID scores at either age. On the Bayley Infant Behavior Record, activity level in boys, but not girls, decreased with increasing mercury exposure. The only subjective observation correlated with prenatal mercury exposure was a slight decrease in activity level in boys (but not girls) as determined by the Bayley Infant Behavior Record.

The overall study cohort was broken down into sub-groups based upon maternal hair mercury concentration as follows: $3 ppm (n=164), 4–6 ppm (n=215), 7–9 ppm (n=161), 10–12 ppm (n=97), and

>12 ppm (n=99). No significant or remarkable effect on the activity of the respective groups of children was observed outside the highest concentration group (i.e., maternal hair concentrations >12 ppm). When boys were examined separately, there appeared to be a trend toward decreased activity with increasing mercury concentrations, most visible above the group median value of 5.9 ppm. The mercury effect was highly significant in males (p=0.0004), but it was not statistically significant (p=0.87) in females. The activity level scores for males decreases 1/10 point (on a 9-point scale) for every ppm of mercury in maternal hair. While the activity score for the overall cohort was comparable to the mode of 5 for U.S. children, those children born to mothers with hair mercury levels of 20 ppm, males scored >1 point below the U.S. mode value. Scores of females remained at the comparable value for U.S. children, regardless of the magnitude of maternal hair mercury level. When the subjective activity scores for male and female children are evaluated collectively, no significant/remarkable decrease in activity is apparent outside the >12 ppm maternal hair concentration group. The affect on activity level in boys is not considered an adverse effect, and the 5.9 ppm level is categorized as a NOAEL. Since the children had been exposed *in utero*, they represent the most sensitive subpopulation.

Myers et al. (1997) evaluated the population of the SCDS for developmental milestones similar to those determined in Iraq. As part of this ongoing study, cohort children were evaluated at 6.5, 19, 29, and 66 months of age. At 19 months care-givers were asked at what age the child walked (n=720 out of 738) and talked (n=680). Prenatal mercury exposure was determined by atomic absorption analysis of maternal hair segments corresponding to hair growth during the pregnancy.

The median mercury level in maternal hair for the cohort in this analysis was 5.8 ppm with a range of 0.5–26.7 ppm. The mean age (in months) at walking was 10.7 (SD=1.9) for females and 10.6 (SD=2.0) for males. The mean age for talking (in months) was 10.5 (SD=2.6) for females, and 11.0 (SD=2.9) for males. After adjusting for covariates and statistical outliers, no association was found between the age at which Seychellois children walked or talked and prenatal exposure to mercury. The ages for achievement of the developmental milestones were normal for walking and talking in the Seychellois toddlers following prenatal exposure to methylmercury from a maternal fish diet. The 5.8 ppm NOAEL of this study is thus considerably below the one derived from the dose-response analysis of the data for the Iraqi methymercury poisonings (10 ppm).

The SCDS cohort continues to be monitored and evaluated for developmental effects. In an analysis of the latest round of outcome measures for children at age 66 months (n=708), Davidson et al. (1998) report no

adverse developmental effects associated with prenatal and postnatal exposure to methylmercury in fish at the levels experienced in this cohort. The actual exposure is reflected in a mean maternal hair level of 6.8 ppm for the prenatal exposure (SD=4.5, n=711, range, 0.5-26.7) and in a mean children's hair level of 6.5 ppm (SD=3.3, n=708, range, 0.9-25.8) for both the prenatal and subsequent postnatal exposure. The age-appropriate main outcome measures included: (1) the McCarthy Scales of Children's Abilities, (2) the Preschool Language Scale, (3) the Woodcock-Johnson Tests of Achievement—Letter and Word Recognition, (4) Woodcock-Johnson Tests of Achievement—Applied Problems and, (5) the Bender Gestalt test, and (6) the Child Behavior Checklist. The test results were similar to what would be expected from a healthy, well-developing U.S. population. No test indicated a deleterious effect of methylmercury from the exposure levels received in this population. Four of the six measures showed better scores in the highest MeHg groups compared with lower groups for both prenatal and postnatal exposure. The authors conclude that this result is likely due to the benefits of increased levels of fish in the diet, possibly because of increased consumption of omega-3-fatty acids. Serum from a subset of 49 of the children was sampled for polychlorinated biphenyl (PCB) levels. None of the samples had detectable levels (detection limit 0.2 ng/mL) for any of the 28 congeners assayed (from congener 28 to 206), indicating that was no concurrent (i.e, potentially confounding) exposure to PCBs in this population. The median level of total mercury for each of 25 species sampled was 0.004–0.75 ppm, with most medians in the range of 0.05–0.25 ppm, levels that are comparable to fish in the U.S. market. The authors conclude, that this most recent NOAEL of 6.8 ppm for the SCDS cohort at 66 months of age strongly supports the findings at earlier ages, and that the benefits of eating fish outweigh the small risk of adverse effects from an increased exposure to methylmercury for this exposure pathway.

Weihe et al. (1996) began a long-term evaluation of the health implications for people living in the Faroe Islands who are exposed to heavy metals and polychlorinated biphenyls (PCBs) from the consumption of fish and pilot whales. A birth cohort of 1,000 children was examined at approximately 7 years of age for neurobehavioral dysfunctions associated with prenatal exposure to mercury and PCB. Preliminary analyses of the data show that several neurobehavioral tests are associated with mercury exposure parameters. With emphasis on prenatal exposures to PCB, another cohort was generated during 1994–1995, and this cohort will be followed closely during the next years. In the Faroe Islands, marine food constitutes a considerable part of the diet. In addition to fish, both meat and blubber from pilot whales are included in the diet. Muscle tissue of pilot whales caught in the Faroe Islands contains an average mercury concentration of 3.3 μg/g (16 nmol/g), about half of which is methylmercury. In some years an evenly distributed annual catch of pilot whales would make the average dietary intake of mercury close to more than the Provisional

Temporary Weekly Intake of 0.3 mg recommended by WHO. In 1 of 8 consecutive births, the mercury concentration in maternal hair exceeded a limit of 10 µg/g, a level where neurobehavioral dysfunction in the child may occur. The maximum level was 39.1 µg/g. Mercury concentrations in umbilical cord blood showed a similar distribution with a maximum of 351 µg/L. The large variation in mercury exposure is associated with differences in the frequency of whale dinners. The average PCB concentration in pilot whale blubber is very high (about 30 µg/g). With an estimated daily consumption of 7 g of blubber, the average daily PCB intake could therefore exceed 200 µg (i.e., close to the Acceptable Daily Intake). In Scandinavia, the average daily PCB intake is about 15–20 µg.

In the continuation of this work, Grandjean et al. (1997b, 1998) studied a cohort of 1,022 consecutive singleton births generated during 1986–1987 in the Faroe Islands. Increased methylmercury exposure from maternal consumption of pilot whale meat was estimated from mercury concentrations in cord blood and maternal hair. At approximately 7 years of age, 917 of the children underwent detailed neurobehavioral examination. Neuropsychological tests included Finger Tapping; Hand-Eye Coordination; reaction time on a Continuous Performance Test; Wechsler Intelligence Scale for Children-Revised Digit Spans, Similarities, and Block Designs; Bender Visual Motor Gestalt Test; Boston Naming Test; and California Verbal Learning Test (Children). Neurophysiological tests emphasized motor coordination, perceptual-motor performance, and visual acuity; pattern reversal visual evoked potentials (VEP) with binocular full-field stimulation, brain stem auditory evoked potentials (BAEP), postural sway, and the coefficient of variation for R-R interpeak intervals (CVRR) on the electrocardiogram were measured. Mercury in cord blood, maternal hair (at parturition), child hair at 12 months, and child hair at 7 years of age were measured. The geometric average mercury concentrations were 22.9, 4.27, 1.12, and 2.99 µg/g, respectively. Mercury concentrations in cord blood were most closely associated with the concentrations in maternal hair at parturition and less so with children's hair at 12 months and 7 years. Clinical examination and neurophysiological testing did not reveal any clear-cut mercury-related abnormalities. However, mercury-related neuropsychological dysfunctions were most pronounced in the domains of language, attention, and memory, and to a lesser extent in visuospatial and motor functions. The authors state that these associations remain after adjustment for covariates and after exclusion of children with maternal hair mercury concentrations above 10 µg/g (50 nmol/g). They further conclude that the effects on brain function associated with prenatal methylmercury exposure appear diverse, with early dysfunction in the Faroe Island population detectable at exposure levels currently considered to be safe.

In animals, there is evidence of developmental effects following oral exposure to organic mercury during gestation, lactation, and/or postweaning. Increases in several parameters indicative of developmental toxicity have been observed. Not all studies have examined neurological end points, but developmental neurotoxicity has been observed at very low exposure levels.

Methylmercuric chloride administered via gavage to pregnant rats at 8 mg Hg/kg on Gd 10 resulted in increased skeletal variations (incomplete fusion of the sternebrae) (Fuyuta et al. 1979). At higher doses, decreased fetal weight and increased malformations (cleft palate) were observed. Administration of lower doses of methylmercury (4 mg Hg/kg/day) for a longer duration of gestation (Gd 7–9 or 6–14) resulted in an increased incidence of rat fetuses with incomplete ossification or calcification (Nolen et al. 1972). The incidence of skeletal variations at 0.2 mg/kg/day was not significantly different from controls. Methylmercuric chloride administered to pregnant rats (n=10) via gavage at 2 mg Hg/kg/day throughout gestation (Gd 0–20) resulted in increased numbers of malformed fetuses (Inouye and Murakami 1975). The most common malformations were generalized edema and brain lesions. When methylmercuric chloride was administration to pregnant rats at 4 mg Hg/kg/day during Gd 7–14, there was a decreased fetal weight and an increased number of total malformations, hydrocephalus, and wavy ribs (Fuyuta et al. 1978). At 6 mg Hg/kg/day, increased resorptions, fetal deaths, cleft palate, generalized edema, brain lesions, absence of vertebral centra, and defects of the sternum were observed. Skeletal variations seen at 6 mg Hg/kg/day included absence of one or more sternebrae, bipartite sternebrae, and bilobed vertebral centra. Administration of a single dose of 24 mg Hg/kg as methylmercuric chloride to pregnant rats during Gd 6–12 resulted in decreased fetal weights and increased malformations (Inouye and Murakami 1975). The incidence of malformations (hydrocephalus, cleft palate, micrognathia, microglossia, generalized edema, subcutaneous bleeding, and hydronephrosis and hypoplasia of the kidneys) increased with later treatments (after Gd 7). Hydrocephalic brains had lesions in the brain mantle, corpus callosum, caudate putamen, and primordial cerebellum. Brains without hydrocephalus had lesions in similar brain areas, as well as dilation of the third ventricle and partial ablation of the ependymal lining.

Groups of 30 pregnant Fischer 344 rats were orally administered 10, 20, or 30 mg/kg methylmercuric chloride dissolved in saline on Gd 7. Controls were given saline only (n=30). Maternal body weight gain and deaths were monitored. Maternal body weights were decreased for 2 days in rats given 10 mg/kg as methylmercuric chloride and for 6 days in rats given 20 mg/kg and were continuously decreased for those given 30 mg/kg. Maternal death rates were 6.7, 16.7, and 30% in the 10, 20, and 30 mg/kg methylmercuric chloride dose groups; no control dams died. Survival rates of fetuses were 19.2, 41.4, and 91.1%

less than controls for the 10, 20, and 30 mg/kg methylmercuric chloride groups, respectively. The LD_{50} of methylmercuric chloride for fetuses was determined to be 16.5 mg/kg. The backbones of fetuses were severely curved at the high-dose level; mean fetal body lengths were reduced by 9.6, 21.7, and 48.8% in the 10, 20, and 30 mg/kg methylmercuric chloride groups, respectively, as compared to controls. Mercury content in maternal organs was highest in kidneys, followed by blood, spleen, liver, and brain, while in fetal organs it was highest in liver. Fetal liver and brain contained more mercury than maternal liver and brain; however, fetal kidneys retained less mercury than maternal kidneys. The fetal ossification center was not completely formed in sternebrae, particularly in the fifth and second bones, pelvic bones, and pectoral phalanges of fetuses in rats treated with 30 mg/kg methylmercuric chloride. The ossified lengths of skeletal bone stained with alizarin red S were developed least in the fifth sternebrae, metacarpals in the pectoral girdle, and ischium in the pelvic girdle, and were severely retarded in development as position of the ribs goes from the sixth bone (center) to the first and 13th bone (each edge) (Lee and Han 1995).

Four groups of 12 pregnant Sprague-Dawley rats were exposed to methylmercury or elemental mercury alone or in combination as follows: one group was administered 2 mg/kg/day methylmercury via gavage during Gd 6–9; another was exposed by inhalation to 1.8 mg/m^3 metallic mercury (elemental Hg) vapor for 1.5 hours per day during Gd 14–19; a third was exposed to both methylmercury by gavage (2 mg/kg/day, Gd 6–9) and elemental Hg vapor by inhalation (1.8 mg/m^3, Gd 14–19) (methylmercury + elemental Hg); a fourth group was given combined vehicle administration for each of the 2 treatments (control). The inhalation regimen corresponded to an approximate dose of 0.1 mg Hg/kg/day. Maternal body weights were monitored. At postpartum day 3, each litter was reduced to 4 male offspring. Body weight, pinna unfolding, tooth eruption, and eye opening were monitored. Testing of behavioral function was performed between 4 and 5 months of age and included spontaneous motor activity, spatial learning in a circular bath, and instrumental maze learning for food reward. Surface righting reflex and negative geotaxis were measured before weaning. There were no differences between any of the groups in maternal body weight gain or in body weight, pinna unfolding, tooth eruption, surface righting reflex, and negative geotaxis in offspring. Offspring of dams exposed to elemental mercury showed hyperactivity in the spontaneous motor activity test chambers over all three of the following parameters: locomotion, rearing, and total activity. This effect was potentiated in the animals of the methylmercury + elemental mercury group. In the swim maze test, the methylmercury + elemental mercury and elemental mercury groups evidenced longer latencies to reach a submerged platform, which they had learned to mount the day before, compared to either the control or methylmercury groups. In the modified, enclosed radial-arm maze, both the methyl-mercury + elemental Hg and elemental Hg groups showed more ambulations and rearings in the activity

test prior to the learning test. During the learning trial, the same groups (i.e., methylmercury + elemental mercury and elemental mercury) showed longer latencies and made more errors in acquiring all eight pellets. Generally, the results indicate that co-exposure to methylmercury, which by itself did not alter these functions at the dose given in this study, served to significantly enhance the effects of prenatal exposure to elemental mercury (i.e., alterations to both spontaneous and learned behaviors). Brain mercury concentrations in offspring were 1 ng/g w/w in the controls, 4 ng/g in the methylmercury group, 5 ng/g in the elemental mercury group, and 12 ng/g in the methylmercury + elemental mercury group (Fredriksson et al. 1996).

Pregnant Sprague-Dawley rats were treated by gavage with a single oral dose of 8 mg/kg of methylmercury chloride or saline on Gd 15. Within 24 hours after birth, litters were reduced to 6 pups per litter. Pups were weighed weekly and weaned 21 days after birth. Offspring of control and treated rats were killed at 14, 21, and 60 days of age. The binding characteristics of muscarinic receptors labeled in cortical membrane preparation by [3H]-L-quinuclidinyl benzilate were studied, and the mercury level in the same brain area was assessed. Total mercury content in cortical tissues was determined at 21 and 60 days of age. Furthermore, the performance in passive avoidance tasks was evaluated in 10 rats from each group at 8 weeks of age. No differences in mortality or weight gain were observed in methylmercury-exposed pups compared to controls. At 21 days of age, the level of mercury in the cortex was about 30 times higher in exposed rats than in controls (190.2 ng/g w/w versus 6.4 ng/g); at 60 days, mercury levels did not differ significantly (7.4 versus 5 ng/g, respectively). Perinatal exposure to methylmercury significantly reduced the maximum number of muscarinic receptors (Bmax) in the brain of 14-day-old (53%) and 21-day-old (21.3%) rats, while there was no notable difference in 60-day-old rats. This phenomenon seems to be strictly related to the presence of mercury in the cortex since it disappeared with the normalization of mercury levels in the brain. Despite the recovery of muscarinic receptor densities in methylmercury-exposed rats at 8 weeks of age, the avoidance latency was reduced in passive avoidance tests, indicating learning and memory deficits in these animals (Zanoli et al. 1994).

Similar effects were observed in mice exposed to organic mercury. Methylmercuric chloride administered orally by gavage to mice at 5 mg Hg/kg/day during Gd 6–17 resulted in 100% stillbirths or neonatal deaths and the failure of 6 of 9 dams to deliver, with no apparent maternal toxicity (Khera and Tabacova 1973). At lower doses (2 and 4 mg Hg/kg/day) for a shorter duration during gestation (days 6–13), no increase in deaths or resorptions was observed, but increases in malformations, skeletal variations, and delays in ossification were observed (Fuyuta et al. 1978). A higher dose of methylmercuric chloride (16 mg Hg/kg)

administered to mice by gavage on either Gd 10 or 12 resulted in decreased fetal weight, cleft palate, and dilation of the renal pelvis (Yasuda et al. 1985).

Thuvander et al. (1996) evaluated the immunomodulation of methylmercury from perinatal exposure in mice. Offspring from Balb/c mice were exposed to methylmercuric chloride in the diet. Dams (16.0±0.5g) were exposed to 0 (n=72), 0.5 (n=27), or 5 (n=37) mg Hg/kg for 10 weeks prior to mating, and during gestation and lactation. Pups were exposed to mercury until day 15 of lactation; thereafter, the pups were given control milk and control diet. Samples for mercury analysis were collected from the pups on days 22 and 50, and for immunological studies on days 10, 22, and 50. Immunological parameters included numbers of splenocytes and thymocytes, proportions of lymphocyte subpopulations within the thymus, the proliferative response of splenocytes to the B-cell mitogen LPS, NK-cell activity of splenocytes, and the primary antibody response to a viral antigen. Eight pups (n=8NS) were taken from at least three different litters for the immune function analysis.

No disturbances in the behavior of dams or pups were observed for any of the dose groups. All dams gave birth to normal sized litters (8–10 animals/litter). The high dose dams did have a small (4%) but significant increase in body weight (weeks 4, 5, 9 $p<0.05$, week 8 $p<0.01$). The exposure resulted in significantly increased total Hg concentrations in whole blood of offspring on day 22 and 50 from the 5 mg Hg/kg group (170 and 22 ng Hg/g blood in 5 mg/kg dose group compared to 7 and 5 ng Hg/g in control, respectively), and of offspring from the 0.5 mg Hg/kg group on day 22 (24 ng Hg/g compared to 7 ng Hg/g in control). On day 50, blood mercury levels in the 0.5 mg Hg/kg group had decreased to 11 ng Hg/g compared to 5 ng Hg/g in controls. Pups showed a decreased body weight (8%) in the 5 mg/kg group at 10 days of age. Significantly increased numbers of splenocytes were found only in offspring from the 0.5 mg Hg/kg group at 10 and 22 days, and increased number of thymocytes in the 0.5 mg/kg group at 22 days. Flow cytometry analysis of thymocytes revealed increased numbers and altered proportions of lymphocyte subpopulations within the thymus in offspring from both of the exposed groups at 22 days. The only sign of immunosuppression was a decrease in the proportion of CD4+ thymocytes at 10 days, but this was seen in both mercury groups so was probably not related to a decrease in body weight. The proliferative response of splenocytes to the B-cell mitogen LPS was increased in offspring from dams exposed to 5 mg Hg/kg, and the primary antibody response to a viral antigen was stimulated in pups from dams exposed to 0.5 mg Hg/kg. No significant differences were observed in the NK-cell activity of splenocytes except for a transient increase in activity at 22 days in the 5 mg/kg group at one of the two effector-to-target-cell ratios tested. The present results indicate that placental and lactational transfer of

low dose mercury affects thymocyte development and stimulates certain mitogen- or antigen-induced lymphocyte activities in mice. The authors note that these results, in the context of other studies where methylmercury was observed to have suppressive effects, suggests that methylmercury enhances immune function within a narrow dose range. The blood levels of mercury in the present study are close to the levels found in fish-eating populations. The authors note that the clinical relevance of slight stimulation of some immune functions is unknown, but the induction of autoimmunity by methylmercury can not be excluded.

Groups of guinea pigs exposed to a single dose of 11.5 mg Hg/kg as methylmercuric chloride at various times during gestation (66–69 days) showed differences in the manifestation of developmental neurotoxicity, depending on the period of development when exposure occurred (Inouye and Kajiwara 1988). Primarily developmental disturbances of the brain (e.g., smaller brains, dilated lateral ventricles, reduced size of hippocampus and nucleus caudate-putamen) occurred with exposures at 3, 4, or 5 weeks of pregnancy. Exposure during a later pregnancy stage (6 or 7 weeks) produced widespread focal degeneration of neurons in the neocortical region of fetal brains. In hamsters, methylmercuric chloride administered as a single dose of 8 mg on Gd 10 or of 1.6 mg Hg/kg/day on Gd 10–15 resulted in degeneration of cerebellar neurons in neonates (Reuhl et al. 1981a). Examination of offspring 275–300 days after birth showed similar degeneration (Reuhl et al. 1981b). It was not reported whether these histopathological changes correlated with behavioral changes.

Functional disturbances have also been observed following exposure to methylmercuric chloride during gestation. A single dose of 16 mg Hg/kg as methylmercuric chloride administered on Gd 13, 14, 15, 16, or 17 resulted in decreased spontaneous locomotor activity at 5 weeks of age, decreased righting response, abnormal tail position during walking, flexion, and crossing of the hindlimbs (Inouye et al. 1985). Histopathological examination of these animals showed dilated lateral ventricles, decreased caudate putamen, and a slightly simplified cerebellar pattern. Neonates in this study were cross-fostered within 24 hours after birth to prevent intake of mercury through the milk. The offspring of mice receiving 3, 5, or 10 mg Hg/kg/day as methylmercuric hydroxide on day 8 of gestation exhibited a decreased number of avoidances, an increased number of escapes, and an increased trials to reach the criterion on a 2-way avoidance task (Hughes and Annau 1976). No effects were present in the 2 mg Hg/kg dose group. Offspring of rats exposed to 4 mg Hg/kg/day as methylmercuric chloride on Gd 6–9 showed impaired swimming behavior, increased passiveness, and an increased startle response (Stoltenburg-Didinger and Markwort 1990). At 0.4 mg Hg/kg/day, the offspring showed an increased startle response, but at 0.04 mg

Hg/kg/day, no effects were observed. Exposure to 6.4 mg Hg/kg as methylmercuric chloride on Gd 15 resulted in decreases in spontaneous locomotor activity, increased sensitivity to pentylenetetrazol-induced convulsions, and a transient increase in γ-aminobutyric acid (GABA) and benzodiazepine receptors (Guidetti et al. 1992). Using the same exposure paradigm, shorter avoidance latency was observed in 14-, 21-, and 61-day-old rats (Cagiano et al. 1990). Glutamate receptor binding affinity and dopamine receptor number were also significantly affected in the brains of these offspring. Thus, multiple neurotransmitter systems may participate in the neurological effects observed.

A sensitive test for neurological effects of gestational exposure to methylmercury is operant behavioral performance (i.e., rewarded responses to total lever presses). Bornhausen et al. (1980) reported a significant reduction in operant behavioral performance in 4-month-old rat offspring exposed to methylmercuric chloride at 0.008 mg Hg/kg/day on Gd 6–9. A dose of 0.004 mg Hg/kg/day did not alter the behavioral performance of the offspring. No other studies have confirmed this result to date.

Pregnant hamsters received single oral gavage doses of 2.5–63 mg Hg/kg as mercuric acetate on Gd 8 (Gale 1974). Decreased crown-rump length was observed at 5 mg Hg/kg, although this effect did not increase linearly with the dose level. The incidence of resorptions increased at 22 mg Hg/kg and occurred in a dose-related manner. Other effects that occurred at higher dose levels included growth-retarded or edematous embryos. No significant developmental effects were evident at 2.5 mg Hg/kg.

Developmental neurotoxicity and changes in tissues, including the liver and immune system, have been observed in studies in which exposure occurred prior to gestation and/or was continued after gestation for intermediate durations. Retarded behavioral maturation (swimming behavior, righting reflexes) and learning disability (maze learning) were demonstrated in rat offspring receiving a diet of 0.1 mg Hg/kg/day (unspecified forms of mercury) in a contaminated fish diet from Gd 1 to postnatal day 42 (Olson and Boush 1975). Decreased performance in a paradigm intended to assess tactile-kinesthetic function (use of too much force) was observed in offspring of rats exposed to 0.08 mg Hg/kg/day as methylmercuric chloride for 2 weeks prior to mating through weaning (Elsner 1991). No morphological changes were observed in the brains of the offspring of maternal rats given 0.195 mg Hg/kg/day as methylmercuric chloride for 14 weeks prior to mating through postpartum day 50 (Lindstrom et al. 1991). However, norepinephrine levels in the cerebellum of offspring were significantly increased. Methylmercuric chloride at doses of 0.25 mg Hg/kg/day administered beginning several weeks prior to gestation resulted in an increase in the incidence of unilateral or bilateral ocular lesions in the neonates, associated with histological changes in the

Harderian, exorbital lachrymal, and parotid salivary glands (Khera and Tabacova 1973). No effects occurred at the lower dose of 0.05 mg Hg/kg/day. Fetal liver injury at the ultrastructural level (e.g., decreased mitochondrial volume density, enzyme activity, and protein synthesis in fetal hepatocytes) was reported after chronic exposure to low doses of 0.7–1.4 mg Hg/kg/day as methylmercury in the drinking water of rats for 1 month before mating and up to the end of pregnancy (Fowler and Woods 1977). The developing immune system was affected in newborn Sprague-Dawley rats exposed to 0.5 mg Hg/kg/day as methylmercury through the placenta and/or milk (Ilback et al. 1991). Results showed that exposure caused increased thymus lymphocyte activity in offspring exposed during gestation and lactation, while decreased spleen lymphocyte activities were observed in offspring exposed during lactation only. Cell-mediated cytotoxicity was decreased by 41% ($p<0.01$) in offspring exposed during gestation and lactation.

In chronic-duration studies, impaired visual function has been reported. Impaired visual recognition memory was reported for 50-to-60-day-old monkeys born to mothers that received 0.04 or 0.06 mg Hg/kg/day as methylmercury in apple juice for an average of 168 or 747 days prior to mating (Gunderson et al. 1988). In this study, neonates were separated from their mothers at birth to prevent intake of mercury while nursing. Impaired spatial visual function was observed in another study in which infant monkeys were exposed to 0.01, 0.025, or 0.05 mg Hg/kg/day as methylmercuric chloride throughout gestation, followed by gavage doses 5 days a week until 4–4.5 years of age (Rice and Gilbert 1990). The study was limited, however, because only 1–5 animals were tested at each dose level. Furthermore, two of the high-dose animals were unavailable for testing as a result of overt mercury intoxication, and thus the two most affected animals were eliminated. Slight changes in temporal discrimination were also observed in these monkeys at 2–3 years of age (Rice 1992). However, no LOAEL can be determined for this effect because results from monkeys at the mid- and high doses were pooled.

The highest NOAEL values and all reliable LOAEL values for developmental effects in each species and duration category are recorded in Table 2-3 and plotted in Figure 2-3 for organic mercury.

2.2.2.7 Genotoxic Effects

Several studies were located regarding genotoxic effects in humans after oral exposure to organic mercury. A positive correlation between blood mercury levels and structural and numerical chromosome aberrations was found in the lymphocytes of 23 people who consumed mercury-contaminated fish (Skerfving et al. 1974). However, several factors preclude acceptance of these findings as valid. With respect to the

increased incidence of structural aberrations, smokers were not identified, it was unclear whether chromatid and chromosome gaps were excluded from the evaluation, and significant effects were obtained only from lymphocyte cultures initiated several days after collection. The more reliable approach of initiating cultures on the day of collection did not yield significant results. Similarly, the evidence of aneuploidy is suspect. Considering the age of the subjects (54–89 years in the control group and 47–84 years in the exposure group), the average incidence of aneuploidy in the control (1.8%) and exposed (2.8%) groups was lower than would be expected, according to results indicating that aneuploidy in humans increases with age (Cimino et al. 1986). Skerfving et al. (1970) also reported that a significant ($p<0.05$) correlation was found between mercury concentrations and chromosome breaks in the lymphocytes of 9 subjects who had consumed fish contaminated with methylmercury. For reasons similar to those listed for the evaluation of the report by Skerfving et al. (1974), there is not yet a scientific basis to support an association between consumption of fish containing high methylmercury and clastogenesis in human lymphocytes. In addition, one of the test subjects was regularly medicated with isoproterenol, a known clastogen for mammalian cells. Although an increased incidence of sister chromatid exchange was reported in humans who ate mercury-contaminated seal meat (Wulf et al. 1986), data on smoking and consumption of other heavy metals (lead and cadmium) were not provided. Therefore, the possible relevance of the increase in sister chromatid exchanges (SCEs) cannot be determined. A statistical correlation between micronucleus frequency in peripheral blood lymphocytes and total mercury concentration in blood ($p=0.00041$), as well as between micronucleus frequency and age ($p=0.017$), was found in a population of fishers who had eaten mercury contaminated seafood (Franchi et al. 1994).

A single oral gavage administration of mercuric chloride to male Swiss albino mice (5 per group) at doses of 2.2, 4.4, or 8.9 mg Hg/kg induced a dose-related increase in the frequency of chromosome aberrations and the percentage of aberrant cells in the bone marrow (Ghosh et al. 1991). Chromatid breaks were the most common aberration. There was no clear evidence of unscheduled DNA synthesis (UDS) in lymphocytes harvested from male and female cats (3 per group) chronically exposed (39 months) to dietary concentrations of 0.0084, 0.020, or 0.046 mg Hg/kg/day as methylmercury (Miller et al. 1979). In a parallel study, significant increases in nuclear abnormalities were scored in bone marrow cells collected from the three treatment groups (5–8 cats per group); the response, however, was not dose-related. Signs of compound toxicity (slight neurological impairment and minimal central nervous system pathology) were seen in the high-dose group, but these animals yielded the lowest number of abnormal chromosome figures.

Other genotoxicity studies are discussed in Section 2.5.

2.2.2.8 Cancer

Inorganic Mercury. No studies were located regarding cancer in humans after oral exposure to inorganic mercury.

Results of a 2-year National Toxicology Project (NTP 1993) study indicated that mercuric chloride may induce tumors in rats. Fischer 344 rats (60 per sex per group) received 0, 1.9, or 3.7 mg Hg/kg/day as mercuric chloride by gavage for 2 years. There were increases in the incidence of forestomach squamous cell papillomas and an increase in the incidence of thyroid follicular cell carcinomas in males in the 3.7 mg/kg group (NTP 1993). In $B6C3F_1$ mice exposed to 0, 3.7, or 7.4 mg Hg/kg/day as mercuric chloride, renal tubule tumors were evident in 3 of the 49 high-dose males (NTP 1993), but the incidence of these tumors was not significantly increased. There was no evidence of carcinogenicity in the exposed female mice. The cancer effect level (CEL) from this study is recorded in Table 2-2 and is plotted in Figure 2-2.

Organic Mercury. No studies were located regarding cancer in humans following oral exposure to organic mercury.

Significant increases in renal tumors have been observed in rodents exposed either to methylmercuric chloride or phenylmercuric acetate. Dietary exposure of both ICR and $B6C3F_1$ mice for 2 years has resulted in significant increases in renal epithelial cell tumors (Hirano et al. 1986; Mitsumori et al. 1981, 1990). At the highest dose of 0.69 mg Hg/kg/day (dose levels 0, 0.03, 0.14, 0.69), only male $B6C3F_1$ mice (n=60M,60F) showed significant increases in the incidence of renal epithelial cell adenomas and carcinomas (Mitsumori et al. 1990). No tumors were observed in the females $B6C3F_1$ mice exposed to up to 0.6 mg Hg/kg/day. The high dose in males and females also resulted in chronic nephropathy and regeneration of the proximal tubules (more severe in males). At 0.73 mg Hg/kg/day, male ICR mice showed significant increases in the incidence of epithelial cell adenocarcinomas (Hirano et al. 1986). Similar effects were observed in the ICR male mice at the highest dose of 1.6 mg Hg/kg/day (Mitsumori et al. 1981). No increase in tumor incidence was observed in rats exposed via the diet for 2 years to methylmercuric chloride at doses as high as 0.1 mg Hg/kg/day (Verschuuren et al. 1976).

Exposure of male Wistar rats to phenylmercuric acetate in the drinking water at 4.2 mg Hg/kg/day for 2 years resulted in a significant increase in renal cell adenomas (Solecki et al. 1991). However, this report

is limited because the assay was not intended as a carcinogenicity assay, and too few animals were used (20 per dose) to adequately assess the carcinogenicity of the phenylmercuric acetate.

No tumors or precancerous lesions were reported in rats administered 0.04–66.0 mg Hg/kg/day as phenylmercuric acetate in the diet for 2 years (Fitzhugh et al. 1950). As discussed above for mercuric acetate, no conclusions can be drawn from this study because of its limitations.

In a 2-year oral chronic-duration feeding study, no tumors or precancerous lesions were noted in rats administered mercuric acetate in the diet at doses of 0.2–66 mg Hg/kg/day (Fitzhugh et al. 1950); no conclusions could be derived on the carcinogenicity of mercuric acetate. The study was limited because the group sizes were small (10–12 rats per group); survival data were not reported; a considerable but unspecified number of rats reportedly died from pneumonia, which reduced the sensitivity of the study to detect a carcinogenic response; and only limited histopathological analyses were performed.

The CELs from these studies are recorded in Table 2-3 and plotted in Figure 2-3.

2.2.3 Dermal Exposure

Occupational exposure to both inorganic and organic mercury compounds may result in dermal as well as inhalation exposure to these chemicals. The results reported in Section 2.2.1 regarding the effects associated with occupational exposure to mercury-containing chemicals will not be repeated here. The studies discussed below concern reports in which dermal exposure was expected to be the primary route of exposure.

2.2.3.1 Death

Inorganic Mercury. A case study reported that a 27-year-old woman died 4 days after inserting an 8.75-g tablet of mercuric chloride (93 mg Hg/kg assuming 70-kg weight) into her vagina (Millar 1916). Another case study described the death of a man who had been receiving treatment for a wound with daily applications for approximately 2 months of a Chinese medicine containing mercurous chloride (Kang-Yum and Oransky 1992). The patient was reported to have died from renal failure.

An early study conducted by Schamberg et al. (1918) reported death in rabbits after an ointment containing 50% mercury was “rubbed” into the skin for 5 minutes; however, inadequate experimental methodology and an absence of study details prevent a determination of the amount of mercury involved.

Organic Mercury. No studies were located regarding death in humans or animals after dermal exposure to organic mercury.

2.2.3.2 Systemic Effects

No studies were located regarding respiratory, hematological, musculoskeletal, or hepatic effects in humans or animals after dermal exposure to inorganic or organic mercury.

Cardiovascular Effects

Inorganic Mercury. A number of children who were treated with an ammoniated mercury ointment or whose diapers had been rinsed in a mercuric chloride solution experienced tachycardia and elevated blood pressure (Warkany and Hubbard 1953).

No studies were located regarding cardiovascular effects in animals after dermal exposure to inorganic mercury.

Organic Mercury. No studies were located regarding cardiovascular effects in humans or animals after dermal exposure to organic mercury.

Gastrointestinal Effects

Inorganic Mercury. Patients who were hypersensitive to mercury (indicated by positive patch tests) developed stomatitis at the sites of contact with amalgam fillings (Veien 1990). The contact stomatitis faded when amalgam fillings were removed but persisted in a patient who chose to leave them in place. Abdominal pain, nausea, vomiting, and black stools were seen in a man who had been receiving treatment for a wound with daily applications for about 2 months of a Chinese medicine containing mercurous chloride (Kang-Yum and Oransky 1992). Anorexia was reported in a child who had been treated with an ammoniated mercury-containing ointment (Warkany and Hubbard 1953). Extensive necrosis, swelling, and

ulceration in the intestinal mucosa, vomiting, and diarrhea occurred in a woman who inserted a mercuric chloride tablet into her vagina (Millar 1916).

No studies were located regarding gastrointestinal effects in animals following dermal exposure to inorganic mercury.

Organic Mercury. No studies were located regarding gastrointestinal effects in humans or animals after dermal exposure to organic mercury.

Renal Effects

Inorganic Mercury. Congested medulla; pale and swollen cortex; and extensive necrosis, degeneration, and calcification of tubular epithelium were reported in the kidneys of a 27-year-old woman after inserting an 8.75-g tablet of mercuric chloride (93 mg Hg/kg assuming 70-kg weight) into her vagina (Millar 1916). Decreased renal output and renal failure were reported in a man who had been receiving daily applications for 2 months of a Chinese medicine containing mercurous chloride (Kang-Yum and Oransky 1992). A woman who used a depigmenting cream containing mercuric ammonium chloride for approximately 18 years developed an impaired renal function (Dyall-Smith and Scurry 1990). Similarly, a man who used an ointment containing ammoniated mercury for psoriasis for more than 10 years developed a nephrotic syndrome with severe edema (Williams and Bridge 1958). A study of young African women who used skin lightening creams containing ammoniated mercuric chloride for 1–36 months (average, 13 months) showed a nephrotic syndrome among a large portion of the women (Barr et al. 1972). The syndrome was characterized by elevated urinary protein, edema, decreased serum albumin, alpha-1-globulin, beta-globulin, and gamma globulin and increased alpha-2-globulin. Remission was observed in 77% of those who discontinued use of the creams.

No studies were located regarding renal effects in animals after dermal exposure to inorganic mercury.

Organic Mercury. No studies were located regarding renal effects in humans or animals after dermal exposure to organic mercury.

Endocrine Effects

No studies were located regarding endocrine effects in humans or animals after dermal exposure to inorganic or organic mercury.

Organic Mercury. No studies were located regarding endocrine effects in humans or animals after dermal exposure to organic mercury.

Dermal Effects

Inorganic Mercury. Contact dermatitis caused by acute, longer-term, or occupational inorganic mercury exposure has been described in a number of case reports (Bagley et al. 1987; Biro and Klein 1967; Faria and Freitas 1992; Goh and Ng 1988; Handley et al. 1993; Kanerva et al. 1993; Nordlind and Liden 1992; Pambor and Timmel 1989; Skoglund and Egelrud 1991; Veien 1990). Patch tests conducted in many of the cases show some cross-reactivity between various inorganic and organic forms of mercury (Faria and Freitas 1992; Handley et al. 1993; Kanerva et al. 1993; Pambor and Timmel 1989; Veien 1990). In these studies, dermal exposure occurred as a result of the breakage of mercury-containing thermometers or sphygmomanometers, dental amalgams containing elemental mercury, inoculation with vaccines containing merthiolate preservatives, or mercuric sulfide in tattoos. One report of contact dermatitis caused by a mercuric sulfide-containing tattoo suggested that the reaction was not to mercuric sulfide itself but to a mercury derivative that was formed in the skin (Biro and Klein 1967).

Excluding reports of contact dermatitis, limited information was obtained regarding the dermal effects of inorganic mercury. Application of an ammoniated mercury ointment to the skin of children or exposure to diapers that had been rinsed in a mercuric chloride-containing solution resulted in itching, flushing, swelling, and/or desquamation of the palms of the hands and soles of the feet (Warkany and Hubbard 1953). In addition, rashes, conjunctivitis, and/or excessive perspiration were observed. These dermal reactions were not attributed to allergic-type reactions to the mercury. A 23-month-old boy who was exposed to an unspecified form of mercury also developed a "diffuse, pinpoint, erythematous, papular rash" and bright red finger tips "with large sheets of peeling skin" (Tunnessen et al. 1987).

Application of a 1% solution of ammoniated mercuric chloride to the skin resulted in only minor irritation in 2 of 11 exposed subjects (Kawahara et al. 1993). After 18 years of using a mercury-containing cream, a

patient exhibited blue-black pigmentation in a perifollicular distribution on the chin and glabella (Dyall-Smith and Scurry 1990). A skin biopsy revealed black nonrefractile granules in the cytoplasm of macrophages in the papillary dermis and around the upper part of hair follicles. A boy who broke a thermometer in his mouth developed a mass consisting of hyperplasia of the epidermis, necrosis, and ulceration (Sau et al. 1991). This effect may have resulted from a combined effect of the physical injury and the mercury metal.

No studies were located regarding dermal effects in animals after dermal exposure to inorganic mercury.

Organic Mercury. Case report studies suggest that dermal exposure to methylmercury or phenylmercury in humans can cause rashes and blisters on the skin (Hunter et al. 1940; Morris 1960). A 33-year-old male worker exposed to methylmercury nitrate dust for 2 years developed burns and blisters on his forearm (Hunter et al. 1940). These effects healed within 9 days. Sensitivity to phenylmercuric salts is shown by individuals who developed itchy, pruritic, papular eruptions or rashes on their skin following acute dermal exposure (Morris 1960). A 54-year-old woman with a family history of atopy was found to display erythema (at 30 minutes postexposure) and urticaria (at 60 minutes) when treated topically with a 0.01% solution of phenylmercuric acetate (Torresani et al. 1993). This positive reaction was associated with aggravation of facial edema and an attack of bronchospasm. The woman, who was a farmer, was believed to have been previously exposed to phenylmercuric acetate during contact with pesticides and herbicides used on farm crops.

No studies were located regarding dermal effects in animals following dermal exposure to organic mercury.

Ocular Effects. No studies were located regarding ocular effects in humans or animals after dermal exposure to inorganic or organic mercury.

Body Weight Effects. No studies were located regarding body weight effects in humans or animals after dermal exposure to inorganic or organic mercury.

2.2.3.3 Immunological and Lymphoreticular Effects

Inorganic Mercury. As indicated above, contact dermatitis may develop as a result of acute or occupational exposure to inorganic mercury (Anneroth et al. 1992; Bagley et al. 1987; Biro and Klein

1967; Faria and Freitas 1992; Goh and Ng 1988; Nordlind and Liden 1992; Pambor and Timmel 1989; Skoglund and Egelrud 1991; Veien 1990). Patch tests conducted in many of the cases show some cross-reactivity between various inorganic and organic forms of mercury (Faria and Freitas 1992; Pambor and Timmel 1989; Veien 1990). In these studies, dermal exposure occurred as a result of the breakage of mercury-containing thermometers or sphygmomanometers, dental amalgams containing elemental mercury, or mercuric sulfide in tattoos. One report of contact dermatitis caused by mercuric sulfide in a tattoo suggested that the reaction was not to mercuric sulfide itself but to a mercury derivative that was formed in the skin (Biro and Klein 1967).

No studies were located regarding immunological or lymphoreticular effects in animals following dermal exposure to inorganic mercury.

Organic Mercury. No studies were located regarding immunological or lymphoreticular effects in humans or animals after dermal exposure to organic mercury.

2.2.3.4 Neurological Effects

Inorganic Mercury. DeBont et al. (1986) described a 4-month-old boy who had signs of acrodynia accompanied by coma, paralysis of one side of the body, generalized muscle stiffness, and muscular tremors 12 days after he was treated with yellow mercuric oxide ointment for eczema. Topical application of a depigmenting cream containing 17.5% mercuric ammonium chloride for 18 years resulted in mild tremors, anxiety, depression, and paranoid delusions in a 42-year-old woman (Dyall-Smith and Scurry 1990). Children who were treated with an ointment containing ammoniated mercury or who were exposed to diapers that had been rinsed in a mercuric chloride-containing solution experienced irritability, fretfulness, and sleeplessness (Warkany and Hubbard 1953).

No studies were located regarding neurological effects in animals after dermal exposure to inorganic mercury.

Organic Mercury. No studies were located regarding neurological effects in humans or animals after dermal exposure to organic mercury.

No studies were located regarding the following effects in humans or animals after dermal exposure to inorganic or organic mercury:

2.2.3.5 Reproductive Effects

2.2.3.6 Developmental Effects

2.2.3.7 Genotoxic Effects

Genotoxicity studies are discussed in Section 2.5.

2.2.3.8 Cancer

No studies were located regarding cancer in humans or animals after dermal exposure to inorganic or organic mercury.

2.3 TOXICOKINETICS

Absorption is high (approximately 70–80%) for inhaled metallic mercury vapor, and negligible for oral exposure to liquid metallic mercury. Absorption of inorganic mercuric salts may range from 2 to 38% depending upon the form and test conditions. Oral absorption of organic mercury is nearly complete, but respiratory absorption data are lacking, particularly for the alkyl mercurials.

The distribution data for metallic, inorganic, and organic mercury are consistent in identifying the kidney as the organ with the highest mercury bioaccumulation. Because of its high lipophilicity, metallic mercury can also be transferred readily through the placenta and blood-brain barrier. The oxidation of metallic mercury to inorganic divalent cation in the brain can result in retention in the brain. Inorganic mercury compounds can reach most organs; however, their low lipophilicity reduces their ability to penetrate barriers to and accumulate in the brain and fetus. The distribution of methylmercury is similar to that of metallic mercury; a relatively large amount of mercury can accumulate in the brain and fetus (compared to inorganic mercury) because of its ability to penetrate the blood-brain and placental barriers and its conversion in the brain and fetus to the inorganic divalent cation.

Metallic mercury can be oxidized to inorganic divalent mercury by the hydrogen peroxidase-catalase pathway, which is present in most tissues. The inorganic divalent cation can, in turn, be reduced to

metallic mercury. The mercurous ion is unstable in the presence of sulfhydryl groups, and undergoes disproportionation into one atom of metallic mercury and one ion of mercuric mercury. As with metallic mercury, organic mercury can also be converted to inorganic divalent mercury; however, the extent of conversion is less than with metallic mercury.

Following exposure to metallic mercury, the elimination of mercury can occur via the urine, feces, and expired air. Following exposure to inorganic mercury (mercuric), mercury is eliminated in the urine and feces. Organic mercury compounds are excreted predominantly via the feces in humans. In animals, methylmercury is excreted in the feces, and phenylmercury compounds are initially excreted in the feces and then in the urine. Organic mercury compounds are excreted predominantly in the inorganic form. Both inorganic mercury and methylmercury are excreted in breast milk.

Absorption of metallic mercury vapor is believed to occur by rapid diffusion through the lungs. Oral absorption of inorganic mercuric mercury compounds may also involve rapid diffusion through the gastro-intestinal tract. The mechanism for oral absorption of mercurous mercury compounds is not known. Oral absorption of organic mercury is believed to depend on the ability of the organic mercury molecule to bind to molecules such as cysteine. The mechanism of action of inorganic and organic mercury compounds may involve the affinity of these chemicals for sulfhydryl or thiol groups of proteins and other biological compounds.

2.3.1 Absorption

Absorption following inhalation of metallic mercury vapors is relatively high. Absorption following acute oral exposure to metallic mercury is negligible in both humans and animals. Methyl- and phenylmercury compounds are absorbed much more readily than inorganic mercury. Animal studies suggest oral absorption of both organic and inorganic mercury may be influenced by age and diet. Limited information was located regarding dermal absorption of inorganic or organic mercury compounds in humans or animals.

2.3.1.1 Inhalation Exposure

Metallic and Inorganic Mercury. There are limited quantitative data on the absorption of metallic mercury vapor by humans after inhalation exposure, although it is the most common route of inorganic mercury uptake. Metallic mercury is highly lipophilic, and absorption of the inhaled vapor, followed by rapid diffusion across the alveolar membranes of the lungs into the blood, has been reported to be substantial. Exposure to 0.1–0.2 mg/m^3 elemental mercury vapor resulted in approximately 74–80% of inhaled elemental mercury vapor being retained in human tissues (Hursh et al. 1976; Teisinger and Fiserova-Bergerova 1965). Indirect evidence of rapid absorption was provided by elevated mercury levels found in red blood cells, plasma, and excreta of 5 volunteers who inhaled radiolabeled mercury for 14–24 minutes (Cherian et al. 1978). Elevated blood levels of mercury were also observed in humans following a brief occupational exposure (3 days) to less than 0.1 mg/m^3 metallic mercury vapor (Barregard et al. 1992).

Recently, Sandborgh-Englund et al. (1998) evaluated the absorption, blood levels, and excretion of mercury in humans after a single dose of mercury vapor. Nine healthy volunteers (2M, 7F) were exposed to 400 µg Hg/m^3 mercury vapor in air (median 399 µg Hg/m^3; range, 365–430 µg Hg/m^3) for 15 minutes. This dose corresponded to 5.5 nmol Hg/kg body weight. Samples of exhaled air, blood, and urine were collected for 30 days after exposure. The median retention of elemental Hg was 69% of the inhaled dose. During the first 3 days after exposure 7.5–12% of the absorbed dose was lost by exhalation, with the median half-time of Hg in expired breath being 2 days. In blood and plasma, a rapid absorption phase of Hg was seen, followed by a biexponential decline of the curves in both media. A substantial interindividual variation was observed in the area under the concentration-time curves of Hg in blood and plasma. In plasma, the median half-time of the second phase was 10 days. About 1.0% of the absorbed Hg was excreted via the urine during the first 3 days after exposure whereas the estimated amount excreted during the 30 days ranged from 8 to 40%. In order to evaluate the chronic exposure to mercury from dental amalgam in the general population, the daily Hg dose from the fillings was estimated based on the plasma Hg levels of subjects with amalgam fillings and the plasma clearance obtained in this study. The daily dose was estimated to be from 5 to 9 µg/day in subjects with an average number of amalgam fillings.

There are few reports regarding the respiratory absorption of elemental and inorganic mercury compounds in animals. Elevated levels of mercury were detected in blood and tissues of pregnant or nursing guinea pigs after short-term exposure (2–2.5 hours) to metallic mercury vapors (6–10 mg/m^3) (Yoshida et al.

1990, 1992). Following repeated exposure (5 weeks) of rats to mercury vapor (1 mg/m^3), high levels were detected in the blood and brain (Warfvinge et al. 1992). The absorption of inorganic divalent mercury has not been measured, but it is estimated to be approximately 40% in dogs (Morrow et al. 1964).

Organic Mercury. No studies were located regarding absorption in humans or animals after inhalation exposure to compounds of phenyl- or methylmercury. However, indirect evidence indicates organic mercury can be absorbed readily through the lungs. Following inhalation of ^{203}Hg-labeled dimethyl-mercury, radioactivity was excreted within 6 hours, followed by a slower excretion phase (Ostlund 1969).

2.3.1.2 Oral Exposure

Metallic and Inorganic Mercury. Few studies in humans were located regarding absorption of ingested metallic or inorganic mercury. For metallic mercury, ingesting small amounts such as contained in a standard thermometer (about 0.1 mL or about 1 g) does not produce symptoms of intoxication (Wright et al. 1980). Reports of ingestion of substantial amounts of elemental mercury indicate that absorption is negligible (Sue 1994; Wright et al. 1980). Two case histories were identified on acute effects of relatively large ingestions of metallic mercury. The first case history was described an ingestion of 15 mL (204 g) of metallic mercury by a 17-year-old male storekeeper who swallowed mercury from the pendulum of a clock (apparently out of curiosity rather than as a suicide attempt). On admission, and 24 hours later, he was symptom free, and physical examination was normal. The patient had no gastrointestinal symptoms, and was treated with a mild laxative and bed rest. The results of serial daily urine mercury estimates were normal (all less than 15 µg), and did not suggest significant absorption. The radiological investigation illustrated a characteristic pattern of finely divided globules of mercury in the gastrointestinal tract (Wright et al. 1980).

A second and massive incidence of ingestion involved a 42-year-old man who had spent much of his life (since the age of 13) repairing instruments that contained mercury. He intentionally ingested an estimated 220 mL (about 3,000 g) of mercury (Lin and Lim 1993). Upon admission, the patient presented with significantly elevated mercury blood levels (103 µg/L, normal <10 µg/L) and urine levels (73 µg/L, normal <20 µg/L). It is not known how much the occupational exposure had contributed to these levels. The patient was treated with immediate gastric lavage and cathartics. He also received D-penicillamine 1 g/day orally for 7 days. Blood and urine mercury levels obtained 3 days after chelation therapy were 116.9 and 22.9 µg/L, respectively. By 2 weeks postexposure, most of the mercury had been excreted in the feces and

was measured at a total volume of 220 mL (this number was used to estimate the amount initially ingested). The patient was lost to follow-up for 6 months, but at 10 months following the incident, blood mercury had decreased to 1 μg/L and urine mercury to μg/L.

Approximately 15% of a trace dose of mercuric nitrate in an aqueous solution or bound to calf liver protein was absorbed by the gastrointestinal tract of humans (Rahola et al. 1973). The mercurous ion demonstrated limited absorption. No information was located regarding the percentage of absorption of mercuric chloride by the gastrointestinal tract of humans. However, an extremely high serum inorganic mercury concentration (116.5 nmol/mL) was found in a woman who ingested a potentially lethal dose of powdered mercuric chloride (13.8 mg Hg/kg) (Suzuki et al. 1992). Similarly, no information was located regarding the percentage of absorption of mercuric sulfide by the gastrointestinal tract in humans. However, elevated mercury was detected in the urine of two subjects who ingested an unspecified amount of mercuric sulfide (Yeoh et al. 1989).

A number of animal studies indicate absorption of inorganic mercury in the 10–30% range. In earlier studies, absorption rate was reported as low. Only 1–2% of an orally administered dose of mercuric chloride was absorbed in mice (Clarkson 1971). In rats, using whole-body retention data, estimated mercuric chloride absorptions of 3–4, 8.5, and 6.5% were calculated for single oral doses of 0.2–12.5, 17.5, and 20 mg/kg, respectively (Piotrowski et al. 1992). More recent studies using whole-body retention data, however, indicate absorption of 20–25% calculated from single oral doses of 0.2–20 mg Hg/kg as mercuric chloride in mice. Comparison was made of retention data after oral and intraperitoneal dosing, taking excretion and intestinal reabsorption into account (Nielsen and Andersen 1990). In a subsequent study, the whole-body retention of mercury after mercuric chloride administration was observed to initially decline rapidly, indicating incomplete intestinal absorption (Nielsen and Andersen 1992). Mercury was rapidly cleared from the gastrointestinal tract (to <30% of the initial dose within 2 days), and relative carcass retention increased throughout the experimental period, reaching levels around 40% of initial whole-body retention. Blood levels of mercury were closely correlated to whole-body retention of mercury during the first 3 days after administration of mercuric chloride (1 mg Hg/kg). After the initial 3 days, the amount of mercury in the blood declined more rapidly than the whole-body burden.

Morcillo and Santamaria (1995) report absorption of 30–40% for radiolabeled mercuric chloride when administered in drinking water at 5, 50, and 500 μM Hg for 8 weeks to male rats. The percentage of total

mercury excreted by the fecal route was significantly lower in the 500 compared to the 5 and 50 μM Hg group.

The rate of oral absorption of mercuric mercury compounds in rats is dependent on several factors (e.g., intestinal pH, compound dissociation) (Endo et al. 1990). Age and diet also can influence the extent of absorption in mice (Kostial et al. 1978). One-week-old suckling mice absorbed 38% of the orally administered mercuric chloride, whereas adult mice absorbed only 1% of the dose in standard diets. When the adult mice received a milk diet instead of the standard diet, absorption increased to 7% of the administered dose (Kostial et al. 1978).

Several studies suggest that the bioavailability of mercuric sulfide in animals is less than that of mercuric chloride (Sin et al. 1983, 1990; Yeoh et al. 1986, 1989). For example, Sin et al. (1983) found an increase in tissue levels of mercury in mice orally exposed to low doses of mercuric chloride, but elevated levels of mercury were not found in the tissues of mice fed an equivalent weight of mercury as mercuric sulfide. This finding indicates a difference in bioavailability between $HgCl_2$ and HgS in mice. However, a quantitative determination of the relative bioavailabilities of mercuric sulfide versus mercuric chloride has not been derived in the available studies. Furthermore, the relative bioavailability of mercuric sulfide in humans has not been examined.

Organic Mercury. Organic mercury compounds are more readily absorbed by the oral route than inorganic mercury compounds. Based on retention and excretion studies in humans, approximately 95% of an oral tracer dose of aqueous methylmercuric nitrate was absorbed (Aberg et al. 1969). Absorption of mercury was also reported in studies in which volunteers received doses of methylmercury bound to protein (Miettinen 1973) or ate bread contaminated with a fungicide that contained methylmercury (Al-Shahristani et al. 1976); however, no quantitative data regarding the percentage of absorption were available.

In vitro evidence suggests that organic mercury is also readily absorbed in the gastrointestinal tract and that methylmercuric chloride is absorbed to a greater extent than phenylmercuric chloride (Endo et al. 1989). Complexing of methylmercury with nonprotein sulfhydryls also may play a role in intestinal absorption and reabsorption (Urano et al. 1990). Phenylmercuric salt in the diet was completely absorbed in mice (Clarkson 1972a) and readily absorbed in rats following long-term oral administration (Fitzhugh et al. 1950). Absorption was nearly complete within 6 hours after female cynomolgus monkeys were given 0.5 mg Hg/kg as methylmercuric chloride by gavage (Rice 1989b). Following a single oral exposure

(1 mg/kg) of methylmercuric chloride, the level of mercury in the blood of mice declined slowly. At day 14 post-dosing, the blood level was still around 25% of the value at day 1 (Nielsen 1992). Blood levels of mercury were closely correlated to whole-body retention of mercury during the first 3 days after administration of methylmercuric chloride (1 mg Hg/kg) (Nielsen and Andersen 1992). However, at later times after administration, the amount of mercury in the blood declined more rapidly than the whole-body burden. The gastrointestinal retention of mercury slowly decreased in mice given organic mercury. This phenomenon is probably the result of biliary excretion and reabsorption of mercury (Nielsen and Andersen 1992).

Bioavailability of methylmercury in food. Measurements of absorption and toxicity have generally been made using aqueous solutions of methylmercury. The absorption and bioavailability of methylmercury in food, specifically fish and bread, may be affected by dietary components. Potential confounders that may affect bioavailability of methylmercury are dietary phytate and other dietary fibrous materials found in bread and the complexation of methylmercury with selenium in fish.

Dietary fiber and phytate. Dietary fiber and phytate are known as potential inhibitors of the absorption of divalent cations; however, the literature regarding the effect of dietary fiber and phytate on the bioavailability of minerals is contradictory. Data by Yannai and Sachs (1993) indicate that phytate does not affect methylmercury absorption. Yannai and Sachs (1993) compared the absorption by rats of mercury found intrinsically in experimental fish meal with and without added phytate and found no significant differences in the absorption of Hg (93±5%) between 2 experimental fish meal diets (containing 1.4 μmol Hg/kg diet), with or without added sodium phytate. The authors speculated that phytate might be preferentially bound to zinc, iron, and copper, which were present at much higher concentrations in the diet.

In another experiment by Yannai and Sachs (1993), the absorption of mercury was reduced when rats were fed a mercury-contaminated corn diet and corn silage meal. Mercury was incorporated intrinsically into the corn diet using radioactive isotopes (^{203}Hg) infused by capillary action into the stalks of developing corn plants, which then incorporated trace amounts of isotopes into developing kernels. The corn silage meal was from a crop grown in the vicinity of an industrial zone and contained elevated amounts of mercury. Reduced absorptions of 48 and 51% were found for the corn silage and corn diet experiments, respectively.

The reduced bioavailability of the plant food diet compared with the animal-based diet (fish meal) may be due to the presence of indigestible fibrous materials present in plants. Another factor that might affect absorption is the form of mercury (^{203}Hg and methylmercury in the corn and fish meal diets, respectively). The experiments by Yannai and Sachs (1993) are different from other absorption experiments because mercury was intrinsic to the fish, grain, or silage, while in other studies mercury is simply mixed with the experimental diet, usually as mercury salts. In the Iraqi epidemic, methylmercury fungicide was applied extrinsically to wheat that was made into bread. However, no studies were located that measured the absorption of methylmercury when mixed with grain. It is also not known whether the putative component(s) of grain affecting bioavailability are the same in corn and wheat.

Interaction with selenium in diet. The co-administration of methylmercury and selenium is known to depress methylmercury toxicity (Cuvin-Aralar and Furness 1991; Imura and Naganuma 1991). Furthermore, the level of selenium in human hair has been found to negatively correlate with the level of mercury in brain tissue (Suzuki et al. 1993). Methylmercury forms a bismethylmercury selenide complex. Selenium in foods (especially fish) may also complex with methylmercury and, therefore, may potentially reduce the bioavailability of methylmercury. The available data indicate that neither methylmercury uptake nor bioavailability is affected by its presence in fish. Experimental studies on the metabolism of methylmercury in humans following oral ingestion using methylmercury bound to fish muscle protein have shown that absorption is almost complete (95% absorbed) (Miettinen 1973). Animal studies also support this absorption value. Data on cats given fish homogenates indicate absorptions of $90% of methylmercury added to the homogenate, of methylmercury accumulated by fish *in vivo*, or from methylmercury proteinate (Berglund et al. 1971). Using blood and tissue levels as evidence of absorption, Charbonneau et al. (1976) concluded that there was no difference in the biological availability of methylmercury administered to adult cats (0.003, 0.0084, 0.020, 0.046, 0.074, or 0.176 mg Hg/kg/day 7 days a week for 2 years) either as pure methylmercuric chloride in corn oil added to a diet containing uncontaminated fish or as methylmercury-contaminated fish. In the 2 highest dose groups (0.074 and 0.176 mg Hg), at 100 weeks of exposure no significant differences were seen in total mercury concentrations in blood between groups receiving the dose as methylmercuric chloride or as contaminated fish at the same dose level. In addition, monthly blood levels were comparable for all dose groups. No significant differences were seen at 100 weeks in total mercury concentrations in the nervous system tissue or other tissues (renal cortex, renal medulla, liver, spleen, adrenal, bladder, atria, ventricle, ovaries, testes, muscle) between the 2 highest dose groups receiving the dose as methylmercuric chloride or as contaminated fish at the same dose level.

2.3.1.3 Dermal Exposure

Metallic and Inorganic Mercury. Hursh et al. (1989) conclude that dermal absorption of mercury vapor poses a very minor occupational hazard compared to inhalation exposure. They measured dermal absorption of radiolabeled metallic mercury vapor in five human volunteers, using arm skin as representing the whole body skin. About half of the mercury taken up was shed by desquamation of epidermal cells during the following several weeks. The remainder was slowly and diffusely released into the general circulation in contrast to the rapid release and more focal release from the lungs. When absorption for the total skin area (as represented by the forearm skin) was compared with the inhalation route for the same ambient concentration, the dermal route absorbed was estimated at 2.6% of the amount absorbed by the lung.

There was no information found on the dermal absorption of liquid metallic mercury, but unless the skin surface was damaged or the contaminated surface was occluded, it would not be expected to be high (i.e., in light of the very low absorption rate from the gastrointestinal tract). On the other hand, sloughing from the gastrointestinal tract may account for the low rate of absorption.

Indirect evidence of dermal absorption is provided by clinical case studies in which mercury intoxication was reported in individuals following dermal application of ointments that contained inorganic mercury salts (Bourgeois et al. 1986; DeBont et al. 1986).

Absorption of mercurous salts in animals can occur through the skin (Schamberg et al. 1918); however, no quantitative data are available. The rate of absorption for mercuric chloride was not evaluated in any study. However, skin biopsies taken from 2 to 96 hours after application of a 0.1% solution of mercuric chloride showed electron-dense deposits, tentatively identified as mercury, in the cells in the dermis, indicating that mercuric chloride could penetrate the outer layer of the skin (Silberberg et al. 1969).

Organic Mercury. No information was identified for absorption of methylmercury via dermal absorption. There is extremely important hazard assessment information on the dermal absorption of dialkylmercurials. A case history indicates nearly complete absorption of dimethylmercury through the skin resulting in a highly toxic exposure pathway. The exposure occurred to a 48-year-old female chemistry professor who was admitted to the hospital 5 months (154 days) after she inadvertently spilled several drops (estimated at 0.4–0.5 mL) of dimethylmercury from the tip of her pipette onto the back of her disposable latex gloves

(Blayney et al. 1997; Nierenberg et al. 1998). The spill was cleaned and the gloves disposed of. Hair analysis on a long strand of hair revealed that, after a brief lag time, mercury content rose rapidly to almost 1,100 ppm (normal level <0.26 ppm, toxic level >50 ppm), and then slowly declined, with a half-life of 74.6 days. These results support the occurrence of one or several episodes of exposure, and are consistent with laboratory notebook accounts of a single accidental exposure. Testing of family members, laboratory coworkers, and laboratory surfaces also failed to reveal any unsuspected mercury spills or other cases of toxic blood or urinary mercury levels. Permeation tests subsequently performed on disposable latex gloves similar to those the patient had worn at the time of the lone exposure revealed that dimethylmercury penetrates such gloves rapidly and completely, with penetration occurring in 15 seconds or less and perhaps instantly. Severe neurotoxicity developed 5 months postexposure and the patient died 9 months postexposure. The mercury content of hair, blood, and urine were monitored from 5 months postexposure (i.e., following admission of the patient to the hospital) until the patient died. Based on the half-lives and kinetics of mercury in the body, the hair and blood levels were used to estimate the total body burden and the amount of the initial acute dermal dose. They determined that a dose of 0.44 mL of liquid Dimethylmercury (about 1,344 mg), if completely absorbed, would have been sufficient to have produced the levels observed in the patient. This amount is in good agreement with the patient's account and the laboratory records on the amount spilled. Some inhalation exposure, however, could also have occurred during the cleanup of the spill, so this finding needs additional confirmation.

Infants exposed to diapers that had been treated with a phenylmercury fungicide exhibited higher urinary levels of mercury than unexposed infants (Gotelli et al. 1985). In rats, dermal absorption of phenylmercuric acetate from the vaginal tract was 75% of the dose within 8 hours after administration (Laug and Kunze 1949).

2.3.1.4 Other Routes of Exposure

There is some information on the subcutaneous injection of metallic mercury. Schwarz et al. (1996) describe a case history of a female nurse who accidentally plunged a mercury thermometer into her left hand while shaking it. Radiographic imaging revealed that some liquid metallic mercury had infiltrated into the soft tissues of her palm (amount unspecified). The diffusely distributed mercury could not be removed surgically. No immediate follow-up mercury levels in blood or urine were reported. A slightly elevated blood mercury concentration (15 µg/L, toxic level >50) was reported 2 years after this event, which then

declined (no reason provided). Other sources of mercury could have caused the increase, so little can be concluded about how much of the subcutaneous liquid mercury entered the systemic circulation.

In a much more informative case history, a 19-year-old man had injected mercury subcutaneously (Bradberry et al. 1996). Blood and urine mercury concentrations were followed for 6 years after presentation. Hematological and biochemical profiles were normal. Histological results indicated a chronic inflammatory reaction with granuloma formation, secondary to the globular mercury. A postoperative X-ray of the elbow indicated persistent subcutaneous mercury particles. Apart from the initial local discomfort, the patient remained asymptomatic and clinical examination revealed no abnormality up to 6 years postsurgery. No systemic features of mercury poisoning were evident. Blood mercury levels declined from 60 to 70 µg/L at 1 year postoperation to 10 µg/L at 6 years. Serial sampling results of total mercury in 24 urine collections indicated peaks up to 1.2 mg during the first year postoperation, which declined to 59 µg/L at 6 years. The elevated blood and urine levels indicate some systemic absorption. The effects of the surgery on migration of mercury from the subcutaneous tissue to the systemic circulation are not known.

2.3.2 Distribution

In humans, metallic mercury is distributed throughout the body following inhalation exposure. It can readily cross the blood-brain and placental barriers because of its high lipophilicity. After oxidation to mercuric mercury, it accumulates primarily in the kidneys. Inorganic divalent mercury compounds similarly reach all organs; however, the extent of accumulation in the brain and fetus is lower than metallic mercury because of the lower lipophilicity of inorganic mercury compounds. Organic mercury compounds distribute throughout the body following oral exposure and have the highest accumulation in the kidneys. As with metallic mercury, the ability of methyl- and phenyl mercury compounds to cross the blood-brain and placental barriers allows distribution, and subsequent accumulation, in the brain and fetus.

2.3.2.1 Inhalation Exposure

Metallic Mercury. The lipophilic nature of metallic mercury results in its distribution throughout the body. Metallic mercury in solution in the body is highly lipophilic, thereby allowing it to cross blood-brain and placental barriers with ease (Clarkson 1989). Mercury distributes to all tissues and reaches peak levels within 24 hours, except in the brain where peak levels are achieved within 2–3 days (Hursh et al. 1976).

The longest retention of mercury after inhalation of mercury vapor occurs in the brain (Takahata et al. 1970). Japanese workers who died 10 years after their last exposure to metallic mercury vapors still had high residual levels of mercury in their brains (Takahata et al. 1970). Autopsies of 3 dentists revealed 0.945–2.110 mg Hg/kg in the renal cortex, compared to 0.021–0.810 mg Hg/kg for unexposed controls (Nylander et al. 1989).

In volunteers who inhaled a tracer dose of metallic mercury vapor for 20 minutes, approximately 2% of the absorbed dose was deposited per liter of whole blood after the initial distribution was complete (Cherian et al. 1978). Uptake into the red blood cells was complete after 2 hours, but plasma uptake was not complete until after 24 hours. Mercury concentration in red blood cells was twice that measured in the plasma. This ratio persisted for at least 6 days after exposure. However, the ratios of 1–2 have been reported for metallic mercury vapor (Miettinen 1973).

Exposure of rats to mercury vapor (10–100 μg/m^3) for 6 hours a day, 5 days a week from the 4th through 11th weeks of life resulted in measurable amounts of mercury in the blood, hair, teeth, kidneys, brain, lungs, liver, spleen, and tongue, with the kidney cortex having the highest mercury concentration (Eide and Wesenberg 1993). Further, tissue concentrations were positively and significantly correlated with exposure concentrations. In this study, the rat molars were found to have the highest correlation coefficient with measured kidney mercury values, leading to a suggestion by the authors that human deciduous teeth may be useful indicators of chronic mercury exposure and of the mercury uptake by the kidneys and cerebrum (Eide and Wesenberg 1993). In another study, a 4-hour exposure of mice to metallic mercury vapor produced the highest mercury retention in the brain compared to other organs (Berlin et al. 1966). Exposure of mice to metallic mercury vapor (8 mg/m^3, for 6 hours a day for 10 days) resulted in higher mercury levels in the gray than in the white brain matter (Cassano et al. 1966, 1969). Exposure of rats to 1 mg/m^3 metallic mercury vapor for 24 hours a day every day for 5 weeks or 6 hours a day, 3 days a week for 5 weeks resulted in mean mercury brain concentrations of 5.03 and 0.71 μg/g, respectively (Warfvinge et al. 1992). Mercury was found primarily in the neocortex, basal nuclei, and the cerebellar Purkinje cells.

Mercury also accumulates in several cell types populating the dorsal root ganglia (Schionning et al. 1991). After 12–14 hours of exposure of rats to a relatively small amount of metallic mercury vapor (0.55 mg/m^3), accumulation of mercury was observed within all cell types examined (ganglion cells, satellite cells, fibroblasts, and macrophages). Mercury has also been detected in dorsal root neurons and satellite cells of

primates exposed for one year to mercury through amalgams in dental fillings or the maxillary bone (Danscher et al. 1990).

The kidney is the major organ of mercury deposition after inhalation exposure to metallic mercury vapor. Mercury concentrations in the kidneys are orders of magnitude higher than in other tissues (Rothstein and Hayes 1964). Monkeys exposed for one year to metallic mercury vapor from amalgam in dental fillings accumulated mercury in the spinal ganglia, anterior pituitary, adrenal, medulla, liver, kidneys, lungs, and intestinal lymph glands (Danscher et al. 1990). The largest deposits of mercury were found in the kidneys (2.5–5.2 ppm), specifically in the proximal tubule cells.

The kidney contains metallothionein, a metal-binding protein that is also found in fetal and maternal livers and other organs. In the kidneys, the production of metallothionein is stimulated by exposure to mercury. The increased levels of metallothionein increase the amount of mercuric ion binding in the kidneys (Cherian and Clarkson 1976; Piotrowski et al. 1973). Three classes of sulfhydryl groups have been identified in the kidneys, with metallothionein having the greatest affinity for mercury (Clarkson and Magos 1966). Low molecular-weight complexes of mercury have been identified in the urine, suggesting that they may exist in the kidneys and contribute to the kidneys' accumulation of mercury (Piotrowski et al. 1973).

Metallothionein exists in higher concentration in the fetal liver than in the maternal liver of rats. Exposure to mercury in the pregnant dam results in the binding of mercury to metallothionein in fetal liver initially, followed by a redistribution to other organs (Yoshida et al. 1990). Metallothionein and mercury levels were elevated in the kidneys of guinea pig neonates exposed to 6–10 mg/m^3 mercury vapor (Piotrowski et al. 1973).

After exposure to mercury vapor, mercury is distributed throughout the body in different chemical and physical states. Metallic mercury dissolves in the blood upon inhalation, and some remains unchanged (Magos 1967). Metallic mercury in the blood is oxidized to its divalent form in the red blood cells (Halbach and Clarkson 1978). The divalent cation exists as a diffusible or nondiffusible form. The nondiffusible form is mercuric ions that bind to protein and are held in high-molecular weight complexes, existing in equilibrium with the diffusible form.

In the plasma, the mercuric ion is predominantly nondiffusible and binds to albumin and globulins (Berlin and Gibson 1963; Cember et al. 1968; Clarkson et al. 1961). Following mercuric salt administration,

levels of mercuric ions in the plasma are similar to levels of mercuric ions in the red blood cells. Binding of mercury also occurs in tissues, and retention varies, with the brain retaining mercury the longest.

The influence of age on mercury distribution following exposure to metallic mercury was evaluated in neonatal (12 hours old) and adult guinea pigs exposed to 8 or 10 mg Hg/m^3 vapor for 120 minutes (Yoshida et al. 1989). The mercury concentrations were 28, 58, and 64% higher in the brain, lungs, and heart, respectively, of the neonates compared to the mothers. However, the mercury level in the kidneys was approximately 50% lower in the neonates. The lower uptake of mercury in the kidneys of neonates may be due to the functional immaturity of the kidneys at parturition. The higher levels in other highly perfused tissues suggest that mercury accumulation in organs is dependent on how easily metallic mercury can reach the tissues from blood. Similar findings were reported by Jugo (1976) who found higher mercury concentrations in the liver, blood, and brain, but lower concentrations in the kidneys of 2-week-old rats compared to similar tissues in 21-week-old rats. These results also suggest that infants may accumulate mercury more readily after acute exposure and, therefore, may be more likely to exhibit neurotoxicity from mercury vapors.

The extent of mercury accumulation with aging was studied in mice maintained under normal care conditions in a conventional rodent colony without exposure to known mercury sources other than background concentrations normally found in food, water, and air (Massie et al. 1993). There was no significant change in the total amount of mercury in the organs (lungs, heart, brain, and liver) from male C57BL/6J mice ranging in age from 133 to 904 days. However, the ratios of mercury levels in the brain to mercury levels in the liver and kidneys were found to increase significantly and dramatically with age. The increase with aging in the brain-to-liver and brain-to-kidneys ratios suggests that mercury removal from the brain may be less efficient in some older organisms.

Metallic mercury vapor easily penetrates the placental barrier and accumulates in fetal tissues. The high lipophilicity of metallic mercury favors its penetration across the barrier. The uptake of mercury appears to increase during the later gestation period in mice, as indicated by increased mercury accumulation in the fetus after exposure to metallic mercury (Dencker et al. 1983). Guinea pig fetuses that were exposed to 6–13 mg/m^3 mercury vapor during late gestation had elevated mercury concentrations in the liver, while the levels in other tissues were only slightly increased relative to controls (Yoshida et al. 1990). Newborn guinea pigs that were nursed by their mothers, who had been and exposed to mercury vapor (6–9 mg/m^3) for 120 minutes immediately after parturition, had the highest mercury concentrations in the kidneys,

followed by the liver and lungs (Yoshida et al. 1992). In the brain and whole blood, mercury concentrations were slightly elevated compared to nonexposed controls. Levels of mercury in the fetus were approximately 4 times higher after exposure to metallic mercury vapor than after mercuric chloride administration for mice and 10–40 times higher for rats (Clarkson et al. 1972). The transport of the mercuric ions is limited at the placental barrier by the presence of high-affinity binding sites (Dencker et al. 1983).

Inorganic Mercury. No studies were located regarding the distribution of inorganic mercury in humans or animals following inhalation exposure to inorganic mercury compounds.

Organic Mercury. No studies were located regarding the distribution of organic mercury in humans or animals following inhalation exposure to organic mercury compounds.

2.3.2.2 Oral Exposure

Metallic and Inorganic Mercury. Data on the distribution of ingested elemental mercury were not located, and data on the ingestion of inorganic mercury are limited. The metallic mercury that is absorbed from an oral exposure is expected to resemble many aspects of the distribution of mercuric salts because metallic mercury is oxidized to mercuric ion in biological fluids, and the resulting distribution reflects that of the mercuric ion. Unlike elemental mercury, however, the amount of divalent mercury that crosses the blood-brain and placental barriers is much lower because of its lower lipid solubility (Clarkson 1989).

In some studies there is a combined exposure to both organic and inorganic mercury. Oskarsson et al. (1996) assessed the total and inorganic mercury content in breast milk and blood in relation to fish consumption and amalgam fillings. Total mercury concentrations were evaluated in breast milk, blood, and hair samples collected 6 weeks after delivery from 30 lactating Swedish women. In breast milk, about half of the total mercury was inorganic and half was methylmercury, whereas in blood only 26% was inorganic and 74% was methylmercury. The results of a regression analysis for mercury in hair, blood, and milk indicated that there was an efficient transfer of inorganic mercury from blood to breast milk and that mercury from amalgam fillings was probably the main source of mercury in breast milk, while methylmercury levels in blood did not appear to be efficiently transferred to breast milk. Exposure of the infant to mercury in breast milk was calculated to range up to 0.3 µg/kg/day, of which approximately one-half was inorganic mercury. This exposure corresponds to approximately one-half the tolerable daily

intake of total mercury for adults recommended by the World Health organization. The authors concluded that efforts should be made to decrease total mercury burden in women of reproductive age Oskarsson et al. (1996).

Inorganic Mercury. The liver and kidneys of mice had the highest mercury levels 14 days after exposure to a single oral dose of 0.2–20 mg ^{203}Hg/kg as mercuric chloride (Nielsen and Andersen 1990). The brain has substantially lower mercury levels; however, retention was longest in this tissue. Sin et al. (1983) report that the kidneys also had the highest mercury levels following repeated oral exposure of mice to mercuric chloride (4–5 mg Hg/kg) for 2–8 weeks. Mercuric sulfide did not accumulate in the tissues of mice to any significant extent following exposure to low levels of mercuric sulfide (4–5 mg Hg/kg) for 2–8 weeks (Sin et al. 1983). However, the mercury content in the liver and kidneys of mice treated with higher doses of mercuric sulfide (. 8–200 mg Hg/kg/day) for 7 days was significantly increased compared to the controls (Yeoh et al. 1986, 1989). Mice fed mercuric sulfide (86 mg Hg/kg/day) for 1 week exhibited a 21-fold increase in the kidneys' mercury content ($p<0.001$) and an 8.6-fold increase in the liver content compared to controls (Yeoh et al. 1989). Moderate renal effects, with a corresponding mercury concentration of 50 µg/g in the kidneys, were seen in rats exposed to mercuric nitrate (Fitzhugh et al. 1950).

Mercury can accumulate in human hair following oral exposure to mercuric chloride (Suzuki et al. 1992). Hair mercury levels, determined using segmental hair analysis, can be used to monitor exposure to mercury and may leave a historical record of exposure or uptake. In hair cut 41 days after mercuric mercury ingestion (13.8 mg/kg), a sharp peak (40 nmol/g [8 µg/g]) was found in the 1 cm segment closest to the scalp, while the levels were #5 nmol/g in all other segments. Ninety-five days after ingestion, the peak of inorganic mercury shifted to the 2–3 cm segment, while 160 days after ingestion the peak shifted to the 3–4 cm segment. During this time, the height of the peak decreased. An estimated biological half-life of inorganic mercury in hair was 57.8 days. Inorganic mercury in hair had different patterns of longitudinal variation from that of organic mercury.

Organic Mercury. Distribution of organic mercury compounds in humans and animals is similar to that of metallic mercury. Methylmercury distributes readily to all tissues, including the brain and fetus, after absorption from the gastrointestinal tract. The uniform tissue distribution is due to methylmercury's ability to cross diffusion barriers and penetrate all membranes without difficulty (Aberg et al. 1969; Miettinen 1973). Thus, tissue concentrations tend to remain constant relative to blood levels. About 90% of the

methylmercury in blood is found in the red blood cells (Kershaw et al. 1980). The mean mercury concentrations in red blood cells were 27.5 ng/g and 20.4 ng/g in males and females, respectively, exposed to mercury, primarily from mercury-contaminated fish (Sakamoto et al. 1991). Because of this uniform distribution in tissues, blood levels are a good indicator of tissue concentrations independent of dose (Nordberg 1976).

Although distribution is generally uniform, the highest levels of organic mercury are found in the kidneys (Nielsen and Andersen 1991b; Rice 1989b; Ryan et al. 1991). After a single oral dose of 0.04, 0.2, 1, or 5 mg Hg/kg as methylmercuric chloride administered to mice, mercury was retained mostly in the kidneys and liver at 14 days postexposure (Nielsen and Andersen 1991a). The deposition of mercury in the carcass was about 70%, with retention primarily in the skin, hair, and muscles and to a lower degree in the fat and bones (Nielsen and Andersen 1991b). More than 200 days after cynomolgus monkeys were given 0.025 and 0.05 mg Hg/kg/day as methylmercuric chloride in apple juice for about 2 years, the kidneys contained 10.18–27.89 ppm mercury in the cortex and 1.12–10.11 ppm in the medulla, compared to <2 ppm in the other tissues measured (Rice 1989b).

Demethylation of methylmercury to inorganic mercury is species-, tissue-, dose-, and time-dependent. The demethylated inorganic mercury accumulates in the kidney and liver. Suda et al. (1991) evaluated the transformation of methylmercury to inorganic mercury by phagocytic cells. The liver and kidneys are also potential sites of biotransformation (Lind et al. 1988; Magos et al. 1976; Norseth and Clarkson 1970).

The distribution of mercury in the brain has been studied in humans following oral absorption of organic mercury. It is suggested by Aschner and Aschner (1990) that, following acute exposure to methylmercury, most of the total mercury in the brain is represented by organic mercury; however, after chronic exposure, most of the mercury in the brain is inorganic mercury. An explanation for these findings is that organic mercury is converted into inorganic mercury in the brain. After chronic methylmercury exposure in monkeys, estimated half-lives were considerably longer in brain than in blood, also possibly due to conversion of methylmercury to a form that is highly bound to brain tissue (Rice 1989b).

The autopsy of a man whose first symptoms of methylmercury poisoning occurred 26 years earlier revealed that the highest mercury levels (0.62–1.19 μg Hg/g) were in the gyrus of the cerebral cortex, cerebellum, pallidum, and occipital pole of the brain (Takeuchi et al. 1989). Furthermore, total mercury levels (0.02–1.19 μg/g) were much higher than methylmercury levels (approximately <0.01 μg/g) in the brain.

This finding supports the assumption by Suda et al. (1989) that ingested methylmercury is dealkylated to inorganic mercury in the brain.

Monkeys were fed 0.05 or 0.09 mg Hg/kg/day as methylmercury, containing 5% impurity of inorganic mercury, for 0.5–1.5 years (Lind et al. 1988). The low-dosed monkeys were found to have 10–33% of the total mercury present in the inorganic form in brain cortices, while the high-dosed monkeys had 90% in the inorganic form. Demethylation of methylmercury in the brain, as well as in other organs, including the kidneys and liver, is believed to contribute substantially to the high concentration of inorganic mercury in the brain. Following oral exposure to methylmercuric chloride, regional distribution of total mercury in the brain of monkeys was observed; the highest levels were in the thalamus and hypothalamus (Rice 1989b).

In contrast, in the brain of 21-day-old neonatal rats that had been previously exposed to a gavage dose of 6.4 mg Hg/kg as methylmercury chloride *in utero*, the cerebellum had the highest mercury concentrations and the brainstem had the lowest (Braghiroli et al. 1990). By 60 days of age, concentrations in the brain reached normal values, with an estimated half-life of approximately 37 days (Braghiroli et al. 1990). Therefore, age can affect regional distribution in the brain of animals.

Massie et al. (1993) reported no significant change in the total amount of mercury in the organs (lung, heart, kidney, brain, and liver) of male C57BL/6J mice ranging in age from 133 to 904 days of age maintained under conventional conditions with no known source of mercury exposure other than background concentrations. The ratio of mercury in the brain to that in the liver or to that in the kidney was significantly increased with age, indicating that older mice are less able to maintain a low brain-to-liver ratio of mercury regardless of the total body content of mercury.

In a study of organs from sledge dogs fed methylmercury-laden meat and organs from predatory marine animals (Hansen and Danscher 1995), the highest concentration of total mercury was found in the mesenterial lymph nodes, followed by liver and kidneys, indicating that the lymphatic system may play an important role in the transport of mercury to target organs. The tissue concentrations of mercury observed in this study were found to be age-related, and the results suggest that demethylation takes place in all organs, except the skeletal muscle. Demethylation of methylmercury was found to be lower in the brain than in other organs (Hansen and Danscher 1995).

Mercury accumulates in hair following exposure to methylmercury in humans and mice (Grandjean et al.

1992; Nielsen and Andersen 1991a, 1991b; Soria et al. 1992; Suzuki et al. 1992). Hair mercury levels, determined using segmental hair analysis, can be used to monitor exposure to mercury and may leave a historical record of exposure or uptake (Phelps et al. 1980; Suzuki et al. 1992). The concentration of mercury in the hair is considered proportional to the concentration of mercury in the blood. Correlations can be drawn to determine blood concentrations of mercury relative to its concentration in the hair (see the discussion of the methylmercury MRL in Section 2.5). Mercury concentrations in maternal hair were significantly correlated with cord blood levels of mercury in pregnant women who had frequently ingested whale meat throughout pregnancy (Grandjean et al. 1992). The frequent ingestion of whale meat dinners during pregnancy and, to a lesser degree, the frequent consumption of fish, as well as increased parity or age, were associated with high mercury concentrations in cord blood and hair. The incorporation of mercury into hair is irreversible; the loss of hair mercury occurs as the result of hair loss (Nielsen and Andersen 1991b).

As with metallic mercury, methylmercury can readily traverse the placental barrier. In humans with no known exposure to mercury, blood mercury levels increased with advancing gestation such that the mean blood mercury level on admission for delivery (1.15 ppb) was significantly higher than that of the first prenatal visit (0.79 ppb) (Kuntz et al. 1982). Cord blood levels were similar to maternal blood values in labor and postpartum. Concentrations of methylmercury in the fetal blood are slightly higher than in the maternal blood (Inouye and Kajiwara 1988; Kuhnert et al. 1981). Following an oral dose of methylmercuric chloride during gestation, accumulation of mercury was much greater in the fetal kidneys than in the maternal kidneys of guinea pigs (Inouye and Kajiwara 1988). Mercury levels in the liver were slightly higher in the fetus compared to the dam when exposed to organic mercury at late gestation but were similar at early gestation. Distribution of mercury in the maternal and fetal brains was uneven, with the highest concentrations in the neopallium, diencephalon, and mesencephalon and the lowest in the rhombencephalon. Exposure at later gestational weeks resulted in higher concentrations for both maternal and fetal brains (Inouye and Kajiwara 1988).

Methylmercury may also be secreted in mother's milk (Bakir et al. 1973). Following intravenous dosing of methylmercuric chloride (1.6 mg Hg/kg) to pregnant mice on one of days 9–17 of pregnancy, methylmercury was readily transferred to the fetuses from the mothers more predominantly at the later gestational stage (Inouye and Kajiwara 1990). The placental transfer of methylmercury was more efficient compared to the lactational transfer in rats exposed to methylmercury in the diet during 11 weeks prior to mating, during gestation, and during lactation (Sundberg and Oskarsson 1992). A higher concentration of mercury

in the brain in relation to the blood mercury concentration was found after exposure *in utero* compared to exposure in milk. Mercury was present as methylmercury in the blood of the offspring exposed only during gestation, indicating little or no demethylation during the first 15 days after birth. However, inorganic mercury was present in the blood of offspring exposed only through milk, probably resulting from demethylation of methylmercury in the dam and transport of inorganic mercury to the sucklings through milk.

In animal studies, mercury transfer to and distribution in offspring depends on the form administered to the dam. Yoshida et al. (1994) administered either mercury chloride or methylmercury at 1 mg Hg/kg body weight to maternal guinea pigs (Hartley strain) via intraperitoneal injection 12 hours after parturition. Exposure of the offspring was studied on days 3, 5, and 10 postpartum. Concentrations of mercury were lower in the milk than in maternal plasma regardless of the form of administered mercury, but total milk mercury was higher in the dams given mercury chloride. While the ratio of methylmercury to total mercury decreased in plasma from dams, it did not decrease in the milk. Regardless of the form of mercury given to the dams, the highest concentration of mercury in the offspring was found in the kidney, followed by the liver and the brain. Brain mercury, however, was significantly higher in the offspring of methylmercury-treated dams. Mercury levels in major organs of the offspring peaked at 5 days from mercury-chloride-treated dams and at 10 days from methylmercury-treated dams.

Tissue distribution of phenylmercury is initially similar to methylmercury. One week after administration, the distribution pattern resembles that seen after administration of inorganic compounds (Nordberg 1976). Once in the blood, phenylmercury distributes to a greater extent into the red blood cells than the plasma. Phenylmercury also predominantly distributes to the liver (Berlin 1963). It is less permeable to the placental and blood-brain barriers than methylmercury (Yamaguchi and Nunotani 1974). Phenylmercury also accumulates in the fur of rats but to a lesser extent than detected with methylmercury exposure (Gage 1964).

2.3.2.3 Dermal Exposure

No information was identified for distribution of metallic, inorganic, or methylmercury via dermal absorption. A case history for a dermal absorption of dimethylmercury (see Section 2.3.1.3) does provide some information on distribution (Blayney et al. 1997; Nierenberg et al. 1998). A 48-year-old female absorbed approximately 0.4–0.5 mL of dimethylmercury (about 1,500 mg) through the skin on the dorsal

side of her hand. A preliminary laboratory report at 5 months after exposure indicated that the whole-blood mercury concentration was more than 1,000 µg/L (normal range, 1–8 µg/L; toxic level, >200 µg/L). Chelation therapy with oral succimer (10 mg/kg orally every 8 hours) was begun on day 168 after exposure. Whole blood concentrations rose to 4,000 µg/L after one day of chelation, and urinary mercury levels were 234 µg/L (normal range, 1–5 µg/L; toxic level, >50 µg/L). Chelation therapy continued up to the time of the patients death 298 days postexposure, with blood mercury level falling to around 200 µg/L. Metal analysis of the patient's tissues revealed extremely high levels of mercury in the frontal lobe and visual cortex (average value, 3.1 µg/g [3,100 ppb]), liver (20.1 µg/g), and kidney cortex (34.8 µg/g). The mercury content in the brain was approximately 6 times that of the whole blood at the time of death, and was much higher than levels in the brains of nonmercury exposed patients (2–50 ppb).

2.3.2.4 Other Routes of Exposure

Strain and sex differences were observed in renal mercury accumulation 4 hours after a subcutaneous methylmercuric chloride injection (1 µmol/kg) to 5 strains (BALB/cA, C57BL/6N, CBA/JN, C3H/HeN, and ICR) of male mice and 3 strains (BALB/cA, C57BL/6N, and ICR) of female mice (Tanaka et al. 1991). Mercury was distributed to the kidneys, brain, heart, lungs, liver, spleen, carcass, plasma, and red blood cells of all mice tested. Strain and sex differences were found in renal mercury content. In three strains (ICR, BALB/cA, and C57BL/6N), males showed higher renal mercury levels than females.

Differences in tissue concentrations in different inbred mice strains were evaluated by Griem et al. (1997). Female mice from five different strains (C57BL/6, B10.D2, B10.S, A.SW, and DBA/2) received 3 weekly subcutaneous injections of 0.5 mg Hg/kg body weight for up to 12 weeks. Except for the thymus, in which mercury concentrations continued to increase, steady state levels were obtained in blood and liver after 4 weeks and in spleen and kidney after 8 weeks. In the closely related strains C57BL/6, B10.D2, and B10.S, which differ only or primarily at the major histocompatibility complex, mercury concentrations in blood and liver were about 2-fold lower and renal concentrations were from 3- to 5-fold lower than measured in A.SW, and DBA/2 strains. Mercury concentrations in the spleen of C57BL/6, B10.D2, B10.S mice were significantly higher than in the spleen of A.SW, and DBA/2 mice. The higher concentration of Hg in this immune system organ concentration of C57BL/6, B10.D2, B10.S correlates with the increased susceptibility of these strains to a mercury chloride-induced systemic autoimmune syndrome. The strains with lower splenic mercury are more resistant.

Treatment of mice with ethanol results in increased accumulation of mercury in the fetus (Khayat and Dencker 1982). The concurrent generation of NADPH during the oxidation of alcohol enhances the reduction of mercuric ion to metallic mercury, making it more favorable for permeating the placenta. Mercuric chloride's limited ability to cross the placental barrier was also demonstrated in an intravenous study using mice (Inouye and Kajiwara 1990). Following intravenous dosing of mercuric chloride (1.4 mg/kg) to pregnant mice on 1 day between days 9 and 17 of pregnancy, mercuric chloride was transferred inefficiently to the fetus, being blocked almost completely by the fetal membrane. The mercury accumulated in the placenta and yolk sac but not in the amnion or fetal body (Inouye and Kajiwara 1990). A histochemical study demonstrated that mercuric mercury (Hg^{+2}) was blocked in the proximal wall of the yolk sac.

2.3.3 Metabolism

The available evidence indicates that the metabolism of all forms of mercury is similar for humans and animals. Once absorbed, metallic and inorganic mercury enter an oxidation-reduction cycle. Metallic mercury is oxidized to the divalent inorganic cation in the red blood cells and lungs of humans and animals. Evidence from animal studies suggests the liver as an additional site of oxidation. Absorbed divalent cation from exposure to mercuric mercury compounds can, in turn, be reduced to the metallic or monovalent form and released as exhaled metallic mercury vapor. In the presence of protein sulfhydryl groups, mercurous mercury (Hg^{+}) disproportionates to one divalent cation (Hg^{+2}) and one molecule at the zero oxidation state (Hg^{0}). The conversion of methylmercury or phenylmercury into divalent inorganic mercury can probably occur soon after absorption, also feeding into the oxidation-reduction pathway.

Metallic and Inorganic Mercury. Metallic mercury vapor is inhaled through the lungs and rapidly enters the bloodstream. The dissolved vapor can undergo rapid oxidation, primarily in the red blood cells, to its inorganic divalent form by the hydrogen peroxide-catalase pathway (Clarkson 1989; Halbach and Clarkson 1978). It is believed that the rate of oxidation is dependent on: (1) concentration of catalase in the tissue; (2) endogenous production of hydrogen peroxide; and (3) availability of mercury vapor at the oxidation site (Magos et al. 1978). In red blood cells *in vivo*, hydrogen peroxide production is probably a rate-determining step because Nielsen-Kudsk (1973) found that stimulation of hydrogen peroxide production in red cells increased the uptake of mercury vapors in red blood cells. After a low dose, the total mercury content in the blood is proportionately higher than (to the administered dose) after a high dose, indicating that a higher proportion of the lower dose is oxidized (Magos et al. 1989). The hydrogen peroxide-catalase

pathway in red cells may become saturated at higher dose levels (Magos et al. 1989). This oxidation pathway of metallic mercury can be inhibited by ethanol since ethanol is a competitive substrate for the hydrogen peroxide catalase and, consequently, can block mercury uptake by red blood cells (Nielsen-Kudsk 1973).

The oxidation of metallic mercury may also occur in the brain, liver (adult and fetal) (Magos et al. 1978), lungs (Hursh et al. 1980), and probably all other tissues to some degree (Clarkson 1989). In rat liver homogenates, hydrogen peroxide catalase is the predominant oxidative pathway in tissues. Its capacity is very high. Unlike oxidation in red cells, the rate-limiting step in *in vitro* oxidation in the liver is dependent on the rate of mercury delivery to the enzyme (Magos et al. 1978). Unoxidized metallic mercury can still reach the brain because the oxidation of metallic mercury is a slow process compared with the circulation time from the lungs to the brain (Magos 1967). In the brain, unoxidized metallic mercury can be oxidized and become trapped in the brain because it is more difficult for the divalent form to cross the barrier. Autoradiographic studies suggest that mercury oxidation also occurs in the placenta and fetus (Dencker et al. 1983), although the extent of oxidation is not known. The rate of distribution of metallic mercury to the brain and fetus is probably nonlinear because the rate of oxidation in red cells is nonlinear (i.e., can become saturated at higher doses) (Magos et al. 1989).

There is evidence to suggest that the divalent inorganic mercury cation is reduced by mammalian tissue to metallic mercury after its oxidation. Rats and mice pretreated parenterally with mercuric chloride exhale metallic mercury vapor (Clarkson and Rothstein 1964; Dunn et al. 1981a). Liver and kidney homogenates in animals also release mercury vapor after exposure to mercuric chloride. The amount of mercury released increases upon treatment with ethanol (Dunn et al. 1981b). This increase suggests that glutathione reductase is responsible for mercuric ion reduction (Williams et al. 1982). Oxidation of alcohol to acetaldehyde stimulates NADPH production, which is required for mercuric ion reduction. However, alcohol is primarily oxidized in the liver, and this location is not consistent with the increases in metallic mercury vapor released from the kidney homogenates (Dunn et al. 1981b).

Organic Mercury. Once absorbed, methylmercury can apparently be converted into inorganic mercury in tissues, specifically the divalent cation (Hg^{+2}) (Dunn and Clarkson 1980). Several investigators have reported high levels of inorganic mercury in tissues (Magos and Butler 1972; WHO 1990) and feces after methylmercury exposure (Turner et al. 1975). Rat liver microsomes can degrade methylmercury into inorganic mercury. Inorganic mercury production from methylmercury paralleled the hydroxyl radical

production (Suda and Hirayama 1992). The promotion and inhibition of the hydroxyl radical formation and the hydroxyl radical scavenger, affected inorganic mercury production. These results suggest that hydroxyl radicals produced from microsomes may play a predominant role in alkyl mercury degradation. NADPH-cytochrome P-450 reductase is known to be responsible for hydroxyl radical production in liver microsomes. Alkyl mercury degradation varied in proportion to the enzyme activities and hydroxyl radical production. These results suggest that hydroxyl radicals produced by cytochrome P-450 reductase might be the primary reactive species that induces alkyl mercury degradation. *In vitro* studies using a peroxidase-hydrogen peroxide-halide system indicated that besides the hydroxyl radical, hypochlorous acid (HOCl) scavengers are also capable of degrading methylmercury (Suda and Takahashi 1992). Also, metallic mercury exhaled in mice dosed with methylmercury was dependent on the level of inorganic mercury present in the tissue (Dunn and Clarkson 1980). The cation then enters the oxidation-reduction cycle, and metabolism occurs as discussed previously under Inorganic Mercury.

A small amount of an oral dose of methylmercuric chloride can also be converted into inorganic mercury in the intestinal flora (Nakamura et al. 1977; Rowland et al. 1980). However, inorganic mercury is poorly absorbed across the intestinal wall and, therefore, most of it is excreted.

Phenylmercury also rapidly metabolizes to inorganic mercury (Nordberg 1976). The metabolism of phenyl-mercury involves hydroxylation of the benzene ring to an unstable metabolite that spontaneously releases inorganic mercury. Consequently, its tissue disposition following initial metabolism resembles that seen after the administration of inorganic salts (Gage 1973).

Studies in mice indicate that toxicity from exposure to dimethylmercury is the result of metabolic conversion of dimethylmercury to methylmercury, and that dimethylmercury does not enter the brain until it has been metabolized to methylmercury, which occurs over the first several days following absorption (Ostland 1969). Nierenberg et al. (1998) report the results of an analyses of mercury content in the hair of a 48-year-old female who died subsequent to an acute exposure to dimethylmercury. The results are consistent with the kinetic profiles for methylmercury, and support the hypothesis of a rapid conversion of dimethylmercury to a methylmercury metabolite.

2.3.4 Elimination and Excretion

Elimination of metallic mercury occurs through the urine, feces, and expired air, while inorganic mercury is excreted in the urine and feces in humans. Animal data on excretion are limited but indicate that excretion is species and dose dependent. The feces are a major elimination route for inorganic mercury compounds, but high acute doses increase the percentage of excretion via the urine. Excretion of organic mercury is predominantly thought to occur through the fecal (biliary) route in humans. In animals, phenylmercury is excreted initially though the bile and then shifts to urine, whereas methylmercury is primarily excreted in the bile and then the feces. Age is a factor in the elimination of mercury in rats following inorganic and organic mercury exposure, with younger rats demonstrating significantly higher retention than older rats. Both inorganic and organic mercury compounds can be excreted in breast milk. There are no data suggesting that the route of exposure affects the ultimate elimination of inorganic and organic mercury that is absorbed into the body.

Metallic and Inorganic Mercury. The urine and feces are the main excretory pathways of metallic and inorganic mercury in humans, with a body burden half-life of approximately 1–2 months (Clarkson 1989). In a study of former chloralkali workers exposed to metallic mercury vapor for 2–18 years (median, 5 years), Sallsten et al. (1995) found that the elimination of mercury in urine was well characterized by a one-compartment model, which estimated a half-life of 55 days. There was a tendency toward longer half-lives with shorter duration exposures than with long-term exposure, when uptake and elimination have reached a steady state. This might be due to the induction of a higher metabolic rate after a longer exposure time, but there is no experimental evidence to support such an effect (Sallsten et al. 1995). For high-level exposure to inorganic divalent mercury, the urine is probably the major elimination route, with a half-life similar to that of metallic mercury (Clarkson 1989). An elimination half-life from urine was estimated to be 25.9 days following an acute exposure to a high level of mercuric chloride (13.8 mg/kg) (Suzuki et al. 1992). Exhalation in the lungs and secretion in saliva, bile, and sweat may also contribute a small portion to the excretion process (Joselow et al. 1968b; Lovejoy et al. 1974). After an acute mercury exposure in humans, urinary excretion accounts for 13% of the total body burden. After long-term exposure, urinary excretion increases to 58%. Humans inhaling mercury vapor for less than an hour expired approximately 7% of the retained dose of mercury (Cherian et al. 1978; Hursh et al. 1976). The half-life for this elimination pathway was 14–25 hours; therefore, excretion through expired air is negligible 5–7 days after exposure (Cherian et al. 1978). Using a two-compartment model, elimination half-lives in the urine of workers exposed for 20–45 hours to >0.1 mg/m^3 metallic mercury vapor were

estimated to be 28 and 41 days for a fast and slow phase, respectively (Barregard et al. 1992). Mercury is excreted in the urine following oral exposure to mercuric sulfide (0.5 mg Hg/kg) (Yeoh et al. 1989).

The overall elimination rate of inorganic mercury from the body is the same as the rate of elimination from the kidneys, where most of the body burden is localized (see Table 2-4). Inorganic mercury is also readily cleared from the lung. Elimination from the blood and the brain is thought to be a biphasic process with an initial rapid phase in which the decline in the body burden is associated with high levels of mercury being cleared from tissues, followed by a slower phase of mercury clearance from the same tissues (Takahata et al. 1970). An even longer terminal-elimination phase is also possible because of persistent accumulation of mercury, primarily in the brain (Takahata et al. 1970). Following a single oral dose of divalent mercury in 10 volunteers, 85% of the ^{203}Hg activity was excreted within 4–5 days, predominantly in the feces (Rahola et al. 1973).

Following acute mercury vapor intoxication of two humans, it was found that, despite chelation therapy with multiple chelators (2,3-dimercaptopropanol [BAL] followed by 2,3-dimercaptosuccoinic acid [DMSA]), relatively high concentrations of mercury remained in the plasma for a very long time (Houeto et al. 1994). The authors suggested that this could be explained by the progressive release of mercury from red blood cells and tissues after oxidation. In a group of chloralkali workers exposed to metallic mercury vapor for 1–24 years (median, 10 years), a decrease in the mercury concentration (following temporary discontinuation of exposure) in whole blood, plasma, and erythrocytes was found to be best characterized by a two-compartment model (Sallsten et al. 1993). Using a two-compartment model, half-lives were estimated, respectively, to be 3.8 and 45 days for the fast and slow phase in whole blood; plasma, 2 and 36 days in plasma, and 3.6 and 16 days in erythrocytes. The half-lives for the slow phases in whole blood and plasma were longer, and the relative fractions of the slow phases were higher (about 50%) after long-term exposures than after brief exposures (Sallsten et al. 1993).

Workers exposed to vapors of 0.016–0.68 mg Hg/m^3 had detectable levels of mercury in the urine (>2 μg Hg/L) (Stopford et al. 1978). Metallic mercury accounted for <1% of the total mercury in the urine. The rapid appearance of metallic mercury in the urine is probably due to mercury filtered directly from the blood through the glomerulus, whereas mercuric ions found in the urine are attributable to the mercury taken up by the kidneys prior to excretion. Therefore, urinary metallic mercury provides a relative index for blood levels of metallic mercury, and urinary mercuric ions provide a relative index for kidney levels of inorganic mercury. Three different forms of mercury have been identified in the urine from

workers occupationally exposed to mercury: a metallic form, a mercuric-cysteine complex that is reducible, and a large complex in which the mercury can only be released by organic destruction (Henderson et al. 1974).

Data are limited on elimination of metallic and inorganic mercury in animals. Initial excretion of mercury is predominantly in the fecal matter following inhalation of metallic mercury vapor, but as mercury concentrations increase in the kidneys, urinary excretion increases (Rothstein and Hayes 1964). After inhalation, approximately 10–20% of the total excreted metallic mercury is by exhalation (Rothstein and Hayes 1964). Mercury is excreted in the urine of mice exposed orally to mercuric sulfide (. 8–200 mg Hg/kg) (Yeoh et al. 1986, 1989). The amount of mercury in the urine of the treated group was 4.5–15-fold greater than the control levels. Urinary rates of mercury excretion were 1.6–2.2 ng/hour. Neonatal rats (1, 8, and 15 days old) eliminated mercury slower than older rats (22 and 29 days old) given mercuric chloride subcutaneously (Daston et al. 1986).

Inorganic mercury is also excreted in breast milk (Yoshida et al. 1992). Newborn guinea pigs were exposed to inorganic mercury in breast milk from mothers exposed to mercury vapor (6–9 mg/m^3) for 120 minutes after parturition (Yoshida et al. 1992). Mercury concentrations in breast milk were slightly lower than plasma mercury concentrations of the maternal guinea pigs over the observation period. Ratios of milk to plasma were 0.24–0.44 on day 3, 0.45–0.46 on day 5, and 0.46–0.66 on day 10. The decrease in the mercury concentration in breast milk with time was slower than that in maternal plasma. The distribution of mercury to organs in the suckling neonates indicated that they were exposed to the inorganic rather than to elemental mercury.

Sundberg et al. (1998) studied the elimination of radiolabeled inorganic mercury in lactating and nonlactating mice exposed to mercuric chloride via a single intravenous injection at 0.5 mg Hg/kg body weight. A three-compartment pharmacokinetic model was used to fit the data. The study was designed to provide additional information on the speciation of mercury in breast milk and the differences between methylmercury and inorganic mercury migration into milk. Unlike placenta, where methylmercury moves more easily across the placental border than inorganic mercury, inorganic mercury is more readily eliminated in milk than methylmercury. For inorganic mercury, no significant differences were observed between lactating and nonlactating mice for plasma clearance (43.3 and 44.4 mL/hour/kg, respectively) and volume of distribution (4,950 and 3,780 mL/kg). The terminal half-lives of inorganic mercury in plasma were 297 hours for lactating, and 162 hours for nonlactating mice. The milk-to-plasma concentration ratio

for inorganic mercury varied between 0.1 and 3.6, with a mean of 0.64 at plasma levels below 300 ng Hg/g (in the linear region of the relationship) and a mean of 0.17 at higher plasma mercury levels. In contrast, the values for the methylmercury kinetic parameters were significantly higher in lactating than nonlactating mice: plasma clearance (93.5 and 47.1 mL/hour/kg, respectively) and volume of distribution (18,500 and 9,400 mL/kg, respectively). The terminal half-life of methylmercury in plasma was 170 hours for lactating and 158 hours for nonlactating mice. The milk-to- plasma concentration ratios for total mercury after methylmercury administration were lower than those seen with inorganic mercury, and varied between 0.1 and 0.7 with a mean of 0.20. The nearly five-fold higher peak value for plasma to blood mercury levels observed for inorganic mercury reflects the more efficient migration of inorganic mercury from blood to milk compared with that for methylmercury. Mercury concentrations in milk also decreased more quickly for inorganic (terminal half-life of 107 hours) than for methylmercury (constant levels throughout the 9-day follow-up period postexposure). The authors hypothesize that the nonlinear relationship between mercury in milk and plasma following inorganic mercury administration suggests that inorganic mercury enters the mammary gland via a carrier-mediated transport system that is saturated at high plasma levels of inorganic mercury. The results suggest that the physiological changes during lactation alter the pharmacokinetics for methylmercury in mice, but not for inorganic mercury.

Organic Mercury. The fecal (biliary) pathway is the predominant excretory route for methylmercury, with less than one-third of the total mercury excretion occurring through the urine, following oral and inhalation exposure (Norseth and Clarkson 1970). In humans, nearly all of the total mercury in the feces after organic mercury administration is in the inorganic form. The conversion of methylmercury to inorganic mercury is a major step that is dependent on the duration of exposure and/or the duration after cessation of exposure.

In rats and nonhuman primates, methylmercury is secreted in the bile and can be reabsorbed in the intestine (Berlin et al. 1975; Norseth and Clarkson 1971; Urano et al. 1990). It is believed that methylmercury is complexed to nonprotein sulfhydryl compounds in the bile and reabsorbed in this form by a transport system (Ballatori and Clarkson 1982; Urano et al. 1990). In guinea pigs, hamsters, and monkeys, methylmercury, but not inorganic mercury, is extensively reabsorbed from the gall bladder, providing evidence for the biliary-hepatic recycling of this metal (Dutczak et al. 1991). The biliary-hepatic cycle probably contributes to the long biological half-life and toxicity of methylmercury. However, methylmercury can be converted into its inorganic form in the gastrointestinal lumen by intestinal flora (Nakamura et al. 1977;

Rowland et al. 1980), thus decreasing reabsorption and increasing the rate of fecal excretion (Berlin et al. 1975).

During the first few days after intravenous dosing, phenylmercury compounds are also eliminated primarily in the feces as a result of biliary secretion and its concentration in the gastrointestinal tract (mucosa and lumen) (Berlin and Ullberg 1963). The initial urinary excretion of phenylmercury represents primarily the parent compound (Gage 1964). Several days after exposure, however, elimination is primarily in the urine, which contains predominantly inorganic mercury (Gotelli et al. 1985).

Clearance half-times are longer with methylmercury than with inorganic compounds (see Table 2-5). Elimination of methylmercury compounds generally follows first-order kinetics because excretion is directly proportional to body burden and independent of the route of administration (oral or intraperitoneal) (Nielsen and Andersen 1991a). Furthermore, duration of exposure may affect the excretion process of mercury. A two-compartment model was established by Rice et al. (1989) for a single oral dose study in monkeys because of the appearance of an initial rapid elimination phase followed by a slower elimination phase. However, following repeated dosing for 2 years, a one-compartment model was considered a more reasonable fit for the data. Therefore, it was concluded that the average steady-state blood levels of mercury after chronic-duration exposure should not be estimated on the basis of short-term exposure data.

Elimination rates for methylmercury vary with species, dose, sex, and strain (Nielsen 1992). There is also evidence of sex-related differences in the elimination of methylmercury in humans (Miettinen 1973). The direction of the sex-related difference may differ for the fast and slow components of methylmercury elimination, with males excreting faster during the fast component and females excreting faster during the slow component. The net difference in elimination rates at time points distant from exposure indicates that females excrete methylmercury slightly faster than males. This net difference is seen in whole-body biological half-time derived by combining both fast and slow elimination components (Miettinen 1973). Clear sex-related differences were not reported for these volunteers for time points soon after exposure. In contrast, male mice excreted methylmercury much faster than females did for the first 14 days (i.e., primarily the fast component) (Nielsen 1992). Significant sex-related differences in elimination were also observed in rats dosed at 56 days of age (Thomas et al. 1982). As is apparently the case in humans, the difference was measured in the slow component only, with males excreting slightly slower than females. It should be noted that an insignificant difference in elimination was measured for the fast component of

excretion in the rats, with males excreting slightly faster than females. Interestingly, a sex-difference elimination rate was not observed in rats dosed at 24 days or younger (Thomas et al. 1982).

The rate of mercury excretion was also slower in younger animals (7 or 15 days) than in older animals (24 and 56 days) (Thomas et al. 1982). This age-dependent difference in the rate of mercury excretion may reflect differences in the sites of mercury deposition (i.e., hair, red blood cells, skin). In neonatal rats, the excretion of methylmercury is longer than in adult rats because of the inability of the neonatal liver to secrete the toxicant into the bile. Therefore, the immaturity of the transport system in neonatal rats affects the elimination of mercury.

Methylmercury is also excreted in the breast milk of rats, humans, and guinea pigs (Sundberg and Oskarsson 1992; Yoshida et al. 1992). In pups exposed only through milk, approximately 80% of the total mercury in blood was present as methylmercury. Because suckling animals have a limited ability to demethylate methylmercury, the inorganic mercury present in the blood of the offspring probably originated from inorganic mercury in the milk. Since the dams were exposed only to methylmercury in their diet, some demethylation occurred in the dams, followed by the transport of the inorganic mercury to the sucklings via milk.

Sundberg et al. (1998) studied the elimination of radiolabeled methylmercury in lactating and nonlactating mice exposed to methylmercuric chloride via a single intravenous injection at 0.5 mg Hg/kg body weight. A comparison of the results for methylmercury with results for inorganic mercury is discussed in the section above on elimination of “Inorganic Mercury.” A three compartment pharmacokinetic model was used to fit the data. The values for the methylmercury kinetic parameters were significantly higher in lactating than nonlactating mice: plasma clearance (93.5 and 47.1 mL/hour/kg, respectively) and volume of distribution (18,500 and 9,400 mL/kg, respectively). The terminal half-life of methylmercury in plasma was 170 hours for lactating and 158 hours for nonlactating mice. The milk-to- plasma concentration ratios for total mercury after methylmercury administration were lower than those seen with inorganic mercury, and varied between 0.1 and 0.7, with a mean of 0.20. Mercury concentrations in milk were constant throughout the 9-day follow-up period postexposure. The results indicate that physiological changes during lactation alter the pharmacokinetics for methylmercury in mice.

2.3.5 Physiologically based Pharmacokinetic (PBPK)/Pharmacodynamic (PD) Models

Physiologically based pharmacokinetic (PBPK) models use mathematical descriptions of the uptake and disposition of chemical substances to quantitatively describe the relationships among critical biological processes (Krishnan et al. 1994). PBPK models are also called biologically based tissue dosimetry models. PBPK models are increasingly used in risk assessments, primarily to predict the concentration of potentially toxic moieties of a chemical that will be delivered to any given target tissue following various combinations of route, dose level, and test species (Clewell and Andersen 1985). Physiologically based pharmacodynamic (PBPD) models use mathematical descriptions of the dose-response function to quantitatively describe the relationship between target tissue dose and toxic end points.

PBPK/PD models refine our understanding of complex quantitative dose behaviors by helping to delineate and characterize the relationships between: (1) the external/exposure concentration and target tissue dose of the toxic moiety, and (2) the target tissue dose and observed responses (Andersen et al. 1987; Andersen and Krishnan 1994). These models are biologically and mechanistically based and can be used to extrapolate the pharmacokinetic behavior of chemical substances from high to low dose, from route to route, between species, and between subpopulations within a species. The biological basis of PBPK models results in more meaningful extrapolations than those generated with the more conventional use of uncertainty factors.

The PBPK model for a chemical substance is developed in four interconnected steps: (1) model representation, (2) model parametrization, (3) model simulation, and (4) model validation (Krishnan and Andersen 1994). In the early 1990s, validated PBPK models were developed for a number of toxicologically important chemical substances, both volatile and nonvolatile (Krishnan and Andersen 1994; Leung 1993). PBPK models for a particular substance require estimates of the chemical substance-specific physicochemical parameters, and species-specific physiological and biological parameters. The numerical estimates of these model parameters are incorporated within a set of differential and algebraic equations that describe the pharmacokinetic processes. Solving these differential and algebraic equations provides the predictions of tissue dose. Computers then provide process simulations based on these solutions.

The structure and mathematical expressions used in PBPK models significantly simplify the true complexities of biological systems. If the uptake and disposition of the chemical substance(s) is adequately described, however, this simplification is desirable because data are often unavailable for many biological processes. A simplified scheme reduces the magnitude of cumulative uncertainty. The adequacy of the

model is, therefore, of great importance, and model validation is essential to the use of PBPK models in risk assessment.

PBPK models improve the pharmacokinetic extrapolations used in risk assessments that identify the maximal (i.e., the safe) levels for human exposure to chemical substances (Andersen and Krishnan 1994). PBPK models provide a scientifically sound means to predict the target tissue dose of chemicals in humans who are exposed to environmental levels (for example, levels that might occur at hazardous waste sites) based on the results of studies where doses were higher or were administered in different species. Figure 2-4 shows a conceptualized representation of a PBPK model.

PBPK models for mercury exist, and the overall results and individual models are discussed in this section in terms of their use in risk assessment, tissue dosimetry, and dose, route, and species extrapolations.

2.3.5.1 Summary of PBPK Models

Two physiologically based pharmacokinetic models have been developed recently that model the kinetics of methylmercury in rats. Farris et al. (1993) developed a PBPK model that simulates the long-term disposition of methylmercury and its primary biotransformation product, mercuric mercury, in the male Sprague-Dawley rat following a single oral nontoxic exposure. Gray (1995) developed a PBPK model that simulates the kinetics of methylmercury in the pregnant rat and fetus. The Gray model was developed to provide fetal and maternal organ methylmercury concentration-time profiles for any maternal dosing regimen. These model provide useful insight into the key physiological processes that determine the distribution and fate of mercury in the body, but neither model is currently being used in human risk assessment.

2.3.5.2 Mercury PBPK Model Comparison

Both the Farris et al. (1993) and the Gray (1995) PBPK models address the kinetics of methylmercury in rats. Both models provide useful insights into important physiological processes determining methyl-mercury distribution and changes in tissue concentrations. Also, both studies suggest further work to enhance the utility and accuracy of the models The Farris et al. model dealt more effectively with the conversion of methylmercury to mercuric mercury, while the Gray model specifically addressed fetal tissue concentrations as a function of maternal exposures and the extrapolation from short-term to continuous

dosing. The latter is of direct relevance to methylmercury risk assessments currently based on human studies of short-term exposures, while the general public exposure is more typically continuous. Neither model ran simulations nor validated against data for other species (including human). Nor did the models address high-to-low dose extrapolations or different routes of exposure.

2.3.5.3 Discussion of Models

The Farris et al. Model for Methylmercury. The Farris et al. (1993) model is a physiologically based model that simulates the long-term disposition of methylmercury and its primary biotransformation product, mercuric mercury, in growing mammals following a single nontoxic oral dose of the parent compound. The test animal used to develop and validate the model was the male Sprague-Dawley rat. A tracer dose was used in the validation studies to preclude the possibility that the results would be biased by toxic or saturation effects. The model incorporates a number of features, including a time-dependent compartment for volume changes (i.e., the rats grew from 300 to 500 g in body weight over the 98-day time course of the validation study), compartment volume-dependent clearances, and the recycling of mercury from ingestion of hair by rats during grooming.

Risk assessment. The Farris et al. model has not been used in human risk assessment. The authors, however, suggest that the model would be useful in developing a better understanding of species differences and in predicting the affects of altered biochemical or physiological states on methylmercury pharmaco-kinetics. For example, the authors suggest that the model can be adapted to simulate data for neonatal animals or humans that are known to secrete glutathione poorly. It could also help elucidate the mercury kinetics for animals that have altered bile flow or that have nonabsorbable sulfhydryl-containing resins.

Description of the model. The Farris et al. model consists of nine lumped compartments, each of which represent a major site of mercury accumulation, elimination, or effect in mammals. The compartment labeled “carcass” is a residual compartment and consists of all tissues and organs not specifically represented by the other eight compartments in the model. A flow diagram of the model is shown in Figure 2-5. The interdepartmental mass transport parameters used in the model are shown in Table 2-6.

Methylmercury transport between all compartments except brain and hair is modeled as plasma flow limited (i.e., plasma levels rapidly equilibrate with erythrocytes). Mercuric mercury transport parameters

the carcass, gastrointestinal tissue, skin, and kidneys are assumed to follow a common mechanism and are based on the empirically estimated parameter for the kidneys. Transport of both organic and inorganic mercury to brain and hair compartments is assumed to be limited by the blood-brain barrier and the rate of hair growth. Recycled mercury from ingested hair during grooming was assumed available for reabsorption from the gut lumen at 100% for methylmercury and 10% for inorganic mercury.

The authors make the assumption that all of the inorganic mercury resulting from the demethylation of methylmercury is mercuric mercury. Farris et al. (1993) note that the precise site of demethylation is unknown, although the body's tissues and the lumen of the gastrointestinal tract seem most likely. For convenience, however, they modeled demethylation entirely in the liver compartment. Bidirectional and symmetric transport of methylmercury between the gut tissue and lumen is assumed and modeled accordingly. Biliary secretion of both methylmercury and inorganic mercury are modeled as undergoing low-molecular weight nonprotein sulfhydryl (NPSH) secretion d-dependent transport. Methylmercury secreted into the gut lumen, either from biliary secretion or from the gut tissue, is modeled as being readily reabsorbed. In line with previous studies, the model sets a value of 10% for resorption of inorganic mercury secreted into the lumen from bile or from exfoliation of the gastrointestinal mucosal cells.

The assumptions in the model were incorporated into a series of mass-balance differential equations that account for the changes in the amount of methylmercury and mercuric mercury in each compartment. The entire equation set was solved numerically using Gear's method for stiff differential equations (Gear 1971). The initial mercury dose was administered at 100% methylmercury, administered as a bolus to the gut lumen compartment. The mass transport parameters listed in Table 2-6 were multiplied by the time-dependent compartment volumes to give the mass transport parameters used in the model equations.

Validation of the model. The Farris et al. model simulations were compared to an extensive set of data collected by the authors on the metabolism and distribution of an orally dosed bolus of radiolabeled methylmercury in male Sprague-Dawley rats. In a distribution study, tissue samples were collected on days 3, 7, 14, 21, 28, 35, 42, 49, 56, 63, 70, 77, 84, 91, and 98 post-dosing. In a metabolism study with the same dosing regimen, whole body counts and 24-hour feces and urine samples were collected daily for 15 days post-dosing, and then twice weekly.

The model simulations were in close agreement with the observed results from the distribution and metabolism studies. Physiological processes that were highlighted by the results and the discrepancies that

did occur include the probable active transport into the brain (versus passive diffusion) of a methylmercury-cysteine complex, the bidirectional transport of methylmercury between the gut lumen and gut tissue as a more important determinant of methylmercury fecal excretion than biliary secretion, the importance for the determination of methylmercury half-life in rats of the recycling of mercury from ingested hair, and the need for better estimates of the rate constants for the demethylation of methylmercury in order to adapt the model to other species.

No human data were presented to validate the model, and validation was not performed for other routes or duration of mercury exposure.

Target tissues. The target tissues for this model included the blood, liver, gut, kidneys, and brain.

Species extrapolation. The model was developed and validated using the male Sprague-Dawley rat. No other species were tested and data from other species were not used to validate the model. The authors, however, suggest that this model would prove useful in developing better rate constants or other important determinants of species differences (for example, demethylation rates, which differ based on differences in gut flora and tissue enzyme levels).

High-low dose extrapolations. Only the single nontoxic dose was evaluated. No data were presented to evaluate the utility of the model for high-to-low dose extrapolations.

Interroute extrapolation. Only the single oral dose was evaluated. No data were presented to evaluate the validity of the model in extrapolating from an oral to an inhalation or dermal dose. No compartment was included for the lungs. Although a skin compartment was included in the model, absorption from a dermal application of methylmercury was not addressed.

The Gray Model for Methylmercury.

The Gray (1995) PBPK model simulates the kinetics of methylmercury in the pregnant rat and fetus. The Gray model was developed to provide fetal and maternal organ methylmercury concentration-time profiles for any maternal dosing regimen.

Risk assessment. The Gray model has not been used in human risk assessment. The author, however, suggests that the model would be useful to incorporate rat developmental toxicity data into the assessment of methylmercury risk. Specifically, the author suggests the model be used to convert the short-term exposure data from studies presently being used in risk assessments into continuous-exposure scenarios, which are more typical of the general public's likely exposure pattern.

Description of the model. The Gray model is a membrane-limited PBPK model for methylmercury developed using experimental data from the literature. The model parameters include constants for linear binding, membrane transfer, biliary transport, and gut reabsorption; and physiological parameters for tissue cellular and extracellular volumes and plasma flow rates. Mass balance equations were developed that describe the transport to all organ systems important to the distribution or toxicity of methylmercury to the pregnant rat or fetus. Mass balance equations were solved using an Advanced Continuous Simulation Language (ACSL) program developed by Mitchell and Gauthier Associates.

The compartments and barriers to methylmercury transport in the tissue compartments and placenta are shown in Figure 2-6. The cell membrane is assumed to be the barrier for methylmercury transport for all tissues except the brain and placenta. The barrier to methylmercury transport to the brain is the endothelial cell wall of the cerebral vascular system (the blood-brain barrier). The placenta is modeled as four compartments, with separate transfer constants for placental barrier and placental tissue transport. There is a tissue compartment for both the maternal and fetal sides of the placenta.

The flow chart shown in Figure 2-7 illustrates the transport pathways among the 8 compartments of the pregnant rat, the 5 compartments of the fetus, and the placental interface. The linear binding, membrane transfer transport, and secretion/reabsorption constants used in the Gray model are shown in Tables 2-7 and 2-8. The linear binding constants were estimated directly from *in vivo* tissue distribution studies using the ratio of tissue to plasma concentrations at pseudoequilibrium. They represent the degree to which methylmercury binds to intracellular sites. Because the skin (which includes the outer layers of hair and the pelt) contained excreted methylmercury that does not exchange with plasma, the linear binding constant for a typical organ (in this case the liver) was used as the constant for skin. No experimental data were available for fetal red blood cell (RBC) binding, so the author made the assumption that the fetal RBC binding constant would be equal to the maternal RBC binding constant. The conversion of methylmercury into mercuric mercury in the gut is not explicitly calculated in the Gray model; instead, the calculated

reabsorption rate of secreted or shed methylmercury in the gut implicitly accounts for the amount converted (i.e., the amount of demethylated mercury that subsequently would not be reabsorbed).

Published data were used directly or to estimate values for the maternal and fetal extracellular space, maternal plasma volume and flow expansion during pregnancy, and maternal and fetal organ volumes and plasma flow.

The model was run with a single intravenous bolus dose of 1 mg/kg at various times during a 22-day rat gestation period and compared with previously published (different author) maternal and fetal organ concentrations. The model was also run with a daily dosing for 98 days, ending on Gd 20, to simulate a typical human dietary exposure pattern for a frequent consumer of methylmercury-contaminated food.

Validation of the model. The Gray model simulations were validated against published values in the literature for mercury concentrations in maternal and fetal rat tissue from a variety of dosing patterns over the 22-day rat gestation period. Model-derived estimates of methylmercury half-life in red blood cells of 14.8 days for the rat were consistent with published values from 14 to 16 days. Consistent values were also obtained for the timing of the peak mercury concentration in the brain. Model estimates were in agreement with published values for most tissue mercury concentrations for dosing at various times, with percent differences generally <25%. Model estimates of maternal kidney methylmercury concentrations were consistently below published values, possibly due to an underestimate of the inorganic fraction of mercuric mercury in the kidneys.

The model results for a total fetal methylmercury concentration of 0.79% 24 hours after maternal methyl-mercury dosing on Gd 19 compare favorably with published values of 0.6 and 0.88% for administered doses on Gd 19 and 20, respectively.

No human data were presented to validate the model, and validation was not performed for other routes of mercury exposure.

Target tissues. The target tissues for this model included the blood, liver, gut, kidneys, and brain.

Species extrapolation. The model validated the use of published data for the rat. No other species were tested, and data from other species were not used to validate the model. The author, however,

suggests that generally good agreement between the model simulated results and the published values indicate that the model accurately reflects the underlying biological processes and that scaling factors for species-to-species extrapolations should be considered.

High-low dose extrapolations. No data were presented to evaluate the utility of the model for high-to-low dose extrapolations. A continuous exposure was simulated, but it was not validated against published data.

Interroute extrapolation. Only the intravenous route of exposure was evaluated. No data were presented to evaluate the validity of the model in extrapolating to an oral, inhalation, or dermal route of exposure. No compartment was included in the model for the lungs. Although a skin compartment was included in the model, absorption from a dermal application of methylmercury was not addressed.

2.4 MECHANISMS OF ACTION

2.4.1 Pharmacokinetic Mechanisms

The absorption of metallic mercury through the lungs is by rapid diffusion. It is suggested that oral absorption of inorganic mercury compounds depends on their dissociation in the intestinal tract. In several cases, the underlying mechanism for the toxic effects of mercury has been attributed to the high affinity of mercury for protein-containing sulfhydryl or thiol groups.

The mechanism of absorption for metallic mercury vapors is rapid diffusion across alveolar membranes (Berlin et al. 1969; Clarkson 1989). Mercury distribution in the brains of mercury-sensitive SJL/N mice exposed for 10 weeks (5 days per week) to relatively high concentrations (0.5–1.0 mg/m^3) of mercury vapor was found to be affected by the magnitude of exposure (Warfvinge 1995). In animals exposed to 0.5 mg/m^3 for 19 hours a day or 1 mg/m^3 for 3 hours a day, mercury was found in almost the entire brain, whereas in those exposed to 0.3 mg/m^3 for 6 hours a day, mercury was primarily found in the neocortical layer V, the white matter, the thalamus, and the brain stem. In mice exposed to 1 mg/m^3 for just 1.5 hours a day, the white matter and brain stem were the targets for mercury accumulation. These findings in mice were generally in agreement with brain distribution patterns observed in mercury-sensitive rats (Schionning et al. 1991; Warfvinge et al. 1992), except that the white matter was not found to be a target for mercury accumulation.

Oral absorption of metallic mercury is low, possibly because of an *in vivo* conversion to divalent mercury and subsequent binding to sulfhydral groups, or possibly because of poor absorption of the elemental form. For inorganic mercuric compounds, the low absorption in the lungs is probably due to the deposition of particles in the upper respiratory system that should be cleared rapidly (Friberg and Nordberg 1973). Solubility and other chemical properties may also be factors in the absorption. The mechanism for intestinal absorption of inorganic mercuric mercury may also involve the process of diffusion, and the absorption rate is proportional to the concentration of mercury in the lumen of the intestines (Piotrowski et al. 1992). The extent of transport of inorganic mercury across the intestinal tract may depend on its solubility (Friberg and Nordberg 1973) or on how easily the compounds dissociate in the lumen (Endo et al. 1990). Absorption of mercurous compounds is less likely, probably because of solubility (Friberg and Nordberg 1973) or its conversion into the divalent cation in the gastrointestinal tract.

The divalent cation exists in both a nondiffusible form (tissues) and a diffusible form (blood) (Halbach and Clarkson 1978; Magos 1967) (see Section 2.3.2). The mechanism for the distribution of mercury and its compounds probably depends on the extent of uptake of the diffusible forms into different tissues or on the mercury-binding to protein-binding sites (sulfhydryl groups) in red cells and plasma proteins (Clarkson 1972b).

Mechanisms for the toxic effects of inorganic and organic mercury are believed to be similar. It has been suggested that the relative toxicities of the different forms of mercury (e.g., metallic, monovalent, and divalent cations and methyl- and phenylmercury compounds) are related, in part, to its differential accumulation in sensitive tissues. This theory is supported by the observation that mercury rapidly accumulates in the kidneys and specific areas of the central nervous system (Rothstein and Hayes 1960; Somjen et al. 1973).

The accumulation of methylmercury and inorganic mercury in the brain of female monkeys *(Macaca fascicularis)* was studied by Vahter et al. (1994). In this study, animals received oral doses of 50 µg/kg/day for either 6, 12, or 18 months. In normal-weight monkeys (2.4–4.1 kg), a steady-state blood concentration for total mercury was attained in approximately 4 months. The elimination half-life in the blood was found to be 26 days. Accumulation in the brain appeared to be biphasic, with an elimination half-life of 35 days for brain methylmercury in those monkeys exposed for 12 months. The elimination half-life of inorganic mercury, on the other hand, was reported be on the order of years. It was also found that inorganic mercury accounted for approximately 9% of the total brain mercury at 6–12 months, 18% at

18 months, and 74% 6 months after termination of exposure. The authors stated that the presence of inorganic mercury in the brain was thought to be the result of demethylation of methylmercury in the brain. In heavier monkeys, there was a limited distribution of mercury in the fat. A finding of higher brain concentrations in the heavy monkeys than in those of normal weight was probably due to higher blood mercury levels and a higher brain-to-blood distribution ratio. *In vivo* methylation of inorganic mercury, on the other hand, was not shown to occur in occupationally exposed workers (Barregard et al. 1994a, 1994b), contrary to the findings of previous *in vitro* studies.

Distribution of organic mercury is believed to involve complexes with proteins in the body. Methylmercury associates with water-soluble molecules (e.g., proteins) or thiol-containing amino acids because of the high affinity of the methylmercuric cation (CH_3Hg^+) for the sulfhydryl groups (SH-) (Aschner and Aschner 1990). Complexes of methylmercury with cysteine or glutathione have been identified in blood, liver, and bile (Aschner and Aschner 1990). The transport of methylmercury to the brain after subcutaneous injection appears to be closely linked to thiol-containing amino acids (Aschner and Clarkson 1988). The methylmercury cation can bind to the thiol group of the amino acid cysteine, forming a complex in which the valence bonds link the mercury atom to adjacent iron and sulfur atoms at an 180E angle, creating a chemical structure similar to that of the essential amino acid methionine (Clarkson 1995). In such a manner, methylmercury can cross the blood-brain barrier "disguised" as an amino acid via a carrier-mediated system (i.e., transport is not solely the result of methylmercury's lipid solubility). The uptake of methylmercury by the brain is inhibited by the presence of other amino acids such as leucine, methionine, phenylalanine, and other large neutral amino acids (Clarkson 1995).

The mechanism by which methylmercury crosses the blood-brain barrier has also been examined in the rat using a rapid carotid infusion technique (Kerper et al. 1992). The results of this study also showed that methylmercury may enter the brain as a cysteine complex. The uptake of Me_20_3Hg complexed with either L- or D-cysteine was measured as a function of Me_20_3Hg-cysteine concentration in the injection solution. There was a faster rate of uptake of Me_20_3Hg-L-cysteine as compared to the D-cysteine complex. The nonlinearity of Me_20_3Hg-L-cysteine uptake with the increasing concentration suggests that transport of this complex is saturable, while the D-cysteine complex is taken up by simple diffusion. The mechanism for the distribution in the brain of inorganic mercury (resulting from the demethylation of organic mercury) is not well understood.

Strain and sex differences in renal mercury content in mice are attributable, in part, to differences in tissue glutathione content and to differences in renal γ-glutamyltranspeptidase activity, which is controlled, at least in part, by testosterone (Tanaka et al. 1991, 1992). The correlation of hepatic glutathione (or plasma glutathione) with the rate of renal uptake of methylmercury suggests that methylmercury is transported to the kidneys as a glutathione complex (Tanaka et al. 1991). In addition to strain and sex differences in renal mercury content, it has also been demonstrated using mice (133–904 days old) that the ratio of mercury in the brain to that in the liver and the kidneys increased significantly with age (Massie et al. 1993).

In a study of the absorption of inorganic mercury by the rat jejunum, Foulkes and Bergman (1993) found that while tissue mercury could not be rigorously separated into membrane-bound and intracellular compartments (as can the heavy metal cadmium), its uptake into the jejunum includes a relatively temperature-insensitive and rapid influx into a pool readily accessible to suitable extracellular chelators. A separate, slower and more temperature-sensitive component, however, leads to the filling of a relatively chelation-resistant compartment. Nonspecific membrane properties, such as surface charge or membrane fluidity, might account for mucosal mercury uptake (Foulkes and Bergman 1993).

2.4.2 Mechanisms of Toxicity

High-affinity binding of the divalent mercuric ion to thiol or sulfhydryl groups of proteins is believed to be a major mechanism for the biological activity of mercury (Clarkson 1972a; Hughes 1957; Passow et al. 1961). Because proteins containing sulfhydryl groups occur in both extracellular and intracellular membranes and organelles, and because most sulfhydryl groups play an integral part in the structure or function of most proteins, the precise target(s) for mercury is not easily determined, if indeed there is a specific target. Possibilities include the inactivation of various enzymes, structural proteins, or transport processes (Bulger 1986); or alteration of cell membrane permeability by the formation of mercaptides (Sahaphong and Trump 1971). Binding may also occur to other sites (e.g., amine, carboxyl groups) that are less favored than sulfhydryl groups. A variety of mercury-induced alterations are being investigated, including increased oxidative stress, disruption of microtubule formation, increased permeability of the blood-brain barrier, disruption of protein synthesis, disruption of DNA replication and DNA polymerase activity, impairment of synaptic transmission, membrane disruption, impairment of the immune response, and disruption in calcium homeostasis. These alterations may be acting singly or in combination.

Mercury has been shown to affect hepatic microsomal enzyme activity (Alexidis et al. 1994). Intra-peritoneal administration of mercuric acetate (6.2 µmol/kg/day) once daily for 6 days or once as a single dose of 15.68 µmol/kg resulted in an increase in kidney weight and a significant decrease in total cytochrome P-450 content. The single 15.68 µmol/kg injection resulted in the reduction of both microsomal protein level and P-450 content, possibly resulting from the generation of free radicals during the Hg^{++} intoxication process.

Through alterations in intracellular thiol status, mercury can promote oxidative stress, lipid peroxidation, mitochondrial dysfunction, and changes in heme metabolism (Zalups and Lash 1994). $HgCl_2$ has been shown to cause depolarization of the mitochondrial inner membrane, with a consequent increase in the formation of H_2O_2 (Lund et al. 1993). These events are coupled with a Hg^{++}-mediated glutathione depletion and pyridine nucleotide oxidation, creating an oxidant stress condition characterized by increased susceptibility of the mitochondrial membrane to iron-dependent lipid peroxidation. Lund et al. (1993) further postulated that mercury-induced alterations in mitochondrial calcium homeostasis may exacerbate Hg^{++}-induced oxidative stress in kidney cells. As a result of oxidative damage to the kidneys, numerous biochemical changes may occur, including the excretion of excess porphyrins in the urine (porphyrinuria). In a study of the mechanism of porphyrinogen oxidation by mercuric chloride, Miller and Woods (1993) found that mercury-thiol complexes possess redox activity, which promotes the oxidation of porphyrinogen and possibly other biomolecules.

The steps between thiol binding and cellular dysfunction or damage have not been completely elucidated, but several theories exist. Conner and Fowler (1993) have suggested that following entry of the mercuric or methylmercuric ion into the proximal tubular epithelial cell by transport across either the brush-border or basolateral membrane, mercury interacts with thiol-containing compounds, principally glutathione and metallothionein. This interaction initially produces alterations in membrane permeability to calcium ions and inhibition of mitochondrial function. Through unknown signaling mechanisms, mercury subsequently induces the synthesis of glutathione, various glutathione-dependent enzymes, metallothionein, and several stress proteins (Conner and Fowler 1993). In the kidneys, epithelial cell damage is believed to occur as the result of enhanced free radical formation and lipid peroxidation (Gstraunthaler et al. 1983). Treatment with mercury results in depletion of cellular defense mechanisms against oxidative damage such as glutathione, superoxide dismutase, catalase, and glutathione peroxidase (Gstraunthaler et al. 1983). Further, enhancement of glutathione peroxidase has been observed in mercury-treated rats in direct relationship with kidney mercury content (Guillermina and Elias 1995), but inhibition of renal redox cycle

enzymes *in vivo* did not appear to be a significant determinant of the increased lipid peroxidation observed during $HgCl_2$-induced nephrotoxicity. The selenium-dependent form of glutathione peroxidase is highly sensitive to inhibition by mercury, and it has been proposed that mercury interactions with selenium in the epithelial cells limit the amount of selenium available for this enzyme (Nielsen et al. 1991). Depletion of mitochondrial glutathione and increases in mitochondrial hydrogen peroxide at the inner mitochondrial membrane (Lund et al. 1991) may contribute to acceleration of the turnover of potassium and magnesium observed at this membrane (Humes and Weinberg 1983). Acute renal failure resulting from mercury exposure has been proposed to result from decreased renal reabsorption of sodium and chloride in the proximal tubules and increased concentrations of these ions at the macula densa (Barnes et al. 1980). This increase in ions at the macula densa, in turn, results in the local release of renin, vasoconstriction of the afferent arteriole, and filtration failure. These authors based this hypothesis on the observation that saline pretreatment of rats prior to mercuric chloride treatment did not prevent the proximal tubular damage but did prevent the acute renal failure. The saline pretreatment was suggested to have depleted the glomerular renin and thereby prevented the cascade of events occurring after accumulation of sodium and chloride ions at the glomerular macula densa (Barnes et al. 1980). A pivotal role for extracellular glutathione and membrane-bound γ-glutamyltransferase has also been identified in the renal incorporation, toxicity, and excretion of inorganic mercury ($HgCl_2$) in rats (Ceaurriz et al. 1994).

A similar mechanism for the promotion of neuronal degeneration by mercury has been proposed (Sarafian and Verity 1991). Increases in the formation of reactive oxygen species in several brain areas have been observed following intraperitoneal administration of methylmercuric chloride to rodents (Ali et al. 1992; LeBel et al. 1990, 1992). A dissociation between increases in lipid peroxidation and cytotoxicity has been demonstrated by showing inhibition of the lipid peroxidation with α-tocopherol without blocking the cytotoxicity (Verity and Sarafian 1991). These authors were able to show partial protection against the cytotoxicity with ethylene glycol tetra-acetate (EGTA), suggesting that increases in intracellular calcium may play a role in the cytotoxicity. They ultimately concluded that a synergistic interaction occurred between changes in intracellular calcium homeostasis and intracellular thiol status, culminating in lipoperoxidation, activation of Ca2+-dependent proteolysis, endonuclease activation, and phospholipid hydrolysis (Verity and Sarafian 1991). It has been suggested that neurons are highly sensitive to mercury either because of their low endogenous glutathione content or their inefficient glutathione redox activity. Inhibition of protein synthesis has been reported in neurons from rats exposed to methylmercury (Syversen 1977). However, it is unknown whether this inhibition is secondary to neuronal cytotoxicity.

At the functional level, both mercuric chloride and methylmercury have been shown to induce a slow inward current in patch-clamped dorsal root ganglion cells (Arakawa et al. 1991). The current does not appear to be mediated by either the sodium or calcium channels, but it may be activated by increases in intracellular calcium. Such slow inward currents suppress voltage- and neurotransmitter-activated currents. Studies of the effects of inorganic mercury, methylmercury, and phenylmercuric acetate on synaptic transmission in rat hippocampal slices (Yuan and Atchison 1994) revealed that the mechanisms that underlie the effects of various mercurials on central synaptic transmission differ with respect to the sites of action, the potency, and the reversibility of the effect. Inorganic mercury (Hg^{++}) appeared to act primarily on the postsynaptic neuronal membrane, whereas the action of methylmercury and phenylmercuric acetate was at both the pre- and postsynaptic sites but primarily on the postsynaptic membranes. Yuan and Atchison (1994) suggested that these differences may result, in part, from the differences in lipophilicity among the different mercurials studied. Differences in lipophilicity were also implicated by Roed and Herlofson (1994) as playing a role in the different effects produced by methylmercuric chloride and mercuric chloride. Roed and Herlofson (1994) suggested that the high lipid solubility of methylmercuric chloride may divert that organomercurial to the myelin of the nerve, where it very efficiently inhibits neuronal excitability. Further, they suggested that mercuric chloride probably causes inhibitory activity by binding to sulfhydryl groups in transport proteins that convey the messenger function of intracellular Ca^{++}. This, in turn, leads to both inhibition of muscle contraction and enhancement of $HgCl_2$-induced neuronal inhibition. The authors further suggest that $HgCl_2$ inhibits an internal Ca^{++} signal necessary for choline re-uptake and acetylcholine resynthesis.

Gallagher and Lee (1980) evaluated the similarity of inorganic and organic mercury toxicity to nervous tissue by injecting equimolar concentrations of both mercuric chloride and methylmercuric acetate directly into the cerebrum of rats, thereby circumventing systemic metabolic conversion pathways. The lesions induced by mercuric chloride were expected to have been much greater after the mercuric chloride injection, since this process circumvents the necessity for biotransformation. However, the lesions were only slightly larger than those seen after methylmercury injection, suggesting that there is a mechanism for organic mercury neurotoxicity that does not involve conversion into inorganic mercury. This suggestion is supported by the findings of Magos et al. (1985) who failed to establish a correlation between neuronal, cytoplasmic, mercuric ions and neuronal degeneration, or clinical evidence of neurotoxicity. These results do not, however, preclude the possibility that intracellular transport of mercuric mercury may be limited, and the limitations on transport may determine the effects observed.

Recent data from an *in vitro* study suggest that mercuric mercury may be more effective than methylmercury in some paradigms. Using patch-clamped dorsal root neurons, Arakawa et al. (1991) showed augmentation of the GABA-activated chloride current at extremely low mercuric chloride concentrations (0.1 μM), while a 1,000-fold higher concentration of methylmercury showed no such effect. The correlation between these effects observed *in vitro* and what may be occurring *in vivo*, however, is not known.

The experimental data concerning the mechanism of action of methylmercury on the developing nervous system indicate that effects on the microtubules and amino acid transport are disrupted in neuronal cells before overt signs of intoxication are observed. Vogel et al. (1985) demonstrated the potent inhibitory effects of methylmercury on microtubule assembly at ratios stoichiometric with the tubulin dimer. The effects were thought to be mediated through MeHg binding to free sulfhydryl groups on both ends and on the surface of microtubules, which would provide multiple classes of binding sites for MeHg. In subsequent *in vitro* studies, Vogel et al. (1989) identified a single high affinity class of binding sites on tubulin for methylmercury with 15 sites. The authors report that MeHg binds to tubulin stoichiometrically within microtubules, and does not induce microtubule disassembly at this low binding ratio. Free subunits of tubulin, however, will act as uncompetitive inhibitors for MeHg binding to the polymer, and MeHg binding to the multiple sites in the free dimer blocks subsequent assembly. In contrast, the stoichiometric polymer surface binding sites for MeHg in microtubules apparently do not interfere with subsequent polymerization. Mitotic inhibition from damage to microtubulin and binding to tubulin has also been reported by Sager et al. (1983).

Comparison of the effects of mercury on structural elements and enzyme activities (Vignani et al. 1992) suggests that effects on cytoskeletal elements may be observed at lower concentrations than on enzyme activities. In the *in vivo* study by Sager et al. (1982), it was concluded that methylmercury may be acting on mitotic spindle microtubules leading to cell injury in the developing cerebellar cortex. Cell injury observed in the external granular layer of the cerebellar cortex of 2-day-old rats was attributed to a reduced percentage of late mitotic figures (arrested cell division) due to the loss of spindle microtubules. Mitosis and migration of granule cells in the cerebellum end within weeks following birth; therefore, this observation may suggest potential differences in the sensitivities of children and adults to mercury-induced neurotoxicity. The toxic effects of methylmercury on the developing nervous system may also be due to deranged neuronal cell migration (Choi et al. 1978; Matsumoto et al. 1965). Examination of the brains of two infants who died following *in utero* exposure to methylmercury revealed an abnormal pattern in the

organization and a distorted alignment of neurons in the cerebral cortex. Exposures first occurred during the critical period of neuronal migration (from gestation week 7 into the third trimester) in the fetus. Both could result from a direct effect of mercury on microtubule proteins. Cell division and cell migration both require intact microtubules for normal functioning and, therefore, have been suggested as primary targets for methylmercury disruption in the developing nervous system. It is hypothesized by Aschner and Clarkson (1988) that the uptake of methylmercury through the blood-brain barrier in developing and mature animals is closely linked to amino acid transport and metabolism because of the infusion of L-cysteine enhanced ^{203}Hg uptake. The enhanced transport in the fetus may be a result of the immaturity of the transport systems in the blood-brain barrier or of possible physical immaturity of the barrier itself. Methylmercury has also been shown to increase intracellular Ca^{++} and inositol phosphate levels (Sarafian 1993). The observed stimulation of protein phosphorylation in rat cerebral neuronal culture was believed to be the result of elevation of intracellular second messengers (Ca^{++}, inositol phosphate) rather than to a direct interaction between methylmercury and protein kinase enzymes. This observation was considered to suggest a specific interference with neuronal signal transduction.

The mercuric ion is also an extremely potent inhibitor of microtubule polymerization, both *in vivo* and *in vitro* (Duhr et al. 1993). Duhr and his colleagues further reported that the ability of Hg^{++} to inhibit microtubule polymerization or to disrupt already formed microtubules not only cannot be prevented by binding with the chelating agents EDTA and EGTA, but that the binding of these two potent chelators potentiates the Hg^{++}-induced inhibition of tubulin polymerization by disrupting the interaction of GTP with the E-site of brain beta-tubulin, an obligatory step in the polymerization of tubulin.

Mercury has been shown to inhibit a variety of enzymes in the nervous system. The effects of mercuric chloride and methylmercuric chloride on the activity of protein kinase C in rat brain homogenate were studied by Rajanna et al. (1995). In this study, it was found that both forms of mercury inhibited protein kinase C activity in a dose-dependent manner at micromolar concentrations, with methylmercury being a more potent inhibitor than $HgCl_2$. Mercuric chloride has also been shown to cause the inhibition and ultrastructural localization of cerebral alkaline phosphatase (Albrecht et al. 1994) following a single intraperitoneal injection of 6 mg $HgCl_2$/kg body weight. The observed inhibition and subsequent translocation of alkaline phosphatase activity from the luminal to abluminal site and the accompanying ultrastructural alterations were reported to be typical of the formation of "leaky" microvessels known to be associated with damage to the blood-brain barrier. Mercuric chloride has also been demonstrated to block the uptake of [3H]-histamine by cultured rat astroglial cells and brain endothelial cells (Huszti and Balogh

1995). This effect was seen at mercury concentrations as low as 1 μM, and the inhibition was greater in astroglial cells than in the cerebral endothelial cells. At a concentration of 100 μM, however, $HgCl_2$ caused the stimulation of histamine uptake, which was greater in the cerebral endothelial than in the astroglial cells. The mechanisms of these dose-dependent effects were considered to be different, with the inhibition of histamine uptake associated with the loss of the transmembrane Na+ and/or K+ gradient and the stimulation of histamine uptake by the higher mercury concentration being possibly related to a direct effect on the histamine transporter.

Sekowski et al. (1997) used an intact human cell multiprotein complex (which they call a DNA synthesome) to evaluate the effects of mercuric chloride on DNA synthesome-mediated *in vitro* DNA replication and DNA synthesis. The authors state that the DNA synthesome has the advantage of providing the highly ordered environment in which DNA replication occurs while allowing more precise identification of the mechanism or site of effects than possible from the use of whole cells. The results showed that DNA replication and DNA polymerase activity, as well as DNA replication fidelity of the human cell synthesome, were specifically inhibited by mercuric ion at physiologically attainable concentrations. The results suggest that mercuric ions (at concentrations above 10 μM) actively inhibit the elongation stage of DNA replication.

It has been shown that Hg^{++} promotes dose-dependent toxic effects on heart muscle through actions on the sarcolemma, the sarcoplasmic reticulum, and contractile proteins (Oliveira et al. 1994). In this study, inorganic mercury ($HgCl_2$) was shown to have a dose-dependent effect on rat papillary muscle, with a concentration of 1 μM causing a small increase in the force of isometric contraction. Concentrations of 2.5, 5, and 10 μM produced a dose-dependent decrease in contractile force. The rate of force development, however, was effected differently, increasing at 1 and 2.5 μM Hg^{++} but decreasing to control levels at 5 and 10 μM concentrations. Oliveira et al. (1994) suggested that this response was due to an observed progressive reduction in the time to peak tension with increasing mercury concentrations, an effect they attributed to the binding of mercuric ions to SH groups inducing Ca^{++} release from the sarcoplasmic reticulum, the activity of which itself was depressed by mercury in a dose-dependent fashion. Further, tetanic tension did not change during treatment with 1 μM Hg^{++} but decreased with 5 μM, suggesting a toxic effect on the contractile proteins only at high Hg^{++} concentrations (Oliveira et al. 1994).

The molecular events leading to activation of the autoimmune response in susceptible individuals have yet to be fully elucidated. However, chemical modification of major histocompatibility complex (MHC) class

II molecules or modification of self peptides, T-cell receptors, or cell-surface adhesion molecules has been suggested (Mathieson 1992). The immune suppressive effect of mercury has been examined in human B-cells (Shenker et al. 1993). This study showed inhibition of B-cell proliferation, expression of surface antigens, and synthesis of IgG and IgM by both methylmercury and mercuric mercury. These chemicals caused a sustained elevation of intracellular calcium. Based on concurrent degenerative changes in the nucleus (hyperchromaticity, nuclear fragmentation, and condensation of nucleoplasm) in the presence of sustained membrane integrity, the author suggested that the increase in intracellular calcium was initiating apoptic changes in the B-cells, ultimately resulting in decreased viability.

The glomerulopathy produced by exposure of Brown-Norway rats to mercuric chloride has been related to the presence of antilaminin antibodies (Icard et al. 1993). Kosuda et al. (1993) suggest that both genetic background and immune regulatory networks (possibly acting through T-lymphocytes of the RT6 subset) may play an important role in the expression of autoimmunity after exposure to mercury. A strain (Brown-Norway) of rats known to be susceptible to mercury-induced production of autoantibodies to certain renal antigens (e.g., laminin) and autoimmune glomerulonephritis was compared to a nonsusceptible strain (Lewis). Different responses to subcutaneous injections of mercuric chloride regarding $RT6^+$ T-lymphocytes (a subpopulation of lymphocytes considered to have possible immunoregulatory properties) were observed. While a relative decrease in $RT6^+$ T-cells occurring with the development of renal autoantigen autoimmune responses was observed in the mercury-treated Brown-Norway rats, the Lewis rats did not develop renal autoimmunity and were found to have undergone significant change in the $RT6^+$-to-$RT6^+$ T-lymphocyte ratio. When Brown-Norway-Lewis F_1 hybrid rats were similarly dosed, effects similar to those in the Brown-Norway strain were seen, with the autoimmune responses to kidney antigens occurring concomitantly with a change in RT6 population proportionally in favor of T-lymphocytes that do not express the RT6 phenotype. Kosuda et al. (1993) proposed that there are both endogenous and exogenous components of mercury-induced autoimmunity. The endogenous (a genetically determined) component includes T-cell receptors, the major histocompatibility complex, and an immuno-regulatory network based upon a rather delicate balance between helper and suppressor (e.g., the $RT6^+$ T-lymphocytes) cells; whereas the exogenous component is represented by an environmental factor (e.g., mercury) capable of altering the balance within the immunoregulatory network. The manifestation of autoimmunity requires the presence and interaction of both of these components. In a similar study, Castedo et al. (1993) found that mercuric chloride induced $CD4^+$ autoreactive T-cells proliferate in the presence of class II^+ cells in susceptible Brown-Norway rats as well as in resistant Lewis rats. However, while those cells were believed to collaborate with B-cells in Brown-Norway rats to produce

autoantibodies, in Lewis rats they apparently initiate a suppressor circuit involving antiergotypic $CD8^+$ suppressor cells.

In Brown-Norway rats given 5 subcutaneous 1 mg/kg injections of mercuric chloride over a 10-day period, tissue injury (including vasculitis) was seen within 24 hours of the first injection (Qasim et al. 1995). The rapid onset of tissue injury suggests that cells other than T-cells may be involved in the primary induction of vasculitis typically seen as a response to mercuric chloride in this species. It is possible that this injury occurs through a direct action of $HgCl_2$ on neutrophils or through activation of mast cells, resulting in the release of TNF and IL8, which promote chemotaxis and activation of neutrophils. However, the changes in the Th2-like (CD4+CD45) T-cell subsets seen in this study were considered to provide support for the hypothesis that a rise in T helper cells drives the observed autoimmune syndrome, providing B-cell help, which leads to polyclonal activation and production of a range of antibodies.

Jiang and Moller (1995) found that mercuric chloride induced increased DNA synthesis *in vitro* (peak activity between days 4 and 6) in lymphocytes from several mouse strains and suggested a crucial role for helper T-cells in $HgCl_2$-induced immunotoxicity. The results of this study indicated that: (1) mercuric chloride activated CD4+ and CD8+ T-cells *(in vitro)* in a manner analogous to a specific antigen-driven response; (2) activation was dependent upon the presence of accessory cells; and (3) helper T-cells were induced to divide and transform in responder organ cells. This led Jiang and Moller (1995) to hypothesize that mercury binds to molecules on the antigen-presenting cell (APC) and transforms molecules on these cells to superantigens capable of activating T-cells with a particular set of antigen-binding receptors. In this manner, mercury could induce an internal activation of the immune system, which would in turn result in a variety of symptoms in predisposed individuals.

Both mercuric chloride (1 μM) and methylmercury (2 μM) have been shown to increase intracellular Ca^{++} concentrations in splenic lymphocytes in a concentration-dependent manner (Tang et al. 1993). The time course for the effect was, however, different for the two mercurials. In the case of methylmercury, the increase in intracellular Ca^{++} was rapid and the increased level was sustained over time, whereas the Ca^{++} rise caused by $HgCl_2$ was slower. While the effects of those mercurials did not appear to be associated with alterations of membrane integrity, both $HgCl_2$ and methylmercury did appear to cause membrane damage when the incubation time was extended. This study also found that methylmercury and mercuric chloride appear to exert their effects on internal lymphocyte Ca^{++} levels in different ways. Methylmercury increases intracellular Ca^{++} by both an apparent increase in the permeability of the membrane to

extracellular Ca^{++} and the mobilization of Ca^{++} from intracellular stores (perhaps the endoplasmic reticulum and mitochondria), whereas $HgCl_2$ causes only an increased influx of extracellular Ca^{++}.

2.4.3 Animal-to-Human Extrapolations

Mechanisms for the end toxic effects of inorganic and organic mercury are believed to be similar, and the differences in parent compound toxicity result from difference in the kinetics and metabolism of the parent compound. Animal models generally reflect the toxic events observed in humans (i.e., neurological for methylmercury toxicity and the kidneys for inorganic mercury); however, there are species and strain differences in response to mercury exposure. Prenatal exposures in animals result in neurological damage to the more sensitive developing fetus as is the case in humans. The observed inter- and intraspecies differences in the type and severity of the toxic response to mercury may result from differences in the absorption, distribution, transformation, and end tissue concentration of the parent mercury compound. For example, C57BL/6, B10.D2, B10.S inbred mice accumulated higher concentrations of mercury in the spleen than A.SW, and DBA/2 strains, subjected to the same dosage regimen. The higher concentration of splenic mercury in C57BL/6, B10.D2, B10.S correlated with the increased susceptibility of these strains to a mercury chloride-induced systemic autoimmune syndrome. The lower splenic mercury in A.SW, and DBA/2 strains resulted in more resistance to an autoimmune response (Griem et al. 1997).

A better understanding of certain physiological and biochemical processes affecting mercury kinetics may help explain these species differences. Specific processes that appear likely determinants include differences in demethylation rates affecting methylmercury fecal secretion, reabsorption, and membrane transport (Farris et al. 1993); differences in tissue glutathione content and renal γ-glutamyltranspeptidase activity (Tanaka et al. 1991, 1992), differences in antioxidative status (Miller and Woods 1993), differences in plasma cysteine concentrations compared with other thiol-containing amino acids (Aschner and Clarkson 1988; Clarkson 1995), and differences in factors that could affect gut lumenal uptake (Foulkes and Bergman 1993; Urano et al. 1990). Better controls and reporting of dietary factors, volume and timing of doses, and housing conditions would assist in the comparisons of effects among species and strains.

Further development of PBPK/PBPD models will assist in addressing these differences and in extrapolating animal data to support risk assessments for mercury exposure in humans.

2.5 RELEVANCE TO PUBLIC HEALTH

OVERVIEW

The nature and severity of the toxicity that may result from mercury exposure are functions of the magnitude and duration of exposure, the route of exposure, and the form of the mercury or mercury compound to which exposure occurs. Since the ultimate toxic species for all mercury compounds is thought to be the mercuric ion, the kinetics of the parent compound are the primary determinant of the severity of parent compound toxicity. It is differences in the delivery to target sites that result in the spectrum of effects. Thus, mercury, in both inorganic and organic forms, can be toxic to humans and other animals.

Ingestion of methylmercuric chloride, for example, is more harmful than ingestion of an equal amount of inorganic salts (e.g., mercuric chloride or mercuric acetate), since methylmercury is more readily absorbed through the intestinal tract (about 95%) than are mercuric salts (about 10–30%). In turn, ingestion of inorganic mercury salts is more harmful than ingestion of an equal amount of liquid metallic mercury, because of negligible absorption of liquid metallic mercury (about 0.01%) from the gastrointestinal tract. There is insufficient information to develop a complete matrix of effects for different mercury forms by route of exposure. The information on inhalation exposure to mercury is limited primarily to metallic mercury; only a few case studies are available for exposure to inorganic dusts or volatile organomercurials.

Inorganic salts of mercury do not readily cross the blood-brain barrier or the placenta. They are, therefore, ultimately less toxic to the central nervous system and the developing fetus than either absorbed metallic mercury or organic mercury compounds. Metallic mercury is more readily oxidized to mercuric mercury than is methylmercury, so its transport across the placenta and into the brain may be more limited than that of methylmercury. Once in the central nervous system, however, metallic mercury vapor is oxidized to the mercuric ion (Hg^{++}), which is then trapped in the central nervous system due to the limited ability of the mercuric ion to cross the blood-brain barrier. Mercurous salts are relatively unstable in the presence of sulfhydryl groups and readily transform to metallic mercury and mercuric mercury. Thus, mercurous forms of mercury will possess the toxic characteristics of both metallic and mercuric mercury. All mercury compounds may ultimately be oxidized to divalent (or mercuric) mercury, which preferentially deposits in the kidneys, and all mercury compounds may cause some degree of renal toxicity. While this is not

typically the first effect noted in all forms of mercury exposure, it can be an ultimate effect of either low-dose chronic intake or high-dose acute mercury exposure.

The most sensitive end point following oral exposure of any duration to inorganic salts of mercury appears to be the kidneys. Liquid metallic mercury can volatilize at ambient temperatures. The absorption of metallic mercury vapors from lungs is high (about 80%) (Hursh et al. 1976), and the most sensitive target following inhalation exposure to metallic mercury is the central nervous system. Absorbed metallic mercury crosses the placenta, and the fetal blood may concentrate mercury to levels 10 or more times the levels found in the maternal blood. Therefore, the developing fetal nervous system may be quite sensitive to maternal exposures to mercury vapors.

Salts of mercury and organic mercury compounds are far less volatile than liquid mercury under most conditions. Inhalation of mercury vapors from these forms is not considered a major source of exposure. While inhalation of particulate matter containing mercury salts and/or organic compounds is possible, intestinal absorption is a more likely route of exposure. The most sensitive end point for oral exposure to alkyl mercury compounds (e.g., methylmercuric chloride or ethylmercurials) is the developing nervous system, but toxicity to the adult nervous system may also result from prolonged low-dose exposures. Mercury may adversely affect a wide range of other organ systems, if exposures are sufficiently high. These effects may result from the mercuric ion’s affinity for sulfhydryl groups, which are ubiquitous in animal tissue.

Pharmacokinetic studies indicate that repeated or continuous exposure to any form of mercury can result in the accumulation of mercury in the body. Numerous studies using laboratory animals have shown that retention of mercury in the brain may persist long after cessation of short- and long-term exposures. Mercury is unusual in its ability to induce delayed neurological effects. This is especially prevalent with exposure to alkyl mercury compounds. In such cases, the onset of adverse effects may be delayed for months after the initial exposure. The delayed effects of methyl- and dimethylmercury reported in human poisonings are thought, in part, to result from binding to red blood cells, and subsequent slow release. Methylmercury also forms a complex in plasma with the amino acid cysteine, which is structurally similar to the essential amino acid methionine (Aschner and Clarkson 1988). Clarkson (1995) proposed that methylmercury can cross the blood-brain barrier "disguised" as an amino acid via a carrier-mediated system (i.e., transport is not solely the result of methylmercury’s lipid solubility).

Phenylmercuric acetate is another form of organic mercury to which the general public may be exposed. Although phenylmercury compounds are considered organomercurials, they are absorbed less efficiently by the gastrointestinal tract than is methylmercury. Once inside the body, phenylmercury is rapidly metabolized to Hg^{++}, and its effects are, therefore, similar to those of mercuric salts.

Dimethylmercury is an extremely toxic form of organic mercury, and very small exposures can cause se vere and irreversible delayed neurotoxicity, including death. Dimethylmercury is thought to be metabolized to methylmercury prior to crossing the blood-brain barrier. Dimethylmercury is used in the calibration of laboratory equipment, as a reagent, and in the manufacture of other chemicals. Unlike other forms of mercury, dimethylmercury is quickly absorbed through intact skin, and it will penetrate latex or polyvinyl gloves. It is highly volatile, will readily evaporate, and can be inhaled. Based on its vapor pressure of 58.8 mm at 23.7 EC, Toribara et al. (1997) estimated that a cubic meter of saturated air could hold more than 600 g of dimethylmercury. A recent case history of a chemist who died from an accidental spill of dimethylmercury is prompting calls for its removal as an analytical standard as a safety precaution to prevent further accidents.

Upon significant inhalation exposure to metallic mercury vapors, some people (primarily children) may exhibit a syndrome known as acrodynia, or pink disease. Acrodynia is often characterized by severe leg cramps; irritability; and erythema and subsequent peeling of the hands, nose, and soles of the feet. Itching, swelling, fever, tachycardia, elevated blood pressure, excessive salivation or perspiration, morbilliform rashes, fretfulness, sleeplessness, and/or weakness may also be present. It was formerly thought that this syndrome occurred exclusively in children, but recent reported cases in teenagers and adults have shown that these groups are also susceptible.

Occupational mercury exposures generally occur when workers inhale metallic mercury vapors. Some dermal absorption may occur from skin contact with contaminated air, but the rate is low (less than 3% of the inhaled dose). Dialkyl mercury compounds, which are not normally found in hazardous waste sites, are rapidly and extensively absorbed from both dermal and inhalation routes of exposure.

Mercury is a naturally occurring element in the earth’s crust. It is considered to have been a component of the lithosphere since the planet was formed approximately 4.5 billion years ago. However, levels of mercury at or near the earth’s surface (environmental background levels) are increasing as mercury continues to be released from the earth’s crust by both natural (weathering, volcanoes) and human (mining,

burning of fossil fuels) activities. Background levels, however, are considerable below harmful levels. There are a number of possible pathways for exposure to mercury. For a hazardous waste site that contains mercury that is being released to the environment, pathways that could result in human exposure to mercury include: (1) eating fish or wild game near the top of the food chain (i.e., larger fish, larger mammals) that have accumulated mercury in their tissues from living at or near the site; (2) playing on or in contaminated surface soils; (3) playing with liquid mercury from broken electrical switches, thermometers, blood pressure monitors etc.; or (4) bringing any liquid mercury or broken mercury device into the home, where vapors might build up in indoor air. Other potentially harmful exposure pathways include the excessive use of skin ointments or creams (e.g., skin lightening creams, antiseptic creams) that contain mercury compounds, the use of mercury fungicides (breathing vapors or contact of the skin with the fungicide), or the use of liquid mercury in herbal remedies or religious practices, especially if used indoors. If swallowed, liquid mercury is not very harmful, because it is not easily absorbed into the body from the gastrointestinal tract. However, small amounts of liquid mercury evaporate at room temperature, and the inhaled vapors are harmful.

The developing fetus and breast-fed infants are vulnerable to the harmful effects of mercury. The fetus can be exposed to mercury from the pregnant woman's body through the placenta, and infants may be exposed from the nursing woman's milk. Both inhaled mercury vapors and ingested methylmercury can cross the placenta. Inorganic mercury, and to a lesser extent elemental mercury and methylmercury, will move into breast milk. Pregnant women and nursing women need to be extra cautious in their use of consumer products containing mercury (such as some religious or herbal remedies or skin lightening creams); they should also pay attention to possible exposures to mercury at work and at home.

The primary pathways of mercury exposure for the general population are from eating fish or marine mammals that contain methylmercury, or from breathing in or swallowing very small amounts of mercury that are released from the dental amalgam used for fillings. The relative contribution of mercury from these two main sources will vary considerably for different individuals, depending upon the amount of fish consumed, the level of mercury in the fish, the number of amalgam fillings, eating and chewing habits, and a number of other factors.

Methylmercury levels vary considerably between species and within species of fish (depending on water conditions and size), so there are wide ranges in estimates of the average exposure levels to mercury in the general population from consumption of fish. Some researchers estimate that the typical daily exposure to

mercury is 0.49 µg/day for infants (aged 6–11 months), 1.3 µg/d for 2-year-old children, 2.9 µg/day for females aged 25–30 years, and 3.9 µg/day for males 25–30 years of age. Expressed on a per body weight basis, the intake for all age groups, except for 2-year-old children, was approximately 0.05 µg/kg/day (Clarkson 1990; Gunderson 1988). More recently, MacIntosh et al. (1996) estimated mean dietary exposure of 8.2 µg/d (range, 0.37–203.5 µg/day) for females and 8.6 µg/day (range, 0.22–165.7 µg/day) for males. For an average body weight of 65 kg for women and 70 kg for men, the daily intakes of mercury would be 0.126 µg/kg/day (range, 5.7–3,131 ng/kg/day) for women and 0.123 µg/kg/day (range, 3.1–2,367 ng/kg/day) for men, respectively. Lack of data about the actual amount of food consumed accounted for 95% of the total uncertainty for mercury. This was especially true for consumption levels of canned tuna and other fish (MacIntosh et al. 1996)

The Food and Drug Administration (FDA, 1996) has posted on the Internet advice for consumers recommending that pregnant women and women of childbearing age, who may become pregnant, limit their consumption of shark and swordfish to no more that one meal per month. This advice is given because methylmercury levels are relatively high in these fish species. The FDA's advice covers both pregnant women and women of child-bearing age who might become pregnant, since dietary practices immediately before the pregnancy could have a direct bearing on fetal exposure, particularly during the first trimester of pregnancy. The FDA also states that nursing women who follow this advice will not expose their infants to increased health risks from methylmercury (FDA 1996). For the general population (other than pregnant women and women of child-bearing age), the FDA advises limiting the regular consumption of shark and swordfish (which typically contain methylmercury at 1 ppm) to about 7 ounces per week (about one serving). This level of consumption results in methylmercury exposures below the U.S. FDA acceptable daily intake level for mercury. For fish species with methylmercury levels averaging 0.5 ppm, regular consumption should be limited to 14 ounces per week. Recreational and subsistence fishers who eat larger amounts of fish than the general population and routinely fish the same waters may have a higher exposure to methylmercury if these waters are contaminated (EPA 1995). People who consume greater than 100 grams of fish per day are considered high-end consumers. This is over 10 times the amount of fish consumed by members of the general population (6.5 g/day) (EPA 1995). No consumption advice is necessary for the top 10 seafood species, which make up about 80% of the seafood market: canned tuna, shrimp, pollock, salmon, cod, catfish, clams, flatfish, crabs, and scallops. The methylmercury in these species are generally less than 0.2 ppm, and few people eat more than the suggested weekly limit of fish (i.e., 2.2 pounds). More information on exposure to methylmercury and the levels in fish can be found in Section 5.5, General Population and Occupational Exposures.

Estimating mercury exposure from dental amalgams is also difficult because of high variability in the number of amalgam fillings per individual and the differences in chewing, eating, and breathing habits. Dental amalgams, however, would be the most significant source of mercury exposure in the absence of fish consumption or proximity to a waste site or incinerator. A report from the Committee to Coordinate Environmental Health and Related Programs (CCEHRP) of the Department of Health and Human Services determined a level of from 1 to 5 µg Hg/day from dental amalgam for people with 7–10 fillings (DHHS 1993). The World Health Organization reported a consensus average estimate of 10 µg amalgam Hg/day (range: 3–17 µg/day) (WHO 1991). Weiner and Nylander (1995) estimated the average uptake of mercury from amalgam fillings in Swedish subjects to be within the range of 4–19 µg/day. Skare and Engqvist (1994) estimated that the systemic uptake of mercury from amalgams in middle-aged Swedish individuals with a moderate amalgam load (30 surfaces) was, on the average, 12 µg/day, an amount said to be equivalent to a daily occupational air mercury exposure concentration of 2 $\mu g/m^3$. Other researchers have estimated the average daily absorption of Hg from amalgam at 1–27 µg/day, with levels for some individuals being as high as 100 µg/day (Björkman et al. 1997; Lorscheider et al. 1995).

Richardson et al. (1995) estimated total mercury exposure for Canadian populations of different ages to be 3.3 µg/day in toddlers (3–4 years old), 5.6 µg/day in children (5–11 years old), 6.7 µg/day in teens (12–19 years old), 9.4 µg/day in adults (20–59 years old), and 6.8 µg/day in seniors (aged 60+). Of this exposure, amalgam was estimated to contribute 50% to the total Hg in adults and 32–42% for other age groups. Estimates based on 2 independent models of exposure from amalgam alone were 0.8–1.4 µg/day in toddlers), 1.1–1.7 µg/day in children, 1.9–2.5 µg/day in teens; 3.4–3.7 µg/day in adults, and 2.1–2.8 µg/day in seniors (Richardson 1995).

Higher levels of mercury exposure can occur in individuals who chew gum or show bruxism, a rhythmic or spasmodic grinding of the teeth other than chewing and typically occurring during sleep (Barregard et al. 1995; Enestrom and Hultman 1995). Richardson (1995) reported a transient 5.3-fold increase in levels of mercury upon stimulation by chewing, eating, or tooth brushing. Sallsten et al. (1996) also reported over a 5-fold increase in plasma and urinary mercury levels (27 and 6.5 nmol/mmol creatinine versus 4.9 and 1.2 nmol/mmol creatinine, respectively) in a sample of 18 people who regularly chewed nicotine chewing gum (median values of 10 sticks per day for 27 months), compared to a control group.

Berdouses et al. (1995) studied mercury release from dental amalgams using an artificial mouth under controlled conditions of brushing and chewing and found that although the release of mercury during initial

nonsteady-state conditions was influenced by both the age of the amalgam and the amalgam type, the steady-state value of the mercury dose released by the amalgam was only 0.03 µg/day.

Sandborgh-Englund et al. (1998) evaluated the absorption, blood levels, and excretion of mercury in nine healthy volunteers (2 males, 7 females) exposed to 400 µg /m^3 mercury vapor in air for 15 minutes. This exposure corresponded to a dose of 5.5 nmol Hg/kg body weight. Samples of exhaled air, blood and urine were collected for 30 days after exposure. The median retention of elemental Hg was 69% of the inhaled dose. To evaluate the chronic exposure to mercury from dental amalgam in the general population, the daily Hg dose from fillings was estimated based on the plasma Hg levels of subjects with amalgam fillings and the plasma clearance obtained in this study. The daily dose was estimated to be from 5 to 9 µg/day in subjects with an “average” number (20–35 amalgam surfaces) of amalgam fillings (Sandborgh-Englund et al. 1998)

Halbach (1994) examined the data from 14 independent studies and concluded that the probable mercury dose from amalgam is less than 10 µg/day. When combined with the 2.6 µg/day background intake estimated by WHO (1990) for persons without amalgam fillings and with an estimated methylmercury intake of 5 µg/day from food, Halbach noted that the sum of all those inputs still falls within the WHO's 40 µg/day acceptable daily intake (ADI) level for total mercury. For the ADI of 40 µg total mercury exposure inhaled, approximately 30 µg would be absorbed, assuming 80% absorption (Halbach 1994; WHO 1976).

Whether adverse health effects result from exposure to mercury from amalgams at the levels reported above is currently a topic of on-going research and considerable discussion. A thorough review of this subject is beyond the scope of this profile. Readers are referred to the end of this section (see More on the Effects of Dental Amalgam) for a discussion of some recent reviews of this topic, and a few examples of studies on the putative toxic effects or the lack thereof from continued use of amalgam.

Other Uses of Metallic Mercury

A less well-documented source of exposure to metallic mercury among the general population is its use in ethnic religious, magical, and ritualistic practices, and in herbal remedies. Mercury has long been used for medicinal purposes in Chinese herbal preparations and is also used in some Hispanic practices for medical and/or religious reasons. Espinoza et al. (1996) analyzed 12 types of commercially produced herbal ball preparations used in traditional Chinese medicine. Mercury levels were found to range from 7.8 to

621.3 mg per ball. Since the minimum recommended adult dosage is 2 such balls daily, intake levels of up to 1.2 mg of mercury (presumed to be mercury sulfide) might be a daily dosage.

Some religions have practices that may include the use of metallic mercury. Examples of these religions include Santeria (a Cuban-based religion that worships both African deities and Catholic saints), Voodoo (a Haitian-based set of beliefs and rituals), Palo Mayombe (a secret form of ancestor worship practiced mainly in the Caribbean), and Espiritismo (a spiritual belief system native to Puerto Rico). Not all people who observe these religions use mercury, but when mercury is used in religious, folk, or ritualistic practices, exposure to mercury may occur both at the time of the practice and afterwards from breathing in contaminated indoor air. Metallic mercury is sold under the name "azogue" (pronounced ah-SEW-gay) in stores called “botanicas.” Botanicas are common in Hispanic and Haitian communities, where azogue may be sold as an herbal remedy or for spiritual practices. The metallic mercury is often sold in capsules or in glass containers. It may be placed in a sealed pouch to be worn on a necklace or carried in a pocket, or it may be sprinkled in the home or car. Some store owners may also suggest mixing azogue in bath water or perfume, and some people place azogue in devotional candles. The use of metallic mercury in a home or apartment not only threatens the health of the current residents, but also poses health risks to future residents, who may unknowingly be exposed to further release of mercury vapors from contaminated floors, carpeting, or walls.

Due to the increased number of reported metallic mercury poisonings and to the widespread potential for exposure to liquid/metallic mercury in school chemistry and science laboratories and other places accessible to the general public, the EPA and ATSDR issued a joint mercury alert in June 1997, alerting school and public health officials to the potential toxicity of this substance. This joint mercury alert also advised restricting access to mercury-containing spaces and storage rooms, and the use of alternative substances or chemicals for purposes for which liquid/metallic mercury is currently used.

Issues relevant to children are explicitly discussed in Sections 2.6, Children’s Susceptibility, and 5.6, Exposures of Children.

Minimal Risk Levels for Mercury

A common misconception is that health guidance values, such as the MRL, represent a level above which toxicity is likely to occur. This misconception has occasionally led to unwarranted concern and public

apprehension about relatively benign exposures to environmental substances. The MRL is neither a threshold for toxicity, nor a level beyond which toxicity is likely to occur. MRLs are established solely as screening tools for public health officials to use when determining whether further evaluation of potential exposure at a hazardous waste site is warranted. The relevance of the MRL to public health is discussed further in the following sections concerning the derivation of the respective mercury MRLs.

ATSDR has established a chronic inhalation MRL of 0.2 µg/m^3 for metallic mercury. Assuming a ventilation rate of 20 m^3/day for an average adult, and assuming complete absorption, exposure at the level of the MRL would result in a daily dose of 4 µg. This level of exposure is thought to represent no health risk to any element of the human population. No other inhalation MRLs have been derived for mercury or its compounds.

Oral MRLs have been established for acute (0.007 mg/kg/day) and intermediate (0.002 mg/kg/day) duration exposures to inorganic mercury. ATSDR has also established a chronic oral MRL of 0.0003 mg/kg/day (equivalent to 21 µg/day for a 70-kg adult) for methylmercury. This MRL is at least four times the estimated average daily intake level for methylmercury from the diet. The FDA has estimated that, on average, the intake rate for total mercury (both inorganic and organic) is 50–100 ng/kg/day (equivalent to 0.05–0.1 µg/kg/day or 3.5–7 µg/day for a 70-kg adult). This figure is based on the FDA total diet study of 1982–1984 (Gunderson 1988). Approximately 80–90% of the mercury in the FDA estimate would be expected to be in the form of methylmercury. A separate estimate of the average intake of methylmercury alone, based on a survey of fish eaters and on average levels of methylmercury in fish, places the average intake of methylmercury at 36 ng/kg/day (equivalent to 0.036 µg/kg/day or 2.52 µg/day for a 70-kg adult), with a 99% upper-bound estimate at 243 ng/kg/day (equivalent to 0.243 µg/kg/day or 17 µg/day for a 70-kg adult) (Clarkson 1990). These results indicate that an assessment of total methylmercury intake and body burden should be conducted when estimating exposure to mercury in populations (especially sensitive populations) living near hazardous waste sites that have the potential to release mercury to the environment.

Inhalation MRLs

No inhalation MRLs were derived for inorganic mercury salts or organic mercury compounds due to the absence of data or to the lack of sufficient information regarding exposure levels associated with the reported observed effects.

No MRLs were derived for acute- or intermediate-duration inhalation exposure to metallic mercury vapors. Available studies were either deficient in their reporting of details of experimental protocols and results, used an insufficient number of experimental animals, or tested only one dose/concentration level.

- An MRL of 0.0002 mg/m^3 has been derived for chronic-duration inhalation exposure (365 days or longer) to metallic mercury vapor.

A significant increase in the average velocity of naturally occurring tremors compared to controls was observed in a group of 26 mercury-exposed workers (from 3 industries) exposed to low levels of mercury for an average of 15.3 years (range, 1–41 years) (Fawer et al. 1983). To estimate an equivalent continuous exposure concentration, the average concentration assumed for the 8 hour/day exposures was multiplied by 8/24 and 5/7 (0.026 mg/m^3 x 8/24 hours/day x 5/7 days/week=0.0062 mg/m^3). Uncertainty factors of 10 for variability in sensitivity to mercury within the human population and 3 for use of a minimal-effect LOAEL in MRL derivation were then applied to the calculated 0.0062 mg/m^3 value, yielding a chronic inhalation MRL of 0.2 µg/m^3. Although this MRL is based on experimental data from an adult working population, there is no experimental or clinical evidence to suggest that it would not also be sufficiently protective of neurodevelopmental effects in developing embryos/fetuses and children, the most sensitive subgroups for metallic mercury toxicity.

Inhaled metallic mercury is quickly absorbed through the lungs into the blood, and 70–80% is retained. Its biological half-life in humans is approximately 60 days. The half-life is different for different physiological compartments (e.g., 21 days in the head versus 64 days in the kidneys) (Hursh et al. 1976). Since the duration of exposure influences the level of mercury in the body, the exposure level reported in the Fawer et al. (1983) occupational study was extrapolated from an 8-hour day, 40-hour workweek exposure to a level equivalent to a continuous 24 hour/day, 7 day/week exposure, as might be encountered near a hazardous waste site containing metallic mercury.

Gentry et al. (1998) used the neurobehavioral information on a control group and one exposure group from the Fawer et al. (1983) study to derive an inhalation MRL for elemental mercury based upon a benchmark dose (BMD) analysis. Dose-response analysis could be performed on four measures of hand tremor, with tasks performed both at rest and with a load. The exposure level of the exposed group to metallic mercury was assumed to be the mean TWA exposure of 0.026 mg/m^3. A physiologically based pharmacokinetic model for metallic mercury vapor was found to be linear through the region of concern from the LOAEL to

the MRL; that is, the relationship between inhaled concentration and target tissue concentration at the LOAEL and at lower levels (including the MRL) did not differ. Therefore, exposure concentrations were used directly for the analysis. Gentry et al. (1998) also assumed that 1% of the unexposed population would be considered in the adverse response range. The BMD_{10} was the dose at which the probability of exceeding the 1% adverse response level was 10% greater than in unexposed individuals, and the $BMDL_{10}$ is the 95% lower bound confidence level on that dose. A simple linear model sufficed to describe the dose-response. A $BMDL_{10}$ of 0.017 mg metallic mercury/m^3 was derived as a reasonable representation of the sparse data. This level would be equivalent to a NOAEL (i.e., no LOAEL to NOAEL uncertainty factor is needed). Using a PBPK model to estimate target tissue doses from inhaled mercury vapor and adjusting for continuous exposure and interhuman variability (with an uncertainty factor of 10), an MRL of 0.0004 mg/m^3 (based on target tissue dose) was derived which is about two times the ATSDR derived MRL of 0.0002 mg/m^3 based upon the Fawer et al. (1983) LOAEL.

The ability of long-term, low-level exposure to metallic mercury to produce a degradation in neurological performance was also demonstrated in other studies. One such study (Ngim et al. 1992) attributed adverse neurological effects to a lower average level of exposure than did the Fawer et al. (1983) study; however, this study was not used in deriving a chronic inhalation MRL due to uncertainties concerning the study protocol, including methodological and reporting deficiencies. In the Ngim et al. (1992) study, dentists with an average of 5.5 years of exposure to low levels of metallic mercury were reported to have impaired performance on several neurobehavioral tests. Exposure levels measured at the time of the study ranged from 0.0007 to 0.042 mg/m^3, with an average of 0.014 mg/m^3. Mean blood mercury levels among the dentists ranged from 0.6 to 57 μg/L, with a geometric mean of 9.8 μg/L. The performance of the dentists on finger tapping (measures digital motor speed), trail-making (measures visual scanning and motor speed), digit symbol (measures visuomotor coordination and concentration), digit span, logical memory delayed recall (measure of verbal memory), and Bender-Gestalt time (measures visual construction) tests was significantly poorer than controls. The exposed dentists also showed higher aggression than did controls. Furthermore, within the group of exposed dentists, significant differences were observed between a subgroup with high mercury exposure compared to a subgroup with lower exposure. These exposure severity subgroups were not compared to controls, and average exposure levels for the subgroups were not reported. The design and reporting of this study limits its usefulness in deriving an MRL for metallic mercury. The exposure status of the subjects was known to the investigator during testing, mercury levels were not reported for controls, and methods used to adjust for potential contributions other than mercury from amalgams to the study results (such as the possible use in this population of traditional medicines

containing mercury) were not reported. It was also unclear whether the results for the mercury exposure group were inordinately influenced or skewed by the individual dentists with the highest exposures and/or blood levels. These confounding factors precluded the use of the Ngim et al. (1992) study for the derivation of an MRL, but the study does provide support both for the premise that low-dose chronic exposure to metallic mercury can result in adverse health sequelae and for the chronic inhalation MRL that is based on the Fawer et al. (1983) study of occupationally exposed individuals.

Other occupational studies further support the ability of metallic mercury to induce neurological deficits. Several studies have reported significant effects on tremor or cognitive skills among groups exposed occupationally to comparable or slightly higher (up to 0.076 mg/m^3) levels (Ehrenberg et al. 1991; Piikivi et al. 1984; Roels et al. 1982). Difficulty with heel-to-toe gait was observed in thermometer plant workers subjected to mean personal breathing zone air concentrations of 0.076 mg/m^3 (range, 0.026–0.27 mg/m^3) (Ehrenberg et al. 1991). Tremors have also been reported in occupationally exposed workers with urinary mercury concentrations of 50–100 µg/g creatinine and blood levels of 10–20 µg/L (Roels et al. 1982). By comparison, blood mercury levels in the Fawer et al. (1983) study averaged 41.3 and 16.6 µmol Hg/L for the exposed and control groups, respectively. Urinary mercury levels for the exposed workers in the Fawer et al. (1983) study averaged 11.3 µmol Hg/mol creatinine (about 20 µg/g creatinine), compared with 3.4 µmol/mol creatinine in the controls. Piikivi et al. (1984) found decreases in performance on tests that measured intelligence (based upon a similarities test) and memory (evaluating digit span and visual reproduction) in chloralkali workers exposed for an average of 16.9 years (range, 10–37 years) to low levels of mercury, when compared to an age-matched control group. In this study, significant differences from controls were observed on these tests among 16 workers with blood levels ranging from 75 nmol/L to 344 nmol/L and urine levels ranging from 280 nmol/L (about 56 µg/L) to 663 nmol/L. Abnormal nerve conduction velocities have also been observed in chloralkali plant workers at a mean urine concentration of 450 µg/L (Levine et al. 1982). These workers also experienced weakness, paresthesias, and muscle cramps. Prolongation of brainstem auditory evoked potentials was observed in workers with urinary mercury levels of 325 µg/g creatinine (Discalzi et al. 1993). Prolonged somatosensory-evoked potentials were found in 28 subjects exposed to airborne mercury concentrations of 20–96 mg/m^3 (Langauer-Lewowicka and Kazibutowska 1989). All of these studies substantiate the ability of chronic, low- to moderate-level exposure to metallic mercury vapors to cause neurological deficiencies.

Employment of the Chronic Inhalation MRL for Metallic Mercury

ATSDR emphasizes that the MRL is not intended to be used as an estimation of a threshold level. Exceeding the MRL does not necessarily mean that a health threat exists. However, the greater the amount by which the MRL is exceeded and the longer or more frequent the individual exposures, the greater the likelihood that some adverse health outcome may occur. Secondly, the chronic inhalation MRL is, by definition, a level that is considered to be without appreciable (or significant) health risk over a lifetime of exposure at that level. It is further considered to be a "safe" level for all factions of the exposed human population, when exposure exists for 24 hours a day, 7 days a week for an extended period of years. The employment of the MRL, therefore, must be geared to the particular exposure scenario at hand. For example, people may be able to "tolerate" metallic mercury levels above the MRL for intermittent periods of exposure (e.g., 1 or 2 hours per day, 5 days per week) without any adverse health sequelae, either overt or covert. The use of the "contaminated area" (e.g., storage versus exercise room versus day care) will largely influence the use of the MRL. Finally, the MRL is intended primarily as a "screening value" for public health officials to use in their assessment of whether further evaluation of the potential risk to public health is warranted in a hazardous waste site scenario. The MRL is *not* intended, nor should it be indiscriminately used, as a clean-up or remediation level, or as a predictor of adverse health effects. While it is considered to afford an adequate degree of protection for the health of all potentially exposed individuals, it might be unnecessarily stringent for application to some exposure situations (i.e., higher air concentrations might afford a similar degree of protection in some exposure scenarios); thus, its relevance in any specific environmental situation is intended to be determined by an experienced public health or medical official.

Oral MRLs

Metallic Mercury

No oral MRLs were derived for metallic (elemental) mercury due to the lack of data. Oral exposure to liquid metallic mercury would be expected to present little health risk, since it is so poorly absorbed (<0.01%) through the healthy intestine. Sufficiently large quantities could, however, present a risk of intestinal blockage, and some could enter the systemic circulation (blood or lymphatic) through open lesions, presenting a risk of occlusion of smaller arteries, especially within the pulmonary circulation.

Inorganic Mercury

The acute- and intermediate-duration MRLs for oral exposure to inorganic mercury are based on kidney effects reported in a 1993 NTP study of mercuric chloride (NTP 1993). Most of the supporting studies of oral exposure to inorganic mercury also use mercuric chloride.

Mercuric sulfide (also known as cinnabar) is the predominant natural form of mercury in the environment and is a common ore from which metallic mercury is derived. Mercury released to the environment may be transformed into mercuric sulfide. Several studies suggest that the bioavailability of mercuric sulfide in animals is less than that of mercuric chloride (Sin et al. 1983, 1990; Yeoh et al. 1986, 1989). For example, Sin et al. (1983) found an increase in tissue levels of mercury in mice orally exposed to low doses of mercuric chloride, but elevated levels of mercury were not found in the tissues of mice fed an equivalent weight of mercuric sulfide. This finding indicates a difference in bioavailability between $HgCl_2$ and HgS in mice. However, a quantitative determination of the relative bioavailabilities of mercuric sulfide versus mercuric chloride has not been derived in the available studies, nor has the relative bioavailability of mercuric sulfide in humans been examined.

- An MRL of 0.007 mg Hg/kg/day has been derived for acute-duration oral exposure (14 days or less) to inorganic mercury.

The MRL was based on a NOAEL of 0.93 mg Hg/kg/day for renal effects in rats administered mercuric chloride 5 days a week for 2 weeks. The dose used in this study was duration-adjusted for a 5-day/week exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animals to humans and 10 for human variability). Increased absolute and relative kidney weights were observed in male rats exposed to 1.9 mg Hg/kg/day as mercuric chloride (NTP 1993). At higher doses, an increased incidence and severity of tubular necrosis was observed.

Several other studies examining the effects of oral exposure to inorganic mercury salts have also shown renal toxicity in humans as a result of acute oral exposures. Kidney effects (i.e., heavy albuminuria, hypoalbuminemia, edema, and hypercholesterolemia) have been reported after therapeutic administration of inorganic mercury (Kazantzis et al. 1962). Acute renal failure has been observed in a number of case studies in which mercuric chloride had been ingested (Afonso and deAlvarez 1960; Murphy et al. 1979; Samuels et al. 1982). The autopsy of a 35-year-old man who ingested a lethal dose of mercuric chloride

and exhibited acute renal failure showed pale and swollen kidneys (Murphy et al. 1979). A case study reported acute renal failure characterized by oliguria, proteinuria, hematuria, and granular casts in a woman who ingested 30 mg Hg/kg body weight as mercuric chloride (Afonso and deAlvarez 1960). Another case study reported a dramatic increase in urinary protein secretion by a patient who ingested a single dose of 15.8 mg Hg/kg body weight as mercuric chloride (assuming a body weight of 70 kg) (Pesce et al. 1977). The authors of the report surmised that the increased excretion of both albumin and β_2-microglobulin were indicative of mercury-induced tubular and glomerular pathology. Acute renal failure that persisted for 10 days was also observed in a 19-month-old child who ingested an unknown amount of powdered mercuric chloride (Samuels et al. 1982). Decreased urine was also observed in a 22-year-old who attempted suicide by ingesting approximately 20 mg Hg/kg (Chugh et al. 1978).

- An MRL of 0.002 mg Hg/kg/day has been derived for intermediate-duration oral exposure (15–364 days) to inorganic mercury.

This MRL was based on a NOAEL of 0.23 mg Hg/kg/day for renal effects in rats administered mercuric chloride 5 days a week for 6 months (Dieter et al. 1992; NTP 1993). This dose was duration-adjusted for a 5 day/week exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animals to humans and 10 for human variability). Increased absolute and relative kidney weights were observed in rats exposed to 0.46 mg Hg/kg/day, the next higher treatment level. At higher doses, an increased incidence of nephropathy (described as foci of tubular regeneration, thickened tubular basement membrane, and scattered dilated tubules containing hyaline casts) was observed. Renal toxicity is a sensitive end point for inorganic mercury toxicity, as seen in other intermediate-duration oral studies on rats and mice exposed to inorganic mercury (Carmignani et al. 1992; Jonker et al. 1993a; NTP 1993), as well as case reports of humans ingesting inorganic mercury for acute and chronic durations (Afonso and deAlvarez 1960; Davis et al. 1974; Kang-Yum and Oransky 1992; Nielsen et al. 1991; Pesce et al. 1977).

The relatively small difference between the acute-duration MRL (0.007 mg/kg/day) and the intermediate-duration MRL (0.002 mg/kg/day) is not meant, nor is it considered, to imply a high level of precision in the calculation of these health guidance values. Rather, this difference of 5 μg/kg/day reflects the increased toxicity of continued low-dose exposure for longer periods of time and is consistent with the known build-up of mercury levels in body tissues over a prolonged course of continued exposure. The actual precision of any derived (actually estimated) MRL is dependent upon an encompassing, but not sharply defined, area of uncertainty based upon the database used in its determination.

As a method of comparison to evaluate whether use of another method to derive an MRL might result in a different intermediate oral MRL value for inorganic mercury, ATSDR used the same data (Dieter et al. 1992; NTP 1993) to calculate a benchmark dose for inorganic mercury. Using the most sensitive end point identified in this study (relative kidney weight changes in rats), the experimental data were used to obtain a modeled dose-response curve. Benchmark doses were then determined for the 10% response level (0.38 mg/kg/day) and the 5% response level (0.20 mg/kg/day). After adjusting the 5-days/week exposures in the study to 7-days/week equivalent doses, the 10 and 5% response-base benchmarks became 0.27 and 0.15 mg/kg/day, respectively. Application of 10-fold uncertainty factors for each inter- and intraspecies variability resulted in estimated human benchmark doses of 0.003 mg/kg/day for the 10% response level and 0.002 mg/kg/day for the 5% response level. These values strongly support the current existing intermediate oral MRL of 0.002 mg/kg/day for inorganic mercury.

No MRL for chronic-duration oral exposure to inorganic mercury was derived, because the study results showed decreased survival rate for male rats at all LOAELs.

Organic Mercury

Acute, Intermediate, or Chronic Inhalation MRLs: No inhalation MRLs were derived for organic mercury compounds, due to the absence of data or to the lack of sufficient information regarding exposure levels associated with the reported observed effects.

Acute and Intermediate Oral MRLs: No MRLs were derived for acute or intermediate oral exposure to organic mercury compounds due to the absence of data or to the lack of sufficient information regarding exposure levels associated with the reported observed effects.

Chronic Oral MRL for Methylmercury: Hair levels are typically used as an index of exposure to methymercury. A number of studies report that hair mercury levels correlate with total intake levels and with organ-specific levels of mercury. Suzuki et al. (1993) analyzed 46 human autopsies in Tokyo, Japan and reported that hair mercury levels were highly significantly correlated with organ Hg levels in the cerebrum, cerebellum, heart, spleen, liver, kidney cortex, and kidney medulla, when the total mercury or methyl mercury value in the organ was compared with the hair total mercury or organic mercury, respectively.

Nakagawa (1995) analyzed total mercury in hair samples from 365 volunteers in Tokyo, and reported higher mercury levels in those who preferred fish in their diet, compared to those who preferred other foods (preference choices were fish, fish and meat, meat, and vegetables). The mean hair mercury levels were 4 ppm in men who preferred fish and 2.7 ppm in women who preferred fish. The lowest hair mercury levels were seen in men and women who preferred vegetables, 2.27 and 1.31 ppm, respectively. The mean hair level for the whole group was 2.23 ppm (median 1.98).

Drasch et al. (1997) assayed tissue samples of 150 human cadavers (75 males, and 75 females) from a "normal" European (German) population, i.e., there were no occupational or higher than average exposures to metals found in any of the biographies of the deceased. The objective was to evaluate the validity of blood, urine, hair, and muscle as biomarkers for internal burdens of mercury, lead, and cadmium in the general population. All individuals died suddenly and not as a result of chronic ailments. Age ranged from 16 to 93 years, and every decade was represented by approximately 10 males and 10 females. Tissues sampled included kidney cortex, liver, cerebral cortex, cerebellum, petrous portion of the temporal bone, (pars petrosis ossis temporalis), pelvic bone (spina iliaca anterior-superior), muscle (musculus gluteus), blood (heart blood), urine, and hair (scalp-hair). Statistically significant rank correlations between biomarker levels and tissues were observed, but with large confidence intervals for the regressions. The authors conclude that specific biomarkers relative to each metal are useful in estimating body burdens and trends in groups, but are not useful for determining the body burden (and therefore the health risks) in individuals. A notable exception was for correlation to brain mercury. By comparison to a generally poor correlation of cadmium, lead, and mercury between hair and tissue, there was a strong correlation between mercury in hair and mercury in brain (cerebrum and cerebellum). The authors state that this may be due to the high lipophilicity of elemental and short-chain alkyl mercury compounds. As seen in other studies comparing European to Japanese hair mercury levels, the mercury hair levels reported by Nakagawa (1995) of 2–4 ppm for a Japanese population are 10–20 times higher than total mercury levels observed in the Drasch et al. (1997) study (median, 0.247 μg/g in hair; range, 0.43–2.5 μg/g).

Other studies have confirmed a good correlation between hair mercury and brain mercury levels. In a study on the Seychelles Islands cohort, Cernichiari et al. (1995b) compared maternal hair levels, maternal blood levels, fetal blood levels, and fetal brain levels. Autopsy brains were obtained from infants dying from a variety of causes. The concentrations of total mercury in six major regions of the brain were highly correlated with maternal hair levels. This correlation was confirmed by a sequence of comparisons among the four measurements. Maternal hair levels correlated to maternal blood levels (r=0.82) and infant brain

levels (r=0.6–0.8). Concentrations in maternal blood correlated with infant blood levels (r=0.65); and infant blood levels correlated to infant brain levels (r=0.4–0.8).

Accordingly, ATSDR used maternal hair mercury levels as the exposure measurement to derive a chronic MRL for methylmercury. While hair analysis can be confounded by outside sources of contamination (e.g., as might occur in certain occupational settings) (Hac and Krechniak 1993), the study population used as the basis of the chronic oral MRL for methylmercury is far removed from external or industrial sources of mercury, effectively eliminating this as a consideration for the following analysis.

- An MRL of 0.0003 mg Hg/kg/day has been derived for chronic-duration oral exposure (365 days or longer) to methylmercury.

The chronic oral MRL for methylmercury is based upon the Seychelles Child Development Study (SCDS), in which over 700 mother-infant pairs have, to date, been followed and tested from parturition through 66 months of age (Davidson et al. 1998). The SCDS was conducted as a double-blind study and used maternal hair mercury as the index of fetal exposure. Enrollees were recruited by the head nurse/hospital midwife by asking the mothers if they wished to participate in the study when they arrived at the hospital for delivery. The first 779 who did not decline participation became the mothers in the study cohort. Of the initial 779 mothers enrolled in the study at parturition, 740 remained at the pre-determined child testing age of 6.5 months, 738 remained in the 19-month cohort, 736 remained at 29 months, and 711 remained for the 66-month neurobehavioral and developmental examinations.

The Seychellois were chosen as a study population for a number of reasons.

- (1) All fish contain some level of methylmercury (Davidson et al. 1998); and the Seychellois regularly consume a high quantity and variety of ocean fish, with 12 fish meals per week representing a typical methylmercury exposure.

- (2) The median total mercury concentration in 350 fish sampled from 25 species consumed by the Seychellois was <1 ppm (range, 0.004–0.75 ppm), comparable to the mercury concentration in commercially obtainable fish in the U.S. (It should be noted here that while the methylmercury levels in the Seychellois population are 10–20 times those in the U.S., it is not because they consume more highly contaminated fish, but rather because they consume more fish than the U.S. population.)

- (3) The Seychelles represents a relatively pristine environment, with no local industry for pollution, and are situated more than 1,000 miles from any continent or large population center.

- (4) The population is highly literate, cooperative, and has minimal immigration and emigration.
- (5) The Seychellois constitute a generally healthy population, with low maternal alcohol consumption and tobacco use (<2%).
- (6) The large sample size/study population (>700 mother-infant pairs).
- (7) Excellent retention of mother-infant pairs throughout the study (i.e., 711 of the initially enrolled 778 mother-infant pairs still participating at 66 months post-partum.
- (8) The use of standardized neurobehavioral tests.

The results of the 66-month testing in the SCDS revealed no evidence of adverse effects attributable to chronic ingestion of low levels of methylmercury in fish (Davidson et al. 1998). In this study, developing fetuses were exposed *in utero* through maternal fish ingestion before and during pregnancy (Davidson et al. 1998). Neonates continued to be exposed to maternal mercury during breastfeeding (i.e., some mercury is secreted in breast milk), and methylmercury exposure from the solid diet began after the gradual post-weaning shift to a fish diet. In the 66-month study cohort, the mean maternal hair level of total mercury during pregnancy was 6.8 ppm (range, 0.5–26.7 ppm; n=711), and the mean child hair level at the 66-month testing interval was 6.5 ppm (range, 0.9–25.8 ppm; n=708). The mean maternal hair mercury level in the highest exposed subgroup in the study was 15.3 ppm (range, 12–26.7; n=95). The 66-month test battery, which was designed to test multiple developmental domains, included the following primary measurements:

- (1) General Cognitive Index (GCI) of the McCarthy Scales of Children's Abilities (to estimate cognitive ability);
- (2) the Preschool Language Scale (PLS) total score (to measure both expressive and receptive language ability);
- (3) the Letter and Word Recognition and
- (4) Applied Problems subtests of the Woodcock-Johnson (W-J) Tests of Achievement (to measure reading and arithmetic achievement);
- (5) the Bender-Gestalt test (to measure visual-spatial ability); and
- (6) the total T score from the Child Behavior Checklist (CBCL) (to measure the child's social and adaptive behavior). Serum sampling revealed no detectable levels of PCBs (detection limit=0.2 ng/mL).

None of the tests indicated an adverse effect of methylmercury exposure. As evaluated through regression analyses, there was no reduction in performance with increasing maternal hair mercury levels for the neurobehavioral parameters examined. In contrast, scores were better for four of the six tests in the highest MeHg-exposed groups, compared with lower exposure groups for both prenatal and postnatal exposure (the four test were the (1) General Cognitive Index (GCI) of the McCarthy Scales of Children's `Abilities (to estimate cognitive ability); (2) the Preschool Language Scale (PLS) total score (to measure both expressive and receptive language ability); (3) the Letter and Word Recognition and (4) Applied Problems subtests of the Woodcock-Johnson (W-J) Tests of Achievement (to measure reading and arithmetic achievement). While the positive outcomes are not considered to indicate any beneficial effect of methylmercury on neurological development or behavior, they might be more appropriately attributed to the beneficial effects of omega-3 fatty acids or other constituents present in fish tissue, since the methylmercury levels in hair are known to correlate closely with fish intake. The slight decreases in the subjectively reported activity level of boys reported in the 29-month observations were not seen during the 66-month tests. The mean maternal hair level of 15.3 ppm in the highest exposed group in the 66-month test cohort is, therefore, considered a NOAEL for SCDS and is used by ATSDR as the basis for derivation of a chronic oral MRL for methylmercury. A related study (Myers et al. 1997) by some members of the same team of researchers from the University of Rochester examined the Seychellois children for attainment of the same developmental milestones reported to have been delayed in the Iraqi poisoning incident in the early 1970s (Cox et al. 1989); however, unlike the Iraqi study, no delays in the age of first walking and talking was seen in the Seychellois children exposed in utero.

Sensitivity of Neurobehavioral Measures /Reliability of Tests

The neurobehavioral test battery used in the 66-month Seychelles study was designed to assess multiple developmental domains (Davidson et al. 1998). The tests were considered to be sufficiently sensitive and accurate to detect neurotoxicity in the presence of a number of statistical covariates. On-site test administration reliability was assessed by an independent scorer, and mean interclass correlations for interscorer reliability were 0.96–0.97 (Davidson et al. 1998). The sample size was determined to be sufficient to detect a 5.7-point difference on any test with a mean (SD) of 100 (16) between low (0–3 ppm) and high (>12 ppm) hair mercury concentration groups for a 2-sided test (A = 0.05 at 80% power).

Supporting Studies

Crump et al. (1998) conducted benchmark dose (BMD) calculations and additional regression analyses of data collected in a study in which a series of scholastic and psychological tests were administered to children whose mothers had been exposed to methylmercury during pregnancy. Hair samples were collected from 10,970 new mothers in New Zealand in 1977 and 1978. High hair mercury levels were considered to be those over 6 ppm, which was the hair level predicted to result at steady state from consumption of mercury at the WHO/FAO Provisional Tolerable Weekly Intake of 0.3 mg total mercury/week and 0.2 mg methylmercury/week. By this criterion, 73 of approximately 1,000 mothers who had consumed fish more than 3 times/week during pregnancy were determined to have high hair mercury levels. In 1985, when the children were 6 to 7 years of age, 61 children (1 set of twins) of the 73 mothers in the high hair mercury group were located; these children constituted the high exposure group, which was matched with three control groups (one with 3–6 ppm maternal hair mercury levels, one with 0–3 ppm whose mothers had been high fish consumers, and one with 0–3 ppm whose mothers had not been high fish consumers). The entire study cohort consisted of 237 children. A battery of 26 psychological and scholastic tests were administered to the children at school during the year 1985. Mothers were interviewed at the time of test administration to obtain additional data on social and environmental factors. In the high exposure group of children, one boy's mother had a hair mercury level of 86 ppm, which was more than four times higher than the next highest hair mercury level of 20 ppm. BMDs (10% response rate) calculated from five tests ranged from 32 to 73 ppm, when the 86 ppm mother's child was included. This corresponded to a BMDL range of 17 to 24 ppm. Although none of the 86 ppm child's test scores was an outlier according to the definition used in the analyses, his scores were significantly influential in the analyses. When this child was omitted from the analyses, BMDs ranged from 13 to 21, with corresponding BMDLs of 7.4 to 10 ppm.

Developing fetuses in the SCDS were exposed through maternal fish ingestion before and during pregnancy. Each child was evaluated at 19 months and again at 29 months (±2 weeks) for infant intelligence (Bayley Scales of Infant Development [BSID] Mental and Psychomotor Scales), with a modified version of the BSID Infant Behavior Record to measure adaptive behaviors at 29 months (Davidson et al. 1995b). Testing was performed by a team of Seychellois nurses extensively trained in administration of the BSID. Maternal hair concentrations, measured in hair segments that corresponded to pregnancy, ranged from 0.5 to 26.7 ppm, with a median exposure of 5.9 ppm for the entire study group. The mean BSID Mental Scale Indices determined at both 19 and 29 months were found to be comparable

to the mean performance of U.S. children. The BSID Psychomotor Scale Indices at both measurement intervals were two standard deviation units above U.S. norms, but were still consistent with previous findings of motor precocity in children reared in African countries. The study found no effect that could be attributed to mercury on the BSID scores obtained at either the 19- or 29-month measurement/testing interval. The 29-month cohort represented 94% of the 779 mother-infant pairs initially enrolled in the study, and approximately 50% of all live births in the Seychelles in 1989.

The only observation in the 29-month testing that might be attributable to prenatal mercury exposure was a slight decrease in the activity level in boys (but not girls) as determined by the Bayley Infant Behavior Record (subjective observation). Whereas this decrease was significant in males ($p = 0.0004$), it was not statistically significant in females ($p = 0.87$). When the subjective activity scores for male and female children were evaluated collectively, no statistically significant or remarkable decrease in activity was apparent outside the >12 ppm maternal hair concentration group. The affect on activity level in boys is not considered an adverse effect by the authors of the study.

Grandjean et al. (1997b, 1998) reported another epidemiological study of methylmercury exposure for a population in the Faroe Islands. Although the Faroese are a fishing culture, the major source of methylmercury exposure for this population is pilot whale meat, which is intermittently consumed as part of the cultural tradition. The initial study cohort consisted of 1,022 singleton births occurring in a 21-month window during 1986-1987. At approximately 7 years of age, neurobehavioral testing was conducted on 917 of the remaining cohort members. No abnormalities attributable to mercury were found during clinical examinations or neurophysiological testing. A neuropsychological test battery was also conducted, which included the following: Finger Tapping; Hand-Eye Coordination; reaction time on a Continuous Performance Test; Wechsler Intelligence Scale for Children - Revised Digit Spans, Similarities, and Block Designs; Bender Visual Motor Gestalt Test; Boston Naming Test; and California Verbal Learning Test (Children). Neuropsychological tests emphasized motor coordination, perceptual-motor performance, and visual acuity. Pattern reversal visual evoked potentials (VEP) with binocular full-field stimulation, brain stem auditory evoked potentials (BAEP), postural sway, and the coefficient of variation for R-R inter-peak intervals (CVRR) on the electrocardiogram were all measured. The neuropsychological testing indicated mercury-related dysfunction in the domains of language, attention, memory, and visuospatial and motor function (to a lesser extent), which the authors considered to remain after the children of women with maternal hair mercury concentrations above 10 µg/g (10 ppm) were excluded. While this study represents a significant contribution to the human database for methylmercury exposure

and effects, a number of potentially influential factors not fully considered as possible covariates somewhat cloud the interpretation of the results.

These differences between the neuropsychological effects observed in the Faroe Island cohort and the absence of effects reported in the Seychelles Island cohort might result from a variety of factors. The Faroe Island children were older (7–8 years versus 5.5 in the SCDS). Some of the measurement instruments (i.e., the neuropsychological test administered) were also different. Since the first neuropsychological testing in the Faroe study was not conducted until 7 years of age, it is not known whether the observed effects might have been apparent at an earlier age. Ongoing and planned future testing of the Seychelles population will provide additional information on the progression of any observed effects. Further examination of the Seychelles population using the neuropsychological test that showed positive results in the Faroe Island population will also allow a more direct comparison of results.

The diet in the two studies was also considerably different. The majority of the mercury exposure to the Faroe Island population came from whale meat (estimated at about 3 ppm in muscle tissue) with a relatively small portion coming from fish. Some of the mercury in whale meat is in the form of inorganic mercury. In the Seychelles study, all of the mercury came from fish as methylmercury with concentrations of around 0.3 ppm. Whale meat blubber is widely consumed in the Faroe Islands and also contains polychlorinated biphenyls (PCBs). Grandjean et al. (1995b) estimated a daily intake of 200 µg of PCB. This value can be compared to the Tolerable Daily Intake of PCBs established by the FDA of 60–70 µg/day for an adult. Further statistical analysis of the possible influence of PCBs on the observed study results needs to be conducted (see the discussion below on Peer Panel 1Review of Key Studies for additional comments).

The primary biomarker used to estimate mercury exposure was also different in the two studies. The Faroe Island analysis used cord blood, and the Seychelles study used maternal hair level. The use of mercury in cord blood has the advantage of being a more direct measure of exposure to the fetus, but the levels at term may not reflect exposures at earlier developmental stages. While Grandjean et al. (1997) did report maternal hair mercury levels, the mean hair level for the interquartile range of 2.6–7.7 ppm was reported only as a geometric average (4.27 ppm). In contrast, the Seychelles study reported only an arithmetic mean level for the entire study population (6.8 ppm). While both are valid measures, a direct comparison of “average” values for the two studies is not possible without further statistical analysis of both data sets.

In the case of the Faroe study, no data were presented in the peer-reviewed publications to address variability of food/whale meat or blubber intake among the Faroe Islanders, making it difficult to evaluate the possibility of peak intake levels during critical development phases. Consumption data were reported only as <1 pilot whale meat meal/month and 1–2 fish meals per week. In contrast, the Seychelles dietary habits provide a relatively stable intake, and a high degree of correlation was found between mean hair levels in samples covering each trimester and levels in samples for the entire pregnancy (Cernichiari et al. 1995a). Cernichiari et al. (1995b) also report a good correlation between levels of total mercury in neonatal brain and levels in the corresponding maternal hair. While the contribution of continued mercury exposure through breast feeding or post-weaning diet was not fully addressed in the Seychelles study reports (Davidson 1995, 1998), that is not considered a significant drawback of the study, since no effects on neurobehavioral/neuropsychological testing were seen at any maternal hair level. In the Faroese assessment of latent neuropsychological effects from an *in utero* exposure to mercury, however, the role of continuing postnatal exposure to mercury either from breast milk or from ingestion of methylmercury-containing foods (e.g., pilot whale meat) is less clear. Specifically, it is not known what proportion, if any, of the neuropsychological effects reported in the Faroe Islands population could be attributed to 7 years of postnatal exposure to methylmercury in food. The variability and magnitude of this postnatal exposure should, therefore, be further evaluated.

Peer Panel Review of Key Studies

In addition to the traditional peer review process that precedes publication in most scientific journals, the studies considered by ATSDR for use in estimating a chronic oral MRL for methylmercury underwent two stringent reviews by recognized experts in the environmental health field.

On July 20 and 21, 1998, ATSDR assembled a panel of 18 experts from the scientific and medical communities to review current issues and the relevant literature on mercury and its compounds, including methylmercury (ATSDR 1999). Several members of each of the respective research teams that conducted the Iraqi, Seychelles, Faroe, and Madeira studies were included among the expert panelists, and provided extensive overviews of their studies. The presentations were followed by an open, wide-ranging scientific discussion of the merits and interpretations of the currently available studies. Topics of significant discussion included the relative merits of the respective study populations, exposure regimens, sensitivity of neurobehavioral measures, and determination of an uncertainty factor. While it was unanimously agreed that the Seychelles and Faroe studies were both excellent studies that provided a significant contribution to

the human database for methylmercury exposure and effects, a number of factors that could have contributed to the study results, but were not considered as possible statistical covariates, were discussed. In the case of the Faroe study, the consumption of whale blubber, which is known to be contaminated with PCBs, DDT, and possibly other organochlorines, introduces a potentially significant influence on the study results. Weihe et al. (1996) reported that the PCB and DDT concentrations in blubber of pilot whales taken in Faroese waters are about 30 ppm and 20 ppm, respectively. In contrast, the Seychellois population does not eat marine mammals at all. In addition, the Faroe study did not address other possible statistical covariates, such as the dietary and nutritional status of the study population and the use of tobacco during pregnancy, further complicating the interpretation of the neuropsychological test results.

On November 18–20, 1998, a workshop on Scientific Issues Relevant to the Assessment of Health Effects from Exposure to Methylmercury was conducted in Raleigh, North Carolina. The workshop was jointly sponsored by the U.S. Department of Health and Human Services (DHHS), the National Institute of Environmental Health Sciences (NIEHS), the Centers for Disease Control and Prevention (CDC), the Food and Drug Administration (FDA), the U.S. Environmental Protection Agency (EPA), the National Oceanic and Atmospheric Administration (NOAA), the Office of Science and Technology Policy (OSTP), the Office of Management and Budget (OMB), and ATSDR. The purpose of this workshop was to discuss and evaluate the major epidemiologic studies that associated methylmercury exposure and the results of an array of developmental measures in children. These studies monitored and evaluated exposed populations in Iraq, the Seychelles Islands, the Faroe Islands, and the Amazon River Basin. A number of animal studies were also considered in support of a human health risk assessment. Presentation of each study by the research team that conducted the study was followed by an expert panel evaluation that examined each study, taking into consideration the exposure data, experimental design and statistical analysis, potential confounders and variables, and neurobehavioral end points evaluated. A fifth panel evaluated the results of relevant animal studies. Significant issues that were discussed included the use of umbilical cord blood mercury levels versus hair mercury concentrations as an index of methylmercury exposure during pregnancy, the patterns of exposure, the dietary/health status of study populations, other potentially relevant exposures, other confounding influences, and the adjustments made for statistical covariates. All five panels at this workshop commended the efforts of the investigators and respective staffs of the Seychelles and Faroe studies for conducting highly sophisticated investigations under difficult conditions. However, specific findings of several of the panels raise issues that, at present, preclude the Faroe data from consideration as a starting point for MRL derivation.

In addressing the potential influence of concurrent PCB exposure on the Faroe results, the Confounders and Variables (Epidemiology) panel indicated that with respect to four of the prenatal outcomes (related primarily to verbal and memory performance), when PCBs were included in the model, only one of these outcomes is specifically related to mercury exposure. Concerning this matter, the panel wrote that "... the most likely explanation is that both (mercury and PCBs)... affect these three outcomes, but their relative contributions cannot be determined given their concurrence in this population." The Neurobehavioral Endpoints Panel also looked at this issue, and noted that "PCB exposure might act as an effect modifier, increasing the susceptibility to MeHg"; however, this panel further indicated that it did not believe that the effects seen in the Faroe Islands were due to uncontrolled confounding by PCBs. A third panel that addressed the issue of concurrent PCB exposures, the Statistics/Design Panel, noted that only 3 of 208 PCB congeners were measured in the Faroe study, and stated that it "seems likely that mercury was measured more accurately than the biologically relevant PCB exposure. Consequently even if the neurological effects seen in this study were caused entirely by PCBs, it is possible that mercury would still be more highly correlated with these effects than PCBs." The Statistics/Design Panel also said that "the best method to deal with this problem would be to study a population where exposure to PCBs is not an issue." This statement points directly to the Seychelles study as the study most appropriate for MRL derivation.

Another issue raised at Raleigh workshop concerned the taking of hair samples for determining pre-natal exposure. In the Seychelles, hair samples were collected 6 months post-partum, and segments corresponding to pregnancy were selected for analysis. In the case of the Faroese, hair samples were taken at the scalp. Regarding that, the Confounders and Variables (Epidemiology) panel stated that "Given the time it takes the Hg to be excreted into the hair, we can assume that samples collected at parturition do not cover the last 6 weeks of gestation, during which critically important neuronal proliferation and differentiation is taking place."

Regarding the Seychelles and Faroe studies, the Neurobehavioral Endpoints Panel found "no specific neurobehavioral signature injury from MeHg" in the data from either study (Seychelles or Faroe). The same panel also noted that episodic exposure in the Faroese (1–2 fish meals/week and <1 pilot whale meal/month) "may reduce the likelihood of detecting a consistent 'neurobehavioral signature injury' specific to MeHg and may account for different observations in children with the same average exposure."

Based upon the discussions at the Raleigh workshop and the individual panel findings, as well as the aforementioned Atlanta expert panel review, ATSDR has determined that the Seychelles study represents

the most appropriate and reliable database currently available for calculation of a chronic oral MRL from a population exposed only to methylmercury by a relevant route of exposure for the overall U.S. population.

Again, ATSDR would like to strongly emphasize that both the Seychelles study and the Faroese study represent credible scientific contributions by widely respected research teams. Similarly, both studies extend our knowledge base well beyond that provided by the Iraqi study and make significant contributions to our understanding of the effects of low-level exposure to methylmercury by an exposure route and vehicle (i.e., food) relevant to U.S. populations. The continuing monitoring and evaluation of the Seychellois and Faroese populations with more comparable neurobehavioral indices should help strengthen our understanding of the effects of low-level chronic methylmercury exposure and should reduce the uncertainty regarding the public health implications of exposure.

Other Key Studies Reviewed by ATSDR

Other epidemiology studies were also considered by ATSDR in evaluating the database on human exposure to methylmercury. Lebel et al. (1996) evaluated a fish-eating populations in the Amazon River Basin with a neurofunctional test battery and clinical manifestations of nervous system dysfunction in relation to hair mercury concentrations. The villagers examined live along the Tapajos River, a tributary of the Amazon. The study population consisted of 91 adult inhabitants 15–31 years of age. Hair mercury levels were below 50 µg/g (ppm). Clinical examinations were essentially normal, although persons displaying disorganized movements on an alternating movement task and those with restricted visual fields generally had higher hair mercury levels. Near visual contrast, sensitivity, and manual dexterity (adjusted for age) were found to decrease significantly with increasing mercury levels, while a tendency for muscular fatigue and decreasing strength were observed in women. The authors suggested that dose-dependent nervous system alterations might be associated with hair mercury levels below 50 ppm. This study, however, also had a number of potentially confounding factors. The impact of parasitic and other diseases endemic to the study area is of primary concern in the interpretation of the Lebel et al. (1996) results. In addition, the overall nutritional status of the study population was not known or reported, and the use of neuroactive drugs (from local herbs, plants, roots, or mushrooms) was not considered as a potential confounder or covariate. The previous mercury exposure history of the study cohort is also unclear. This is of particular importance because gold mining procedures that use metallic mercury have been commonly practiced along the Amazon Basin for decades. Finally, the end points of the Lebel et al. (1977) study evaluated adult toxicity and not effects in the developing fetus or the newborn (i.e., the most sensitive human population).

Myers et al. (1997) evaluated the population of the SCDS for developmental milestones similar to those determined in Iraq. As part of this ongoing study, cohort children were evaluated at 6.5, 19, 29, and 66 months of age. At 19 months care-givers were asked at what age the child walked (n=720 out of 738) and talked (n=680). Prenatal mercury exposure was determined by atomic absorption analysis of maternal hair segments corresponding to hair growth during the pregnancy. The median mercury level in maternal hair for the cohort in this analysis was 5.8 ppm, with a range of 0.5–26.7 ppm. The mean age (in months) at walking was 10.7 (SD=1.9) for females and 10.6 (SD=2.0) for males. The mean age for talking (in months) was 10.5 (SD=2.6) for females, and 11 (SD=2.9) for males. After adjusting for covariates and statistical outliers, no association was found between the age at which Seychellois children walked or talked and prenatal exposure to mercury. The ages for achievement of the developmental milestones were normal for walking and talking in the Seychellois toddlers following prenatal exposure to methylmercury from a maternal fish diet.

Clarkson (1995) raised some interesting issues concerning whether is it reasonable to apply health effects data based on an acute exposure to methylmercury fungicide eaten in homemade bread (in the 1971–1972 Iraq incident) to fish-eating populations having chronic exposure to much lower concentrations of methylmercury. He addressed two specific issues. The first regards the body's "defense mechanisms" that serve to mitigate the potential damage from mercury. One such mechanism in the case of methylmercury involves an enterohepatic cycling process in which methylmercury from dietary sources absorbed through the intestine is carried to the liver, where substantial quantities are secreted back into the bile and returned to the intestinal tract. During the residence time in the gut, microflora break the carbon-mercury bond, converting methylmercury into inorganic mercury, which in turn is poorly absorbed and is excreted in the feces. This creates an effective detoxification pathway for low-dose dietary exposures to methylmercury, but probably not for acute, high-dose exposures, such as occurred in Iraq. Secondly, the transport of methylmercury into brain tissue is inhibited by the presence of many amino acids, including leucine, methionine, and phenylalanine. Thus, it is possible that the rising plasma concentrations of amino acids from ingestion of fish protein may serve to depress the uptake of methylmercury by the brain.

While both of these issues need further laboratory/clinical investigation, they do raise appropriate questions concerning the relevance of the relatively short-term (i.e., about 6 weeks), high-level contaminated grain exposure scenario encountered in Iraq to the dietary methylmercury exposure scenarios encountered in many fish-eating populations (e.g., the Seychelles Islanders, Faroe Islanders, Peruvian villagers, and Inuit native people of Greenland). This position is supported by Cicmanec (1996), who reviewed data from the

Iraqi study, as well as data from studies of fish-consuming populations in the Faroe Islands, Seychelles Islands, and Peruvian fishing villages. Cicmanec concluded that the Iraqi population does not represent a sensitive subpopulation within a perinatal group; rather, the relatively lower threshold identified in that study was the result of confounders. Crump et al. (1995) reanalyzed the dose-response data from the Cox et al. (1989) report of the Iraqi incident and found the results to be potentially skewed by inadequacies in the study design and data-collection methods. Shortcomings or potentially confounding factors include: (1) the retrospective recall of developmental milestones by mothers and other family members; (2) the lack of precision in the determination of birth and other milestone dates; (3) and the possible biasing of the dose-response analysis by variation in symptom reporting and infant sex composition in the two study subcohorts. Crump et al. (1995) noted that perhaps the most serious limitation of the Iraqi study is the inability to assess the potential effects of low-level chronic-duration exposure to methylmercury, as these particular data are based on very high intake levels over a relatively brief period of time.

No increase in the frequency of neurodevelopmental abnormalities in early childhood was observed in a cohort of 131 infant-mother pairs in Mancora, Peru (Marsh et al. 1995b). The mean concentration of mercury in maternal hair was determined to be 8.3 ppm (range, 1.2–30 ppm), and the source of the mercury was believed to be from consumption of marine fish. Similarly, a study of 583 Faroe Island infants for the first 12 months after birth found no decrease in the age of attainment of sitting, creeping (crawling), and standing developmental milestones (Grandjean et al. 1995a). The age at which a child reached a particular developmental milestone was not only not found to be associated with prenatal mercury exposure, but infants that reached a milestone early were found to have significantly higher mercury concentrations in their hair at 12 months of age. It was also found that early milestone attainment was clearly associated with breast-feeding, which was in turn related to higher infant hair mercury levels. The authors (Grandjean et al. 1995a) concluded that the beneficial effects associated with breast-feeding seemed to overrule, or to compensate for, any neurotoxic effects on milestone development that could be due to the presence of contaminants (e.g., mercury) in human milk.

Additional studies have shown developmental toxicity after oral exposure of humans and animals to organic mercury compounds (Amin-Zaki et al. 1974; Bakir et al. 1973; Bornhausen et al. 1980; Cagiano et al. 1990; Elsner 1991; Engleson and Herner 1952; Fowler and Woods 1977; Guidetti et al. 1992; Harada 1978; Hughes and Annau 1976; Ilback et al. 1991; Inouye and Kajiwara 1988; Khera and Tabacova 1973; Lindstrom et al. 1991; McKeown-Eyssen et al. 1983; Nolen et al. 1972; Olson and Boush 1975; Rice 1992; Rice and Gilbert 1990; Snyder and Seelinger 1976; Stoltenburg-Didinger and Markwort 1990).

The accumulation of mercury is greater in larger fish and in fish higher in the food chain. The tendency for increased mercury concentration with increasing fish body weight is particularly noticeable in carnivorous fish species. Malm et al. (1995) analyzed mercury concentrations in 16 species of carnivorous fish from the Tapajos River basin in Brazil and hair samples from local populations who regularly ate such fish. Mercury levels in the fish averaged 0.55 ppm (range, 0.04–3.77 ppm), and the mercury levels in the hair of the affected fish-eating populations averaged approximately 25 ppm. In one population that consumed higher quantities of large carnivorous fish at the end of the local rainy season, 8 of 29 persons evaluated had hair mercury levels above 40 ppm, and one individual had a hair mercury concentration of 151 ppm. Some villages along the river can have per capita daily fish consumption rates around 200 g or more, which would greatly impact the human body burden and hair levels of mercury in such populations.

Hair-to-Blood Concentration Ratio

The hair:blood concentration ratio for total mercury is frequently cited as 250. However, a precise basis for this particular value is unclear. Ratios reported in the literature range from 140 to 416, a difference of more than a factor of 2.5 (see Table 2-9). Differences in the location of hair sampled (head versus chest, distance of sample from head or skin) may contribute to differences in observed ratios between studies. For example, as much as a 3-fold seasonal variation in mercury levels was observed in average hair levels for a group of individuals with moderate-to-high fish consumption rates, with yearly highs occurring in the fall and early winter (Phelps et al. 1980; Suzuki et al. 1992). Thus, it is important to obtain hair samples as close to the follicle as possible to obtain an estimate of recent blood levels. Large errors (the direction of which depends on whether samples were taken while blood levels were falling or rising) could result if hair samples are not taken close to the scalp. Several studies did not report the distance to the scalp for the hair samples taken. The high slope reported by Tsubaki (1971a) may have reflected the fact that mercury levels were declining at the time of sampling (Berglund et al. 1971), so the hair levels may reflect earlier, higher blood levels. Hair taken from different parts of the body also may yield different ratios. In 26 subjects with moderate-to-high fish consumption, axillary hair (i.e., from the armpit area) was found to contain an average of 23% less mercury than head hair (Skerfving et al. 1974).

Phelps et al. (1980) obtained multiple blood samples and sequentially analyzed lengths of hair from 339 individuals in Northwestern Ontario. The large sample size and the attention to sampling and analysis with regard to the hair:blood relationship make the results from this study the most appropriate to use for estimating the mercury blood levels of the Seychellois women during pregnancy. The actual ratio Phelps et

al. (1980) observed between the total mercury concentration in hair taken close to the scalp and simultaneous blood sampling for this group was 296. To estimate the actual ratio, the authors assumed that blood and hair samples were taken following complete cessation of methylmercury intake. They also assumed a half-life of methylmercury in blood of 52 days and a lag of 4 weeks for appearance of the relevant level in hair at the scalp. Based on these assumptions, they calculated that if the actual hair:blood ratio were 200, they would have observed a ratio of 290 (i.e., essentially equivalent to the observed value of 296). Based on these and other considerations, Phelps et al. (1980) state that the actual ratio is "probably higher than 200, but less than the observed value of 296." As the authors point out, two-thirds of the study population were sampled during the falling phase of the seasonal variation and one-third or less in the rising phase. This fact would tend to result in a lower observed ratio; therefore, the actual average value is likely to be >200.

Phelps et al. (1980) also provide estimates assuming a 2-week lag for the appearance of the relevant level of mercury in the centimeter of hair nearest the scalp. For a 2-week lag time, an actual ratio of 250 would have resulted in an observed ratio of 301 (again, essentially identical to the observed value of 296). A study of ingestion of a large dose of mercuric chloride in one individual suggests that the lag time is longer than 2 weeks (Suzuki et al. 1992). Hair samples were taken at 41 and 95 days following ingestion of the mercuric chloride. In the 41-day hair sample, a large mercury peak occurred in the centimeter of hair closest to the scalp, with no elevation in mercury in the second centimeter of hair. Head hair grows at a rate of about 1.1 cm a month (Al-Shahristani and Shihab 1974; Cox et al. 1989). If emergence had occurred so that the elevation in mercury could be measured in the first centimeter of hair by 2 weeks after exposure, then by day 41 after exposure the peak should have moved into the second centimeter of hair, at least enough to raise the mercury level slightly in the second centimeter. Because no elevation was seen in the second centimeter of hair at 41 days, it would appear that emergence occurred at a lag of >2 weeks. In the hair sample taken at 95 days, the leading edge of the mercury peak occurred in the third centimeter of hair.

Based on the data presented in Phelps et al. (1980) and the lag time indicated in the individual studied by Suzuki et al. (1992), the actual average value is likely to be somewhere between 200 and 250. Because the data do not allow a more accurate determination of an average ratio, the value 250 is acceptable for the purpose of estimating average blood levels in the Seychellois population. Using 250 rather than a lower number results in a lower MRL. It should be noted that a wide range in hair:blood ratios has been reported for individuals in various studies: 137–342 in Soria et al. (1992), 171–270 in Phelps et al. (1980), 416 in

Cernichiari et al. (1995), and 137–585 in Birke et al. (1972). Therefore, this ratio (250) should not be used as the sole basis for determining levels of exposure and potential effect for individuals.

Calculation of dietary intake of mercury from blood concentration.

Fraction of mercury in diet that is absorbed (A_D). Radiolabeled methyl-mercuric nitrate was administered in water to three healthy volunteers (Aberg et al. 1969). The uptake was >95%. Miettinen et al. (1971) incubated fish liver homogenate with radiolabeled $MeHgNO_3$ to yield a methylmercury proteinate. The proteinate was then fed to fish that were killed after a week, cooked, and fed to volunteers after confirmation of the methylmercury in the fish. Mean uptake exceeded 94%. For the derivation of an MRL, an absorption factor of 0.95 is used.

Fraction of the absorbed dose that is found in the blood (A_B). The value 0.05 has been used for this parameter in the past (Berglund et al. 1971; WHO 1990). Three studies report observations of the fraction of the absorbed methylmercury dose distributed to blood volume in humans. Kershaw et al. (1980) report an average fraction of 0.059 of the absorbed dose in the total blood volume, based on a study of 5 adult male subjects who ingested methylmercury-contaminated tuna. In a group of 9 male and 6 female volunteers who had received ^{203}Hg-methylmercury in fish, approximately 10% of the total body burden was present in 1 L of blood in the first few days after exposure, dropping to approximately 5% over the first 100 days (Miettinen et al. 1971). In another study, an average value of 1.14% for the percentage of absorbed dose in 1 kg of blood was derived from subjects who consumed a known amount of methyl-mercury in fish over a period of 3 months (Sherlock et al. 1984). Average daily intake for the 4 groups observed in the study ranged from 43 to 233 µg/day. The authors report a dose-related effect on the estimated percentage of the absorbed dose in 1 kg of blood, with 1.26% of the absorbed dose in 1 kg of blood at an average daily intake of 43 µg/day and 1.03% of the absorbed dose in 1 kg of blood at an average daily intake of 233 µg/day. The average for all subjects in the study was 1.14%. When individual values for distribution to one kilogram of blood reported in the study are converted into the percentage of the absorbed dose in the total blood volume (assuming that blood is 7% of body weight [Best 1961] and using body weights reported for individuals in the study), the average value for A_B for all individuals is 0.056 (0.057 using the values for percentage in 1 kg normalized for body weight as reported in the study). The average value for A_B for 6 women as reported in Sherlock et al. (1984) is 0.048 (0.047 using values normalized for body weight). The average for 14 men is 0.059 (0.061 using values normalized for body weight).

The average values for A_B for all studies ranged from 0.047 to 0.061 (the values for women and men reported in Sherlock et al. [1984]). The data suggest that the average value of A_B for women may be lower than that for men, and they further suggest that 0.05 may be appropriate for modeling intake in a group of women (Sherlock et al. 1984). Based on these studies, the best estimate of A_B based on the available data is 0.05. Use of a higher value (i.e., 0.06 instead of 0.05) for this parameter would result in a lower MRL, but the sensitive populations are pregnant women and developing fetuses, making the 0.5 value more appropriate for the Seychelles study population.

Elimination constant (b). Reported clearance half-times for methylmercury from blood or hair range from 48 to 65 days (Table 2-5). The average elimination constant based on the six studies listed in Table 2-5 is 0.014. The average of the individual values for *b* reported for 20 volunteers ingesting 42–233 µg Hg/day in fish for 3 months (Sherlock et al. 1984) is also 0.014. Use of the value 0.014 for this parameter, rather than 0.01 (as used by WHO 1990), results in a higher MRL.

Volume of blood in body (V), and body weight. Blood volume is assumed to be 7% of body weight, with an increase to about 9% during pregnancy (Best 1961). Data for the body weight of the Seychelles Islands women were not found. Assuming an average body weight of 60 kg for women, the blood volume is 4.2 L (60 kg x 0.07 L/kg). The 9% of body weight value is not used because it is not representative of the blood volume throughout pregnancy. Blood volume does not begin to increase significantly from the 7% pre-pregnancy level until around the 27th week of pregnancy. It then sharply rises until week 40 or parturition (Guyton 1996). To use the 9% value would, therefore, be representative of the blood volume late in pregnancy (i.e., mid- to late- third trimester), but not throughout most of pregnancy. In contrast, the hair mercury level to which it is compared represents an average value throughout pregnancy. The use of the 9% value would result in a higher MRL, and is not considered appropriate in this instance.

Calculation of Exposure Dose

The concentration of mercury in hair is assumed to be 250 times the concentration in blood. ATSDR's peer-reviewed, published guidance for MRL derivation (Chou et al. 1998) calls for the use of the highest value at which no adverse effects were observed in the critical study. Using, therefore, the 15.3 ppm mean maternal hair (taken at parturition) value from the highest exposure group (range, 12–26.7 ppm) in the Seychellois test population as a NOAEL for the 66-month Seychelles testing (Davidson et al. 1998), the corresponding methylmercury concentration in blood would be: 1/250 x 15.3 µg/g x 1 mg/1,000 µg x 1,000 g/L = 0.061 mg/L.

Converting blood mercury concentration to daily intake.

The concentration of mercury in the blood may be converted to a daily intake by using the following equation from WHO (1990):

$$C = \frac{f \cdot d}{b \cdot V} \cdot \frac{A_D \cdot A_B \cdot d}{b \cdot V}$$

Where:

C	=	concentration in blood
f	=	fraction of the daily intake taken up by the blood
d	=	daily dietary intake
b	=	elimination constant
A_D	=	percent of mercury intake in diet that is absorbed
A_B	=	percent of the absorbed amount that enters the blood
V	=	volume of blood in the body

where:

C = (0.95 x 0.05 x d)/(0.014 x 4.2)

C = 0.81 d

0.061 mg = 0.81 d

d = 0.075 mg/day

Using the assumed body weight of 60 kg for women, the estimated dose that would result in a hair level of 15.3 ppm is 0.075/60 kg = 0.0013 mg/kg/day. Therefore, the NOAEL derived from the highest exposure group (n = 95) at 66 months is 0.0013 mg/kg/day.

Consideration of Uncertainty

The standard/traditional areas of uncertainty addressed in any duration-specific MRL are: (1) interspecies variability (i.e., cross-species extrapolation of a NOAEL or LOAEL); (2) intra-human variability (i.e., differences in susceptibility to a substance or effect within the human population); (3) use of an LOAEL for MRL derivation when an NOAEL for the critical effect is not available; and (4) extrapolation from subchronic to chronic duration. In addition, a modifying factor may also be used when special circumstances exist that may contribute to, or introduce, uncertainty into the calculated health guidance value (MRL) in an area not typically covered by the traditional uncertainty factor approach.

The NOAEL of 15.3 ppm mercury in maternal hair from Davidson et al. (1998) used as the starting point for MRL derivation was based upon an unusually large study cohort of the population considered most sensitive to the neurodevelopmental effects of methylmercury, i.e., pregnant women and their developing fetuses. The negative results of this study are strongly supported by the BMD NOAEL range of 13 to 21 ppm calculated for the New Zealand cohort of 237 mother-child pairs (Crump et al. 1998). Consequently, much of the uncertainty normally present in the MRL derivation process does not exist in the case of methylmercury. Nonetheless, in view of the nature of the most susceptible group (developing fetuses) and some questions raised in the vast human data base for this chemical, an aggregate value of 4.5 was employed.

This value (4.5) was based upon three separate components, two of which are interrelated and the other independent. For the Seychelles data, a value of 1.5 was used to address variability in hair-to-blood ratios among women and fetuses in the U.S. population, as determined by pharmacokinetic modeling of actual data by Clewell et al. (1998); a second value of 1.5 was applied to address the remainder of any inter-individual variability (i.e., pharmacodynamics) in the U.S. population. A third, and independent, factor of 1.5 was employed to account for the possibility that the domain-specific tests, as employed extensively in the Faroe Islands, but not the Seychelles (which used primarily neurobehavioral tests of global function) might be able to detect very subtle neurological effects not tested for in the 66-month Seychelles cohort.

The World Health Organization (WHO, 1993, 1996) has defined the -kinetic and -dynamic components of intrahuman variability as being equal contributors to, and collectively constituting the total of, human variability. In order to assure a conservative approach, these two interdependent components were added to give a composite uncertainty factor of three (i.e., 1.5 + 1.5 = 3) to account for the full range of variability attributable to mercury in the Seychelles study. A modifying factor of 1.5 was also used to account for the possibility of domain-specific effects, as were seen in the Faroe study, being attributable to mercury. Since these effects were considered to be entirely separate or “independent” events, this modifying factor of 1.5 was multiplied by the uncertainty factor of 3.0 (for uncertainty attributable solely to the Seychelles study) to yield an aggregate uncertainty of 4.5 for chronic oral exposure to methylmercury.

While domain-specific tests from the Seychelles were reviewed at the North Carolina meeting in November 1998 and the results failed to demonstrate effects, the tests do not represent the full range of domain-specific tests that were administered in the Faroe Islands. For these reasons, and based on our consultation with our Board of Scientific Counselors about concerns for “missing” data sets (i.e., in relation to the Executive Order of children’s health and the agency’s efforts to protect the health of children, including the developing fetus), ATSDR determined that an additional factor of 1.5 should be used since the full range of

domain-specific neuropsychological test results from the Seychelles are not yet available. When these results become available and if they fail to show domain-specific effects, this additional factor of 1.5 would no longer be needed. At that time ATSDR will re-evaluate its MRL, as well as all other relevant data, in compliance with the agency's mandates and authorities.

Therefore, in the calculation of the chronic oral MRL for methylmercury, the NOAEL of 0.0013 mg/kg/day from the 66-month study (Davidson et al. 1998) is divided by 4.5, giving a chronic oral MRL for methylmercury of 0.0003 mg/kg/day [0.0013 mg/kg/day / 4.5 (UF) = 0.0003 mg/kg/day].

Alternative Derivations of the MRL

To ensure a health guidance value based upon the best use of the Seychelles study data (widely considered the most relevant data available), ATSDR evaluated alternate MRL derivation methods for methylmercury. One such approach is to use the mean total mercury level of 6.8 ppm in maternal hair for the entire Seychellois study cohort. Using the same formula as in the previous MRL calculation,

C = (0.95 x 0.05 x d) / (0.014 x 4.2)
C = 0.81 d
(1/250 x 6.8) = 0.027
0.027 mg/L = 0.81 d
d = 0.034 mg/day
0.034 mg/day / 60 kg = 0.0006 mg/kg/day

In consideration of uncertainty factors for this MRL approach, multiple factors also apply. In this case, the mean value of 6.8 ppm for the NOAEL is for the entire study cohort at 66 months (n = 711). An uncertainty factor of 1.5 was used to account for the pharmacokinetically based variability of hair-to-blood ratios (95% confidence level) in pregnant women and fetuses in the U.S. population (Clewell et al. 1998, 1999). The extremely large size of the study population (n=711), in combination with an uncertainty factor of 1.5, is considered adequate to encompass the full range of pharmacokinetic and pharmacodynamic variability within the human population. An independent modifying factor of 1.5 was also used to take into consideration the positive results of the domain-specific tests administered in the Faroe study (Grandjean et al. 1997, 1998). The uncertainty factor of 1.5, multiplied by the modifying factor of 1.5, yields a total aggregate value of 2.25. Applying the factor of 2.25 to the daily intake calculated from the 6.8 ppm

NOAEL yields a chronic oral MRL value of 0.0003 mg/kg/day for methylmercury (0.0006 mg/kg/day divided by 2.25 = 0.0003 mg/kg/day).

A third approach to deriving a health guidance value is the use of bench mark dose (BMD) modeling. Clewell et al. (1998) used a benchmark dose analysis to determine a reference dose (RfD, a health guidance value used by the Environmental Protection Agency and, in some ways, the equivalent of ATSDR's chronic oral MRL). Clewell et al. (1998) used the data from the 29-month test in the Seychellois population (Davidson et al. 1995b) for their analysis (i.e., the 66-month study had not been published at the time of their benchmark dose analysis). The BMD is calculated by fitting a mathematical dose-response model to dose-response data. The bench mark dose level (BMDL) is a lower statistical confidence bound on the BMD and replaces the NOAEL in the calculation of a health guidance value. The BMD approach has been proposed as superior to the use of "average" or "grouped" exposure estimates when dose-response information is available, as is the case for the Seychelles study. Clewell et al. (1998) note that the Faroe Islands study reported by Grandjean et al. (1997b) could not be used for dose-response modeling due to inadequate reporting of the data and the confounding influence of co-exposure to PCBs.

For the 29-month Seychelles data, Clewell et al. (1998) used the 95% lower bound on the 10% benchmark dose level (BMDL), which represents a conservative estimate of the traditional NOAEL. The benchmark dose modeling over the entire range of neurological endpoints reported by Davidson et al. (1995b) yielded a lowest $BMDL_{10}$ of 21 ppm methylmercury in maternal hair. This $BMDL_{10}$ was then converted to an expected distribution of daily ingestion rates across a population of U.S. women of child-bearing age by using a Monte Carlo analysis with a physiologically based pharmacokinetic (PBPK) model of methyl-mercury developed by Gearhart et al. (1995). This analysis addresses the impact of interindividual pharmacokinetic variability on the relationship between ingestion rate and hair concentration for methyl-mercury. The resulting distribution had a geometric mean value of 0.00160 mg/kg/day (S.D. 0.00133). The 1st, 5th, and 10th percentiles of that distribution were 0.00086, 0.00104, and 0.00115 mg/kg/day, respectively. Clewell et al. (1998) suggested that the 5th percentile of 0.00104 mg/kg/day provides a scientifically based, conservative basis that incorporates the pharmacokinetic variability across the U.S. population of child-bearing women and that no other uncertainty factor for interindividual variability would be needed. To the benchmark-estimated NOAEL of 21 ppm derived from the Seychelles 29-month data, Clewell et al. (1998) applied an uncertainty factor of 3 to account for data base limitations. (Note: The 66-month Seychelles data was not yet published at the time; hence the reliance on the 29-month Seychelles

data for the benchmark analysis.) Consequently, Clewell et al. (1998) concluded that using a NOAEL of 7 ppm (21 ppm / 3 (UF) provides additional protection against the possibility that effects could occur at lower concentrations in some populations. Based upon this reasoning, Clewell and his colleagues recommended a health guidance value (i.e., an RfD) of 0.0004 mg/kg/day. If a modifying factor of 1.5 is used to further address the domain-specific findings in the Faroe study, a final MRL of 0.3 µg/kg/day results.

The above benchmark analysis of 29-month data from the Seychelles Child Development Study strongly supports the MRL of 0.0003 mg/kg/day calculated by ATSDR in this profile. Similarly, addressing the Seychellois 66-month data from the perspective of using the mean value (15.3 ppm) of the highest exposure group in the study, a method prescribed in ATSDR's published guidance for MRL development (Chou et al. 1998), also results in an identical MRL. ATSDR therefore has high confidence that this level is protective of the health of all potentially exposed human populations.

Employment of the Chronic Oral MRL for Methylmercury

It should be emphasized that the MRL is considered by ATSDR to be a level of exposure to a single chemical/substance which is considered "safe" for all potentially exposed populations for a specified duration of time (acute, intermediate, or chronic). It is not considered be a threshold for adverse effects, and not address the likelihood of adversity at any incremental level above the MRL value. ATSDR notes that the 0.3 µg/kg/day chronic oral MRL for methylmercury is in close agreement with the tolerable daily intake (ADI) levels of 0.47 and 0.48 µg/kg/day established by the FDA and WHO, respectively.

MRLs are, by definition (Chou et al. 1998), substance-specific and do not include effects attributable to interaction (whether additive, synergistic, or antagonistic) with other chemicals or environmental substances. Their relevance to the mission of ATSDR is to assist public health officials in the identification of chemicals/elements of potential health concern at hazardous waste sites. The ATSDR MRL is not intended to be used in the regulatory or site clean-up process, but is instead intended to serve as a basis of comparison with actual measured levels of environmental exposure. Further, the role of informed biomedical judgment is crucial in the application of any MRL, or the media-specific health guidance values (HGVs) derived from them, in any given exposure scenario (Risher and De Rosa 1997). MRLs for a particular substance are based upon the most sensitive effect/endpoint in that portion of the human population considered to be most susceptible to injury from exposure to that substance. Thus, the MRL has never been intended as a one-size-fits-all tool for all hazardous waste site exposure scenarios; rather, it is merely a starting point for further examination of potential health risk. Therefore, at sites

where methylmercury is present in combination with other known or suspected neurodevelopmental toxicants, such as lead or polychlorinated biphenyls (PCBs), and in which exposure is primarily episodic in nature, the health assessor might consider using a value below the chronic oral MRL for methylmercury as a starting point for determination of further site investigation. (A more complete description of the uses of MRLs and other HGVs can be found in Chou et al. 1998 and Risher and De Rosa 1997.)

Background and general population exposures relevant to the oral MRL for methylmercury

Mercury hair levels have been monitored in a variety of populations and generally range from 1 to 4 ppm, depending upon the level of fish consumption. Table 2-10 summarizes the mean (or median) values and the maximum value from a number of these studies.

Diet. Based on the FDA total diet study of 1982–1984 (Gunderson 1988), FDA estimated that the average intake for total mercury (both inorganic and organic) is 50–100 ng/kg/day. Based on the more recent 1989–1990 FDA total diet study, the estimated intake of total mercury is 27–60 ng/kg/day (Cramer 1994). An estimated 86% of the mercury in the total diet study is derived from fish (Tollefson and Cordle 1986). A separate estimate of the average intake of methylmercury alone, based on a survey of fish eaters and average levels of methylmercury in fish, places the average intake of methylmercury at 36 ng/kg/day, with a 99% upper bound at 243 ng/kg/day (Clarkson 1990).

Potential protective effect of selenium in fish. Selenium is known to bioconcentrate in fish, and selenium has been observed to correlate with mercury levels in the blood of fish consumed (Grandjean et al. 1992). Furthermore, there is evidence suggesting that consumption of methylmercury from fish, in conjunction with other beneficial constituents in fish (e.g., omega-3 fatty acids) may not result in the same toxicity dose-response relationship observed with methylmercury exposure from consumption of contaminated grain (as in the Iraqi population) (Davidson et al. 1998).

Regarding the bioavailability of methylmercury in fish, the available data indicate that methylmercury uptake is not affected by its presence in fish. Experimental studies on the metabolism of methylmercury in humans following the ingestion of contaminated fish (using methylmercury bound to fish muscle protein) have shown that absorption is almost complete (95% absorbed) (Miettinen 1973). Animal studies also support this absorption value. Data on cats given fish homogenates indicate absorptions of $90% of methylmercury, whether added to the homogenate, accumulated by fish *in vivo*, or from methylmercury proteinate (Berglund et al. 1971). Using blood and tissue levels as evidence of absorption, Charbonneau et al. (1976) concluded that there was no difference in the biological availability of methylmercury

administered to adult cats (0.003, 0.0084, 0.020, 0.046, 0.074, or 0.176 mg Hg/kg/day, 7 days a week for 2 years), either as pure methylmercuric chloride in corn oil added to a diet containing uncontaminated fish or as methylmercury-contaminated fish. In the two highest dose groups (0.074 and 0.176 mg Hg/kg body weight at 100 weeks of exposure), no significant differences were seen in total mercury concentrations in the blood between groups receiving the dose as methylmercuric chloride or as contaminated fish at the same dose level. In addition, monthly blood levels were comparable for all dose groups. No significant differences were seen at 100 weeks in total mercury concentrations in nervous tissue or other tissues (renal cortex, renal medulla, liver, spleen, adrenal, bladder, atria, ventricle, ovary, testis, muscle) between the two highest dose groups receiving the dose as methylmercuric chloride or as contaminated fish at the same dose level.

Regarding the effect of selenium on methylmercury toxicity, most studies have shown that the simultaneous administration of mercury and selenium in equimolar doses to animals has resulted in decreased toxicity of both elements for acute- and chronic-duration exposures. This effect has been observed with inorganic and organic mercury and with either inorganic or organic selenium compounds, although inorganic forms of selenium appear to be more effective than organic forms (Chang 1983; Skerfving 1978). Selenium protects against the acute nephrotoxicity of the mercuric ion and methylmercuric ion in rats (Ganther et al. 1972, 1980; Hansen 1988; Magos et al. 1987; Parizek and Ostadolva 1967) and possibly against acute neurotoxicity of methylmercuric ion in rats (Ohi et al. 1980).

Somewhat paradoxically, the protective effect of selenium has been associated with a higher whole-body retention of mercury (Hansen 1988; Magos et al. 1987). In a study of selenium excretion in workers exposed to low levels of metallic mercury vapor in a chloralkali plant, Ellingsen et al. (1995) found that even in a low-to-moderate occupational exposure, mercury may reduce the urinary selenium concentration in humans in a manner that is not yet fully known. Evidence from human autopsy tissues suggests that distribution of mercury throughout the body may be influenced by the presence of selenium (Suzuki et al. 1993). In this study, however, the level of selenium was found to negatively correlate with the level of mercury in some tissues including the cerebrum, spleen, and kidney cortex. Suzuki et al. (1993) also report that hair selenium values negatively correlated with total organ mercury and inorganic mercury levels. The association between concentrations of inorganic mercury and selenium in both the occipital lobe and the thalamus of the brains of methylmercury-exposed female monkeys was reported by Bjorkman et al. (1995). These authors observed a tendency to a "hockey stick-shaped" relationship between concentrations of selenium and inorganic mercury in the thalamus of monkeys with ongoing exposure to methylmercury, and

they postulated an important role for selenium in the retention of mercury in the brain. These studies indicate that selenium has an effect on mercury toxicokinetics, although more study is needed to determine the nature of the interaction with respect to different organs and exposure regimens.

Although the specific mechanism for the protection is not well understood, possible mechanisms for selenium's protective effect include redistribution of mercury (Mengel and Karlog 1980), competition by selenium for mercury-binding sites associated with toxicity, formation of a mercury-selenium complex that diverts mercury from sensitive targets (Hansen 1988; Magos et al. 1987; Naganuma and Imura 1981), and prevention of oxidative damage by increasing selenium available for the selenium-dependent glutathione peroxidase (Cuvin-Aralar and Furness 1991; Imura and Naganuma 1991; Nylander and Weiner 1991). One laboratory study showed that selenium-treated animals can remain unaffected, despite an accumulation of mercury in tissues to levels that are otherwise associated with toxic effects (Skerfving 1978). Support for the proposal that an inert complex is formed comes from the 1:1 ratio of selenium and mercury found in the livers of marine mammals and in the bodies of experimental animals administered compounds of mercury and compounds of selenium, regardless of the ratio of the injected doses (Hansen 1988).

Southworth et al. (1994) evaluated the elimination of slurried fly ash discharges into a water-filled quarry and found that the discharge was followed by a steady increase in concentrations of mercury in the axial muscle of resident largemouth bass *(Micropterus salmoides)*, increasing from 0.02 to 0.17 μg/g in a period of just 3 years. These authors also found that while selenium concentrations in the quarry decreased from 25 to <2 μg/L during the same period, selenium concentrations in bass remained at about 3 times the background levels. Southworth and his co-authors concluded that the results of their study suggest that selenium may also be effective at blocking the accumulation of methylmercury in harder, more alkaline waters.

SPECIFIC ADVERSE EFFECTS ATTRIBUTABLE TO MERCURY EXPOSURE

Death. Inhalation of sufficiently high concentrations of metallic and organic mercury vapors, ingestion of sufficiently high doses of organic and inorganic mercury, and exposure to dialkyl mercurials by any route can be fatal to humans and animals. In the cases of both inhalation and dermal exposure to dialkyl-organomercurials (e.g., diethyl- and dimethylmercury), acute exposures that appear innocuous or unremarkable at the time of exposure can result in death following a delay period of weeks or months. The tragic case of a delayed neurotoxicity and ultimately fatal poisoning 9 months after an acute dermal

exposure to only a few drops of dimethylmercury is striking example of the danger of these forms of organic mercury (Blayney et al. 1997; Nierenberg et al. 1998). At least 5 other deaths have been reported due to alkyl mercury exposure since its first synthesis in the mid-19th century (Toribara et al. 1997). These accidental poisoning cases also reveal a latency period of some months between the exposure and the onset of symptoms. In such cases, irreversible brain damage has already occurred by the time the first symptoms appeared.

No information was located regarding specific concentrations of elemental mercury vapor that may be lethal; however, lethal exposures have generally occurred as a result of exposure under conditions in which exposure levels are likely to be quite high (e.g., heating metallic mercury in a closed space). Death in these cases has generally been attributed to respiratory failure (Campbell 1948; Kanluen and Gottlieb 1991; Matthes et al. 1958; Rowens et al. 1991; Soni et al. 1992; Taueg et al. 1992; Teng and Brennan 1959; Tennant et al. 1961). Deaths resulting from inhalation exposure to organic mercury compounds have also been reported (Brown 1954; Hill 1943; Hook et al. 1954; Lundgren and Swensson 1949). Although the cause of death following inhalation of organic mercury was not reported, severe neurological dysfunction was observed prior to death.

Lethal doses for acute oral exposure to inorganic mercury have been estimated to be 29–50 mg Hg/kg (Troen et al. 1951). Deaths resulting from oral exposure to inorganic mercury have been attributed to renal failure, cardiovascular collapse, and severe gastrointestinal damage (Gleason et al. 1957; Kang-Yum and Oransky 1992; Troen et al. 1951).

Deaths from consumption of methylmercury-contaminated foods are well documented in outbreaks in Japan and Iraq, and lethal doses of 10–60 mg Hg/kg have been estimated from tissue concentrations (Bakir et al. 1973; Tsubaki and Takahashi 1986). Fatalities were attributed to central nervous system toxicity (Bakir et al. 1973; Tamashiro et al. 1984). Pneumonia and nonischemic heart disease were prominent secondary causes of death in the Japan epidemic (Tamashiro et al. 1984). Case reports of deaths associated solely with dermal mercury exposure to inorganic mercury are limited to a woman who died after inserting a mercuric chloride tablet into her vagina (Millar 1916) and a man who died after a 2-month treatment with a topical medicine containing mercurous chloride (Kang-Yum and Oransky 1992). Death was attributed to renal failure in one of these cases (Kang-Yum and Oransky 1992), and severe renal damage was noted at the autopsy in the other (Millar 1916).

Animal data support the findings from human studies and provide somewhat more information regarding lethal exposure levels. Deaths associated with acute-duration inhalation exposure to metallic mercury vapor have been observed in rats and rabbits at about 27–29 mg/m^3 (Ashe et al. 1953; Livardjani et al. 1991b). Severe pulmonary edema has been reported as the cause of death following such exposures (Christensen et al. 1937). Severe ataxia occurred prior to death in rats exposed to methylmercury iodide vapor for intermediate durations (Hunter et al. 1940). Acute oral LD_{50} values for inorganic mercury ranged from 25.9 to 77.7 mg Hg/kg, with the most sensitive LD_{50} for 2-week-old rats (Kostial et al. 1978). Increased mortality in chronic-duration oral studies has been observed at 1.9 mg Hg/kg in male rats gavaged 5 days a week (NTP 1993). Early deaths were attributed to renal toxicity. Oral exposure to methylmercuric compounds has resulted in increased mortality at 16 mg Hg/kg (single dose) (Yasutake et al. 1991b), 3.1 mg Hg/kg/day for 26 weeks (Mitsumori et al. 1981), and 0.69 mg Hg/kg/day for up to 2 years (Mitsumori et al. 1990).

Systemic Effects

Respiratory Effects. The evidence from case report studies suggests that inhalation of metallic mercury vapor may result in clinical respiratory symptoms (e.g., chest pains, dyspnea, cough, reduced vital capacity) (Bluhm et al. 1992a; Gore and Harding 1987; Haddad and Sternberg 1963; Hallee 1969; Kanluen and Gottlieb 1991; King 1954; Lilis et al. 1985; Matthes et al. 1958; McFarland and Reigel 1978; Milne et al. 1970; Rowens et al. 1991; Snodgrass et al. 1981; Soni et al. 1992; Taueg et al. 1992; Teng and Brennan 1959; Tennant et al. 1961). In the more severe cases, respiratory distress, pulmonary edema, lobar pneumonia, fibrosis, desquamation of the bronchiolar epithelium, and death due to respiratory failure have been observed (Campbell 1948; Gore and Harding 1987; Jaffe et al. 1983; Kanluen and Gottlieb 1991; Matthes et al. 1958; Taueg et al. 1992; Teng and Brennan 1959; Tennant et al. 1961). Acute- and intermediate-duration studies in rabbits appear to corroborate clinical symptoms observed in humans following inhalation exposure to metallic mercury vapors. Mild-to-moderate pathological changes (unspecified) were exhibited in the lungs of rabbits exposed to 6–28.8 mg/m^3 mercury vapor for up to 11 weeks (Ashe et al. 1953), and death due to asphyxiation has been observed in rats exposed to 27 mg/m^3 for 2 hours (Livardjani et al. 1991b). Lung congestion was observed after 100 hours of continuous exposure of rats to 1 mg/m^3 (Gage 1961). The potential for oral exposure was not quantified; however, it is likely that most of the exposure was through inhalation. Inconclusive evidence is available regarding respiratory effects due to inhalation of organic mercury (Brown 1954; Hunter et al. 1940), and there is no conclusive evidence indicating that oral or dermal exposure to inorganic or organic forms of mercury is

directly toxic to the respiratory system. Based on these results, it would appear that acute inhalation exposure of humans to high levels of metallic mercury may result in pulmonary effects.

Cardiovascular Effects. The evidence from clinical, occupational, and general population studies suggests that inhalation of metallic mercury may affect the cardiovascular system in humans, producing elevations in blood pressure and/or heart rate (Aronow et al. 1990; Bluhm et al. 1992a; Campbell 1948; Fagala and Wigg 1992; Foulds et al. 1987; Friberg et al. 1953; Haddad and Sternberg 1963; Hallee 1969; Jaffe et al. 1983; Karpathios et al. 1991; Siblerud 1990; Smith et al. 1970; Snodgrass et al. 1981; Soni et al. 1992; Taueg et al. 1992; Teng and Brennan 1959). Studies of workers chronically exposed to elemental mercury vapor have shown increased incidences of palpitations (Piikivi 1989), high incidences of hypertension (Vroom and Greer 1972), and increased likelihood of death due to ischemic heart and cerebrovascular disease (Barregard et al. 1990). Of particular interest is the study showing slightly higher blood pressure in persons with dental amalgams than in those with no mercury-containing amalgams (Siblerud 1990). Less information is available regarding inhalation of organic mercury, but one study showed elevated blood pressure in two men occupationally exposed to methylmercury compounds (Hook et al. 1954). Electrocardiographic abnormalities (ventricular ectopic beats, prolongation of the Q–T interval, S–T segment depression, and T-wave inversion) were reported in persons who ate foods contaminated with ethylmercury compounds or who ingested a large dose of mercuric chloride (Chugh et al. 1978; Cinca et al. 1979; Jalili and Abbasi 1961). It is unclear whether these electrocardiographic abnormalities were the result of direct cardiac toxicity or whether they were secondary to other toxicity.

A number of the above cases of mercury-related tachycardia and elevated blood pressure in children inhaling metallic mercury vapors (Aronow et al. 1990; Fagala and Wigg 1992; Foulds et al. 1987; Karpathios et al. 1991) are associated with acrodynia, a nonallergic hypersensitive reaction in children to mercury exposure. Similar elevations in heart rate and blood pressure have been reported in children ingesting mercurous chloride (calomel)-containing medications and in children dermally exposed to ammoniated mercury-containing ointments or diapers that had been rinsed in a mercuric chloride-containing solution (Warkany and Hubbard 1953).

Limited animal data are available regarding inhalation exposure to mercury, but studies indicate that mercury may have a toxic effect on the heart. Effects ranging from mild pathological changes to marked cellular degeneration of heart tissue were exhibited in rabbits inhaling 0.86–28.8 mg/m^3 mercury vapor for

acute and intermediate durations (Ashe et al. 1953). However, it is unclear whether these changes represent direct toxic effects on the heart or whether they were secondary to shock.

The bulk of information available regarding cardiovascular effects after oral exposure of animals to mercury generally supports findings seen in human inhalation studies. Oral administration of 7 mg Hg/kg/day as inorganic mercury (mercuric chloride) for a year or 0.4 mg Hg/kg/day as organic mercury (methylmercuric chloride) for up to 28 days in rats resulted in elevated blood pressure (Carmignani et al. 1989; Wakita 1987). At higher concentrations (28 mg Hg/kg/day as mercuric chloride for 180 days), decreases in cardiac contractility were observed; these effects were suggested to be due to direct myocardial toxicity (Carmignani et al. 1992). Biphasic effects on myocardial tissue have been demonstrated on isolated papillary muscles from rat ventricles (Oliveira and Vassallo 1992). At low mercury concentrations, an increase in contractile force was observed, whereas at 5–10-fold higher concentrations dose-related decreases in contractile force were observed. The increases in contractile force were suggested to be due to inhibition of Na+-K+-ATPase, and the decreases were suggested to be due to inhibition of Ca2+-ATPase of the sarcoplasmic reticulum. Based on these results, it would appear that children with hypersensitivity to mercury may exhibit tachycardia and elevated blood pressure following inhalation, oral, or dermal exposure to mercury or to mercury-containing compounds. In addition, low-level exposure to mercury for extended periods may cause elevated blood pressure in exposed populations. Chronic-duration inhalation exposures or intermediate-duration oral exposures may also be associated with increased mortality due to ischemic heart or cerebrovascular disease; however, the data supporting this conclusion are more limited.

Gastrointestinal Effects. Both inhalation and oral exposures to mercury have resulted in gastrointestinal toxicity. Mercurial stomatitis (inflammation of the oral mucosa, occasionally accompanied by excessive salivation) is a classic symptom of mercury toxicity and has been observed following inhalation exposure to both inorganic and organic mercury (Bluhm et al. 1992a; Brown 1954; Campbell 1948; Fagala and Wigg 1992; Garnier et al. 1981; Haddad and Sternberg 1963; Hallee 1969; Hill 1943; Hook et al. 1954; Karpathios et al. 1991; Sexton et al. 1976; Snodgrass et al. 1981; Tennant et al. 1961; Vroom and Greer 1972).

Mercuric chloride is caustic to the tissues of the gastrointestinal tract, and persons who have ingested large amounts of this form of mercury have exhibited blisters, ulceration, and hemorrhages throughout the gastro-intestinal tract (Afonso and deAlvarez 1960; Chugh et al. 1978; Murphy et al. 1979; Samuels et al.

1982). In some cases, gastrointestinal lesions have been observed after inhalation exposure to high concentrations of metallic mercury vapors. The autopsy of a young child who inhaled metallic mercury vapor revealed necrosis in the mucosa of the stomach and duodenum (Campbell 1948). Irritation of the oral mucosa has also been observed at the site of contact with dental amalgams that contain mercury (Veien 1990). However, this type of response appears to be a combination of stomatitis and a contact dermatitis.

Symptoms of abdominal cramps, diarrhea, and nausea have been reported following acute- and/or intermediate-duration inhalation, oral, and dermal exposures of persons to mercury (Afonso and deAlvarez 1960; Bluhm et al. 1992a; Campbell 1948; Cinca et al. 1979; Haddad and Sternberg 1963; Hallee 1969; Jalili and Abbasi 1961; Kang-Yum and Oransky 1992; Kanluen and Gottlieb 1991; Lilis et al. 1985; Milne et al. 1970; Sexton et al. 1976; Snodgrass et al. 1981; Soni et al. 1992; Taueg et al. 1992; Teng and Brennan 1959; Tennant et al. 1961; Warkany and Hubbard 1953).

Rabbits displayed mild pathological changes to marked cellular degeneration and some necrosis in the colon tissue after inhaling 28.8 mg/m^3 mercury for 4–30 hours (Ashe et al. 1953). Inflammation and necrosis of the glandular stomach were observed in mice given 59 mg Hg/kg as mercuric chloride by gavage 5 days a week for 2 weeks (NTP 1993). An increased incidence of forestomach hyperplasia was observed in male rats exposed to 1.9 mg Hg/kg/day as mercuric chloride for 2 years (NTP 1993). Necrosis and ulceration of the cecum have been observed in rats after chronic-duration exposure to 4.2 mg Hg/kg/day as phenylmercuric acetate in the drinking water (Fitzhugh et al. 1950; Solecki et al. 1991). Ulceration of the glandular stomach was observed in mice after 2 years of exposure to methylmercuric chloride (0.86 mg Hg/kg/day) in the diet. Acute-duration inhalation exposure or chronic-duration oral exposure to inorganic and organic mercury may, therefore, result in various gastrointestinal symptoms in humans, with possible damage to intestinal tissues.

Hematological Effects. Leukocytosis associated with a metal fume fever-like syndrome has been observed in persons exposed to high concentrations of metallic mercury vapor (Campbell 1948; Fagala and Wigg 1992; Haddad and Sternberg 1963; Hallee 1969; Jaffe et al. 1983; Lilis et al. 1985; Matthes et al. 1958; Rowens et al. 1991). It is probable that this effect is specific to inhalation exposure to mercury. Because of the high concentrations of mercury that have been involved in the studies reviewed, it is unlikely that persons exposed to mercury vapors at hazardous waste sites would be exposed to sufficiently high concentrations of mercury to result in leukocytosis. Other hematological effects associated with exposure to mercury include decreased hemoglobin and hematocrit in persons with dental amalgams (Siblerud 1990)

and decreased δ-aminolevulinic acid dehydratase activity in erythrocytes or increased serum proteins involved in the storage and transport of copper in workers exposed to mercury vapor (Bencko et al. 1990; Wada et al. 1969). Anemia was found in a man who ingested a lethal amount of mercuric chloride (Murphy et al. 1979). However, the anemia was most likely the result of massive gastrointestinal hemorrhaging. No reports of effects on blood parameters in humans were located after oral exposure to organic mercury. A decrease in red cell count, hemoglobin, and hematocrit and rupture of erythrocytes were observed after intraperitoneal injection of mice with 19.2 mg Hg/kg as methylmercuric chloride (Shaw et al. 1991). A decrease in hemoglobin, hematocrit, and red blood cell count was observed in rats that received phenylmercuric acetate in their drinking water for 2 years (Solecki et al. 1991). However, this effect was probably due to blood loss associated with intestinal ulcers. Thus, there is limited information that suggests that prolonged exposure of humans to high levels of mercury, possibly from living in the vicinity of hazardous waste sites, may result in hematological changes.

Musculoskeletal Effects. Increases in tremors, muscle fasciculations, myoclonus, or muscle pains have been reported in persons exposed to unspecified concentrations of elemental mercury vapor (Adams et al. 1983; Albers et al. 1982, 1988; Aronow et al. 1990; Barber 1978; Bidstrup et al. 1951; Bluhm et al. 1992a; Chaffin et al. 1973; Chapman et al. 1990; Fawer et al. 1983; Karpathios et al. 1991; McFarland and Reigel 1978; Sexton et al. 1976; Smith et al. 1970; Taueg et al. 1992; Verberk et al. 1986; Vroom and Greer 1972; Williamson et al. 1982), in individuals inhaling alkyl mercury compounds (Brown 1954; Hook et al. 1954; Hunter et al. 1940), and in persons ingesting mercurous chloride (Warkany and Hubbard 1953) or ethyl-mercury compounds (Jalili and Abbasi 1961). These muscular effects are probably the result of peripheral nervous system dysfunction. It is probable that persons exposed to sufficiently high concentrations of mercury in the air or in foodstuffs (e.g., contaminated fish) at hazardous waste sites may also experience symptoms of tremors, myoclonus, muscle fasciculations, or muscle pains. A single report was identified that found evidence of rhabdomyolysis (destruction of the skeletal muscle) in a 22-year-old man who attempted suicide by ingesting 2 g of mercuric chloride (Chugh et al. 1978). It is extremely unlikely that persons at hazardous waste sites would be exposed to similarly high concentrations of mercuric chloride.

Hepatic Effects. Elevated serum glutamic pyruvic transaminase (SGPT), ornithine carbamyl transferase, and serum bilirubin, as well as evidence of decreased synthesis of hepatic coagulation factors, were reported in a case study of a child who inhaled an unspecified concentration of metallic mercury vapor (Jaffe et al. 1983). Similarly, hepatomegaly and hepatocellular vacuolation were observed in a man who

died following acute-duration, high-level exposure to elemental mercury vapor (Kanluen and Gottlieb 1991; Rowens et al. 1991). A lethal oral dose of mercuric chloride in a 35-year-old man also resulted in jaundice, an enlarged liver, and elevated aspartate aminotransferase, alkaline phosphatase, lactate dehydrogenase, and bilirubin (Murphy et al. 1979).

Inhalation of 6–28.8 mg/m^3 mercury vapor for 6 hours to 11 weeks by rabbits produced effects ranging from mild pathological changes to severe necrosis in the liver, including necrosis and degeneration; effects were less severe at the shorter durations (Ashe et al. 1953). Intermediate-duration oral exposure to inorganic mercury has also been associated with increases in hepatic lipid peroxidation (Rana and Boora 1992) and in serum alkaline phosphatase (Jonker et al. 1993a). It is unclear to what extent these effects were due to the direct toxic effects of mercury on the liver or were secondary to shock in the exposed animals. Reliable information regarding hepatic effects following organic mercury exposure was not located. These limited data suggest the potential hepatic toxicity of short-term inhalation of high concentrations of mercury vapor to humans. It is unlikely that persons at hazardous waste sites would ingest sufficiently large amounts of mercuric chloride to cause hepatic toxicity.

Renal Effects. The kidney is one of the major target organs of mercury-induced toxicity. Adverse renal effects have been reported following exposure to metallic, inorganic, and organic forms of mercury in both humans and experimental animals. The nephrotic syndrome in humans associated with the ingestion, inhalation, or dermal application of mercury is primarily identified as an increase in excretion of urinary protein, although depending on the severity of the renal toxicity, hematuria, oliguria, urinary casts, edema, inability to concentrate the urine, and hypercholesterolemia may also be observed (Agner and Jans 1978; Afonso and deAlvarez 1960; Anneroth et al. 1992; Barr et al. 1972; Buchet et al. 1980; Campbell 1948; Cinca et al. 1979; Danziger and Possick 1973; Dyall-Smith and Scurry 1990; Engleson and Herner 1952; Friberg et al. 1953; Hallee 1969; Jaffe et al. 1983; Jalili and Abbasi 1961; Kang-Yum and Oransky 1992; Kanluen and Gottlieb 1991; Kazantzis et al. 1962; Langworth et al. 1992b; Murphy et al. 1979; Pesce et al. 1977; Piikivi and Ruokonen 1989; Roels et al. 1982; Rowens et al. 1991; Samuels et al. 1982; Snodgrass et al. 1981; Soni et al. 1992; Stewart et al. 1977; Tubbs et al. 1982). These effects are usually reversible. However, in the most severe cases, acute renal failure has been observed (Afonso and deAlvarez 1960; Davis et al. 1974; Jaffe et al. 1983; Kang-Yum and Oransky 1992; Murphy et al. 1979; Samuels et al. 1982). Renal biopsies and/or autopsy results have primarily

Rowens et al. 1991), but glomerular changes have also been reported (Kazantzis et al. 1962; Tubbs et al. 1982).

Although the primary effect of mercury on the kidneys appears to be a direct toxic effect on the renal tubules, there is also evidence that implicates an immune mechanism of action for mercury-induced glomerular toxicity in some persons. In support of this theory, a few human case studies have reported deposition of IgG, immune complexes, and/or complement C3 along the glomerular basement membrane (Lindqvist et al. 1974; Tubbs et al. 1982).

Studies in animals support the conclusion that the primary toxic effect of both inorganic and organic mercury in the kidneys is on the epithelial cells of the renal proximal tubules. The changes observed in these studies were comparable with those observed in humans (i.e., proteinuria, oliguria, increases in urinary excretion of tubular enzymes, proteinaceous casts, decreased ability to concentrate the urine, decreased phenolsulfonphthalein excretion, increased plasma creatinine) (Bernard et al. 1992; Chan et al. 1992; Dieter et al. 1992; Girardi and Elias 1991; Jonker et al. 1993a; Kirschbaum et al. 1980; Nielsen et al. 1991; NTP 1993; Yasutake et al. 1991b). In addition, the animal studies provided detailed information regarding the histopathological changes occurring in the kidneys (Carmignani et al. 1989, 1992; Chan et al. 1992; Dieter et al. 1992; Falk et al. 1974; Fitzhugh et al. 1950; Fowler 1972; Goering et al. 1992; Hirano et al. 1986; Jonker et al. 1993a; Klein et al. 1973; Magos and Butler 1972; Magos et al. 1985; Mitsumori et al. 1990; Nielsen et al. 1991; NTP 1993; Yasutake et al. 1991b). The progression of renal toxicity included initial degenerative changes in the epithelial cells of the proximal tubules (nuclear swelling, increased eosinophilia/basophilia, vacuolization, and cellular hypertrophy). In the early stages, these degenerative changes were accompanied by tubular regeneration. Occasionally, when there is minor toxic damage, only the regenerative changes were observed. As the lesions progressed, tubular dilation, desquamation of the epithelial cells, and thickening of the tubular basement membrane were observed. Fibrosis, inflammation, necrosis, and atrophy of the tubules and glomerular changes (i.e., hypercellularity, thickening of the glomerular basement membrane) were then observed.

Several investigators have suggested that the renal toxicity exhibited after administration of organic forms of mercury (e.g., methylmercury) may actually result from the *in vivo* metabolism of this form to inorganic mercury (Fowler 1972; Klein et al. 1973; Magos et al. 1985). This hypothesis is supported by the increase in the smooth endoplasmic reticulum, a potential site for this metabolic conversion, and the measurement of

substantial levels of inorganic mercury in the kidneys following exposure to methylmercury (Fowler 1972; Klein et al. 1973).

In New Zealand White rabbits and in certain strains of mice and rats, a membranous glomerulonephropathy was the predominant finding in the absence of significant tubular damage. This syndrome was characterized by proteinuria, deposition of immune material (i.e., IgG and complement C3) in the renal mesangium and glomerular blood vessels, and minimal glomerular cell hyperplasia (Aten et al. 1992; Druet et al. 1978; Hirszel et al. 1985; Hultman and Enestrom 1992; Matsuo et al. 1989; Michaelson et al. 1985; Pelletier et al. 1990; Pusey et al. 1990; Roman-Franco et al. 1978; van der Meide et al. 1993). Deposition of antiglomerular basement membrane antibodies has been observed in a susceptible strain of rat at subcutaneous doses of mercuric chloride as low as 0.15 mg Hg/kg 4 days a week for 2 weeks (Michaelson et al. 1985). Increases in urinary protein were not observed until 0.74 mg Hg/kg 4 days a week for 2 weeks. In mice, autoantibodies to glomerular basement membrane were not observed, but deposition of IgG in the kidneys occurs as a result of autoantibodies to nucleolar antigens (Hultman and Enestrom 1988). The immune basis for these responses is covered in the section on immunological effects below. The susceptibility to this form of renal toxicity appears to be governed by both MHC genes and nonMHC genes (Aten et al. 1991; Sapin et al. 1984). Among rat strains, Brown-Norway, MAXX, and DZB strains showed susceptibility to renal damage, whereas Lewis, M520, and AO rats did not (Aten et al. 1991; Druet et al. 1978; Michaelson et al. 1985). Among mouse strains, SJL/N mice are susceptible to renal toxicity, whereas DBA, C57BL, and Balb/c mice are not (Hultman and Enestrom 1992; Hultman et al. 1992). The apparent genetic basis for susceptibility to mercury-induced nephrotoxicity in experimental animals has important implications with regard to susceptible subpopulations of humans.

Based on the above information, it is likely that persons exposed to sufficiently high concentrations of mercury may experience renal tubular toxicity. Certain persons who are genetically predisposed may also develop an immunologically based membranous glomerulonephritis.

Dermal Effects. Dermal reactions have been observed in persons exposed to inorganic and organic mercury following inhalation, oral, and/or dermal exposures. The predominant skin reaction is erythematous and pruritic skin rashes (Al-Mufti et al. 1976; Aronow et al. 1990; Bagley et al. 1987; Biro and Klein 1967; Bluhm et al. 1992a; Engleson and Herner 1952; Faria and Freitas 1992; Foulds et al. 1987; Goh and Ng 1988; Hunter et al. 1940; Jalili and Abbasi 1961; Kang-Yum and Oransky 1992; Karpathios et al. 1991; Morris 1960; Pambor and Timmel 1989; Schwartz et al. 1992; Sexton et al. 1976;

Tunnessen et al. 1987; Veien 1990; Warkany and Hubbard 1953). In many of the dermal cases, a contact dermatitis type of response was observed. However, a nonallergic pruritus is characteristic of acrodynia, a hypersensitive reaction to mercury exposure observed primarily in children, and several of the above cases may have been attributable to this syndrome (Aronow et al. 1990; Engleson and Herner 1952; Foulds et al. 1987; Jalili and Abbasi 1961; Karpathios et al. 1991; Tunnessen et al. 1987; Warkany and Hubbard 1953). Other dermal reactions characteristic of acrodynia include heavy perspiration (Aronow et al. 1990; Fagala and Wigg 1992; Karpathios et al. 1991; Sexton et al. 1976; Warkany and Hubbard 1953) and itching, reddened, swollen and/or peeling skin on the palms of the hands and soles of the feet (Aronow et al. 1990; Fagala and Wigg 1992; Jalili and Abbasi 1961; Karpathios et al. 1991; Tunnessen et al. 1987; Warkany and Hubbard 1953). No animal studies were located to support these findings. However, these results demonstrate that two populations may experience dermal effects as a result of mercury exposure. One is those persons who develop an allergic reaction to mercury. The other is those who are hypersensitive to mercury and who develop acrodynia upon exposure. It is unknown whether sufficiently high concentrations of inorganic mercury in soil or methylmercury in fish may exist at hazardous waste sites to trigger allergic dermatitis in sensitive persons or acrodynia in those predisposed to develop this syndrome.

Ocular Effects. Ocular effects have been observed in persons exposed to high concentrations of metallic mercury vapors. These effects are probably due to direct contact of the mercury vapor with the eyes. The observed effects include red and burning eyes, conjunctivitis (Bluhm et al. 1992a; Foulds et al. 1987; Karpathios et al. 1991; Schwartz et al. 1992; Sexton et al. 1976), and a yellow haze on the lenses of the eye (Atkinson 1943; Bidstrup et al. 1951; Locket and Nazroo 1952). The yellow haze was associated with long-term occupational exposures. Animal studies were not available to support these findings. However, the evidence suggests that exposure to high levels of mercury vapor may result in ocular irritation.

Other Systemic Effects. Studies of workers exposed to mercury vapor found no effect on serum levels of thyroid-stimulating hormone (Erfurth et al. 1990; McGregor and Mason 1991). However, an enlarged thyroid, with elevated triiodothyronine and thyroxine, as well as reduced thyroid-stimulating hormone developed in a 13-year-old boy exposed to mercury vapor for 2 weeks (Karpathios et al. 1991). Animal studies generally support an effect of acute-duration high-level exposure on the thyroid, although the results have been somewhat variable (Goldman and Blackburn 1979; Sin and The 1992; Sin et al. 1990). A single intramuscular injection of 14.8 mg Hg/kg in rabbits resulted in increased thyroid peroxidase and triiodo-thyronine and decreased thyroxine (Ghosh and Bhattacharya 1992). A study in which rats received three

daily subcutaneous doses of methylmercuric chloride showed slight increases in thyroid weight and basal levels of thyroid-stimulating hormone and thyroxine (Kabuto 1991). However, it was unclear whether these changes were statistically significant. In contrast, a single subcutaneous dose of 6.4 mg/Hg as methylmercuric chloride resulted in significant decreases in serum thyroxine (Kabuto 1987). At higher doses (9.6 and 12.8 mg mercury), increases in prolactin and thyroid-stimulating hormone were observed. The reason for these differences is unclear, but the data suggest that thyroid function may be affected if persons are exposed to sufficiently high concentrations of mercury.

Animal studies also provide evidence of mercury-induced effects on the corticosteroid levels. Increased adrenal and plasma corticosterone levels were reported in rats receiving 2.6 mg Hg/kg/day as mercuric chloride in drinking water after 120 days (Agrawal and Chansouria 1989). At 180 days of exposure, these effects were not evident in the animals. The investigators suggested that mercuric chloride is a dose- and duration-dependent chemical stressor. Subchronic administration of methylmercury to rats caused a diminished secretory response of corticosterone and testosterone serum levels following adrenocorticotropin (ACTH) and human chorionic gonadotropin (HCG) stimulation, respectively (Burton and Meikle 1980). The adrenal glands showed marked hyperplasia and increased weight, and basal levels of these hormones were also depressed. The treated animals exhibited stress intolerance and decreased sexual activity. These results suggest that methylmercury may have an adverse effect on steroidogenesis in the adrenal cortex and testes. Based on these animal studies, inorganic and organic mercury may also act on the corticosteroid system to alter hormonal levels. It is unclear to what extent the effects observed are the result of generalized stress or direct toxic effects on the endocrine system regulating corticosteroid levels.

Inhalation of metallic mercury vapor may result in a metal fume fever-like syndrome characterized by fatigue, fever, chills, cough, and an elevated leukocyte count (Bluhm et al. 1992a; Garnier et al. 1981; Lilis et al. 1985; McFarland and Reigel 1978; Milne et al. 1970; Schwartz et al. 1992; Snodgrass et al. 1981). Also, children with acrodynia frequently exhibit low-grade intermittent fevers (Aronow et al. 1990; Warkany and Hubbard 1953). Animal data are not available to support this finding, but the human data suggest that exposure to sufficiently high concentrations of metallic mercury vapor may result in transient fever (see Hematological Effects).

Immunological Effects. As indicated in the section on dermal effects, allergic dermatological reactions occurred in persons exposed to inorganic mercury from dental amalgams, tattoos, or breakage of medical instruments (Anneroth et al. 1992; Bagley et al. 1987; Biro and Klein 1967; Faria and Freitas

1992; Goh and Ng 1988; Pambor and Timmel 1989; Skoglund and Egelrud 1991; Veien 1990). Additionally, mercury may cause either decreases in immune activity or an autoimmune response, depending on the genetic predisposition of the individual exposed. The human data are very limited, and only decreased IgG production has been observed in workers chronically exposed to metallic mercury vapor at chloralkali and ore production plants (Bencko et al. 1990; Moszczynski et al. 1990b). Neither of these studies, however, adjusted for smoking or alcohol. Increases in serum immunoglobulins (IgA, IgG, IgE, or IgM) and autoantibody titres (antilaminin or antiglomerular basement membrane antibodies) have not been observed in similarly exposed populations (Bernard et al. 1987; Cardenas et al. 1993; Langworth et al. 1992b). There is limited information in humans that suggests that certain individuals may develop an autoimmune response when exposed to mercury. Deposition of IgG and complement C3 have been observed in the glomeruli of two workers with mercury-induced proteinuria (Tubbs et al. 1982). Also, increased antiglomerular basement membrane antibodies and elevated antinuclear antibodies have been observed in a few persons with exposure to mercury in dental amalgams (Anneroth et al. 1992). After removal of one dental amalgam, a significant decrease in IgE levels was observed. Within the populations described above that showed no overall increase in immune parameters, individuals in these groups showed either increases in anti-DNA antibody titres or antiglomerular basement membrane responses (Cardenas et al. 1993; Langworth et al. 1992b). Moszczynski et al. (1995) studied workers exposed to mercury vapor and reported a positive correlation between the T-helper cell count and the duration of exposure. The combined stimulation of the T-cell line and an observed decrease in the helper/suppressor ratio were suggestive of an autoimmune response.

The immune system reaction to mercury has been extensively studied in animals. Although it has not been completely described, a great deal of information exists about the changes that occur in the immune system in response to mercury exposure (Bigazzi 1992; Goldman et al. 1991; Mathieson 1992). Animal strains that are susceptible or predisposed to develop an autoimmune response show a proliferation of autoreactive T-cells (specifically CD4+ T-cells) (Pelletier et al. 1986; Rossert et al. 1988). The fundamental change caused by mercury that results in the autoimmune response appears to be in these autoreactive T-cells, since transfer of these cells to an unexposed animal results in the development of the autoimmune response in the unexposed animal (Pelletier et al. 1988). A subset of the CD4+ T-cells, the Th2 cells, are activated and induce polyclonal B-cell activation (possibly through the release of interleukin-4 [IL-4]), which results in IgE production by the B-cells (Ochel et al. 1991). The increases in serum IgE are paralleled by increases in MHC molecule expression on the B-cells (Dubey et al. 1991a). These changes are accompanied by enlargement of the spleen and lymph nodes, an increase in the number of spleen cells

(thought to be associated with the B-cell proliferation) (Hirsch et al. 1982; Matsuo et al. 1989), and marked increases in serum levels of IgE (Dubey et al. 1991b; Hirsch et al. 1986; Lymberi et al. 1986; Prouvost-Danon et al. 1981). Increases in the production of autoantibodies (IgG) to glomerular basement membrane, thyroglobulin, collagen types I and II, and DNA also occur (Pusey et al. 1990). Immune complex deposits occur in blood vessels in several organs (Hultman et al. 1992), and deposition of these autoantibodies and complement in the renal glomerulus ultimately lead to membranous glomerulonephropathy, although the deposition of the IgG alone does not appear to be sufficient to induce renal dysfunction (Michaelson et al. 1985). In rodents, the autoimmune response spontaneously resolves within a few weeks. The mechanism underlying the resolution is unknown, but antiidiotypic antibodies and a change in the balance between Th2 and Th1 (another subset of the CD4+ T-cells) cell activation (see below) have been proposed (Mathieson 1992). After this resolution phase has occurred, affected individuals develop a resistance to future autoimmune toxicity (Bowman et al. 1984). The resistance appears to be mediated by CD8+ T-cells, since depletion of these cells reverses the resistance (Mathieson et al. 1991).

The so-called resistant strains, however, show a different response to mercury exposure. These resistant strains also show an increase in MHC expression molecules on B-cells, but this response is extremely short-lived, and increases in serum IgE were not observed (Dubey et al. 1991a; Prouvost-Danon et al. 1981). The difference in the responses of the so-called resistant and susceptible strains may be found in the activation of Th1 cells and the increase in secretion of γ-interferon by the Th1 cells of resistant animals (van der Meide et al. 1993). The susceptible strains do not show an increase in γ-interferon production with mercury exposure. Because γ-interferon inhibits the proliferation of Th2 cells, the absence of this response in the susceptible strains may allow the Th2 cell-stimulated production of autoantibodies to occur, whereas in the resistant strains the production of antibodies is curtailed. Thus, differences in the activation of Th1 versus Th2 cells may underlie the differences in susceptibility of various individuals. Studies using in-bred strains of mice and rats have determined that the susceptibility to the different immune reactions is governed by both MHC genes as well as other genes (Aten et al. 1991; Druet et al. 1977; Mirtcheva et al. 1989; Sapin et al. 1984). As indicated in the section on renal effects, Brown-Norway, MAXX, and DZB rat strains showed susceptibility, whereas Lewis, M520, and AO rats did not (Aten et al. 1991; Druet et al. 1978; Michaelson et al. 1985). Among mouse strains, SJL/N mice are susceptible and DBA, C57BL, and Balb/c mice are not (Hultman and Enestrom 1992; Hultman et al. 1992). In a resistant strain, the Balb/c mouse, immune suppression was manifested as decreased natural killer cell activity in mice administered a diet containing 0.5 mg Hg/kg/day as methylmercury (Ilback 1991).

Neurological Effects. The nervous system is the primary target organ for elemental and methyl-mercury-induced toxicity. Neurological and behavioral disorders in humans have been observed following inhalation of metallic mercury vapor and organic mercury compounds, ingestion or dermal application of inorganic mercury-containing medicinal products (e.g., teething powders, ointments, and laxatives), and ingestion or dermal exposure to organic mercury-containing pesticides or ingestion of contaminated seafood. A broad range of symptoms has been reported, and these symptoms are qualitatively similar, irrespective of the mercury compound to which one is exposed. Specific neurotoxic symptoms include tremors (initially affecting the hands and sometimes spreading to other parts of the body), emotional lability (characterized by irritability, excessive shyness, confidence loss, and nervousness), insomnia, memory loss, neuromuscular changes (weakness, muscle atrophy, and muscle twitching), headaches, polyneuropathy (paresthesias, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory and motor nerve conduction velocities), and performance deficits in tests of cognitive and motor function (Adams et al. 1983; Albers et al. 1982, 1988; Aronow et al. 1990; Bakir et al. 1973; Barber 1978; Bidstrup et al. 1951; Bluhm et al. 1992a; Bourgeois et al. 1986; Chaffin et al. 1973; Chapman et al. 1990; Choi et al. 1978; Cinca et al. 1979; Davis et al. 1974; DeBont et al. 1986; Discalzi et al. 1993; Dyall-Smith and Scurry 1990; Ehrenberg et al. 1991; Fagala and Wigg 1992; Fawer et al. 1983; Foulds et al. 1987; Friberg et al. 1953; Hallee 1969; Harada 1978; Hook et al. 1954; Hunter et al. 1940; Iyer et al. 1976; Jaffe et al. 1983; Jalili and Abbasi 1961; Kang-Yum and Oransky 1992; Karpathios et al. 1991; Kutsuna 1968; Langauer-Lewowicka and Kazibutowska 1989; Kutsuna 1968; Langolf et al. 1978; Langworth et al. 1992a; Levine et al. 1982; Lilis et al. 1985; Lundgren and Swensson 1949; Matsumoto et al. 1965; McFarland and Reigel 1978; Melkonian and Baker 1988; Miyakawa et al. 1976; Ngim et al. 1992; Piikivi and Hanninen 1989; Piikivi and Tolonen 1989; Piikivi et al. 1984; Roels et al. 1982; Sexton et al. 1976; Shapiro et al. 1982; Snodgrass et al. 1981; Smith et al. 1970; Tamashiro et al. 1984; Taueg et al. 1992; Tsubaki and Takahashi 1986; Verberk et al. 1986; Vroom and Greer 1972; Warkany and Hubbard 1953; Williamson et al. 1982). Some individuals have also noted hearing loss, visual disturbances (visual field defects), and/or hallucinations (Bluhm et al. 1992a; Cinca et al. 1979; Fagala and Wigg 1992; Jalili and Abbasi 1961; Locket and Nazroo 1952; McFarland and Reigel 1978; Taueg et al. 1992). Although improvement has often been observed upon removal of persons from the source of exposure, it is possible that some changes may be irreversible. Autopsy findings of degenerative changes in the brains of poisoned patients exposed to mercury support the functional changes observed (Al-Saleem and the Clinical Committee on Mercury Poisoning 1976; Cinca et al. 1979; Davis et al. 1974; Miyakawa et al. 1976). Limited information was located regarding exposure levels associated with the above effects, but increased tremors and cognitive difficulties are sensitive end points for chronic low-level exposure to metallic mercury vapor (Fawer et al.

1983; Ngim et al. 1992). Photophobia has been reported exclusively in children with acrodynia (Fagala and Wigg 1992; Warkany and Hubbard 1953). The physiological basis for the photophobia is unknown.

The neurotoxicity of inorganic and organic mercury in experimental animals is manifested as functional, behavioral, and morphological changes, as well as alterations in brain neurochemistry (Arito and Takahashi 1991; Ashe et al. 1953; Berthoud et al. 1976; Burbacher et al. 1988; Cavanaugh and Chen 1971; Chang and Hartmann 1972a, 1972b; Chang et al. 1974; Charbonneau et al. 1976; Concas et al. 1983; Evans et al. 1977; Fukuda 1971; Fuyuta et al. 1978; Ganser and Kirschner 1985; Inouye and Murakami 1975; Jacobs et al. 1977; Kishi et al. 1978; Lehotzky and Meszaros 1974; Leyshon and Morgan 1991; MacDonald and Harbison 1977; Magos and Butler 1972; Magos et al. 1980, 1985; Mitsumori et al. 1981; Miyama et al. 1983; Post et al. 1973; Rice 1989c; Rice and Gilbert 1982, 1992; Salvaterra et al. 1973; Sharma et al. 1982; Tsuzuki 1981; Yip and Chang 1981).

Animal studies have shown damage to the cerebellar cortex and dorsal root ganglion cells following both mercuric chloride and methylmercuric chloride exposure (Chang and Hartmann 1972b). These structures appear to be especially sensitive to the toxic effects of mercury (Chang and Hartmann 1972a, 1972b; Chang et al. 1974; Charbonneau et al. 1976; Falk et al. 1974; Hirano et al. 1986; Jacobs et al. 1977; Leyshon and Morgan 1991; MacDonald and Harbison 1977; Magos and Butler 1972; Magos et al. 1980, 1985; Mitsumori et al. 1990; Yip and Chang 1981), although other areas (e.g., the cerebral cortex, corpus striatum, thalamus, hypothalamus, organ of Corti, and peripheral nerves) have also shown degenerative changes after exposure to methylmercury (Berthoud et al. 1976; Chang et al. 1974; Charbonneau et al. 1976; Falk et al. 1974; Fehling et al. 1975; Jacobs et al. 1977; Miyakawa et al. 1974, 1976; Yip and Chang 1981). Cats and monkeys appear to be more sensitive to the toxic effects than rodents and have shown signs of neurotoxicity at approximately 10-fold lower doses (0.05 mg Hg/kg/day) following long-term exposure to methylmercuric chloride (Charbonneau et al. 1976; Rice 1989c; Rice and Gilbert 1982, 1992).

Although it is unclear whether changes in neurochemical parameters are primary targets of mercury or whether the changes are secondary to degenerative changes in neurons, several neurotransmitter systems have been shown to be affected by mercury exposure. Cholinergic transmission at the neuromuscular junction has been shown to be affected by mercury exposure (Eldefrawi et al. 1977; Sager et al. 1982). Changes in GABA receptor activity and number have also been observed (Arakawa et al. 1991; Concas et al. 1983). Changes in the activities of enzymes involved in cholinergic, adrenergic, dopaminergic, and

serotonergic synthesis and/or catabolism have also been observed following mercury exposure (Sharma et al. 1982; Tsuzuki 1981).

Collectively, the above information shows the high sensitivity of the nervous system to mercury toxicity and indicates that persons exposed to sufficiently high amounts of mercury may experience adverse neurological symptoms.

Reproductive Effects. Studies in humans indicate that metallic mercury vapor does not cause infertility or malformations following paternal exposure (Alcser et al. 1989; Lauwerys et al. 1985) but may cause an increase in the rate of spontaneous abortions (Cordier et al. 1991). No correlation was observed between levels of testosterone, luteinizing hormone, or follicle-stimulating hormone and occupational exposure to metallic mercury vapor, indicating that the pituitary control of testosterone secretion was not affected (Erfurth et al. 1990; McGregor and Mason 1991). However, *in vitro* studies have shown that mercury can adversely affect human spermatozoa. Inorganic (mercuric chloride) and organic (methylmercuric chloride) mercury decreased the percentage of motile spermatozoa *in vitro* (Ernst and Lauritsen 1991). Incubation of human spermatozoa with inorganic mercury resulted in mercury deposits localized in the membranes of the midpiece and tailpiece. The lack of mercury grains in spermatozoa with methylmercury exposure may be due to the inability of spermatozoa or the semen plasma to demethylate methylmercury in the 15-minute incubation period (Ernst and Lauritsen 1991).

Female dentists and dental assistants exposed to metallic mercury vapors had increased reproductive failures (spontaneous abortions, stillbirths, and congenital malformations) and irregular, painful, or hemorrhagic menstrual disorders (Sikorski et al. 1987). Correlations were observed between the incidence of these effects and hair mercury levels.

Rowland et al. (1994) report that female dental assistants with a high occupational exposure to mercury were found to be less fertile than controls. The probability of conception with each menstrual cycle (called "fecundability" by the authors) in women who prepared 30 or more amalgams per week and who were evaluated as having 4 or more poor mercury-hygiene practices was only 63% of that of unexposed controls. Hygiene was incorporated into the evaluation of the results of this study because occupational groups with roughly the same potential for exposure often contain subjects whose actual exposures are quite different, depending on their particular work environment and their work (and personal) hygiene practices within that environment. Rowland et al. (1994) found that 20% of the women in their final evaluation who prepared

more than 30 amalgams a week had 4 or more poor mercury-hygiene factors. Among women preparing a comparable number of amalgams, there were differences in "fecundability," based on the number of self-reported poor hygiene factors. The study is limited in that a group of unexposed women had lower fertility than the low exposed group suggesting other unaccounted for exposures or confounding factors.

Animal data suggest that mercury may alter reproductive function and/or success when administered to either males or females. In males, mercury exposure results primarily in impaired spermatogenesis, sperm motility, and degeneration of seminiferous tubules. Oral administration of methylmercury to males has resulted in decreases in litter size due to preimplantation loss (presumably due to defective sperm) in rats (Khera 1973), decreases in sperm motility in monkeys (at 0.025 mg Hg/kg/day for 20 weeks) (Mohamed et al. 1987), and tubular atrophy and decreased spermatogenesis in mice after prolonged exposure (Hirano et al. 1986; Mitsumori et al. 1990). Parenteral administration of methylmercury has shown similar results. A single intraperitoneal injection of 10 mg/kg of methylmercury in male mice resulted in decreased implantations in females (Suter 1975), and a single intraperitoneal injection of 1 mg/kg of methylmercury resulted in a reversible failure of spermatogenesis and infertility in male mice (Lee and Dixon 1975). Repeated intraperitoneal injections of methylmercury (3.5 mg Hg/kg/day for 6 weeks) in male rats resulted in decreased sexual activity, depression of testosterone levels (Burton and Meikle 1980), and decreased spermatogenesis (0.004 mg Hg/kg/day for 15–90 days) (Vachhrajani et al. 1992). Less is known about the effects of inorganic mercury on the male reproductive system, but a single intraperitoneal injection of mercuric chloride (1 mg Hg/kg) in male rats resulted in decreased conceptions in females (Lee and Dixon 1975), and 0.74 mg Hg/kg resulted in tubular degeneration (Prem et al. 1992). An *in vitro* study (Mohamed et al. 1987) suggested that the decrease in sperm motility observed in monkeys may be due to interference with microtubule assembly or dynein/microtubule sliding function.

In females, mercury exposure results primarily in increases in resorptions and decreases in implantations. Inhalation exposure of female rats to metallic mercury vapor (2.5 mg/m^3, 6 hours a day, 5 days a week for 21 days) resulted in a prolongation of the estrous cycle (Baranski and Szymczyk 1973). Oral administration of mercuric acetate (22 mg Hg/kg) to pregnant hamsters resulted in an increase in resorptions (Gale 1974). Oral administration of methylmercury to pregnant guinea pigs (11.5 mg Hg/kg) resulted in an increase in abortions (Inouye and Kajiwara 1988), and 3 mg Hg/kg resulted in a decrease in the number of pups in the litter from pregnant mice (Hughes and Annau 1976). Pregnant mice given a single dose of 20 mg Hg/kg as methylmercuric chloride had increased resorptions, decreased live fetuses, and decreased fetuses per litter (Fuyuta et al. 1978). Repeated oral administration of methylmercury

(0.06 mg Hg/kg/day) to female monkeys resulted in an increase in the number of abortions and a decrease in conceptions (Burbacher et al. 1988). No effect on the monkeys' menstrual cycles was observed. Intraperitoneal administration of mercuric chloride (1.48 mg Hg/kg) to female mice resulted in decreases in litter size and number of litters/female and an increase in dead implants in some strains of mice, but these effects were strain-specific (Suter 1975). In female mice administered a single intraperitoneal dose of 1 mg Hg/kg as mercuric chloride, a decrease in mean implantation sites was observed (Kajiwara and Inouye 1992). Subcutaneous injection of female hamsters with 6.2–8.2 mg Hg/kg as mercuric chloride for 1–4 days resulted in a disruption of estrous (Lamperti and Printz 1973). Inhibition of follicular maturation and normal uterine hypertrophy, morphological prolongation of the corpora lutea, and alteration of progesterone levels were observed. Collectively, these results suggest that at sufficiently high mercury concentrations, men may experience some adverse effects on testicular function and women may experience increases in abortions, decreases in conceptions, or development of menstrual disorders.

Developmental Effects. Mercury is considered to be a developmental toxicant. Extremely limited information was located regarding human developmental effects associated with exposure to inorganic mercury (Alcser et al. 1989; Derobert and Tara 1950; Melkonian and Baker 1988; Thorpe et al. 1992). However, developmental toxicity in humans associated with oral exposure to organic forms of mercury is well recognized (Amin-Zaki et al. 1974; Bakir et al. 1973; Cox et al. 1989; Engleson and Herner 1952; Harada 1978; Marsh et al. 1980, 1981, 1987; McKeown-Eyssen et al. 1983; Snyder and Seelinger 1976). The symptoms observed in offspring of exposed mothers are primarily neurological in origin and have ranged from delays in motor and verbal development to severe brain damage. Subtle changes, such as small changes in intelligence or learning capacity are currently being tested in populations with low-level, chronic exposure to mercury in the diet (Davidson et al. 1998; Grandjean et al. 1997b, 1998). MRLs for acute- and intermediate-duration exposure to methylmercury have been developed based on the lowest observed peak hair level in a mother whose child was reported to have a delayed onset of walking (14 ppm in hair) (Cox et al. 1989; WHO 1990).

Animal studies suggest that both inorganic mercury and organic mercury cause developmental toxicity. Metallic mercury vapor may be transferred across the placenta (Greenwood et al. 1972). The placental transport of mercury in pregnant mice and its localization in the embryo and fetus were studied by autoradiography and gamma counting (Khayat and Dencker 1982). Retention of ^{203}Hg vapor following inhalation was compared to intravenous injection of ^{203}Hg as mercuric chloride. The authors reported that inhalation of mercury vapor resulted in a mercury concentration that was four times higher than the

concentration resulting from injection of mercuric chloride. Furthermore, the authors reported that metallic mercury appeared to oxidize to Hg^{+2} in the fetal tissues. Evidence that inhalation exposure may result in developmental toxicity comes from a study in which neonatal rats were exposed to metallic mercury vapor during a period of rapid brain development (this occurs postnatally in rodents but prenatally in humans), resulting in impaired spatial learning (Fredriksson et al. 1992). Oral administration of inorganic mercury salts to pregnant hamsters has been observed to produce an increase in the number of resorptions and small and edematous embryos (Gale 1974). Mercury-induced embryotoxicity in one non-inbred and five inbred stains of female hamsters was investigated by Gale and Ferm (1971). A single subcutaneous injection of 9.5 mg Hg/kg as mercuric acetate to dams on Gd 8 produced a variety of malformations, including cleft palate, hydrocephalus, and heart defects, and statistically significant interstrain differences in the embryotoxic response. Single doses of 1.3–2.5 mg Hg/kg as mercuric acetate injected intravenously into pregnant hamsters on Gd 8 produced growth retardation and edema of the fetuses at all 3 dose levels, while an increase in the number of abnormalities was detected at the two higher doses (Gale and Ferm 1971). The relative effectiveness of different exposure routes in hamsters was compared by Gale (1974). The following sequence of decreasing efficacy was noted for mercuric acetate: intraperitoneal > intravenous > subcutaneous > oral. The lowest doses used (2 mg/kg for intraperitoneal and 4 mg/kg for the other 3 routes) were all effective in causing increased resorption and an increased percentage of abnormalities. Intravenous injection of 1.5 mg Hg/kg/day as mercuric chloride also resulted in a significant increase in the number of abnormal preimplantation embryos (Kajiwara and Inouye 1986).

In animals, embryolethal, anatomical, and behavioral effects have been reported following oral exposure of pregnant dams to methylmercury (Bornhausen et al. 1980; Cagiano et al. 1990; Elsner 1991; Fowler and Woods 1977; Fuyuta et al. 1978, 1979; Guidetti et al. 1992; Gunderson et al. 1988; Hughes and Annau 1976; Ilback et al. 1991; Inouye and Kajiwara 1988; Inouye and Murakami 1975; Inouye et al. 1985; Khera and Tabacova 1973; Lindstrom et al. 1991; Nolen et al. 1972; Olson and Boush 1975; Reuhl et al. 1981a, 1981b; Rice 1992; Rice and Gilbert 1990; Stoltenburg-Didinger and Markwort 1990; Yasuda et al. 1985). Thus far, the most sensitive animal assay for developmental neurotoxicity has been a behavioral paradigm that examined the number of rewarded responses to differential reinforcement at high rates (Bornhausen et al. 1980). At doses of 0.008 mg Hg/kg/day and above, a dose-related decrease in rewarded responses was observed in 4-month-old offspring of rats treated on Gd 6–9. The effect was more pronounced in male offspring than females. Foster mothers were used to preclude consumption of contaminated milk during lactation.

Developmental toxicity has also been observed with parenteral exposure to methylmercury in pregnant dams during gestation. In mice given methylmercuric hydroxide subcutaneously daily from Gd 7–12, significant dose-related increases in the percentage of litters with resorptions were seen in groups receiving 3.45–8.6 mg Hg/kg/day (Su and Okita 1976). The frequency of cleft palate increased significantly in litters of the 3.45 and 4.3 mg Hg/kg/day groups only. A high incidence of delayed palate closure and cleft palate was also reported in mice injected subcutaneously with 5 mg Hg/kg of methylmercuric chloride on Gd 12 (Olson and Massaro 1977). Gross incoordination and decreased frequencies of defecation and urination in pups were observed following intraperitoneal administration of a single dose of methylmercury dicyandiamide (8 mg/kg/day) to pregnant mice on day 7 or 9 of pregnancy (Spyker et al. 1972). Degenerative changes were observed in the cerebellum and cerebral cortex of rat pups of maternal rats injected with 4 mg Hg/kg as methylmercuric chloride on Gd 8 (Chang et al. 1977). Degenerative renal changes (in epithelial cells of proximal tubules and Bowman's capsule of glomeruli) were reported in rat fetuses of dams exposed intraperitoneally to methylmercuric chloride during Gd 8 (Chang and Sprecher 1976). The studies by Spyker and Smithberg (1972) demonstrated strain differences in susceptibility to the developmental effects of methylmercury dicyandiamide. Intraperitoneal administration of single doses of methylmercury dicyandiamide (2, 4, or 8 mg/kg) to pregnant mice of strains 129 Sv/S1 and A/J during gestation resulted in retardation of fetal growth and increased resorption of implants in both strains. Teratogenic effects, primarily of the palate and jaw, were detected at all dose levels in 129 Sv/S1 mice, but only at the highest dose in strain A/J. The differential effects of methylmercury were dependent on the strain, the dose of the agent, and the stage of embryonic development.

Antilaminin antibodies induced by mercuric chloride have been demonstrated to be detrimental to the development of cultured rat embryos (Chambers and Klein 1993). Based upon that observation, those authors suggested that it might be possible for an autoimmune disease induced by a substance such as mercury at an early age to persist into later life, acting as a teratogen independent of both dose-response relationships and time of exposure, but that possibility remains to be experimentally demonstrated.

One developmental study of phenylmercury compounds was reported by Gale and Ferm (1971) in which hamsters were injected intravenously with phenylmercuric acetate at doses ranging from 5 to 10 mg/kg on Gd 8. With the exception of the lowest dose, all other doses induced increased resorption rates and edema, along with a few miscellaneous abnormalities including cleft palate and exencephaly.

The above information clearly indicates the possibility of developmental toxicity in offspring of mothers that ingest sufficient amounts of organic mercury. The animal data also suggest that exposure to sufficient amounts of inorganic mercury by inhalation of metallic mercury vapor or ingestion of inorganic mercury may result in developmental toxicity.

Genotoxic Effects. The overall findings from cytogenetic monitoring studies of workers occupationally exposed to mercury compounds by inhalation (Anwar and Gabal 1991; Barregard et al. 1991; Mabille et al. 1984; Popescu et al. 1979; Verschaeve et al. 1976, 1979) or accidentally exposed through ingestion (Wulf et al. 1986) provided no convincing evidence that mercury adversely affects the number or structure of chromosomes in human somatic cells. Studies reporting a positive result (Anwar and Gabal 1991; Barregard et al. 1991; Popescu et al. 1979; Skerfving et al. 1970, 1974; Verschaeve et al. 1976; Wulf et al. 1986) were compromised either by technical problems, a lack of consideration of confounding factors, or a failure to demonstrate a relationship between mercury exposure and induced aberrations. Therefore, none of these studies can be used to predict the potential genetic hazard to humans associated with exposure to mercury or mercury compounds.

A dose-related increase in chromosome aberrations was observed in the bone marrow of mice administered a single oral dose of mercuric chloride at levels of at least 4.4 mg Hg/kg (Ghosh et al. 1991). By contrast, there was no valid evidence of a genotoxic effect on somatic cells of cats chronically exposed to methyl-mercury orally (Miller et al. 1979). However, only minimal toxicity was observed at the high dose (0.046 mg Hg/kg/day) in this study. Doses of 0.86, 1.7, or 3.4 mg Hg/kg as methylmercury hydroxide administered once by intraperitoneal injection to groups of 2 male CBA mice did not cause an increase in micronucleated polychromatic erythrocytes harvested from bone marrow cells 24 hours after treatment (Jenssen and Ramel 1980). Similarly, there was no increase in structural chromosome aberrations in bone marrow cells collected from male Swiss OF_1 mice (3–4/group) 12, 24, 36, or 48 hours postexposure to single intraperitoneal doses of 0.7, 1.5, 3.0, or 4.4 mg Hg/kg as mercuric chloride (Poma et al. 1981). The lack of a clastogenic response, particularly with mercuric chloride, should not be viewed as a possible inability of this compound to penetrate somatic cell membranes. There are data from the study of Bryan et al. (1974) indicating that mercuric chloride can bind to chromatin in the livers of mice challenged with 38 mg Hg/kg/day as mercuric chloride for 1 month. Although the overall data are mixed, the findings from a well conducted study using oral dosing suggests that mercury can be clastogenic for somatic cells (Ghosh et al. 1991).

The intraperitoneal administration of mercuric chloride at levels comparable to those described above did not induce a clastogenic response in the spermatogonia of the same mouse strain (Poma et al. 1981). Structural chromosome aberrations were not produced in metaphase II oocytes of 15 virgin Syrian hamsters receiving a single intraperitoneal injection of 7.4 mg Hg/kg as methylmercury chloride (Mailhes 1983). However, the frequency of hyperploid cells in the treated animals was significantly ($p<0.01$) increased compared to the control. A borderline significant increase in hypoploid cells was also seen. By contrast, Jagiello and Lin (1973) found no evidence of aneuploidy in the oocytes of Swiss/Webster mice (6–8/group) for 3 days after receiving a single intravenous injection of dimethylmercury (140 mg Hg/kg) or mercuric acetate (2, 5, or 10 mg Hg/kg). The lack of concordance between these two studies could be related to the different mercurials that were utilized, the different routes of exposure, or the possible differences in species sensitivity. There are data from a series of dominant lethal assays suggesting that variable strain sensitivity to mercury compounds can affect the outcome of germinal cell cytogenetic investigations (Suter 1975). In this study, two strains of male mice, $(101\times C3H)F_1$ and $(SEC\times C57BL)F_1$, and one strain of female mice, $(101\times C3H)F_1$, received single intraperitoneal injections of 8.6 mg Hg/kg as methylmercuric hydroxide. An additional group of females was injected intraperitoneally with 1.5 mg Hg/kg as mercuric chloride. Males were sequentially mated with untreated females over the entire spermatogenic cycle; treated females were mated once with untreated males. Methylmercuric hydroxide had no effect on fertility and did not induce a clastogenic response in $(101\times C3H)F_1$ males. However, a comparable dose administered to $(SEC\times C57BL)F_1$ males adversely affected fertility and caused significant reductions in total and live implants accompanied by increases in the percentage of dead implants following the first two mating cycles. Suggestive evidence of poor reproductive performance and a dominant lethal effect was also seen in female $(101\times C3H)F_1$ mice treated with methylmercuric hydroxide (8.6 mg Hg/kg) and mercuric chloride (1.5 mg Hg/kg). It was noteworthy that an independent phase of the investigation examined reproduction in females in two additional strains, $(SEC\times C57BL)F_1$ and a mixed stock obtained by crossing $(SEC\times C57BL)F_1$ females with XGSY males. Neither compound had a detrimental effect on the fertility of these females. The single dominant lethal assay conducted with rats (strain not specified) showed that mercuric chloride, administered orally for 12 months (1.8×10^{-3} to 1.8×10^{-4} mg Hg/kg), induced a dose-related increase in dominant lethal mutations, as indicated by increased embryonic death (Zasukhina et al. 1983).

The overall findings from *in vivo* germinal cell assays suggest that mercury compounds are clastogenic for mammalian germ cells. However, the apparent differences in species sensitivity and, in some cases, strain

sensitivity preclude an extrapolation of the relevance of these findings to humans. Refer to Table 2-11 for a further summary of these results.

Several *in vitro* assays employing human cells were located. Both structural and numerical chromosomal aberrations were observed following the exposure of human lymphocytes to methylmercury chloride or dimethylmercury *in vitro* (Betti et al. 1992). Although the smoking status of the donor was not reported, all of the cells came from the same donor, and no aberrations were observed in the control cultures. Mercuric acetate caused single-strand breaks in DNA from human KB-cells (Williams et al. 1987). Methylmercuric chloride treatment of human lymphocytes resulted in the formation of chromosome and chromatid aberrations (Betti et al. 1993b). Further, it was found to be a weak inducer of sister chromatid exchange, but that effect did not increase with an increasing dosage. Methylmercuric chloride was also found to be capable of producing aneuploidy (particularly hyperdiploidy). At low doses, more chromosomal aberrations were observed in the second metaphases than in the first, suggesting that several premutational lesions induced by that organomercurial survived through one cell cycle. Thus, the damage produced by methylmercuric chloride appeared to be stable and could lead to chromosome segregation errors. Betti et al. (1993b) concluded that methylmercuric chloride was capable of producing long-lasting damage, which in turn gives rise to both structural and numerical chromosome abnormalities. Bala et al. (1993) reported that methyl-mercuric chloride in concentrations of 10^{-5}, 10^{-6}, and 10^{-7} M induced aberrant metaphases (including gaps) in cultured human peripheral lymphocytes in a dose-dependent manner ($p<0.05$). Methylmercuric chloride at the higher concentrations also induced a significant number of breaks. Further, methylmercuric chloride induced a significant number of SCEs per cell in a dose-dependent manner. However, cultures treated with gamma linolenic acid (GLA), a derivative of dietary essential fatty acid, did not differ from controls with respect to aberrations, and GLA reduced the frequency of SCEs induced by methylmercuric chloride in a dose-dependent manner ($p<0.05$).

Mercuric chloride was not mutagenic in the *Salmonella typhimurium* plate incorporation assay (Wong 1988). These negative results are not unexpected because the Ames test is not suitable for the detection of heavy metal mutagens. Oberly et al. (1982) reported, however, that doses of mercuric chloride (4.4 and 5.9 µg Hg/mL) approaching severely cytotoxic levels induced a weak mutagenic response in mouse lymphoma L5178Y cells but only in the presence of auxiliary metabolic activation.

In an *in vitro* study of the clastogenic effects of mercurials in animal cells, Howard et al. (1991) observed a dose-related increase in chromosome aberrations in Chinese hamster ovary (CHO) cells treated with

mercuric chloride. In a study of the potentiating effects of organomercurials on clastogen-induced chromosomal aberrations in cultured Chinese hamster cells, Yamada et al. (1993) investigated the effects of five organomercurial compounds (methylmercuric chloride, ethylmercuric chloride, phenylmercuric chloride, dimethylmercury, and diethylmercury) and found all to produce remarkable cytotoxicity. Fifty percent or more depression in the mitotic index was observed following treatment with methylmercuric chloride (2.5 µg/mL), ethylmercuric chloride (2.5 µg/mL), phenylmercuric chloride (1.25 µg/mL), and HgCl and $HgCl_2$ ($1.25 µg/mL). Post-treatment with methylmercuric chloride and ethylmercuric chloride increased the number of breakage and exchange-type aberrations induced by 4-nitroquinoline 1-oxide and methylmethane sulfonate but they did not show any clastogenic effects by themselves. Dimethylmercury, diethylmercury, mercurous chloride, and mercuric chloride did not show any potentiating effects. Following pretreatment with the 4-nitroquinoline 1-oxide or the DNA cross-linking agent mitomycin C, treatment with methylmercuric chloride during the G1 phase resulted in the enhancement of both breakage- and exchange-type aberrations. Ethylmercuric chloride treatment during the G1 phase also enhanced both types of aberrations induced by 4-nitroquinoline 1-oxide, but did not show any potentiating effect. When treatment was during the G2 phase, however, both methylmercuric chloride and ethylmercuric chloride enhanced breakage-type aberrations only. In the Yamada et al. (1993) study, the dialkyl mercury compounds dimethylmercury and diethylmercury did not show any cytotoxicity at 5–40 µg/mL, but they did cause a significant increase in the frequency of aberrant cells at the 40 µg/mL concentration. The authors of this study suggested three possible mechanisms for the observed potentiation of clastogenicity by monoalkylated mercurials: (1) they interfere with the repair of base lesions induced by 4-nitroquinoline 1-oxide and mitomycin C during the prereplication stage, thus increasing unrepaired DNA lesions that subsequently convert into DNA double-strand breaks in the S phase; (2) methylmercuric chloride (but not ethylmercuric chloride) inhibits the repair of cross-linking lesions during the prereplication stage; and (3) their G2 effects enhance breakage-type aberrations only. Yamada et al. (1993) concluded that because mercury compounds are known to react with protein thiol groups to inhibit protein activity, it is possible that they also inhibit some protein activities involved in the DNA repair process. The specific target protein for organomercurials and why the potentiation activities of methylmercury chloride and ethylmercury chloride differ remain to be identified.

There is a sizable database of studies investigating the DNA-damaging activity of mercuric chloride. The finding that mercuric chloride can damage DNA in rat and mouse embryo fibroblasts (Zasukhina et al. 1983), supports the *in vivo* evidence of species- and intraspecies-specific sensitivity to the genotoxic action of mercuric chloride. Marked conversion of DNA into the single-stranded form occurred at 10^{-6} M

mercuric chloride in rat fibroblasts, while 5×10^{-6} M mercuric chloride produced a comparable response in C57BL/6 mouse cells; at this level, the response in CBA mouse cells was marginal. Mercuric chloride can also bind to the chromatin of rat fibroblasts (Rozalski and Wierzbicki 1983) and Chinese hamster ovary cells (Cantoni et al. 1984a, 1984b; Christie et al. 1984, 1985). Using the alkaline elution assay with intact Chinese hamster ovary cells, several studies have demonstrated that mercuric chloride induces single-strand breaks in DNA (Cantoni and Costa 1983; Cantoni et al. 1982, 1984a, 1984b; Christie et al. 1984, 1985). Furthermore, Cantoni and Costa (1983) found that the DNA-damaging potential of mercuric chloride is enhanced by a concurrent inhibition of DNA repair mechanisms. Methylmercuric chloride induced single-strand breaks in the DNA of intact rat glioblastoma cells, Chinese hamster V79 (fetal lung) cells, human lung cells, and human nerve cells (Costa et al. 1991). Results of the *Bacillus subtilis* rec-assay (Kanematsu et al. 1980) and the sister chromatid exchange assay (Howard et al. 1991) provide additional support to the body of evidence suggesting that mercuric chloride is genotoxic. However, there is no clear evidence that mercury would cause DNA damage *in vivo*.

Two organic mercury compounds (methylmercury chloride at 0.08–0.4 µg Hg/mL and methoxyethyl mercury chloride at 0.04–0.23 µg Hg/mL) induced weak but dose-related mutagenic responses in Chinese hamster V-79 cells near the cytotoxic threshold (Fiskesjo 1979). Methylmercury was neither mutagenic nor caused recombination in *Saccharomyces cerevisiae*, but it did produce a slight increase in the frequency of chromosomal nondisjunction (Nakai and Machida 1973). Both methylmercury and phenylmercuric acetate induced primary DNA damage in the *B. subtilis* rec-assay (Kanematsu et al. 1980).

In contrast, high concentrations of methylmercury (1 or 2 µm) did not increase the frequency of sister chromatid exchanges in cultured blastocysts of early ICR mouse embryos (Matsumoto and Spindle 1982). Severe toxicity, which was more intense in blastocysts than in morulae, consisted of cessation of preimplantation development, blastocoel collapse, and mitotic delay.

In summary, the body of evidence showing the induction of primary DNA damage in mammalian and bacterial cells and weak mutagenesis in mammalian cells suggests that inorganic and organic mercury compounds have some genotoxic potential. Although the data on clastogenesis are less consistent, recent well conducted studies suggest that mercury compounds can be clastogenic. Refer to Table 2-12 for a further summary of these results.

Cancer. Mercury has not been determined to be carcinogenic in humans (Cragle et al. 1984; Kazantzis 1981). An excess of lung cancer (type not specified) was found in Swedish chloralkali workers, but these workers had also been exposed to asbestos (Barregard et al. 1990). A significant association between the farm use of mercury-containing fungicides and lymphocytic leukemia in cattle was presented by Janicki et al. (1987). However, this study is limited because exposure to other chemicals was not adequately addressed and risk estimates were not adjusted for other risk factors for leukemia.

Animal data, however, suggest that mercuric chloride, and methylmercuric chloride, phenylmercuric acetate are tumorigenic in rats and/or mice. In a 2-year NTP (1993) study, male Fischer 344 rats administered mercuric chloride by gavage had an increased incidence of squamous cell papillomas of the forestomach and an increased incidence of thyroid follicular cell carcinomas at 3.7 mg Hg/kg/day. There is equivocal evidence of carcinogenicity in female rats (a nonsignificant incidence of squamous cell papillomas) and in male $B6C3F_1$ mice (a nonsignificant incidence of renal tubule adenomas and carcinomas). Dietary exposure of ICR and $B6C3F_1$ mice to methylmercuric chloride resulted in significant increases in the incidences of renal epithelial cell adenomas and/or carcinomas in males at doses as low as 0.69–0.73 mg Hg/kg/day (Hirano et al. 1986; Mitsumori et al. 1981, 1990). Similar increases were not observed in females. Renal cell adenomas were also significantly increased in male Wistar rats that received 4.2 mg Hg/kg/day as phenylmercuric acetate in their drinking water (Solecki et al. 1991). This study is limited, however, because an insufficient number of animals were tested to adequately assess carcinogenicity.

Swiss mice were exposed for 15 weeks to drinking water containing methylmercuric chloride at concentrations of 0.038, 0.095, and 0.38 mg Hg/kg/day (Blakley 1984). Urethane (1.5 mg/g) was subsequently given intraperitoneally to the mice at week 3 of the study. Methylmercury exposures of 0.038 and 0.095 mg Hg/kg/day produced a significant increase in the incidence of urethane-induced pulmonary adenomas. The author suggested that methylmercury enhances the formation of pulmonary adenomas and that the immunosuppressive activity of methylmercury may be partially responsible for this tumor-enhancing effect. No other studies were located regarding carcinogenic effects in animals following oral exposure to mercury.

The Department of Health and Human Services (DHHS), and the International Agency for Research on Cancer (IARC) have not classified mercury as to its human carcinogenicity. The Environmental Protection Agency has determined that mercury chloride and methylmercury are possible human carcinogens.

More on Health Effects and Dental Amalgam.

A number of government sponsored scientific reviews of the literature on the health effects associated with the use of dental amalgam have concluded that the data do not demonstrate a health hazard for the large majority of individuals exposed to mercury vapor at levels commonly encountered from dental amalgam (DHHS 1993; Health Canada 1997). Governments that have restricted the use of amalgam or recommend limited use (e.g., Germany, Sweden, Denmark, and Canada) cite the need to minimize human exposure to all forms of mercury as much as possible and to reduce the release of mercury to the environment (DHHS 1993; Health Canada 1997). The restrictive actions, however are prospective, and none of the government reports recommend removing existing fillings in people who have no indication of adverse effects attributable to mercury exposure. Removal of existing amalgams, if improperly performed or not indicated, may result in unnecessarily high exposure to mercury. Levels of mercury release for various dental procedures have been reported by Eley (1997). Chelation therapy (used to remove metals from the body tissues) also may have adverse health effects (and varying levels of effectiveness), and should be considered only in consultation with a qualified physician.

In 1990 in the United States, over 200 million restorative procedures were provided of which dental amalgam accounted for roughly 96 million (DHHS 1993). In the 1970s, the use of amalgam was 38% higher. The use of mercury amalgam has been steadily declining and is expected to continue to decline due to improvements in dental hygiene and preventive care. Approximately 70% of the restorations placed annually are replacements. Advocates of the safety of amalgam emphasize the long history of use (over 150 years) and the large exposed population without apparent adverse effects as strong support for their position (ADA 1997). They also underscore the poor quality of the studies in the literature reporting adverse effects attributable to amalgam. Researchers concerned about the safety of mercury amalgams counter that sample sizes in the studies that support the safety of amalgams are also too small to detect low frequency effects in the general population, and that the absence of high quality studies simply reflects the relatively small amount of research effort that has gone into resolving this very important issue (Richardson 1995; Weiner et al. 1990).

The general public is also clearly concerned about the placement of mercury, a substance with demonstrated toxic effects, into their mouths. A survey conducted by the American Dental Association in 1991 demonstrated that nearly half of the 1,000 American adults surveyed believed that health problems could develop as a result of dental amalgam (ADA 1991). Increases in life expectancy and increases in the

numbers of older adults who still have their permanent teeth will result in longer mercury exposure durations from dental amalgam, which may result in new or increased severity of effects. Recent improvements in neurological measures of performance (especially cognitive and behavior tests) as well as immunological assays have also improved the ability to resolve more subtle or preclinical effects. In this context, DHHS (1993) and other summary reports on the health risks from the use of mercury amalgam generally support the need for further investigations.

Additional recommendations concerning the use of dental amalgam include minimizing exposure to populations susceptible to mercury toxicity including pregnant women and nursing women (to minimize the exposure to their developing young), young children up to the age of 6 (and especially up to the age of 3), people with impaired kidney function, and people with hypersensitive immune response to mercury. People who have higher than average exposures to mercury from other sources (e.g., people who consume large quantities of fish or who work in professions that expose them to mercury) should also consider their total mercury exposure in making their life style and health care decisions. In all cases, the choice not to use mercury amalgam should be made in consultation with a qualified dentists (and/or physician) and weighed against the risk of alternative practices and materials.

The DHHS (1993) report also strongly recommends educating the public on the risks and benefits of dental amalgam. To prevent misleading or unduly alarming the public, the layperson should be informed that the presence of metallic mercury in dental amalgams is, in itself, not sufficient to produce an adverse health effect. Toxic levels of mercury must first be released from the filling, absorbed into the body, and transported to target tissues where adverse effects are produced. What constitutes a “toxic level” from an amalgam exposure has been the focus of recent research. Uncertainty continues concerning the presence or absence of a threshold for adverse effects from low level chronic exposure to mercury. The above mentioned inadequacies in study size, the measures used for effects, the reproducibility of the results, and the subjective nature of some of the low level effects have precluded a consensus in the scientific community on the safety of mercury amalgam. In the absence of clearly defined toxicity from low level exposures, one approach has been to focus upon determining exposure levels from mercury amalgam, and whether these levels exceed recommended guidelines or regulations. Since these guidelines and regulations (including the MRL) are themselves extrapolated from the hazardous effects literature, there is some circularity in the argument that exposures of mercury from amalgam that exceed guidelines like the MRL (or other standard) “support” the position that mercury amalgams pose a health risk. This aspect of the

controversy will only be satisfactorily resolved with better toxicity and pharmacokinetic data for chronic low level mercury exposure from amalgams.

People who are concerned that their mercury exposure may be causing adverse effects can be tested for allergies to mercury or to other metals, or for the amount of mercury in their body. Tests that measure the amounts of mercury in hair and urine are available and provide some indication of the potential for adverse effects from mercury. For more information about the tests that are available, see Section 2.7, Biomarkers of Exposure and Effects.

The following studies supporting or refuting the adverse health effects from exposure to dental amalgam provide some examples from the recent literature of effects being evaluated and the procedures that are being used. Some of the studies depend upon the self-reporting of symptoms or may be weakly blinded (i.e., the patients were not completely unaware of the assignment to different exposure groups) which could bias the outcome, especially with respect to some of the end points. An exhaustive analysis of the results presented below, however, is beyond the scope of this profile, and the reader is referred to the cited references for a more complete discussion of the issues concerning the potential adverse effects from exposure to dental amalgam.

Studies reporting no association between adverse effects and mercury amalgam.

Berglund and Molin (1996) evaluated whether a group of patients with symptoms, self-related to their amalgam restorations, experienced an exposure to mercury vapor from their amalgam restorations that reached the range at which subtle symptoms have been reported in the literature. They further evaluated whether the mercury exposure for these patients was significantly higher than for controls with no reported health complaints. The symptom group consisted of 10 consecutively selected patients from a larger group. The larger group consisted of patients who were referred by their physicians for an investigation of a correlation between subjective symptoms and amalgam restorations. The control group consisted of 8 persons with no reported health complaints. The intra-oral release of mercury vapor was measured between 7:45 a.m. and 9:00 p.m. at intervals of 30–45 minutes, following a standardized schedule. The mercury levels in plasma, erythrocytes, and urine were also determined. The calculated daily uptake of inhaled mercury vapor, released from the amalgam restorations, was less than 5% of the daily uptake calculated at the lower concentration range given by the WHO (1991), at which subtle symptoms have been found in particularly sensitive individuals. The symptom group had neither a higher estimated daily uptake

of inhaled mercury vapor nor a higher mercury concentration in blood and urine than in the control group. The study provided no scientific support for the belief that the symptoms of the patients examined originated from an enhanced mercury release from their amalgam restorations.

Bagedahl-Strindlund et al. (1997) evaluated Swedish patients with illnesses thought to be causally related to mercury release during dental restorations, and mapped the psychological/psychiatric, odontological, and medical aspects of the patients and their purportedly mercury-induced symptoms. A total of 67 consecutive patients and 64 controls matched for age, sex, and residential area were included in the study. Questionnaires were completed and a semi-structured psychiatric interview performed. The Comprehensive Psychopathological Rating Scale was used to record psychopatholgical symptoms. The Karolinska Scales of Personality (KSP) set was used to assess personality traits. The Toronto Alexithymia Scale and the Schalling-Sifneos Personality Scale were completed. The Whitely Index was used to assess hypochondriacal attitudes. The type and number of amalgam-filled surfaces was determined. The most striking result was the high prevalence of psychiatric disorders (predominantly somatoform disorders) in the patients (89%) compared to the controls (6%). The personality traits differentiating the patients according to the Karolinska Scales of Personality were somatic anxiety, muscular tension, psychasthenia, and low socialization. More patients than controls showed alexithymic traits. The prevalence of diagnosed somatic diseases was higher, but not sufficiently so to explain the large difference in perceived health. The multiple symptoms and signs of distress displayed by the patients could not be explained either by the odontological data or by the medical examination. These data indicate that the patients show sociodemographic and clinical patterns similar to those of somatizing patients. The number of amalgam-filled surfaces did not differ significantly between patients and controls; 19% of the patients lacked amalgam fillings.

Grandjean et al. (1997a) evaluated the effects of chelation therapy versus a placebo on patient improvement for patients who attribute their environmental illness to mercury from amalgam fillings. Succimer (meso-2, 3-dimercaptosuccinic acid) was given at a daily dose of 30 mg/kg for 5 days in a double-blind, randomized placebo-controlled trial. Treatment of patients who attribute their environmental illness to mercury from amalgam fillings is largely experimental. On the Symptom Check List, overall distress, and somatization, obsessive-compulsive, depression, and anxiety symptom dimensions, were increased in 50 consecutive patients examined, and Eysenck Personality Questionnaire scores suggested less extroversion and increased degree of emotional lability. Urinary excretion of mercury and lead was considerably increased in the patients who received the chelator. Immediately after the treatment and 5–6 weeks later, most distress

dimensions had improved considerably, but there was no difference between the succimer and placebo groups. These findings suggest that some patients with environmental illness may substantially benefit from placebo.

Stoz et al. (1995) studied 185 mothers with tooth amalgam filling surfaces ranging from 0 to 780 mm^2 and found no relationship between the blood values of the women and their children and the size of the surfaces of the amalgam fillings. All mothers gave birth to healthy children. Malt et al. (1997) evaluated the physical and mental symptomatology of 99 self-referred adult patients complaining of multiple somatic and mental symptoms attributed to dental amalgam fillings. These patients were compared with patients with known chronic medical disorders seen in alternative (n=93) and ordinary (n=99) medical family practices and patients with dental amalgam fillings (n=80) seen in an ordinary dental practice. The assessments included written self-reports, a 131-item somatic symptom checklist, Eysenck Personality Questionnaire, the General Health Questionnaire, and Toronto Alexithymia Scale. Somatic symptom complaints were categorized by exhaustion, and musculoskeletal, cardiovascular, and gastrointestinal effects. The mean number of silver fillings surfaces were 40.96 in self-referrents as compared to 36.61 in the dental practice patients. No correlation between number of dental fillings and symptomatology was found. Self-reports suggested that 62% suffered from chronic anxiety. Forty-seven percent suffered from major depression compared with none in the dental control sample. Symptoms suggesting somatization disorder were found in 29% of the dental amalgam sample compared with only one subject in the 272 comparison subjects; 37.5% of the dental amalgam patients reported symptoms of chronic fatigue syndrome compared with none in the dental control sample and only 2 and 6%, respectively, in the two clinical comparison samples. The dental amalgam group reported higher mean neuroticism and lower lie scores than the comparison groups. The authors concluded that self-referred patients with health complaints attributed to dental amalgam are a heterogeneous group of patients who suffer multiple symptoms and frequently have mental disorders. The authors report a striking similarity with the multiple chemical sensitivity syndrome.

An ad hoc review group of the DHHS Working Group on Dental Amalgam examined 175 literature articles concerning mercury amalgam (DHHS, 1997). The articles represented an assortment of literature from peer-reviewed journals and a variety of other print media. None of the 12 expert reviewers evaluating the articles suggested that any study under review would indicate that individuals with dental amalgam restorations would experience adverse health effects. Many of the reviewed articles were reported to suffer from inadequacy of experimental control, lack of dose-response information, poor measurement of exposure, and a variety of other experimental design inadequacies.

Studies that report an association between dental amalgam and adverse effects.

Echeverria et al. (1995) evaluated the behavioral effects of low-level exposure to Hg among dentists who had either been exposed to mercury or not as measured in a selection procedure where the exposed group was defined as those with urinary mercury levels greater than 19 µg/L. Exposure thresholds for health effects associated with elemental mercury exposure were examined by comparing behavioral test scores of 19 exposed (17 males, 2 females) with those of 20 unexposed dentists (14 males, 6 females). The mean urinary Hg of exposed dentists was 36.4 µg/L, which was 7 times greater than the 5 µg Hg/L mean level measured in a national sample of dentists (urinary Hg was below the level of detection in unexposed dentists for this study). To improve the distinction between recent and cumulative effects, the study also evaluated porphyrin concentrations in urine, which are correlated with renal Hg content (a measure of cumulative body burden). Significant urinary Hg dose-effects were found for poor mental concentration, emotional lability, somatosensory irritation, and mood scores (tension, fatigue, confusion). Individual tests evaluating cognitive and motor function changed in the expected directions but were not significantly associated with urinary Hg. However, the pooled sum of rank scores for combinations of tests within domains were significantly associated with urinary Hg, providing evidence of subtle preclinical changes in behavior associated with Hg exposure. Coproporphyrin, one of three urinary porphyrins altered by mercury exposure, was significantly associated with deficits in digit span and simple reaction time. Exposed dentists placed significantly more amalgams per week (28.0) than unexposed dentists (19.8). No significant differences were found between exposed and unexposed dentists for the overall number of years in practice or the number of amalgams removed per week.

Altmann et al. (1998) compared visual functions in 6-year-old children exposed to lead and mercury levels, in a cohort of 384 children (mean age 6.2 years) living in three different areas of East and West Germany. After adjusting for confounding effects, statistically significant lead-related changes were found only for some of the visually evoked potentials (VEP) interpeak latencies, while some of the contrast sensitivity values were significantly reduced with increasing mercury concentrations. All other outcome variables were not significantly related to lead or mercury levels. The authors concluded that even at blood lead levels in the range of 14–174 µg/L and at very low urinary mercury levels subtle changes in visual system functions can be measured. The geometric means of urinary mercury concentrations were 0.161, 0.203, and 0.075 µg Hg/24 hours for subjects of the three study areas (0.157 µg Hg/24 hours for the total study); the average numbers of amalgam fillings were 0.76, 1.10, and 1.88, respectively (1.15 amalgam fillings for the total study).

Siblerud and Kienholz (1997) investigated whether mercury from silver dental fillings (amalgam) may be an etiological factor in multiple sclerosis (MS). Blood findings were compared between MS subjects who had their amalgams removed (n=50) and MS subjects with amalgams (n=47). All subjects filled out a health survey, an MS health questionnaire, and a psychological profile; the MS amalgam removal group completed a health questionnaire comparing their health before and after amalgam removal. MS subjects with amalgams were found to have significantly lower levels of red blood cells, hemoglobin, and hematocrit compared to MS subjects with amalgam removal. Thyroxine (T-4) levels were also significantly lower in the MS amalgam group, which had significantly lower levels of total T-lymphocytes and T-8 (CD8) suppressor cells. The MS amalgam group had significantly higher blood urea nitrogen (BUN) and BUN/creatinine ratio, and lower serum IgG. Hair mercury was significantly higher in the MS subjects compared to the non-MS control group (2.08 versus 1.32 ppm). A health questionnaire found that MS subjects with amalgams had significantly more (33.7%) exacerbations during the past 12 months compared to the MS volunteers with amalgam removal: 31% of MS subjects felt their MS got better after amalgam removal, 7% felt it was eliminated, 33% felt no change, and 29% believed the condition got worse. In addition, 17% of the MS with amalgam group had more neuromuscular symptoms compared to the amalgam removal group.

Björkman et al. (1997) examined the mercury concentrations in saliva, feces, urine, whole blood, and plasma before and after removal of dental amalgam fillings in 10 human subjects. Before removal, the median mercury concentration in feces was more than 10 times higher than in samples taken from an amalgam-free reference group of 10 individuals. Two days following removal of all amalgams, a considerable increase in mercury appeared in the feces. This initial increase was followed by a significant decrease. In saliva, there was an exponential decline in the mercury concentration during the first 2 weeks after amalgam removal ($t_{1/2}$ of 1.8 days). The authors concluded that while mercury amalgam fillings are a significant source of mercury in saliva and feces, those levels decrease considerably following amalgam removal. Further, the gastrointestinal uptake of mercury seen in conjunction with removal of amalgam fillings appears to be low. Of 108 patients (all with amalgam dental fillings) presenting to an environmental toxicology service, the average salivary mercury level was 11 µg/L (range, <1–19 µg/L) before chewing and 38 µg/L (range, 6–500 µg/L) after chewing. Six of the 108 patients had salivary mercury concentrations >100 µg/L. Of 58 patients with suspected allergic disease, an epicutaneous test for amalgam was positive in 32 of them; however, direct involvement of dental amalgams in these sensitivities was not mentioned. Seventy-five of the total patients presenting with symptoms felt that amalgam fillings

or other dental materials were responsible, at least in part, for their symptoms, although no causal relationship was borne out by medical evaluation.

Bratel et al. (1996) investigated (1) healing of oral lichenoid reactions (OLR) following the selective replacement of restorations of dental amalgam, (2) whether there were differences in healing between contact lesions (CL) and oral lichen planus (OLP), and (3) whether there was a difference in healing potential when different materials were selected as a substitute for dental amalgam. Patients included in the study presented with OLR confined to areas of the oral mucosa in close contact with amalgam restorations (CL; n=142) or with OLR which involved other parts of the oral mucosa as well (OLP; n=19). After examination, restorations of dental amalgam which were in contact with OLR in both patient groups were replaced. The effect of replacement was evaluated at a follow-up after 6–12 months. In the CL group, the lesions showed a considerable improvement or had totally disappeared in 95% of the patients after replacement of the restorations of dental amalgam (n=474). This effect was paralleled by a disappearance of symptoms, in contrast to patients with persisting CL (5%) who did not report any significant improvement. The healing response was not found to correlate with age, gender, smoking habits, subjective dryness of the mouth or current medication. However, the healing effect in patients who received gold crowns was superior than in patients treated with metal-ceramic crowns (MC) ($p<0.05$). In the OLP group (n=19), 63% of the patients with amalgam-associated erosive and atrophic lesions showed an improvement following selective replacement. OLP lesions in sites not in contact with amalgams were not affected. Most of the patients (53%) with OLP reported symptoms also after replacement. From these data the authors conclude that in the vast majority of cases, CL resolves following selective replacement of restorations of dental amalgam, provided that a correct clinical diagnosis is established. The authors note that MC crowns did not facilitate healing of CL to the same extent as gold crowns.

Hultman et al. (1994) studied the effects of dental amalgams in in-bred mice genetically susceptible to mercury-induced immunotoxic effects. Following intraperitoneal implantation of a silver amalgam and observation for up to 6 months, chronic hyperimmunoglobulinemia, serum IgG autoantibodies targeting the nucleolar protein fibrillarin, and systemic immune-complex deposits developed in both a time- and dose-dependent manner. The functional capacity of splenic T- and B-cells was affected in a dose-dependent fashion. In this study, not only did the dental amalgam implantation cause chronic stimulation of the immune system with induction of systemic autoimmunity, but the implantation of silver alloy not containing mercury also induced autoimmunity, suggesting that other metals have the potential to induce autoimmunity in that genetically susceptible strain of mice. Accumulation of heavy metals from dental amalgams, as well

as from other sources, may lower the threshold of an individual metal to elicit immune aberrations, which could lead to overt autoimmunity.

2.6 CHILDREN'S SUSCEPTIBILITY

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans, when all biological systems will have fully developed. Potential effects on offspring resulting from exposures of parental germ cells are considered, as well as any indirect effects on the fetus and neonate due to maternal exposure during gestation and lactation. Relevant animal and *in vitro* models are also discussed.

Children are not small adults. They differ from adults in their exposures and may differ in their susceptibility to hazardous chemicals. Children's unique physiology and behavior can influence the extent of their exposure. Exposures of children are discussed in Section 5.6, Exposures of Children.

Children sometimes differ from adults in their susceptibility to hazardous chemicals, but whether there is a difference depends on the chemical (Guzelian et al. 1992; NRC 1993). Children may be more or less susceptible than adults to health effects, and the relationship may change with developmental age (Guzelian et al. 1992; NRC 1993). Vulnerability often depends on developmental stage. There are critical periods of structural and functional development during both pre-natal and post-natal life and a particular structure or function will be most sensitive to disruption during its critical period(s). Damage may not be evident until a later stage of development. There are often differences in pharmacokinetics and metabolism between children and adults. For example, absorption may be different in neonates because of the immaturity of their gastrointestinal tract and their larger skin surface area in proportion to body weight (Morselli et al. 1980; NRC 1993); the gastrointestinal absorption of lead is greatest in infants and young children (Ziegler et al. 1978). Distribution of xenobiotics may be different; for example, infants have a larger proportion of their bodies as extracellular water and their brains and livers are proportionately larger (Altman and Dittmer 1974; Fomon 1966; Fomon et al. 1982; Owen and Brozek 1966; Widdowson and Dickerson 1964). The infant also has an immature blood-brain barrier (Adinolfi 1985; Johanson 1980) and probably an immature blood-testis barrier (Setchell and Waites 1975). Many xenobiotic metabolizing enzymes have distinctive developmental patterns and at various stages of growth and development, levels of particular enzymes may be higher or lower than those of adults and sometimes unique enzymes may exist at particular developmental stages (Komori et al. 1990; Leeder and Kearns 1997; NRC 1993; Vieira et al. 1996).

Whether differences in xenobiotic metabolism make the child more or less susceptible also depends on whether the relevant enzymes are involved in activation of the parent compound to its toxic form or in detoxification. There may also be differences in excretion, particularly in the newborn who has a low glomerular filtration rate and has not developed efficient tubular secretion and resorption capacities (Altman and Dittmer 1974; NRC 1993; West et al. 1948). Children and adults may differ in their capacity to repair damage from chemical insults. Children also have a longer lifetime in which to express damage from chemicals; this potential is particularly relevant to cancer.

Certain characteristics of the developing human may increase exposure or susceptibility while others may decrease susceptibility to the same chemical. For example, the fact that infants breathe more air per kilogram of body weight than adults may be somewhat counterbalanced by their alveoli being less developed, so there is a disproportionately smaller surface area for absorption (NRC 1993).

Adverse health effects from different forms of mercury differ primarily because of differences in kinetics rather than mode of action. As discussed in the introduction to this section, children have different, and sometimes dramatically different, morphology or physiology that alters the way toxic compounds are absorbed and distributed throughout their bodies. For mercury compounds, preventing entry into the systemic circulation is the best means to prevent adverse effects. Once mercury enters the circulation, the tissues that end up as target sites are those that accumulate the most mercuric ion or the ones that are most often exposed to mercuric ion. That is why the kidney is a prime target site, for in fulfilling its major role of filtering and purifying the blood, the kidney is continually exposed to ionic mercury. The central nervous system is a major target site because mercuric ion also concentrates in the brain compartment. Ironically, it may be the blood-brain barrier that contributes to, rather than prevents, mercuric ion "trapping" in the brain. A current hypothesis is that once lipophilic forms of mercury cross the blood-brain barrier, they are oxidized to more hydrophilic species and become trapped inside the brain compartment. This "one way" only kinetic pathway results in continually increasing brain mercuric ion levels, as long as nonpolar forms are in the blood stream. Even small amounts of nonpolar mercury (<2 g) in the body may eventually lead to central nervous system damage (Neirenberg et al. 1998). The low capacity for central nervous system tissues to regenerate and the fact that even subtle damage to small areas of the brain can have profound overall effects, makes this tissue very susceptible to the highly toxic mercuric ion. These factors, and a slow but inevitable trapping of mercuric ions may lead to the mercury-induced delayed central nervous system toxicity observed months to years after exposure ceases (Neirenberg et al. 1998, Rice 1996a). Even potent chelators have not been effective in interfering with progressive central nervous

system damage once a nonpolar mercury compound gains access to the circulatory system and begins to concentrate in tissues (Neirenberg et al. 1998, Taueg et al. 1992).

For similar routes and forms of mercury, the adverse health effects seen in children are similar to the effects seen in adults. For example, a young child who was intoxicated with mercury vapor, died of pulmonary edema and had a grayish, necrotic mucosa of the stomach and duodenum (Campbell 1948). These effects are similar to those seen in adult populations occupationally exposures to inhaled metallic mercury vapors. Respiratory effects in adults from inhalation of metallic mercury vapor include pulmonary edema, lobar pneumonia, fibrosis, desquamation of the bronchiolar epithelium, and death in severe cases due to respiratory failure (Gore and Harding 1987; Jaffe et al. 1983; Kanluen and Gottlieb 1991; Matthes et al. 1958; Taueg et al. 1992; Teng and Brennan 1959; Tennant et al. 1961).

The majority of the information regarding cardiovascular effects comes from reports of children who were treated with mercurous chloride tablets for worms or mercurous chloride-containing powders for teething discomfort (Warkany and Hubbard 1953). These authors described multiple cases in which tachycardia and elevated blood pressure were observed in the affected children.

Electrocardiography in four family members who ate meat from a hog that had consumed seed treated with ethylmercuric chloride showed abnormal heart rhythms (ST segment depression and T wave inversion) (Cinca et al. 1979). Death of the two children in the family was attributed to cardiac arrest, and autopsy of these boys showed myocarditis. Cardiovascular abnormalities were also observed in severe cases of poisoning in the Iraqi epidemic of 1956, when widespread poisoning resulted from eating flour made from seed grains treated with ethylmercury *p*-toluene sulfonanilide (Jalili and Abbasi 1961). These abnormalities included irregular pulse, occasionally with bradycardia, and electrocardiograms showing ventricular ectopic beats, prolongation of the Q-T interval, depression of the S-T segment, and T inversion.

Several children who were treated with mercurous chloride for constipation, worms, or teething discomfort had swollen red gums, excessive salivation, anorexia, diarrhea, and/or abdominal pain (Warkany and Hubbard 1953). They also experienced muscle twitching or cramping in the legs and/or arms, but these muscular effects were probably secondary to changes in electrolyte balance (i.e., potassium imbalance due to fluid loss or renal wasting).

Acute renal failure that persisted for 10 days was observed in a 19-month-old child who ingested an unknown amount of powdered mercuric chloride (Samuels et al. 1982). Several children who were treated with medications containing mercurous chloride for constipation, worms, or teething discomfort exhibited flushing of the palms of the hands and soles of the feet (Warkany and Hubbard 1953). The flushing was frequently accompanied by itching, swelling, and desquamation of these areas. Morbilliform rashes, conjunctivitis, and excessive perspiration were also frequently observed in the affected children. Patch tests conducted in several children revealed that the rashes were not allergic reactions to the mercury. They also had irritability, fretfulness, sleeplessness, weakness, photophobia, muscle twitching, hyperactive or hypoactive tendon reflexes, and/or confusion.

A 13-month-old child who ingested porridge made from flour that had been treated with an alkyl mercury compound (specific mercury compound not reported) developed a measles-like rash, fever, and facial flushing (Engleson and Herner 1952). A 4-year-old boy who had been given a Chinese medicine containing mercurous chloride for 3 months developed drooling, dysphagia, irregular arm movements, and impaired gait (Kang-Yum and Oransky 1992). A number of children who were treated with an ammoniated mercury ointment or whose diapers had been rinsed in a mercuric chloride solution experienced tachycardia and elevated blood pressure, and anorexia (Warkany and Hubbard 1953).

In addition, rashes, conjunctivitis, and/or excessive perspiration were observed. These dermal and ocular reactions were not attributed to allergic-type reactions to the mercury. A 23-month-old boy who was exposed to an unspecified form of mercury also developed a "diffuse, pinpoint, erythematous, papular rash" and bright red finger tips "with large sheets of peeling skin" (Tunnessen et al. 1987).

A woman chronically exposed to an undetermined concentration of mercury vapor reported that her first pregnancy resulted in spontaneous abortion, and her second resulted in the death of the newborn soon after birth (Derobert and Tara 1950). It is unclear whether the reproductive toxicity experienced by the woman was due to the mercury exposure. However, after recovery from overt mercury poisoning, she gave birth to a healthy child. Not all exposures lead to immediate adverse effects. A woman occupationally exposed to mercury vapors for 2 years prior to pregnancy and throughout pregnancy was reported to have delivered a viable infant at term (Melkonian and Baker 1988). Urinary mercury in the woman at 15 weeks of pregnancy was 0.875 mg/L (normal levels are approximately 0.004 mg/L). A case report of a woman exposed to mercury vapors in her home during the first 17 weeks of pregnancy reported that the woman delivered a normal child who met all developmental milestones (although the child was not formally tested

for psychological development) (Thorpe et al. 1992). Mercury exposure was not measured, but the child was born with hair levels of 3 mg/kg (3 ppm) of mercury. This hair level was comparable to that observed in populations consuming fish once a week (WHO 1990) and suggests that exposure in this case may have been relatively low.

In the *in vivo* study by Sager et al. (1982), it was concluded that methylmercury may be acting on mitotic spindle microtubules leading to cell injury in the developing cerebellar cortex. Cell injury observed in the external granular layer of the cerebellar cortex of 2-day-old rats was attributed to a reduced percentage of late mitotic figures (arrested cell division) due to the loss of spindle microtubules. Mitosis and migration of granule cells in the cerebellum end within weeks following birth; therefore, this observation may suggest potential differences in the sensitivities of children and adults to mercury-induced neurotoxicity.

Regardless of whether mercury exposure is through inhalation of mercury vapors, ingestion of organic mercury or mercury salts, or dermal application of mercury-containing ointments, patients (primarily children) may exhibit a syndrome known as acrodynia, or pink disease. Acrodynia is often characterized by severe leg cramps; irritability; and erythema and subsequent peeling of the hands, nose, and soles of the feet. Itching, swelling, fever, tachycardia, elevated blood pressure, excessive salivation or perspiration, morbilliform rashes, fretfulness, sleeplessness, and/or weakness may also be present. It was formerly thought that this syndrome occurred exclusively in children, but recent reported cases in teenagers and adults have shown that these groups are also susceptible.

Developmental effects from prenatal or postnatal exposures to mercury are unique to children. During critical periods of structural and functional development in both prenatal and postnatal life, children are especially vulnerable to the toxic effects of mercury. Inhalation exposures are relatively rare outside of the occupational setting so the exposure route and form of mercury most commonly associated with a risk for development effects is the ingestion of methylmercury on the surface of contaminated foods (methylmercury used as a fungicide on seed grain) or accumulated within the food (methylmercury in fish, wild game, and marine mammals). The exposure route and form of mercury most commonly associated with maternal exposures is to foods contaminated with methylmercury fungicides (Bakir et al. 1973) or foods that contain high levels of methylmercury (Grandjean et al. 1997b, 1998; Tsubaki and Takahashi 1986).

The first such incident was reported in Sweden in 1952 when flour from grain treated with an unspecified alkyl mercury compound ingested by a pregnant woman was associated with developmental toxicity. An

apparently normal infant was born, but the infant later displayed brain damage manifested by mental retardation, incoordination, and inability to move (Engleson and Herner 1952). A 40-year-old woman, 3 months pregnant, consumed methylmercury-contaminated meat for an unspecified duration and subsequently delivered a male infant with elevated urinary mercury levels (Snyder and Seelinger 1976). At 3 months, the infant was hypotonic, irritable, and exhibited myoclonic seizures. At 6 years of age, the child displayed severe neurological impairment (e.g., blindness, myoclonic seizures, neuromuscular weakness, inability to speak) (Snyder and Seelinger 1976).

Another incidence of neurodevelopmental effects occurring as a result of *in utero* exposure to methylmercury was reported by Cox et al. (1989) and WHO (1990). The effect of concern was the delayed onset of walking in offspring in Iraqi children whose mothers were exposed to methylmercury through the consumption of seed grain treated with methylmercury as a fungicide (Al-Mufti et al. 1976; Bakir et al. 1973; Cox et al. 1989; Marsh et al. 1981, 1987).

A New Mexico family, including a pregnant woman, a 20-year-old female, and 2 children (a 13-year-old male and an 8-year-old female) ate meat from a hog inadvertently fed seed grain treated with a fungicide containing methylmercury and experienced severe, delayed neurological effects (Davis et al. 1994). Several months after the exposures, the children developed symptoms of neurological dysfunction. The newborn child of the exposed mother showed signs of central nervous system disorder from birth. Twenty-two years after the 3-month exposure period, the people who were 20 and 13 years old at time of exposure had developed cortical blindness or constricted visual fields, diminished hand proprioception, choreoathetosis, and attention deficits. MRI examination of these two revealed residual brain damage in the calcarine cortices, parietal cortices, and cerebellum. The brain of the person who was exposed at age 8 (who died of aspiration pneumonia with a superimposed *Klebsiella* bronchopneumonia and sepsis at age 29) showed cortical atrophy, neuronal loss, and gliosis, most pronounced in the paracentral and parieto-occipital regions. Regional brain mercury levels correlated with the extent of brain damage. The youngest (*in utero* at the time of exposure) developed quadriplegia, blindness, severe mental retardation, choreoathetosis, and seizures, and died at age 21. Since inorganic mercury crosses the blood-brain barrier poorly, biotransformation of the methylmercury to inorganic mercury may have occurred after the methylmercury crossed the blood-brain barrier, accounting for its observed persistence in the brain and its possible contribution to the brain damage.

More recently, Grandjean et al. (1997b, 1998) evaluated a cohort of 1,022 consecutive singleton births generated during 1986–1987 in the Faroe Islands. Increased methylmercury exposure from maternal consumption of pilot whale meat was indicated by mercury concentrations in cord blood and maternal hair. Neurophysiological tests emphasized motor coordination, perceptual-motor performance, and visual acuity; pattern reversal visual evoked potentials (VEP) with binocular full-field stimulation, brain stem auditory evoked potentials (BAEP), postural sway, and the coefficient of variation for R-R interpeak intervals (CVRR) on the electrocardiogram were measured. Clinical examination and neurophysiological testing did not reveal any clear-cut mercury-related abnormalities. However, mercury-related neuropsychological dysfunctions were most pronounced in the domains of language, attention, and memory, and to a lesser extent in visuospatial and motor functions. These associations remained after adjustment for covariates and after exclusion of children of mothers with maternal hair mercury concentrations above 10 μg/g (50 nmol/g). The effects on brain function associated with prenatal methylmercury exposure appear widespread, and early dysfunction is detectable at exposure levels currently considered safe.

There are differences in the outcomes of these epidemiology studies on low level chronic exposures to methylmercury in foods. Davidson et al. (1998) report no adverse developmental effects associated with prenatal and postnatal exposure to methylmercury in fish in a Seychelles Island cohort of children at age 66 months (n=708). The exposure levels are reflected in maternal hair levels of 6.8 ppm for the prenatal exposure (SD=4.5, n=711) and children's hair levels of 6.5 ppm (SD=3.3, n=708) for both the prenatal and subsequent postnatal exposure. The age-appropriate main outcome measures included: (1) the McCarthy Scales of Children's Abilities, (2) the Preschool Language Scale, (3) the Woodcock-Johnson Tests of Achievement - Letter and Word Recognition, (4) Woodcock-Johnson Tests of Achievement - Applied Problems and, (5) the Bender Gestalt test, and (6) the Child Behavior Checklist. The test results were similar to what would be expected from a healthy, well-developing U.S. population. No test indicated a deleterious effect of methylmercury from the exposure levels received in this population. Four of the six measures showed better scores in the highest MeHg groups compared with lower groups for both prenatal and postnatal exposure. This result is likely due to the benefits of increased levels of fish in the diet, possibly because of increased consumption of omega-3-fatty acids. Serum from a subset of 49 of the children was sampled for polychlorinated biphenyl levels (PCBs). None of the samples had detectable levels (detection limit 0.2 ng/mL) for any of the 28 congeners assayed (from congener 28 to 206) indicating that was no concurrent (i.e, potentially confounding) exposure to PCBs in this population. The median level of total mercury for each of 25 species sampled was 0.004–0.75 ppm, with most medians in the range of 0.05–0.25 ppm, levels that are comparable to fish in the U.S. market. The authors conclude that this

most recent NOAEL of 6.8 ppm for the Seychelles cohort at 66 months of age strongly supports the findings at earlier ages, and that the benefits of eating fish outweigh the small risk of adverse effects from an increased exposure to methylmercury for this exposure pathway.

The differences in these studies highlight the importance of interpreting epidemiology results and, indeed, all study results on mercury toxicity within a fairly comprehensive context of the numerous factors that might affect the toxicokinetics and the amount absorbed (e.g., form of mercury, route of exposure, age, diet of population exposed, health status, other potential sources of exposure to mercury, dose duration, constancy of dose amount over time, etc.)

A route of exposure unique to children is breast milk. Both organic and inorganic mercury can move into breast milk from a nursing woman's body, and children will readily absorb this mercury. Oskarsson et al. (1996) assessed the total and inorganic mercury content in breast milk and blood in relation to fish consumption and amalgam fillings (an exposure source for older children). Total mercury concentrations were evaluated in breast milk, blood, and hair samples collected 6 weeks after delivery from 30 lactating Swedish women. In breast milk, about half of the total mercury was inorganic and half was methylmercury, whereas in blood only 26% was inorganic and 74% was methylmercury. That is because, unlike the placental barrier, which is crossed more easily by methylmercury than by inorganic mercury, inorganic mercury moves more easily into breast milk. Some researchers think that a carrier mediated process is involved (Sundberg et al 1998).

For the Swedish population in the study, Oskarsson et al. (1996) reports that there was an efficient transfer of inorganic mercury from blood to breast milk and that mercury from amalgam fillings was probably the main source of mercury in breast milk, while methylmercury levels in blood did not appear to be efficiently transferred to breast milk . Exposure of the infant to mercury in breast milk was calculated to range up to 0.3 µg/kg/day of which approximately one-half was inorganic mercury. This exposure corresponds to approximately one-half the tolerable daily intake of total mercury for adults recommended by the World Health organization. The authors concluded that efforts should be made to decrease total mercury burden in women of reproductive age Oskarsson et al. (1996).

The metabolism of mercury is relatively straightforward compared, for example, to pesticides or some organic solvents. No information was identified to indicate that metabolic pathways are different for children and adults, or that children have unique metabolites. Once absorbed, metallic and inorganic

mercury enter an oxidation-reduction cycle. Metallic mercury is oxidized to the divalent inorganic cation in the red blood cells and lungs of humans and animals. Evidence from animal studies suggests the liver as an additional site of oxidation. Absorbed divalent cation from exposure to mercuric mercury compounds can, in turn, be reduced to the metallic or monovalent form and released as exhaled metallic mercury vapor. In the presence of protein sulfhydryl groups, mercurous mercury (Hg^{+}) disproportionates to one divalent cation (Hg^{+2}) and one molecule at the zero oxidation state (Hg^{0}). The conversion of methylmercury or phenyl-mercury into divalent inorganic mercury can probably occur soon after absorption, also feeding into the oxidation-reduction pathway.

A number of good physiologically based pharmacokinetic models are currently available for mercury, including some that address developmental toxicity and maternal/fetal transfer. Two models were constructed based upon data from the kinetics of methylmercury in rats. Farris et al. (1993) developed a PBPK model that simulates the long-term disposition of methylmercury and its primary biotransformation product, mercuric mercury, in the male Sprague-Dawley rat following a single oral nontoxic exposure. Gray (1995) developed a PBPK model that simulates the kinetics of methylmercury in the pregnant rat and fetus. The Gray model was developed to provide fetal and maternal organ methylmercury concentration-time profiles for any maternal dosing regimen. Sundberg et al. (1998) fitted a three compartment model to the elimination kinetics of methylmercury and inorganic mercury transfer to milk in lactating and nonlactating mice. Luecke et al. (1997) developed a model based on human physiology but extended to simulate animal data that depict internal disposition of two chemicals (singly or in combination) during pregnancy in the mother and the embryo/fetus. Leroux et al. (1996) developed a biologically based-dose-response model to describe the dynamics of organogenesis, based on the branching process models of cell kinetics. Gearhart et al. (1995) developed a PBPK model to coherently describe methylmercury pharmacokinetics in a variety of species (adult rat, monkey, and human), and to predict fetal levels of methylmercury from an i*n utero* exposure.

No information was identified on biomarkers of exposure for children. Mercury levels in hair, urine, and blood are the standard measures of exposure. There are biomarkers for developmental effects that are unique to specific ages and stages of development throughout the child's developmental process. Developing the best measures for evaluation of cognitive functions is an area of intense debate and on-going research.

Concerning interactions with other chemicals, there is an ongoing debate about the value of fish in the diet versus the risk from increased exposure to methylmercury that may be in the fish. One recent study reported a beneficial effect from increased fish consumption even though mercury body burdens were increased to some extent (Davidson et al. 1998). One possible factor in the fish that could improve health is omega 3-fatty acid. Children and adults both benefit from a healthy diet, but there may more emphasis on the benefits to growing children. Other interactions for mercury include the effect of various substances on its gastrointestinal absorption (e.g., iron, zinc) or possibly protective effects from prevention or repair of mercury related oxidative damage (e.g., interactions with selenium as an antioxidant). No information was identified that specifically addresses differences in these interactions for children compared to adults.

The methods used to reduce peak absorption and to reduce body burdens in exposed adults (i.e., chelation therapy) are also used for exposures in children.

No information was identified on parental exposures affecting children in areas of parental germ cells or germ line mutations. The topic of exposure pathways for mercury via nursing or pregnant women who have been exposed is of main concern and has been addressed earlier in this section.

2.7 BIOMARKERS OF EXPOSURE AND EFFECT

Biomarkers are broadly defined as indicators signaling events in biological systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989).

A biomarker of exposure is a xenobiotic substance or its metabolite(s), or the product of an interaction between a xenobiotic agent and some target molecule(s) or cell(s), that is measured within a compartment of an organism (NAS/NRC 1989). The preferred biomarkers of exposure are generally the substance itself or substance-specific metabolites in readily obtainable body fluid(s) or excreta. However, several factors can confound the use and interpretation of biomarkers of exposure. The body burden of a substance may be the result of exposures from more than one source. The substance being measured may be a metabolite of another xenobiotic substance (e.g., high urinary levels of phenol can result from exposure to several different aromatic compounds). Depending on the properties of the substance (e.g., biological half-life) and environmental conditions (e.g., duration and route of exposure), the substance and all of its metabolites may have left the body by the time samples can be taken. It may be difficult to identify individuals exposed

to hazardous substances that are commonly found in body tissues and fluids (essential mineral nutrients [e.g., copper, zinc, and selenium]). Biomarkers of exposure to mercury are discussed in Section 2.7.1.

Biomarkers of effect are defined as any measurable biochemical, physiological, or other alteration within an organism that, depending on magnitude, can be recognized as an established or potential health impairment or disease (NAS/NRC 1989). This definition encompasses biochemical or cellular signals of tissue dysfunction (e.g., increased liver enzyme activity or pathological changes in female genital epithelial cells), as well as physiological signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are not often substance specific. They also may not be directly adverse, but they can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effect caused by mercury are discussed in Section 2.7.2.

A biomarker of susceptibility is an indicator of an inherent or acquired limitation of an organism's ability to respond to the challenge of exposure to a specific xenobiotic substance. It can be an intrinsic genetic or other characteristic or a pre-existing disease that results in an increase in the absorbed dose, a decrease in the biologically effective dose, or a target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 2.8 (Populations That Are Unusually Susceptible).

2.7.1 Biomarkers Used to Identify or Quantify Exposure to Mercury

Blood and urine mercury concentrations are commonly used as biomarkers of exposure to mercury. Hair has been used as a biomarker of exposure to methylmercury. Occupational studies show that recent mercury exposure is reflected in blood and urine (Naleway et al. 1991; WHO 1991). However, at low exposure levels (<0.05 mg Hg/m^3), correlation to blood or urine mercury levels is low (Lindstedt et al. 1979). Blood levels of mercury peak sharply during and soon after short-term exposures, indicating that measurements should be made soon after exposure (Cherian et al. 1978). The specific time frame at which measurements become less reliable has not been determined. Workers exposed for a chronic duration, however, may have a high body burden of mercury, therefore, mercury levels would probably still be elevated in the urine and blood for a long period of time after cessation of exposure (Lindstedt et al. 1979). The following discussion of blood and urine mercury levels generally refers to measurements taken immediately or within a few days following the last exposure.

The mean total mercury levels in whole blood and urine of the general population are approximately 1–8 µg/L and 4–5 µg/L, respectively (Gerhardsson and Brune 1989; WHO 1990). Recently, the International Commission on Occupational Health (ICOH) and the International Union of Pure and Applied Chemistry (IUPAC) Commission on Toxicology determined that a mean value of 2 µg/L was the background blood level of mercury in persons who do not eat fish (Nordberg et al. 1992). These blood and urine levels are "background" in the sense that they represent the average levels in blood in the general population and are not associated with a particular source for mercury. However, the intra- and inter-individual differences in these biomarkers are substantial, possibly due to dental amalgams (urine) and ingestion of contaminated fish (blood) (Verschoor et al. 1988; WHO 1991). Long-term consumption of fish is the source of nearly all of the methylmercury measured in the general population, and individuals in communities with high fish consumption rates have been shown to have blood levels of 200 µg/L, with daily intake of 200 µg mercury (WHO 1990). However, acute inhalation exposure to low levels of metallic mercury resulted in much lower levels in the blood (0.028 and 0.18 µg/100 mL) and urine (from 94 to >438 µg/L) (Kanluen and Gottlieb 1991; Rowens et al. 1991).

Urine mercury measurement is reliable and simple, and it provides rapid identification of individuals with elevated mercury levels (Naleway et al. 1991). It is a more appropriate marker of inorganic mercury, because organic mercury represents only a small fraction of urinary mercury. Yoshida (1985) found that urinary mercury levels were better correlated with exposure than were blood inorganic mercury concentrations in workers exposed to metallic mercury vapor.

Several studies have reported a correlation between mercury in blood and urine; however, results vary, and it is not known whether the ratio between concentrations in urine and blood remains constant at different exposure levels (Lindstedt et al. 1979; Roels et al. 1987; Smith et al. 1970). Significant correlations between occupational exposure to mercury vapor and mercury levels in the blood and urine of 642 workers in 21 chloralkali facilities were reported by Smith et al. (1970). According to the investigators, an air concentration (8-hour TWA) of 0.1 mg/m^3 was associated with blood levels of 6 µg/100 mL and urine levels of 220 (not corrected for specific gravity), 200, or 260 µg/L (corrected to specific gravities of 1.018 or 1.024, respectively). It is likely that current worker exposure is significantly less than this study indicates, because practices such as requiring showers after workshifts and cleaning work clothes after use have been implemented since 1970, when the Smith study was conducted. Another group of investigators, Henderson et al. (1974), found the concentrations reported in Smith et al. (1970) to be 2–10 times higher than those found 2 years later. As suggested by Roels et al. (1982), the actual mercury absorption by

workers exposed to the same air concentration may vary; therefore, researchers should report urine mercury levels together with estimated exposure concentrations to address the issue of variance between individuals.

Studies assessing mercury vapor exposure have suggested various ratios relating the concentration of mercury in the air (in μg/m^3) to the levels of mercury in the urine (in μg/L). Such estimates include 1:1 (Bell et al. 1973), 1:1.22 (Roels et al. 1987), and 1:2.5 (Lindstedt et al. 1979; Rosenman et al. 1986). Urinary metallic mercury levels ranging from 0.05 to 1.7 μg/L were detected in the urine of workers exposed to mercury vapor (>0.1 mg/m^3); this elemental mercury represented <1% of the inorganic mercury content of the urine (Yoshida and Yamamura 1982). With increased exposure to mercury vapor (0.47–0.67 mg/m^3), the amount of elemental mercury in the urine increased. A "rough" correlation between levels of metallic mercury vapor in air and mercury levels in blood and urine was established by Rosenman et al. (1986). They associated levels of 50 μg/100 mL in blood and 250 μg/L in urine with a mercury level in air of approximately 0.1 mg/m^3 (8-hour TWA), and 28 μg/100 mL in blood and 100 μg/L in urine with a TWA of 0.05 mg/m^3. Roels et al. (1987) found a correlation between daily mercury vapor exposure and blood or urine mercury levels in 10 workers employed for at least 1 year at an alkaline battery plant. The mercury levels in the air and the pre- or post-workshift levels of blood and urinary mercury correlated well (r=0.79–0.86 [blood] and r=0.70–0.80 [urine]). Based on a ratio of 1:0.045:1.22 (mercury in air:blood mercury:urinary mercury), Roels et al. (1987) concluded that exposure to 0.05 mg/m^3 mercury vapor would result in a blood mercury of 2.26 μg/100 mL and a urinary mercury of 61 μg/g creatinine. This correlation differed from that reported by Rosenman et al. (1986), possibly because fewer subjects were evaluated and determination of mercury vapor concentration by Roels et al. (1987) was based on air sampling collection during 5 consecutive days at 10 different workplaces.

Expired air samples have been considered as possible biomarkers of exposure for mercury. Following inhalation of metallic mercury vapor, some of the mercury may be eliminated in the expired air, but excretion from this pathway is negligible 5–7 days after exposure (Cherian et al. 1978; Hursh et al. 1976). Thus, expired air as a measure of mercury exposure can only be used soon after short-term exposure to mercury vapor. There is no information on the amount of mercury in expired air following long-term exposure to mercury.

Nonoccupational exposure to mercury includes the use of mercury-containing products and consumption of mercury-contaminated food. Urine samples from young women using skin-lightening creams containing

5–10% mercuric ammonium chloride had a mean mercury concentration of 109 µg/L, compared to 6 µg/L for urine samples from women who had discontinued use and to 2 µg/L for women who had never used the creams (Barr et al. 1973). Increased urinary excretion and blood levels of mercury were observed in volunteers who used phenylmercuric borate solutions or lozenges intended for the treatment of mouth or throat infections (Lauwerys et al. 1977). Swedes consuming fish contaminated with 0.3–7 mg Hg/kg (0.3-7 ppm) had blood cell levels of total mercury ranging from 8 to 390 ng/g (Skerfving 1974). Long-term exposure to methylmercury at 4 µg Hg/kg/day was associated with a mercury level in blood cells of approximately 300 ng/g (Skerfving 1974). The steady-state concentration of methylmercury in blood may be related to daily intake in the following equation (Task Group on Metal Accumulation 1973; WHO 1990):

$$C = \frac{f \times d}{b \times V} = \frac{A_D \times A_B \times d}{b \times V}$$

Where:

C	=	concentration in blood
f	=	fraction of the daily intake taken up by the blood
d	=	daily dietary intake
b	=	elimination constant
A_D	=	percent of mercury intake in diet that is absorbed
A_B	=	percent of the absorbed amount that enters the blood
V	=	volume of blood in the body

Hair is a biomarker of long-term exposure to methylmercury. Once mercury is incorporated into hair, it remains unchanged (Clarkson et al. 1973; Nielsen and Andersen 1991a, 1991b). A number of studies have examined the level of mercury in hair relative to the amount of fish consumed (see Table 2-10) (Airey 1983b; Haxton et al. 1979; Oskarsson et al. 1990; Sherlock et al. 1982). A fairly strong correlation has been demonstrated by these studies between the amount of fish consumed, the level of mercury in the fish, and the level of mercury in hair. Furthermore, the relationship between hair levels and blood levels has been well studied (see Table 2-9) (Amin Zaki et al. 1976; Den Tonkelaar et al. 1974; Haxton et al. 1979; Kershaw et al. 1980; Phelps et al. 1980; Sherlock et al. 1982; Skerfving 1974; Soria et al. 1992).

A number of studies report that hair mercury levels correlate with total intake levels and with organ-specific levels of mercury. Suzuki et al. (1993) analyzed 46 human autopsies in Tokyo, Japan and reported that hair mercury levels were highly significantly correlated with organ Hg levels in the cerebrum, cerebellum, heart, spleen, liver, kidney cortex, and kidney medulla, when the total mercury or methyl mercury value in the organ was compared with the hair total mercury or organic mercury, respectively.

When the inorganic mercury value was tested, significant correlations remained, with weaker coefficients in all the organs but the spleen. Stepwise multiple regression analysis indicated that hair organic mercury value was the major correlating variable for the organ total mercury or organ methyl mercury value in all the organs. With respect to the organ inorganic mercury value, the hair organic mercury value was the major correlate for the cerebrum and kidney (both cortex and medulla), the hair inorganic mercury value was the major variable for the cerebellum and heart, and the hair phosphorous and hair organic mercury were the major variables for the liver. No explanatory variable existed for the spleen. Auxiliary correlating variables accounted for the organ total mercury and inorganic mercury levels, among which the hair selenium value was conspicuous and with negative regression coefficients.

Nakagawa (1995) analyzed total mercury in hair samples from 365 volunteers in Tokyo, and reported higher mercury levels in those who preferred fish in their diet, compared to those who preferred other foods (preference choices were fish, fish and meat, meat, and vegetables). The mean hair mercury levels were 4 ppm in men who preferred fish and 2.7 ppm in women who preferred fish. The lowest hair mercury levels were seen in men and women who preferred vegetables, 2.27 and 1.31 ppm, respectively. The mean hair level for the whole group was 2.23 ppm (median 1.98).

Drasch et al. (1997) assayed tissue samples of 150 human cadavers (75 males, 75 females) from a “normal” European (German) population, i.e., there were no occupational or higher than average exposures to metals found in any of the biographies of the deceased. The objective was to evaluate the validity of blood, urine, hair, and muscle as biomarkers for internal burdens of mercury, lead, and cadmium in the general population. All individuals died suddenly and not as a result of chronic ailments. Age ranged from 16 to 93 years, and every decade was represented by approximately 10 males and 10 females. Tissues sampled included kidney cortex, liver, cerebral cortex, cerebellum, petrous portion of the temporal bone, (pars petrosis ossis temporalis), pelvic bone (spina iliaca anterior-superior), muscle (musculus gluteus), blood (heart blood), urine, and hair (scalp-hair). Statistically significant rank correlations between biomarker levels and tissues were observed but with large confidence intervals for the regressions. The authors conclude that specific biomarkers relative to each metal are useful in estimating body burdens and trends in groups, but are not useful for determining the body burden (and therefore the health risks) in individuals. A notable exception was, that in comparison to a generally poor correlation of cadmium, lead, and mercury between hair and tissue, there was a strong correlation between mercury in hair and mercury in brain (cerebrum and cerebellum). The authors state that this may be due to the high lipophilicity of elemental and short-chain alkyl mercury compounds. As seen in other studies comparing European to

Japanese hair mercury levels, the hair levels reported by Nakagawa (1995) of 2–4 ppm for a Japanese population are 10–20 times higher than levels observed in the Drasch et al. (1997) study (median, 0.247 µg/g in hair; range, 0.43-2.5 µg/g).

Other studies have confirmed a good correlation between hair mercury and brain mercury levels. In a study on the Seychelles Islands cohort, Cernichiari et al. (1995b) compared maternal hair levels, maternal blood levels, fetal blood levels, and fetal brain levels. Autopsy brains were obtained from infants dying from a variety of causes. The concentrations of total mercury in six major regions of the brain were highly correlated with maternal hair levels. This correlation was confirmed by a sequence of comparisons among the four measures. Maternal hair correlated to maternal blood (r=0.82) and infant brain level (r=0.6–0.8). Maternal blood correlated to infant blood (r=0.65); and infant blood correlated to infant brain (r=0.4–0.8).

There are potential confounding factors and other factors to consider when assessing mercury exposure based upon mercury hair levels. Mercury may be deposited to hair from the air when significant sources of mercury are present in the air or when certain hair treatments are used (Hac and Krechniak 1993; WHO 1991). Potential sources of external mercury exposure should, therefore, be evaluated as part of an exposure assessment. Some studies also report a sex related difference in mercury tissue levels. Nielson et al. (1994) observed a significant sex-related differences in the toxicokinetics of methylmercury in mice following administration of a single radiolabeled dose. Drasch et al. (1997) reported that mercury levels in all tissues assayed in their human cadaver study had higher levels compared to male tissues. The difference was significant for the kidney (median female kidney mercury level=92.0 ng/g, males=40.8 ng/g; p=0.002). In blood and urine there was a similar trend. In contrast, the authors report that mercury hair levels in females were significantly lower than in males (median females=205 ng/g, males 285 ng/g; p=0.02). Nakagawa (1995) also report higher mean mercury hair levels in males (2.98 µg/g) compared with females (2.02 µg/g) in a Japanese population. Further research is, therefore, needed to characterize potential sex related difference in the toxicokinetics of mercury under different exposure scenarios.

Eide and Wesenberg (1993) studied mercury concentrations in various organs and tissues in rats exposed to mercury vapor for approximately 2 months and proposed that human deciduous teeth may be useful indicators of chronic mercury exposure, as well as indicators of mercury uptake in organs such as the kidneys and the brain.

Other potential biomarkers of exposure include renal dysfunction parameters, neurological effects, and increased urinary porphyrins, and are discussed below in Section 2.7.2.

2.7.2 Biomarkers Used to Characterize Effects Caused by Mercury

Several potential biomarkers of effect for mercury have been evaluated, usually for neurological and renal dysfunction. Many of these toxic effects have been correlated with blood and urine levels (see Table 2-13). However, most indicators are nonspecific and may have resulted from other influences. As discussed in Section 2.2, many studies have examined the relationship between urine mercury levels and specific renal and neurological effects. Renal dysfunction has been studied extensively as a potential sensitive measure of mercury exposure. Signs of renal dysfunction at mercury air concentration of 0.1 mg/m^3 were reported by Stewart et al. (1977). Case reports have associated the therapeutic use of inorganic mercury salts with the occurrence of nephrotic syndrome (Kazantzis et al. 1962).

Several different biomarkers have been evaluated for assessing renal damage; however, renal parameters are interdependent (Verschoor et al. 1988). Furthermore, these markers are not specific for mercury exposure and may be a consequence of other concurrent chemical exposures. Markers for renal toxicity may indicate decreased function, cytotoxicity, or biochemical changes (Cardenas et al. 1993). Biomarkers for decreased function include increases in urinary proteins and elevation of serum creatinine or β_2-microglobulin. Biomarkers for renal cytotoxicity include increases in urinary excretion of antigens and enzymes located within renal tissues. Biomarkers for biochemical changes occurring within the kidneys include eicosanoids, fibronectin, kallikrein activity, and glycosaminoglycans in urine. Glomerular changes resulting from mercury exposure have predominantly been reported as increases in high-molecular weight proteinuria (Buchet et al. 1980; Kazantzis et al. 1962; Stonard et al. 1983; Tubbs et al. 1982). Renal tubular changes in workers exposed to mercury include increased urinary excretion of *N*-acetyl-β-D-glucosaminidase (NAG), β-galactosidase, and retinol binding protein (Barregard et al. 1988; Langworth et al. 1992b; Rosenman et al. 1986). Elevated urinary NAG levels occurred with urinary mercury levels of 100–250 μg/L in a study population of mixed ethnic background (Rosenman et al. 1986), with urinary levels of 35 μg/g creatinine in chloralkali workers (Barregard et al. 1988), with urinary mercury levels >25 μg/g creatinine in chloralkali workers (Langworth et al. 1992b), and with urinary mercury levels >50 μg/g creatinine in another group of chloralkali workers (Cardenas et al. 1993). NAG levels were not affected in chloralkali workers with urinary mercury levels of 15 μg/g creatinine (Piikivi and Ruokonen 1989). No significant increase of proteinuria, albuminuria, and other indicators of renal

dysfunction was evident in 62 mercury workers with average blood mercury levels of 1.6 μg/100 mL (range, 0.25–7.56 μg/100 mL) and average urine mercury levels of 56 μg/g creatinine (range, 3–272 μg/g creatinine) (Lauwerys et al. 1983). Another renal parameter evaluated is β-microglobulin, which has a normal range of 0.004–0.37 mg/L (Naleway et al. 1991). No statistically significant relationship was found between urinary β-microglobulin levels and elevated urinary mercury concentrations (Ehrenberg et al. 1991; Naleway et al. 1991). Examination of a wide range of biomarkers for renal toxicity in a group of chloralkali workers identified several other changes at low urinary mercury levels (Cardenas et al. 1993). Workers with urinary mercury levels in the range of 5–50 μg/g creatinine showed statistically significant increases in urinary Tamm-Horsfall glycoprotein (localized in the epithelial cells of the convoluted tubules) and decreases in urinary prostaglandins E2 and F2α. In workers with >50 μg/g creatinine, increased NAG, tubular brush border antigens, alkaline phosphatase, thromboxane B2, and glycosaminoglycans were also observed. Urinary porphyrins, which are intermediates in the biosynthesis of heme, may be another potential biomarker of effect for mercury exposure. A correlation was observed between urinary mercury and urinary coproporphyrin (Wada et al. 1969). Correlations were also observed for decreases in δ-aminolevulinic acid-dehydratase and cholinesterase activity with increases in urinary mercury. Porphyrins are considered a nonspecific measure of effect because they are influenced by other metal exposures. Woods et al. (1991) present data suggesting that there is a specific urinary porphyrin profile that may serve as a biomarker of mercury accumulation in the kidneys during prolonged inorganic and organic mercury exposure. A urinary porphyrin pattern, characterized by elevated coproporphyrin, pentacarboxyl porphyrin, and precoproporphyrin, for methylmercury hydroxide exposure was observed in mice for up to 30 weeks. This profile is observed at variable dose levels, as well as up to at least 40 weeks after cessation of exposure. The time course of the profile during prolonged treatment is closely associated with divalent inorganic mercury (Hg^{+2}), suggesting that the effects are mediated by this cation because it inhibits the heme pathway (Woods et al. 1991). Specificity may be a problem unless the porphyrin levels are analyzed at the same time as urinary mercury measurements.

The neurophysiological and neuropsychological health effects of mercury have been extensively studied in occupationally exposed individuals in an effort to monitor body levels and to determine a threshold value below which these effects are unlikely to occur. As with other biomarkers of effect, neurological changes induced by mercury may resemble exposure to other chemicals that can cause damage to the brain.

Case studies have associated exposure to mercury vapor with neurological effects (e.g., tremors, insomnia, shyness, emotional instability, decreased motor function and muscle reflexes, headaches, and abnormal

EEGs) (Davis et al. 1974; Jaffe et al. 1983; McFarland and Reigel 1978). Some studies have examined the relationship between nerve function and mercury levels in blood, urine, and tissue. Tissue levels of mercury have also been found to correlate with impaired nerve function. Among 23 dentists with mercury levels greater than 20 µg/g (measured in wrist tissue), 30% exhibited reduced nerve conduction velocity when compared with dentists with tissue levels of mercury below 20 µg/g (Shapiro et al. 1982). The decrease in nerve conduction velocity was observed in both sensory and motor nerves.

A dose-response relationship has also been reported for the association between paresthesia and blood mercury concentrations in an Iraqi population exposed to methylmercury. At a blood mercury level of 24 µg/100 mL 65 days after cessation of exposure, the incidence of paresthesia caused by methylmercury rose significantly (Clarkson et al. 1976). Below this concentration, any incidence of paresthesia was assumed to be related to other causes, according to the investigators. As a result of the reported blood mercury half-life of 65 days in this population, the maximum blood mercury concentration was likely to have been 48 µg/100 mL at the end of the exposure. Some evidence of paresthesia, sensory impairment, general ataxia, and visual field effects in exposed Swedes was reported; however, no significant increases in occurrence were found in Swedes with high levels of mercury in blood cells (82–1,100 ng/g) as compared to Swedes with lower blood cell mercury levels (12–75 ng/g) (Skerfving 1974). The study did not include a matched control group.

Many possible biomarkers of effect for mercury exposure have been correlated with urinary mercury levels. Workers exposed to elemental mercury vapor with urinary mercury excretion levels ranging from 7 to 1,101 µg/day exhibited significantly reduced tibial nerve velocity and increased median nerve latency in both motor and sensory nerves as compared with controls (Vroom and Greer 1972). Prolonged motor and sensory nerve latency was also associated with urine mercury levels ranging from 20 to 450 µg/L in 18 male workers exposed to elemental mercury vapor at a mercury cell chlorine plant (Levine et al. 1982). Urine mercury levels exceeding 200 µg/L have been reported to be associated with tremors and poor eye-hand coordination (Williamson et al. 1982). Twelve workers chronically exposed to elemental mercury vapor had urinary mercury levels ranging from <10 to 670 µg/L. A significant relationship between urine mercury and hand steadiness was reported. Increased tremor frequency, increased reaction time, and reduced eye-hand coordination were observed as urine mercury levels increased from 5 to 1,000 µg/L in 77 exposed individuals (Miller et al. 1975). A weak but significant quantitative relationship between urine mercury levels and finger tremors was elucidated by Verberk et al. (1986). The relationship between acceleration of finger tremors and excretion of mercury in the urine of 20 workers exposed to metallic

mercury was expressed by the equation 10 log (acceleration)=G0.888 + 0.0059 (urine mercury) (r=0.39, p<0.05, n=20). Tremors have also been reported in 567 workers from chloralkali production facilities whose blood mercury levels ranged from <1 to >10 µg/100 mL and whose urine mercury levels ranged from <10 to >1,000 µg/L. Increased tremors and reduced eye-hand coordination were associated with blood mercury levels of 1–2 µg/100 mL and urine mercury levels of 50–100 µg/g creatinine (Smith et al. 1970). Cavalleri et al. (1995) have suggested that exposure to elemental mercury vapors at levels producing urine mercury concentrations >50 µg/g creatinine can cause a dose-related loss of color vision.

An association between urine mercury levels and performance on memory tests and verbal intelligence tests has been established. Abnormal results on memory tests were reported for 9 workers exposed to mercury in the production of thermometers; urinary mercury excretion levels were 7–1,101 µg/24 hours (Vroom and Greer 1972). The short-term memory span of 26 workers was examined by Smith et al. (1983) and found to decrease with increasing urine mercury levels. The range of mercury found in the urine of these workers was 0–510 µg/L. A significant linear relationship was reported between subjects' 50% memory threshold spans and 12-month urinary mercury concentrations. Disturbances on tests of verbal intelligence and memory were more frequent among individuals with mercury blood levels above 1.5 µg/100 mL and mercury urine levels above 56 µg/L in 36 male chloralkali workers (Piikivi et al. 1984).

Potential biomarkers for the autoimmune effects of mercury include measurement of antiglomerular basement membrane antibodies, anti-DNA antibodies, serum IgE complexes, and total IgE (Cardenas et al. 1993). Elevated IgE, antiglomerular basement membrane antibodies, and anti-DNA antibodies have been observed in a few persons with exposure to mercury from dental amalgams (Anneroth et al. 1992). Other individuals have also been shown to have elevated anti-DNA or antiglomerular basement membrane antibodies (Cardenas et al. 1993; Langworth et al. 1992b).

Recent data regarding the action of low-level mercury exposure on receptors and signal transduction pathways in peripheral lymphocytes suggest potential applications of certain surrogate markers in mechanistic studies of neurotoxicity and, possibly, in assessing early biochemical effects of neurotoxicants in humans (Manzo et al. 1995). Additional biomarkers for effects on the immune, renal, hepatic, and neurological systems are presented in the CDC/ATSDR (1990) and OTA (1990) reports. See Section 2.2 for a more detailed discussion of the effects caused by mercury.

2.8 INTERACTIONS WITH OTHER CHEMICALS

As with many other metals, both toxic and nontoxic, interrelationships exist that can influence and alter the absorption, distribution, excretion, and toxicity of one or more of the component metals. For example, the zinc status of an individual can affect mercury toxicity. Pretreatment with zinc provides some protection from the nephrotoxic effects of inorganic mercury in rats (Zalups and Cherian 1992). The data indicate that zinc-induced metallothionein binds mercury in the renal cortex and shifts the distribution of mercury from its site of toxicity at the epithelial cells of the proximal tubules. Thus, the renal content of mercury is increased, yet less is available to cause toxicity. In contrast, the renal toxicity of mercuric chloride is exacerbated in zinc-deficient animals (Fukino et al. 1992). In the zinc-deficient state, less mercury accumulates in the kidneys, but the toxicity is greater. The mechanism of the protection appears to involve more than simply a redistribution of renal mercury, because in the absence of mercury exposure, zinc deficiency increases renal oxidative stress (increased lipid peroxidation, decreased reduced ascorbate). When mercury exposure occurs, the oxidative stress is compounded (increased lipid peroxidation and decreased glutathione and glutathione peroxidase). Thus, zinc appears to affect the biochemical protective mechanisms in the kidneys as well.

Similarly, in most studies, the simultaneous administration of mercury and selenium in equimolar doses to animals has resulted in decreased toxicity of both elements in acute and chronic exposure studies. This effect has been observed with inorganic and organic mercury and with either inorganic or organic selenium compounds, although inorganic forms of selenium appear to be more effective than organic forms (Chang 1983; Skerfving 1978). Selenium protects against the acute nephrotoxicity of the mercuric ion and the methylmercuric ion in rats (Ganther 1980; Ganther et al. 1972; Hansen 1988; Magos et al. 1987; Parizek and Ostadolva 1967) and possibly against acute neurotoxicity of methylmercuric ion in rats (Ohi et al. 1980). The protective effect of selenium has been associated with a higher whole-body retention of mercury rather than with increased mercury excretion (Hansen 1988; Magos et al. 1987). Mercury-selenium complexes are formed when these chemicals are co-administered. Mercuric mercury forms a complex with selenium and a high-molecular weight protein (Naganuma and Imura 1981). Methylmercury forms a bismethyl-mercury selenide complex. Although the specific mechanism for the protection is not well understood, possible mechanisms for selenium's protective effect include redistribution of mercury (Mengel and Karlog 1980), competition by selenium for mercury-binding sites associated with toxicity, formation of a mercury-selenium complex that diverts mercury from sensitive targets (Hansen 1988; Magos et al. 1987; Naganuma and Imura 1981), and prevention of oxidative damage by increasing

selenium available for the selenium-dependent glutathione peroxidase (Cuvin-Aralar and Furness 1991; Imura and Naganuma 1991; Nylander and Weiner 1991). Selenium-treated animals can remain unaffected despite an accumulation of mercury in tissues to levels that are otherwise associated with toxic effects (Skerfving 1978). Support for the proposal that an inert complex is formed comes from the 1:1 ratio of selenium and mercury found in the livers of marine mammals and in the bodies of experimental animals administered compounds of mercury and compounds of selenium, regardless of the ratio of the injected doses (Hansen 1988). Mercuric mercury has been shown to form a complex with selenium and a high-molecular weight protein (Naganuma and Imura 1981). Methylmercury forms a bismethyl-mercury selenide complex.

Although the fetotoxicity of methylmercuric chloride has been shown to be enhanced by the feeding of a selenium-deficient diet in mice (Nishikido et al. 1987), additional selenium administration does not appear to protect against teratogenic effects (i.e., cleft palate) of methylmercuric chloride in mice (Lee et al. 1979). High doses of selenium administered as selenite for 30 days prior to gestation and through Gd 18 to mice fed a diet containing high doses of methylmercuric chloride increased the incidence of cleft palate (Nobunaga et al. 1979). It is possible that cleft palate induction by methylmercury is the result of a suppression of growth rather than a tissue-specific teratogenic action (Lee et al. 1979). If this were the case, high doses of selenium that inhibit growth could potentiate the induction of cleft palate by methyl-mercury administration. Further discussion of selenium-mercury interactions can be found in Section 2.3.1.2.

Ethanol promotes an increase in the respiratory excretion of metallic mercury by inhibiting the enzyme catalase, which is responsible for oxidizing metallic mercury to mercuric mercury. This process was shown in workers who ingested a moderate dose of alcohol and experienced a 50% decrease in mercury retention upon inhalation exposure to metallic mercury vapor (Nielsen-Kudsk 1973). Also, ethanol increased the amount of mercury exhaled by people who inhaled metallic mercury vapor or received trace doses of mercuric chloride (Nielsen-Kudsk 1965). Therefore, less mercury should reach the kidneys and less renal toxicity should be observed (Nielsen-Kudsk 1965). However, ethanol also allows elemental mercury to persist longer in the plasma, resulting in prolonged diffusion of elemental mercury throughout the body (Nielsen-Kudsk 1965). Therefore, ethanol can cause mercury to distribute more easily across the blood-brain barrier and the placenta, thereby increasing the risk of mercury toxicity to the brain and the developing fetus. In addition, the oxidation of ethanol with concurrent NADPH generation enhances

the reduction of the mercuric ion to metallic mercury, thereby making it more favorable for permeating the placenta (Khayat and Dencker 1982).

Ethanol also potentiates the toxicity of methylmercury (Rumbeiha et al. 1992; Tamashiro et al. 1986; Turner et al. 1981). Studies in animals have shown increased mortality (Tamashiro et al. 1986), increased severity and decreased time to onset of neurotoxicity (hind-limb ataxia) (Tamashiro et al. 1986; Turner et al. 1981), and increased renal toxicity (increased hematuria, renal weight, blood urea nitrogen, and oliguria) (Rumbeiha et al. 1992; Tamashiro et al. 1986) when methylmercury exposure occurred concomitant with ethanol ingestion. Although increased mercury concentrations were observed in the brain and kidneys, the changes in mercury content were insufficient to fully explain the observed potentiation of toxicity (Tamashiro et al. 1986), suggesting that ethanol may enhance the toxic mechanisms of methylmercury. The mechanism for this enhancement is unknown.

Atrazine and potassium dichromate have also been demonstrated to enhance the toxicity of inorganic mercury. Administration of atrazine, a widely used herbicide, with methylmercury in the diet resulted in a higher deposition of mercury in the liver and an earlier onset of neurotoxicity (Meydani and Hathcock 1984). The mechanism underlying this interaction was unclear. Parenteral administration of minimally toxic doses of potassium dichromate and mercuric chloride resulted in a synergistic inhibition of the renal transport of organic ions *p*-aminohippurate and tetraethylammonium (Baggett and Berndt 1984). Although the mechanism underlying this interaction was not examined, it may be associated with the fact that both mercury and potassium dichromate are both toxic to the renal proximal tubule (Biber et al. 1968).

Agents that deplete nonprotein sulfhydryls may increase the toxicity of mercury. Depletion of glutathione levels with diethylmaleate in rats resulted in greatly increased renal toxicity of mercury chloride (Girardi and Elias 1991). Greater decreases in glomerular filtration and increases in fractional excretion of sodium and lithium, urinary γ-glutamyltransferase, and lipid peroxidation were observed. Conversely, chemicals that protect against oxidative damage may decrease the toxic effects of mercury. Increased survival and decreased toxicity were observed in rats given vitamin E (α-tocopherol) during treatment with methylmercury (Welsh 1979). It is probable that the mechanism for the protection involved the antioxidant properties of vitamin E.

The exogenous application of the monothiols glutathione or its, precursor N-acetyl-DL-homocysteine thiolactone (NAHT), or B-complex and E vitamins to mice exposed to methylmercuric chloride injected

at dosages of 1 mg/kg/day was reported by Bapu et al. (1994). Therapy with both B-complex vitamins and vitamin E was found to mobilize a significant amount of mercury from all tissues examined (brain, spinal cord, liver, and kidneys), with the maximum mobilization (about 63%, compared with controls) being recorded in the spinal cord following vitamin E treatment. NAHT treatment also produced significant mobilization of mercury from nervous tissue but caused an increase in mercury concentration in non-nervous tissue.

Another group of compounds that combines with mercury (and other divalent cation species) is comprised by those used in chelation therapy to reduce the body burden of mercury by enhancing its elimination from the body. Such chelators include: ethylenediaminetetraacetic acid (EDTA); ethylene glycol bis(beta-aminoethyl ether)N,N,N',N'-tetraacetic acid (EGTA); 2,3-dimercaptopropane-1-sulphonate (DMPS); 2,3-dimercaptosuccoinic acid (DMSA); 2,3-dimercaptopropanol (British anti-Lewisite [BAL]; sometimes called dimercaprol); and N-acetylpenicillamine (NAP). While these chelating agents have a very high affinity for Hg^{++}, which makes them effective mercury chelators, they also have an affinity for other divalent cations, many of which are essential for normal physiological function.

BAL was the first chelating agent used for mercury toxicity, and it is still widely used today for inorganic mercury poisoning (ATSDR 1992). BAL is also believed to be effective in treating phenylmercury poisoning, because of the rapid *in vivo* oxidization of phenylmercuric acetate to Hg^{++}, thereby rendering phenylmercury similar in behavior to inorganic mercury. BAL is <u>contraindicated</u> for cases of methyl-mercury poisoning, however, because it has been demonstrated to increase the concentration of methyl-mercury in the brain. Possible side effects of BAL include nausea, vomiting, headache, tachycardia, fever, conjunctivitis, blepharospasm, and lacrimation. As an adjunct or alternative to parenterally administered BAL, oral NAP may be used (ATSDR 1992). Side effects of NAP may include fever, rash, leukopenia, eosinophilia, and thrombocytopenia.

DMPS and DMSA are derivatives of BAL, but they have been found to be more effective than BAL in experimental studies. Although still considered an investigational drug, DMPS decreased the mercury excretion half-life from 33.1 to 11.2 days in 2 workers exposed to high levels of mercury vapor (ATSDR 1992). In a study of the influence of DMPS and DMSA on renal deposition of mercury in rats, both chelating agents were found to cause a significantly increased urinary excretion of mercury (Zalups 1993), although significant differences in the extrarenal handling of these two chelators were found. DMPS was also shown to increase the urinary excretion of mercury 7.6-fold in a group of former chloralkali workers

3 years after cessation of occupational exposure (Sallsten et al. 1994), probably reflecting the excretion of mercury stored in the kidneys. In a case report of two human mercury vapor intoxication incidents, treatment with BAL followed by DMSA was found to decrease plasma inorganic mercury uptake at concentrations <50 µg/L. However, relatively high concentrations of mercury remained in the plasma for a very long time, possibly due to the progressive release of mercury from red blood cells and tissues after oxidation.

EDTA and EGTA also effectively form complexes with Hg^{++}, and enhance its excretion from the body, in what is typically considered a relatively benign or biologically inert fashion. In a study using human brain homogenates from autopsy samples from apparently healthy brains, Duhr et al. (1993) demonstrated that not only is the inhibition of microtubule polymerization and the disruption of already-formed microtubules not prevented by the addition of EDTA and EGTA (which bind Hg^{++} with very high affinity) but, to the contrary, these two chelating agents potentiate the Hg^{++}-induced inhibition of tubulin polymerization. Duhr et al. (1993) further reported that the mercury-EDTA and mercury-EGTA complexes cause the inhibition of tubulin polymerization by disrupting the interaction of GTP with the E-site of brain beta-tubulin, an obligatory step in the polymerization of tubulin.

2.9 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to mercury than will most people exposed to the same level of mercury in the environment. Reasons include genetic makeup, developmental stage, age, health and nutritional status (including dietary habits that may increase susceptibility, such as inconsistent diets or nutritional deficiencies), and substance exposure history (including smoking). These parameters result in decreased function of the detoxification and excretory processes (mainly hepatic, renal, and respiratory) or the pre-existing compromised function of target organs (including effects on clearance rates and any resulting end-product metabolites). Populations more susceptible to the toxic effects of mercury than a healthy young adult include: the elderly because of declining organ function, higher levels of persistent heavy metals (e.g., cadmium) that also accumulate in the kidney, and potentially higher brain to liver or kidney mercury concentrations; people with pre-existing disease (e.g., renal or neurological disease); and the youngest of the population because of their immature and developing organs. Populations at greater risk due to unusually high exposure are discussed in Section 5.7 (Populations With Potentially High Exposure).

Probably the most widely recognized form of hypersensitivity to mercury is the occurrence of acrodynia, or pink disease, in persons exposed to mercury. Acrodynia is characterized by itching, flushing, swelling, and/or desquamation of the palms of the hands or soles of the feet, morbilliform rashes, excessive sweating and/or salivation, tachycardia, elevated blood pressure, insomnia, weakness, irritability, fretfulness, and peripheral sensory disturbances (Warkany and Hubbard 1953). The occurrence of acrodynia was determined to be an idiosyncratic reaction to mercury exposure. Despite widespread exposure of children to mercury-containing laxatives, antiascariasis medications, and teething powders in the 1940s and 1950s, only a few children developed acrodynia. The affected population was not the most highly exposed; numerous reports identified higher exposures in others with no evidence of the disease. The physiological basis for this hypersensitivity is unknown, but patch testing indicated that it is not an allergic response to mercury exposure.

Animal studies (Aten et al. 1992; Druet et al. 1978; Hirszel et al. 1985; Hultman and Enestrom 1992; Matsuo et al. 1989; Michaelson et al. 1985; Pelletier et al. 1990; Pusey et al. 1990; Roman-Franco et al. 1978; van der Meide et al. 1993) and limited human data (Lindqvist et al. 1974; Tubbs et al. 1982) also indicate that there may be persons with a genetic predisposition to develop an autoimmune glomerulo-nephritis upon exposure to mercury. In this form of renal toxicity, proteinuria is observed following the reaction of autoantibodies with renal tissues and deposition of immune material (i.e., IgG and complement C3) in the renal mesangium and glomerular blood vessels. Both susceptible and resistant mouse and rat strains have been identified, and susceptibility appears to be governed by both MHC genes and nonMHC genes (Aten et al. 1991; Druet et al. 1978; Hultman and Enestrom 1992; Hultman et al. 1992; Michaelson et al. 1985; Sapin et al. 1984).

Unborn children are another known susceptible population to the toxic effects of mercury (see Section 2.2.2.4). Data from large-scale poisonings in Japan (Harada 1978) and Iraq (Marsh et al. 1987) indicate that infants exposed *in utero* to alkyl mercury compounds developed severe neurological toxicity whereas their mothers may have experienced no or only mild toxicity. This difference may be due to methylmercury binding to tubulin (Vogel et al. 1985, 1989) and the role of microtubules in neuronal cell division and migration in the developing nervous system (Sager et al. 1982). There is evidence indicating that the developing male fetus may be more susceptible to methylmercury exposure than the female fetus (Buelke-Sam et al. 1985; Grant-Webster et al. 1992; Sager et al. 1984).

Neonates may also be especially susceptible to mercury toxicity. Both inorganic and organic forms of mercury are excreted in the milk (Sundberg and Oskarsson 1992; Yoshida et al. 1992). Furthermore, suckling rats exhibit a very high absorption of inorganic mercury as a percentage of the diet (30–40%) compared to adult rats, which absorb approximately 1% of the inorganic mercury from the diet (Kostial et al. 1978). The highest oral toxicity to inorganic mercury as expressed by the LD_{50} was for 2-week-old rats; by 3–6 weeks of age, rats showed a dramatic drop in sensitivity to inorganic mercury poisoning (Kostial et al. 1978). The transfer of mercury to suckling rats through milk was found to result in greater concentrations of the metal in the brains of the offspring than in the mother (Yang et al. 1973). Developmental neurotoxicity, similar to that seen with *in utero* exposure, has been observed in an infant exposed to alkyl mercury only after birth (Engleson and Herner 1952).

Individuals with diseases of the liver, kidneys, lungs, and nerves are considered to be at greater risk of suffering from the toxic effects of both organic and inorganic mercury. Individuals with a dietary insufficiency of zinc, glutathione, antioxidants, or selenium or those who are malnourished may be more susceptible to the toxic effects of mercury poisoning because of the diminished ability of these substances to protect against mercury toxicity (see Section 2.8).

2.10 METHODS FOR REDUCING TOXIC EFFECTS

This section describes clinical practice and research concerning methods for reducing toxic effects of exposure to mercury. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for the treatment of exposures to mercury. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice.

Although there are a number of treatments currently available, none are completely satisfactory and additional development of treatment drugs and protocols is needed. The recent death of a researcher poisoned with dimethylmercury is a case in point (Nierenberg et al 1998; Toribara et al. 1997). In spite of prompt action and excellent medical care and monitoring, the clinical course in this patient continued to decline, and ultimately ended in death.

In general, even the inorganic mercurials, that are considered to be more easily chelated, are difficult to remove from the body and are not treated without some side effects. Infants and young children are

particularly difficult to treat, sometimes requiring exchange transfusion or other more elaborate measures. Reducing the body burden or toxic effects of mercury in pregnant women presents an even greater challenge (i.e., treatment must be effective for both the mother and the developing child), and specific treatment protocols are needed.

2.10.1 Reducing Peak Absorption Following Exposure

Strategies used to reduce absorption of mercury may differ depending on the route of exposure and the specific chemical to which one is exposed. Elemental mercury and certain organic forms of mercury have high vapor pressures and are readily absorbed by the lungs; inhalation of these chemicals may be the major exposure of concern. Because ingestion of most chemical forms of mercury is possible, strategies for limiting absorption from the gastrointestinal tract would be of utmost concern in such situations. The organic mercury compounds have greater absorption from the gut than elemental and inorganic mercury; thus, strategies differ depending on the form of mercury ingested. Dermal absorption of the various forms of mercury is also possible, so strategies should also consider limiting dermal absorption.

The first step in mitigating the toxic effects of inhalation and dermal exposures to mercury or its compounds is removal from the contaminated area or source (Bronstein and Currance 1988; Gossel and Bricker 1984; Haddad and Winchester 1990; Stutz and Janusz 1988). Since continued exposure may occur when clothing is contaminated, clothing may be removed as well (Bronstein and Currance 1988; Stutz and Janusz 1988). If dermal or ocular exposure has occurred, thoroughly washing the exposed areas with water has been suggested; treatment protocols recommend the use of Tincture of Green® soap a disinfectant) for the skin and normal saline for the eyes (Bronstein and Currance 1988; Stutz and Janusz 1988).

Several treatments have been suggested to reduce absorption of mercury from the gastrointestinal tract; however, most refer to the inorganic forms of mercury. It is likely that strategies that are effective in reducing the absorption of inorganic forms may also have some efficacy for organic forms. Several procedures that have been recommended for trapping mercury in the gastrointestinal tract are based on the mercury's affinity for binding to sulfhydryl groups. For example, oral administration of a protein solution (e.g., milk or egg whites) has been suggested to reduce absorption (Gossel and Bricker 1984; Haddad and Winchester 1990; Stutz and Janusz 1988). Salt-poor albumin administration has also been suggested (Haddad and Winchester 1990). Nonabsorbable agents (e.g., polystyrene resins containing sulfhydryl

groups) have been used to decrease the absorption rate of methylmercury (Clarkson et al. 1973). The oral administration of activated charcoal has also been suggested (Gossel and Bricker 1984; Stutz and Janusz 1988). Rapid removal of mercury from the gastrointestinal tract may be indicated in some acute, high-dose situations. In such situations, immediate emesis or gastric lavage has been suggested (Goldfrank et al. 1990; Haddad and Winchester 1990). Inclusion of salt-poor albumin or sodium formaldehyde sulfoxylate in the lavage fluid to convert the mercuric ion into the less soluble mercurous ion in the stomach has also been recommended (Haddad and Winchester 1990). Emesis is contraindicated following the ingestion of mercuric oxide, presumably because of the risk of damage to the esophagus as the potentially caustic compound is ejected. A saline cathartic, such as magnesium sulfate, to speed removal from the gastrointestinal tract has also been recommended unless diarrhea has already begun (Haddad and Winchester 1990; Stutz and Janusz 1988). Giving CaNa2-EDTA is contraindicated because it binds poorly to mercury, may be toxic to the kidneys, chelates other essential minerals, and may cause redistribution of mercury in the body (Gossel and Bricker 1984).

2.10.2 Reducing Body Burden

Since the main source of mercury exposure for the general public is organic mercury in the diet, minimizing the consumption of mercury-laden fish and shellfish is an effective means of reducing the body burden. The amount of inhaled mercury vapor from accidental spills of metallic mercury (e.g., from broken thermometers or electrical switches) can be minimized by informing the general public of the potential dangers and volatility of liquid mercury, and by prompt and thorough clean-up of liquid mercury spills.

Following exposure and absorption, metallic mercury is distributed primarily to the kidneys. Elemental mercury is highly soluble in lipids and easily crosses cell membranes (Gossel and Bricker 1984), particularly those of the alveoli (Florentine and Sanfilippo 1991). Once in the blood, this form of mercury can distribute throughout the body, as well as penetrate the blood-brain barrier, thus accumulating in the brain (Berlin et al. 1969). The body burden half-life of metallic mercury is about 1–2 months (Clarkson 1989). The kidney is also the primary organ of accumulation for compounds of inorganic mercury, but the liver, spleen, bone marrow, red blood cells, intestine, and respiratory mucosa are target tissues as well (Haddad and Winchester 1990; Rothstein and Hayes 1964). Inorganic mercury is excreted primarily through the kidneys; its half-life ranges from 42–60 days (Hursh et al. 1976; Rahola et al. 1973). As with elemental mercury, organic mercury compounds accumulate throughout the body (Aberg et al. 1969; Miettinen 1973). Accumulation of organic mercury also occurs in the liver, where it

is metabolized, excreted through the bile, and often reabsorbed in the gastrointestinal tract (Florentine and Sanfilippo 1991; Haddad and Winchester 1990). The half-life of lower alkyl mercurials is about 70–79 days (Aberg et al. 1969; Miettinen 1973).

For several years, diaphoresis (excretion through perspiration) was used to lower the body burden of mercury in miners exposed to mercury vapors (Sunderman 1978). Recently, this method of therapy has also been used to lower tissue levels of mercury in a patient exposed to metallic mercury in the manufacture of thermometers (Sunderman 1978).

Chelation therapy is presently the treatment of choice for reducing the body burden of mercury. There are currently a number of chelators that are either in practical use or under investigation in *in vivo* and *in vitro* studies (Florentine and Sanfilippo 1991; Gossel and Bricker 1984; Haddad and Winchester 1990). These chelators differ in their efficacy for various forms of mercury, routes of administration, side effects, and routes of excretion. Depending on the chemical to which one has been exposed and the health status of the individual, different chelators may be indicated. One popularly used chelator, dimercaprol or BAL, has two sulfhydryl groups that can bind mercury and compete with its binding to sulfhydryl groups in body tissues (Florentine and Sanfilippo 1991; Haddad and Winchester 1990). BAL is one of the more effective chelators for inorganic mercury salts. BAL is administered intramuscularly and is the preferred chelator when oral dosing is impractical (Florentine and Sanfilippo 1991; Gossel and Bricker 1984; Haddad and Winchester 1990). Approximately 50% of the dimercaprol-mercury complex is excreted through the kidneys, while the remainder is eliminated in the bile and feces. Thus, this chelator is preferred when renal impairment has occurred. BAL therapy, however, has several limitations. Significant reabsorption of mercury from the bile occurs (Shimada et al. 1993). Also, multiple toxic side effects including urticaria, elevated blood pressure and heart rate, nausea and vomiting, headache, conjunctivitis, lacrimation, and paresthesias have been reported (Goldfrank et al. 1990). Children may develop fevers, and individuals with a glucose-6-phosphatase deficiency may develop hemolysis. BAL treatment is <u>contraindicated</u> for elemental and organic mercury compounds because it has been shown to increase brain levels of mercury in animal studies when used to treat exposures to phenylmercury or methoxyethylmercury compounds (Berlin 1986; Berlin and Rylander 1964) or elemental mercury vapor (Goldfrank et al. 1990), indicating the possibility of increased neurotoxicity.

Another currently used mercury chelator is D-penicillamine. This drug has been used somewhat effectively to reduce the toxicity of elemental and inorganic mercury exposures. It can be taken orally, and its

metabolism is slight in humans. Penicillamine is removed though the kidneys (Florentine and Sanfilippo 1991). However, acute allergic reactions to penicillamine may occur (Goldfrank et al. 1990). An experimental drug, *N*-acetyl-D,L-penicillamine (NAP), is very similar to its analog, penicillamine, in its properties of absorption, metabolism, and excretion; however, it may be more mercury-specific in its chelating abilities and less toxic (Goldfrank et al. 1990; Haddad and Winchester 1990). A high success rate (88%) has been reported by investigators using NAP to treat victims of mercury inhalation (Florentine and Sanfilippo 1991).

2,3-Dimercaptosuccoinic acid (DMSA), an analogue of BAL, is another experimental chelating agent. DMSA can be given orally and is primarily excreted through the kidneys (Aposhian et al. 1992b). It has been shown to be an effective chelator for both inorganic and methylmercury (Magos 1967). Comparative studies have demonstrated that DMSA is as effective, if not more so, as dimercaprol, penicillamine, and NAP. Data also suggest that this chelating drug produces fewer adverse effects than NAP (Florentine and Sanfilippo 1991). 2,3-Dimercaptopropane-1-sulfonate (DMPS) is another BAL analogue that is an orally effective chelator for mercury. Reports differ with respect to which of these analogues is less toxic (Ellenhorn and Barceloux 1988; Goldfrank et al. 1990; Jones 1991; Karagacin and Kostial 1991). Better results were obtained in rats with DMPS than with DMSA when the chelating agent was administered at least 24 hours following exposure to mercuric chloride. However, early oral administration of DMPS (within 24 hours) resulted in increased mercury retention (Karagacin and Kostial 1991). In contrast, DMSA resulted in decreased mercury retention irrespective of when it was administered.

Hemodialysis with infusion of a chelator (cysteine, *N*-acetylcysteine, NAP) has been reported to be effective in some severe cases of poisoning where renal failure is a complication (Berlin 1986; Goldfrank et al. 1990; Haddad and Winchester 1990). It has been reported to be advantageous to begin the hemodialysis before substantial renal damage has occurred (Haddad and Winchester 1990).

Because methylmercury undergoes enterohepatic recirculation, nonabsorbable agents have been used to "trap" methylmercury excreted into the bile (Lund et al. 1984). A polystyrene resin containing sulfhydryl groups added to food at a concentration of 1% doubled the elimination rate of methylmercuric chloride when administered to mice. The elimination half-life decreased from 65 to 20 days (Clarkson et al. 1973). Excretion of methylmercury may also be enhanced by bile drainage either through catheterization and drainage of the choledochal duct or by surgical establishment of gallbladder drainage (Berlin 1986). However, this method has not been used therapeutically.

2.10.3 Interfering with the Mechanism of Action for Toxic Effects

The majority of metallic mercury vapor and organic mercury absorbed by the body is rapidly oxidized to the more toxic and soluble mercuric ion in the blood and tissues through a hydrogen peroxide catalase pathway (Clarkson 1989; Halbach and Clarkson 1978). It is believed that the high affinity of the cation for protein-containing sulfhydryl or thiol groups is the underlying mechanism for the biological activity of mercury (Clarkson 1972a; Hughes 1957; Passow et al. 1961). In a process that is not yet completely understood, mercury disrupts the intracellular sulfhydryl status, resulting in oxidative stress, followed by activation of catabolic enzymes (i.e., proteases, endonucleases), and ultimately in cellular injury (Verity and Sarafian 1991). Treatment with agents that reduce oxygen radical-producing reactions may be effective in reducing mercury-induced oxidative cell damage. For example, pretreatment of rats with deferoxamine, a potent iron chelator and inhibitor of iron-catalyzed oxygen radical-producing reactions, reduced the increase in reactive oxygen species seen in the cerebellum after methylmercury exposure (LeBel et al. 1992; Sarafian and Verity 1991). Similarly, treatment with *N*-acetylcysteine, an antioxidant, resulted in increased survival time and less severe lung lesions in rats following exposure to mercury vapor (Livardjani et al. 1991b). Vitamin E (alpha tocopherol) and *N*,*N*'-diphenyl-*p*-phenylenediamine therapy have antioxidant effects and have been shown to be effective in protecting against methylmercury-induced toxicity (Ganther 1980; Welsh 1979).

Strategies to block the oxidation of elemental mercury to mercuric ion through the hydrogen peroxide catalase pathway do not appear to be a viable method for mitigating the effects of mercury exposure because treatment with chemicals (e.g., ethanol) that have been shown to block this reaction (Nielsen-Kudsk 1965) result in higher levels of blood mercury and increased renal toxicity (Rumbeiha et al. 1992). Another option would be to reduce the oxidized mercury ions to the monovalent mercurous form. A treatment of this nature has been suggested for ingested inorganic mercury.

Metals and chemicals shown to be antagonistic to the toxic effects of mercury may offer a possible method of interfering with the mercury's mechanism of action. Selenium, as sodium selenite, has been used in counteracting mercury poisoning, although the specific mechanism is not understood (Mengel and Karlog 1980; Naganuma and Imura 1981). The efficacy of selenium administration also appears to be dependent on the form of mercury to which one is exposed. Co-administration of sodium selenite with mercuric chloride resulted in decreased renal toxicity, whereas co-administration with methylmercuric chloride had no effect on renal toxicity (Yasutake et al. 1991b). The nephrotoxic effects of inorganic mercury may be

protected against by pretreatment with zinc (Zalups and Cherian 1992). Data in rats suggest that zinc can induce metallothionein in the renal cortex and cause mercury accumulation in the kidneys to shift from the outer medulla to the cortex, where a greater percentage is bound to the induced metallothionein. However, despite its potential use for interfering with the mercury-induced renal effects, zinc also prolongs retention in the body.

2.11 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of mercury is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of mercury.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

2.11.1 Existing Information on Health Effects of Mercury

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to inorganic and organic mercury are summarized in Figures 2-8 to 2-11. The purpose of these figures is to illustrate the existing information concerning the health effects of inorganic and organic mercury. Each dot in the figures indicates that one or more studies provide information associated with that particular effect. The dot does not imply anything about the quality of the study or studies. Gaps in this figure should not be interpreted as "data needs." A data need, as defined in ATSDR's Decision Guide for Identifying Substance-Specific Data Needs Related to Toxicological Profiles (ATSDR 1989), is substance-specific information necessary to conduct comprehensive public health assessments. Generally, ATSDR defines a data gap more broadly as any substance-specific information missing from the scientific literature.

Information concerning metallic mercury exists primarily for the inhalation route of exposure in humans and animals (see Figure 2-8). Human data exist for all categories of effect following inhalation exposure to metallic mercury vapor. The results from inhalation studies in animals have been reported for all end points except immunological and genotoxic effects, and cancer. With the exception of case studies on contact dermatitis and neurological effects after acute and occupational dermal exposure to metallic mercury in humans, no studies were located for either the oral or dermal routes of exposure for either humans or animals.

Existing information on inorganic mercury salts is shown in Figure 2-9. No studies were found on the health effects from inhaled mercury salts in humans or animals. A number of case histories for acute or chronic oral exposure to mercury salts provide information on systemic and neurological effects and death. Some case histories and occupational studies provide information on dermal exposures to mercury salts at acute, intermediate, and chronic exposures leading to death, immunologic, neurologic, and systemic effects. No animal inhalation studies for inorganic mercury salts were identified, and only one acute study provides limited information on death from dermal exposure. A number of animal studies that have investigated the effects from oral exposure to mercury salts provide good information on systemic effects; limited information on cancer, neurologic, immunologic, and genotoxic effects; and no information on reproductive or developmental effects.

Information on methylmercuric and phenylmercuric mercury is presented in Figures 2-10 and 2-11. These two forms of organic mercury were chosen to represent the group of organic mercurials because they have been detected at Superfund sites, and because methylmercury is the predominant form of organic mercury in the environment. There is a paucity of information on phenylmercury. Only a few case histories are available for effects following inhalation exposure (death, acute or chronic systemic effects, and neurologic effects), and the information from these reports is very limited. Only one case history for acute systemic effects following dermal exposure to phenylmercury was identified. One chronic oral study in rats and a cancer study in rats and mice provide the only animal data for phenylmercury. In contrast, there are a number of human studies on systemic, neurologic, and developmental effects resulting from an oral exposure to methylmercury. No human toxicity data were identified for immunologic, reproductive, or genotoxic effect, nor for carcinogenicity. The human data for methylmercury are accompanied by a relatively large number of animal studies representing all three exposure durations and providing some, although often limited, information for all health effects categories. As with phenylmercury, there are only a few case histories for inhalation and dermal exposures, with limited information on neurologic and

systemic effects or death from acute poisonings. The animal data for inhalation exposure to methylmercury is equally scarce and nonexistent for dermal exposures.

2.11.2 Identification of Data Needs

Acute-Duration Exposure. The human toxicity information for acute duration exposures to mercury is limited to qualitative data and case histories following oral, inhalation, and dermal routes of exposure. Several case reports described death due to respiratory impairment from inhaled metallic mercury (Campbell 1948; Kanluen and Gottlieb 1991; Soni et al. 1992; Taueg et al. 1992). Respiratory, cardiovascular, gastrointestinal, hematological, and renal effects have been observed after acute-duration inhalation exposure to metallic mercury vapors (Bluhm et al. 1992a, 1992b; Campbell 1948; Garnier et al. 1981; Haddad and Sternberg 1963; Hallee 1969; Jaffe et al. 1983; Kanluen and Gottlieb 1991; Karpathios et al. 1991; Lilis et al. 1985; McFarland and Reigel 1978; Milne et al. 1970; Snodgrass et al. 1981; Soni et al. 1992; Taueg et al. 1992). Acute exposure to ingested inorganic mercury salts has also resulted in gastrointestinal and renal symptoms (Afonso and deAlvarez 1960; Kang-Yum and Oransky 1992). Tremors, irritability, and decreased motor function and reflexes are common neurological symptoms following high-level acute duration exposures to metallic mercury vapors (Adams et al. 1983; Bluhm et al. 1992a; Hallee 1969; Jaffe et al. 1983; McFarland and Reigel 1978; Snodgrass et al. 1981). Acute exposure to ingested methylmercury has resulted in both neurological and developmental toxicity (Al-Mufti et al. 1976; Amin-Zaki et al. 1974; Bakir et al. 1973; Cox et al. 1989; Engleson and Herner 1952; Harada 1978; Marsh et al. 1980, 1981, 1987; Snyder and Seelinger 1976). Information on short term dermal exposures in humans to inorganic mercury are from case studies, and provide some information on renal, neurological, immunological, and dermatological effects (Bagley et al. 1987; Bourgeois et al. 1986; DeBont et al. 1986; Faria and Freitas 1992; Kawahara et al. 1993; Millar 1916; Pambor and Timmel 1989).

Dermal effects from acute duration dermal exposures to organic mercury compounds have also been reported to a limited extent (Morris 1960). In a highly publicized poisoning, a laboratory researcher was thought to have received a single dermal exposure to the organomercurial, dimethylmercury (estimated at between 0.1 and 0.5 mL at a density of 3 g/mL), that apparently penetrated the researcher's latex safety gloves and resulted in a severe neurotoxicty 5 months later that subsequently ending with death (Blayney et al. 1997; Nierenberg et al. 1998; Toribara et al. 1997). Additional studies on dermal absorption of organic mercury, especially dimethylmercury, are needed to further evaluate the risk to human health.

Acute inhalation exposure to metallic mercury in rats and rabbits have resulted in death, respiratory, gastrointestinal, hepatic, renal, neurological, and/or developmental effects (Ashe et al. 1953; Fredriksson et al. 1992; Livardjani et al. 1991b). Acute oral exposures to inorganic mercury have resulted in renal, gastrointestinal, and thyroid effects in rats and/or mice (Dieter et al. 1992; Nielsen et al. 1991; NTP 1993; Sin et al. 1990) and neurological effects in rats (Chang and Hartmann 1972a, 1972b). An acute oral MRL was derived for inorganic mercury based on renal effects in rats (NTP 1993). Acute oral exposures to organic mercury have resulted in renal, neurological, developmental, and reproductive effects in rats, mice, guinea pigs, and rabbits (Arito and Takahashi 1991; Bornhausen et al. 1980; Cagiano et al. 1990; Chang and Hartmann 1972b; Guidetti et al. 1992; Hughes and Annau 1976; Inouye and Kajiwara 1988; Jacobs et al. 1977; Khera 1973; Khera and Tabacova 1973; Magos et al. 1985; Nolen et al. 1972; Post et al. 1973; Stoltenburg-Didinger and Markwort 1990; Yasutake et al. 1991b). Well conducted animal studies on neurological effects from an acute inhalation exposure to metallic mercury or to an acute dermal exposure to organic mercury are needed because of the potential for these kinds of exposures to populations near hazardous waste sites. The potential for latent or delayed expression of toxicity after an acute exposure to mercury from all the most likely routes and forms (especially for a dermal exposure to dimethylmercury) needs to be addressed.

Intermediate-Duration Exposure. Inhalation data on intermediate-duration exposure to metallic mercury vapors are limited to case reports of individuals exhibiting cardiovascular, gastrointestinal, hematological, renal, dermal, immunological, and neurological effects similar to acute exposures (Anneroth et al. 1992; Barber 1978; Fagala and Wigg 1992; Foulds et al. 1987; Friberg et al. 1953; Schwartz et al. 1992; Sexton et al. 1976; Taueg et al. 1992). Workers inhaling diethylmercury vapors developed gastrointestinal and neurological symptoms prior to death (Hill 1943). No inhalation exposure data are available on intermediate-duration exposure to mercuric mercury. Information on intermediate-duration oral exposure to inorganic mercury is limited to the observation of neurological symptoms in a boy who ingested Chinese medicine containing mercurous mercury for several months (Kang-Yum and Oransky 1992). Intermediate-duration oral exposure to organic mercury has resulted in dermal, neurological, and developmental toxicity (Al-Mufti et al. 1976; Amin-Zaki et al. 1974; Bakir et al. 1973; Cox et al. 1989; Harada 1978; Marsh et al. 1980, 1981, 1987; Snyder and Seelinger 1976). Intermediate-duration dermal exposure to inorganic mercury has resulted in adverse gastrointestinal, renal, and immunological health effects (Anneroth et al. 1992; Kang-Yum and Oransky 1992). No studies were located that examined effects resulting from intermediate-duration dermal exposure to organic mercury.

Inhalation exposure to metallic mercury vapors for an intermediate duration has resulted in renal and/or neurological effects in rabbits (Ashe et al. 1953) and rats (Fukuda 1971; Kishi et al. 1978). No studies were located regarding effects in animals after intermediate-duration inhalation exposure to organic mercury. An intermediate inhalation MRL was not derived for metallic mercury because studies were considered inadequate. Following intermediate-duration oral exposure to inorganic mercury, adverse cardiovascular, hepatic, and renal health effects have were observed in rats and mice exposed to mercuric chloride (Andres 1984; Carmignani et al. 1992; Dieter et al. 1992; Hultman and Enestrom 1992; Jonker et al. 1993a; NTP 1993; Rana and Boora 1992). Immunological and neurological health effects were also observed (Chang and Hartmann 1972a; Dieter et al. 1983; Hultman and Enestrom 1992). An intermediate oral MRL was derived for inorganic mercury based on increased kidney weight in rats (NTP 1993). Intermediate-duration oral exposure to organic mercury has resulted in adverse cardiovascular, renal, immunological, neurological, and developmental health effects in rats, mice, cats, and monkeys (Berthoud et al. 1976; Burbacher et al. 1988; Chang and Hartmann 1972a; Chang et al. 1974; Concas et al. 1983; Elsner 1991; Evans et al. 1977; Fowler 1972; Fowler and Woods 1977; Ganser and Kirschner 1985; Hirano et al. 1986; Ilback 1991; Khera and Tabacova 1973; Leyshon and Morgan 1991; Lindstrom et al. 1991; MacDonald and Harbison 1977; Magos and Butler 1972; Mitsumori et al. 1981; Olson and Boush 1975; Sharma et al. 1982; Tsuzuki 1981; Wakita 1987; Yip and Chang 1981). The data were insufficient to derive an intermediate-duration MRL for oral exposure to organic mercury because serious adverse health effects (e.g., neurological degeneration, behavioral changes) were observed at the lowest doses (Burbacher et al. 1988; Chang et al. 1974; Chang and Hartmann 1972a). No studies were located regarding intermediate-duration dermal exposure in animals. Because populations surrounding hazardous waste sites might be exposed to higher-than-normal levels of mercury for an intermediate duration, more quantitative information on metallic and organic mercury toxicity, specifically neurotoxicity, following inhalation and oral exposure in humans and animals is needed. The potential for latent or delayed expression of toxicity after an exposure of intermediate duration needs to be addressed.

Chronic-Duration Exposure and Cancer. Occupational exposure to metallic mercury vapors has been reported to result in adverse cardiovascular, gastrointestinal, renal, ocular, immunological, and reproductive health effects (Barregard et al. 1988, 1990; Bencko et al. 1990; Bidstrup et al. 1951; Buchet et al. 1980; Cardenas et al. 1993; Cordier et al. 1991; Danziger and Possick 1973; Ehrenberg et al. 1991; Kazantzis et al. 1962; Langworth et al. 1992b; Lille et al. 1988; Moszczynski et al. 1990b; Piikivi 1989; Piikivi and Hanninen 1989; Roels et al. 1982; Schuckmann 1979; Siblerud 1990; Smith et al. 1970; Stewart et al. 1977; Tubbs et al. 1982; Vroom and Greer 1972). Substantial evidence indicates that

chronic inhalation of metallic mercury vapors results in neurotoxicity (Albers et al. 1988; Bidstrup et al. 1951; Chapman et al. 1990; Discalzi et al. 1993; Ehrenberg et al. 1991; Fawer et al. 1983; Langauer-Lewowicka and Kazibutowska 1989; Langworth et al. 1992a; Levine et al. 1982; Melkonian and Baker 1988; Ngim et al. 1992; Piikivi and Hanninen 1989; Piikivi and Tolonen 1989; Piikivi et al. 1984; Shapiro et al. 1982; Smith et al. 1970; Verberk et al. 1986; Vroom and Greer 1972; Williamson et al. 1982). A chronic inhalation MRL was derived for neurological effects observed in workers chronically exposed to metallic mercury (Fawer et al. 1983). Very limited information is available indicating that chronic-duration inhalation of organic mercury (sometimes unspecified) causes adverse cardiovascular, gastrointestinal, renal, and neurological health effects (Brown 1954; Hook et al. 1954; Hunter et al. 1940; Williamson et al. 1982). Chronic-duration ingestion of mercurous chloride resulted in dementia and irritability (Davis et al. 1974). Qualitative and quantitative data on organic mercury exposure are provided by the neurological disorders associated with ingestion of methylmercury-contaminated fish, but the length of exposure is unknown (Kutsuna 1968). Chronic occupational exposure to alkyl mercury compounds caused neurological changes in humans (Lundgren and Swensson 1949). The available evidence indicates that the differences in toxicity between inorganic and organic mercury forms are largely the result of the differences in their distribution in the body. Information concerning methyl-mercury is much more extensive than that for phenylmercury, especially considering the outbreaks of methylmercury poisoning that have occurred in Japan and Iraq.

Cardiovascular and renal health effects in rats and mice after chronic-duration ingestion of inorganic mercury have been reported (Carmignani et al. 1989; Fitzhugh et al. 1950; NTP 1993). An intermediate oral MRL based on renal effects was derived for intermediate oral exposure to inorganic mercury (NTP 1993). Chronic-duration oral exposure to organic mercury has resulted in adverse gastrointestinal, renal, developmental, neurological, and reproductive health effects in rats, mice, cats, and monkeys (Charbonneau et al. 1976; Fitzhugh et al. 1950; Hirano et al. 1986; Mitsumori et al. 1981, 1990; Rice 1989c, 1992; Rice and Gilbert 1982, 1990, 1992; Solecki et al. 1991). A chronic MRL for oral exposure to organic mercury was derived based on a study of prenatal exposures in a fish-consuming population on the Seychelles Islands (Davidson et al. 1998). Additional chronic-duration data on neurological disorders following metallic and organic mercury exposure are needed because they are a sensitive end point. Furthermore, there is a potential for chronic exposure to higher-than-normal levels of mercury in populations living in the vicinity of hazardous waste sites.

Additional chronic-duration oral exposure information in animals concerning renal effects following inorganic mercury exposure is needed to evaluate the threshold of this effect in humans following chronic exposure. The data would be useful if populations living near hazardous waste sites were to be exposed chronically to inorganic mercury that leached into near-by wells or water supplies.

Forestomach squamous cell papillomas and thyroid follicular cell carcinomas have been observed in rats and renal tubule tumors have been observed in mice following oral exposure to mercuric chloride (NTP 1993). Renal tumors have also been observed in rats and mice after oral exposure to organic mercury (Hirano et al. 1986; Mitsumori et al. 1981, 1990; Solecki et al. 1991). These results suggest the potential carcinogenicity of mercury to humans. Therefore, additional chronic-duration animal studies on metallic, inorganic, and organic mercury are needed to confirm the findings of the NTP study. Additional long-term follow-up studies examining carcinogenicity in highly exposed populations (i.e., those involved in mercury mining, or the exposed Iraqi or Japanese populations) are needed to evaluate the likelihood of tumors appearing in humans.

Genotoxicity. Although there are data from several *in vivo* studies on rats (oral exposure) and mice (intraperitoneal) indicating that inorganic and organic mercury compounds can cause clastogenic effects in mammalian germinal cells, the differences in species sensitivity, and in some cases strain sensitivity, do not permit the use of these findings for predicting a potential hazard to human genetic material (Suter 1975; Zasukhina et al. 1983). Epidemiological studies of humans occupationally or accidentally exposed to mercurials were inconclusive, but the combined results from these studies did not suggest that metallic mercury and organic mercury are clastogens for human somatic cells (Anwar and Gabal 1991; Barregard et al. 1991; Mabille et al. 1984; Popescu et al. 1979; Verschaeve et al. 1976, 1979; Wulf et al. 1986). There is, however, convincing evidence that inorganic and organic mercury compounds can interact with and damage DNA *in vitro* (Williams et al. 1987). The outcome of this damage has not been characterized, but there is some indication that mercury compounds are weak mutagens for cultured mammalian cells. In addition, *in vitro* results with human cells (Betti et al. 1992) and animal cells (Howard et al. 1991) and *in vivo* data in mice (Ghosh et al. 1991) suggest that mercury compounds can cause clastogenic effects in somatic cells. Considering the problems stated above in using the whole animal data, and the apparent species- and strain-specific responses noted in the DNA damage tests with cultured mammalian cells, the *in vitro* data, while of interest, are probably not reliable indicators of potential adverse effects in humans exposed to mercury. Well controlled human epidemiological studies are needed to determine the genetic hazard of mercury compounds to humans.

Reproductive Toxicity. Occupational exposure to metallic mercury has not been shown to result in statistically significant effects on male fertility (Alcser et al. 1989; Lauwerys et al. 1985). However, an increase in the rate of spontaneous abortions may occur (Cordier et al. 1991). A spontaneous abortion occurred in a female after ingesting an acute dose of mercuric chloride (Afonso and deAlvarez 1960). There were no studies available on dermal exposure to metallic, inorganic, or organic mercury. Additional epidemiological studies on inhalation and dermal exposure to mercury are needed to evaluate the threshold of reproductive effects in workers (including dentists and dental assistants).

Inorganic mercury exposure caused a significant increase in the incidence of resorptions in hamsters (Gale 1974). Abortions and decreased mean litter size have been observed in rats, mice, guinea pigs, and monkeys following oral exposure to organic mercury (Burbacher et al. 1988; Hughes and Annau 1976; Inouye and Kajiwara 1988; Khera 1973). There was a decrease in conceptions and an increase in early abortions and stillbirths in female monkeys exposed orally to methylmercury for 4 months, but the menstrual cycle length was not affected (Burbacher et al. 1988). However, prolonged estrous cycles were found in rats inhaling metallic mercury (Baranski and Szymczyk 1973). Adverse effects on spermatogenesis and on histopathology of the testes have been reported in several studies in animals exposed to methylmercury (Hirano et al. 1986; Mitsumori et al. 1990; Mohamed et al. 1987). There was no information on reproductive effects following dermal exposure to mercury in animals. A 90-day study is needed to provide reproductive organ pathology data on male and female animals. Multigenerational studies for inorganic and organic mercury are also needed. Additional reproductive studies are needed because reproductive-aged populations near hazardous waste sites might be exposed to mercury.

Developmental Toxicity. Occupational exposure to metallic mercury in males did not result in statistically significant effects on malformations or the number of children born (Alcser et al. 1989; Lauwerys et al. 1985). The results from an inhalation developmental rat study (Baranski and Szymczyk 1973) suggest that metallic mercury vapors may cause a higher incidence of fetal malformations, resorptions, and deaths. Dermal studies on metallic mercury in humans and animals were not available. Additional well-conducted inhalation and dermal studies on metallic mercury in animals are needed to evaluate the potential for adverse developmental effects to humans from mercury.

Inorganic mercury exposure caused a significant increase in the incidence of resorptions in hamsters (Gale 1974). No other human or animal studies were available on developmental effects following inorganic mercury exposure. Therefore, additional studies for inhalation, oral, and dermal exposures are

needed to evaluate the potential developmental toxicity of inorganic mercury to populations, specifically young children, living near hazardous waste sites. Longitudinal studies for higher dose level acute and intermediate exposures are needed to determine the potential delayed expression of toxicity.

Prenatal exposure to methylmercury from contaminated food during the early stages of pregnancy has caused neurological damage in humans (Amin-Zaki et al. 1974; Bakir et al. 1973; Choi et al. 1978; Cox et al. 1989; Engleson and Herner 1952; Harada 1978; Marsh et al. 1980, 1981, 1987; Matsumoto et al. 1965; McKeown-Eyssen et al. 1983; Snyder and Seelinger 1976). Severe neurological impairment developed in a child exposed *in utero* to methylmercury, and effects were still present at 6 years of age (Snyder and Seelinger 1976). In animals, numerous oral exposure studies on the developmental effects of organic mercury have been conducted. Disruptions in the development of the nervous system in rats, mice, hamsters, and guinea pigs (Chang et al. 1977; Inouye and Kajiwara 1988; Khera and Tabacova 1973; Reuhl et al. 1981a, 1981b) and in the immune system in rats (Ilback et al. 1991) have been reported. Behavioral changes were also observed in rats and mice (Bornhausen et al. 1980; Hughes and Annau 1976; Olson and Boush 1975). Additional long-term inhalation, oral, and dermal studies for inorganic and organic mercury are needed to evaluate the threshold of developmental effects in workers chronically exposed to mercury or in populations living near hazardous waste sites.

Immunotoxicity. The results from two occupational studies indicate a decreased serum IgG levels in workers to inhaled metallic mercury vapors (Bencko et al. 1990; Moszczynski et al. 1990b), but these studies are limited and did not evaluate potential confounders (smoking and alcohol). Other studies in similarly exposed populations did not observe an increases in serum immunoglobulins (IgA, IgG, IgE, or IgM) and autoantibody titres (antilaminin or antiglomerular basement membrane antibodies) (Bernard et al. 1987; Cardenas et al. 1993; Langworth et al. 1992b). There is limited information in humans that suggests that certain individuals may develop an autoimmune response (Tubbs et al. 1982; Moszczynski et al. 1995). Data on immunological effects following oral exposure to organic mercury compounds in humans are not available. Oral exposures to inorganic and organic mercury in animals indicate that the immune system may be a target organ for mercury. Immune deposits were observed in the intestines and kidneys of rats exposed to mercuric chloride for 2 months, but no functional changes were evident in these tissues (Andres 1984). Suppression of the lymphoproliferative response occurred at a higher dose of mercury in mice exposed to mercuric chloride for 7 weeks (Dieter et al. 1983). Reduced natural killer cell activity in spleen and blood was exhibited in mice administered a diet containing methylmercury for 12 weeks (Ilback 1991). It is unknown how an adverse effect on the immune system from exposure to one form of mercury

might affect the response to other forms or other routes of exposure (e.g., how an adverse immune effect induced by inhalation of mercury vapor from dental amalgam might effect the dose-response from exposure to ingested methylmercury). Therefore, the potential for immunotoxic effects from exposure to mercury vapor, mercury salts, or methylmercury separately or in combination is of considerable importance and warrants further research, especially from low level chronic exposures.

Neurotoxicity. The nervous system is the major target organ for metallic and organic mercury through inhalation and oral routes, respectively. In humans, the neurological effects of metallic mercury have been observed primarily after acute high-concentration exposures (accidental) to intermediate and chronic low-concentration exposures (occupational). Tremors and irritability are the most prominent symptoms of inhaled metallic mercury in humans (Albers et al. 1988; Bidstrup et al. 1951; Fawer et al. 1983; Piikivi et al. 1984). Information on effects in humans from oral exposure includes case histories, for example, a chronic oral exposure to a laxative containing mercurous chloride (Davis et al. 1974), acute to intermediate duration ingestion of high levels of methylmercury-contaminated food (Bakir et al. 1973; Kutsuna 1968), or to chronic low-level exposures from fish or marine mammals containing methylmercury (Davidson et al. 1995aa, 1995b; Grandjean et al. 1997b, 1998). Case histories of dermal exposure to inorganic mercury cite similar neurological effects from acute (Bourgeois et al. 1986; DeBont et al. 1986) or chronic exposures (Dyall-Smith and Scurry 1990).

The neurotoxicity of inhaled metallic mercury has been studied in animals for acute and intermediate exposures (Ashe et al. 1953; Ganser and Kirschner 1985; Kishi et al. 1978). Behavioral, motor, and cognitive effects, as well as histopathological changes in the brain, were reported in rats, rabbits, and mice. Neurological disturbances in rats and mice resulted from acute, intermediate, and chronic oral exposures to mercuric mercury (Chang and Hartmann 1972b; Ganser and Kirschner 1985). Oral exposure to organic mercury in animals produced a range of neurological changes (Charbonneau et al. 1976; Evans et al. 1977; Magos and Butler 1972; Rice and Gilbert 1982; Sharma et al. 1982; Tsuzuki 1981). A chronic inhalation MRL was derived for metallic mercury. Additional animal studies are needed, however, to evaluate the neurotoxicity of inorganic mercuric salts to resolve some of the conflicting findings from pervious work (Chang and Hartmann 1972b; Ganser and Kirschner 1985; Goldman and Blackburn 1979; NTP 1993). *In vivo* studies are needed to evaluate the mechanisms of neurotoxic effects seen in *in vitro* studies, i.e., the lipoperoxidation and cell injury in methylmercury-exposed cerebellar granule cells (Sarafian and Verity 1991). Further evaluation is needed in humans and animal models of the potential for neurological effects and delayed neurotoxicity from chronic low level exposures to organic and inorganic mercury, especially

from multiple sources (i.e., organic mercury from fish consumption in conjunction with metallic mercury released from dental amalgam).

Epidemiological and Human Dosimetry Studies. There have been a number of occupational studies on workers chronically exposed to metallic mercury vapors. Mercury exposure (as measured by urine or blood mercury levels) and neurological effects have been evaluated (Adams et al. 1983; Miller et al. 1975; Roels et al. 1982; Smith et al. 1970). The most obvious deficiency in these epidemiological studies is the absence of good measures of exposure. Additional data are needed on the potential health effects for populations near hazardous waste sites based upon specific identification of the form of mercury and the pathways of exposure (i.e., the levels of exposure that populations near waste sites may actually experience from inorganic mercury in the air, water, and soil, or methylmercury in contaminated food). An area of considerable controversy, which is in need of good epidemiological data, is the potential for adverse effects from the mercury released from dental amalgam. Although this is not an exposure pathway associated with hazardous waste sites, mercury from amalgam represents a major contributor to the total body burden for a large percentage of the population, and thus must be factored into an assessment of the toxicokinetic behavior and toxic effects of mercury originating from a waste site. Long term longitudinal studies are needed for all dose durations and forms to evaluate delayed or persistent expression of mercury toxicity.

Biomarkers of Exposure and Effect

Exposure. Blood and urine mercury levels have been used as biomarkers of high level exposure in acute and chronic studies for both inorganic and organic mercury (Akesson et al. 1991; Naleway et al. 1991; Verschoor et al. 1988). Hair has been used as a biomarker for chronic low level organic mercury exposure (Nielsen and Andersen 1991a, 1991b; Oskarsson et al. 1990), with an awareness of the potential for external contamination (Clarkson et al. 1983). Further development of more sensitive tests to measure mercury in expired air and retention in hair are needed for monitoring short- and long-term exposures, respectively, for populations at risk.

As seen in other studies comparing European to Japanese hair mercury levels, the hair levels reported by Nakagawa (1995) of 2–4 ppm for a Japanese population are 10–20 times higher than levels observed in the Drasch et al. (1997) study (median, 0.247 μg/g in hair; range, 0.43–2.5 μg/g). These differences in the mercury exposure may affect not only the mercury hair levels but also the mercury hair-to-tissue

correlations. Further study is needed on the effects that the exposure level of methylmercury (as well as other forms of mercury) has on tissue distributions and the correlation to biomarkers of exposure.

There are potential confounding factors and other factors to consider when assessing mercury exposure based upon mercury hair levels. Mercury may be deposited to hair from the air when significant sources of mercury are present in the air or when certain hair treatments are used (Hac and Krechniak 1993; WHO 1991). Potential sources of external mercury exposure should, therefore, be evaluated as part of an exposure assessment. Some studies also report a sex related difference in mercury tissue levels. Nielson et al. (1994) observed a significant sex-related differences in the toxicokinetics of methylmercury in mice following administration of a single radiolabeled dose. Drasch et al. (1997) reported that mercury levels in all tissues assayed in their human cadaver study had higher levels compared to male tissues. The difference was significant for the kidney (median female kidney mercury level=92.0 ng/g, males=40.8 ng/g; p=0.002). In blood and urine there was a similar trend. In contrast, the authors report that mercury hair levels in females were significantly lower than in males (median females=205 ng/g, males 285 ng/g; p=0.02). Nakagawa (1995) also report higher mean mercury hair levels in males (2.98 µg/g) compared with females (2.02 µg/g) in a Japanese population. Further research is, therefore, needed to characterize potential sex related difference in the toxicokinetics of mercury under different exposure scenarios.

Further research on other biomarkers of mercury does not warrant a high priority.

Of particular importance is the collection of pharmacokinetic data showing the relationship between low-level exposure (acute, intermediate, and chronic) and blood and urine levels throughout the study .duration. Also tissue levels at necropsy should be taken immediately after cessation of dosing. In animal studies, a similar group of animals should be followed for urine (and blood, but not as important here) mercury levels for periods of 30, 60, 90, and 120 days postdosing to examine whole-body excretion, and necropsy tissue samples should also be taken from several animals at 30, 60, 90, and 120 days postdosing. Primates would be the best animal model, but rodent models could suffice.

A needed study is a longitudinal epidemiology study that tracked daily individual exposure levels in chloralkali industry workers, fluorescent lightbulb manufacturers, or other mercury utilizing industries, and associated these exposure levels with weekly urine and blood samples for a period of 1–2 years. Neurobehavioral testing (using tests from ATSDR’s recommended test battery for adults) should be used

conducted at 6-month intervals. Workers new to these industries would be the best subjects, since their pre-exposure blood and urine levels could be used as reference values.

A biomarker/exposure could also be conducted in persons with dental amalgam fillings. Urine levels should be tracked in those with fillings and in those with removed or replaced amalgam fillings. There are a number of confounding factors and logistical difficulties in conducting such studies, and new study protocols should be developed to address the problems encountered in previous studies.

Effect. Potential biomarkers of effect for mercury-induced renal toxicity have been well described (Cardenas et al. 1993; Lauwerys et al. 1983; Rosenman et al. 1986; Verschoor et al. 1988). Biomarkers for neurological changes (e.g., paresthesia, decreased motor function, and impaired nerve conduction) have also been described (Clarkson et al. 1976; Shapiro et al. 1982). There is long history of evaluation of the neurophysiological and neuropsychological effects associated with mercury levels in blood, urine, and (Levine et al. 1982; Vroom and Greer 1972; Williamson et al. 1982). More recently, studies are evaluating cognitive and neurobehavioral effects with increasing sophistication in the assays and analyses that are used (Davidson et al. 1998; Grandjean et al. 1997b, 1998). Additional biomarkers are needed in this continuing effort to resolve subtle cognitive or neurobehavioral effects, and immune system effects from chronic low level exposures to methylmercury in food or metallic mercury released from dental amalgam, especially in sensitive populations.

Absorption, Distribution, Metabolism, and Excretion. Limited data are available to assess the relative rate and extent of absorption in humans following inhalation exposure to metallic mercury (Barregard et al. 1992; Berlin et al. 1969; Friberg and Vostal 1972; Hursh et al. 1976; Teisinger and Fiserova-Bergerova 1965) and in humans and animals following oral exposure to both inorganic salts and organic mercury (Aberg et al. 1969; Clarkson 1971, 1972a, 1989; Endo et al. 1989, 1990; Fitzhugh et al. 1950; Friberg and Nordberg 1973; Kostial et al. 1978; Miettinen 1973; Nielsen 1992; Nielsen and Andersen 1992; Rice 1989b; Suzuki et al. 1992; Urano et al. 1990; Weiss et al. 1973; Yeoh et al. 1989). Indirect evidence of absorption following inhalation exposure in humans and animals is reported for inorganic and organic mercury (Clarkson 1989; Ostlund 1969; Warfvinge et al. 1992; Yoshida et al. 1990, 1992). Only limited quantitative data were located regarding dermal uptake of metallic mercury in humans (Hursh et al. 1989). Information is needed regarding the rate and extent of dermal absorption of inorganic and organic mercury in humans and animals. Quantitative information concerning the inhalation and oral absorption of mercury (all forms)are needed.

In general, quantitative data are available to evaluate the rate and extent of distribution, metabolism, and elimination of mercury in humans and animals following inhalation and oral exposure. Data on distribution, metabolism, and excretion following dermal exposure are lacking for all forms of mercury. The distribution data for metallic, inorganic and organic mercury are similar in humans and animals (Aschner and Aschner 1990; Berlin 1963; Cherian and Clarkson 1976; Cherian et al. 1978; Clarkson 1989; Clarkson and Magos 1978; Danscher et al. 1990; Grandjean et al. 1992; Nielsen and Andersen 1990, 1991a, 1991b; Nordberg 1976; Schionning et al. 1991; Sin et al. 1983; Suzuki et al. 1992; Warfvinge et al. 1992; Yeoh et al. 1989; Yoshida et al. 1990, 1992). No quantitative distribution data were located for organic mercury compounds following inhalation exposure. The oxidation and reduction reactions that control the disposition of elemental mercury were identified in both animals and humans (Clarkson 1989; Halbach and Clarkson 1978; Nielsen-Kudsk 1973). Quantitative data on the biotransformation of organic mercury are limited (Norseth and Clarkson 1970). Reliable quantitative evidence on excretion of metallic and inorganic mercury in humans and animals following inhalation exposure is available (Cherian et al. 1978; Hursh et al. 1976; Joselow et al. 1968b; Lovejoy et al. 1974).

As discussed in the section on data needs for biomarkers, further study is needed on the effects that the exposure level of methylmercury (as well as other forms of mercury) has on tissue distributions and the correlation to biomarkers of exposure. Age appears to be a factor in the elimination of mercury in rats following inorganic and organic mercury exposures (Daston et al. 1986; Thomas et al. 1982). Elimination of methylmercury in rats may also be sex-related (Ballatori and Clarkson 1982). Nielson et al. (1994) observed a significant sex-related differences in the toxicokinetics of methylmercury in mice following administration of a single radiolabeled dose. Drasch et al. (1997) reported that mercury levels in all tissues assayed in their human cadaver study had higher levels compared to male tissues. Nakagawa (1995) also report higher mean mercury hair levels in males (2.98 μg/g) compared with females (2.02 μg/g) in a Japanese population. Further research is, therefore, needed to characterize potential sex related difference in the toxicokinetics of mercury under different exposure scenarios.

Insufficient data are available to assess whether or not there are any differences in absorption, distribution, metabolism, and excretion of mercury with respect to time or dose (i.e., if saturation phenomena occur). The majority of the available toxicokinetic data involve acute exposures to single doses. For all three routes, studies are needed that compare various dose levels and durations in order to determine if there are any differences in the toxicokinetics of mercury. Little is known about how mercurials are eliminated from specific organs. In particular, the mechanism by which mercury is eliminated from the brain is

unknown. This information is needed to design better treatment drugs and protocols. Mechanistic studies are needed on how mercury (in its various forms) is excreted and how such activities can be enhanced.

An important priority research and data need is a study of the effects of dietary selenium on the absorption and toxicity of methylmercury. Primates would be the most appropriate species for such a study. Oral dosage levels (in food) should cover an sufficient dose range to provide useful information for high fish consuming populations. Mercury excretion should also be measured and compared with controls at least weekly, with the entire study length being not less than 6 months, and preferably one to two years in duration. Concurrent neurobehavioral testing should be included, if possible, and be conducted at fixed intervals depending upon the duration of the study.

Comparative Toxicokinetics. There is only limited data available on species differences in absorption rates following oral exposures to all forms of mercury, and the results are negative (i.e., no differences) (Clarkson 1971, 1972a; Friberg and Nordberg 1973; Nielsen and Andersen 1990; Rice 1989b). There are data concerning inhalation absorption of metallic and inorganic mercury (Berlin et al. 1969; Cherian et al. 1978; Clarkson 1989; Hursh et al. 1976); however, the data are insufficient to allow for interspecies comparisons (Ostlund 1969). Studies comparing the inhalation absorption of all forms of mercury in humans and animals are needed to improve the utility of animal data in assessing human risk. The limited information available on dermal exposure suggests that dermal absorption of both inorganic and organic mercury compounds occurs in humans and animals, although no comparison of the rate or extent of absorption can be made between species (Gotelli et al. 1985; Hursh et al. 1989; Laug and Kunze 1949; Schamberg et al. 1918). As with inhalation exposure, studies comparing the dermal absorption of all forms of mercury in humans and animals are needed to improve the utility of animal data for assessing human risk.

The distribution of mercury in humans and animals appears to be similar. The lipophilic nature of metallic mercury results in its distribution throughout the body in humans (Takahata et al. 1970) and in animals (Berlin and Johansson 1964; Berlin et al. 1966). Distribution of inorganic mercury compounds resembles that of metallic mercury; however, human distribution is preferentially to the kidneys, liver, and intestines. Also, levels in the brain are substantially lower, as these compounds have a lower lipophilicity. Distribution of organic mercury compounds is also similar to that of metallic mercury. The ability of methylmercuric compounds to cross the blood-brain and placental barriers enables ready distribution to all tissues, although, again, the highest levels are found in the kidneys. Phenylmercuric compounds are

initially distributed in a similar manner to methylmercury; however, the distribution eventually resembles that of inorganic mercury.

The available evidence suggests that feces and urine constitute the main excretory pathways of metallic mercury and inorganic mercury compounds in both humans and animals. Additional excretory routes following metallic and inorganic mercury exposure include exhalation and secretion in saliva, sweat, bile, and breast milk (Joselow et al. 1968b; Lovejoy et al. 1974; Rothstein and Hayes 1964; Sundberg and Oskarsson 1992; Yoshida et al. 1992). Excretion following exposure to organic mercury is considered to be predominantly through the fecal route in humans. Evidence from studies in humans and animals (mice, rats) suggests that exposure to methylmercury leads primarily to biliary secretion, while excretion is initially through the bile; it then shifts to the urine following phenylmercury exposure (Berlin and Ullberg 1963; Berlin et al. 1975; Gotelli et al. 1985; Norseth and Clarkson 1971). No further comparative studies on excretion are warranted because there is no apparent difference in the excretion of mercury in any form in humans and animals.

Two PBPK models have recently been published on the pharmacokinetics of methylmercury in rats (Farris et al. 1993; Gray 1995). Additional PBPK studies are needed to support species and dose extrapolations, and a better understanding of the underlying toxic and kinetic mechanisms is needed in support of human risk assessments.

Validation of *in vitro* data is a major need. Much of the data from *in vitro* experimentation is based on unrealistic concentrations of the toxicant or is derived from studies using non-physiological designs. In particular, more validation is needed for immunotoxicity studies and biochemical studies.

Methods for Reducing Toxic Effects. Nonspecific methods or treatments for reducing absorption following mercury exposure include the administration of chelators or protein solutions to neutralize and bind to inorganic mercury compounds (Bronstein and Currance 1988; Florentine and Sanfilippo 1991; Gossel and Bricker 1984). The use of a particular chelator is dependent upon the type of mercury exposure (Gossel and Bricker 1984). Chelation therapy is the treatment of choice for reducing the body burden of mercury (Florentine and Sanfilippo 1991; Gossel and Bricker 1984; Haddad and Winchester 1990). However, chelation releases mercury from soft tissues that can then be redistributed to the brain. Additional research is needed to elucidate the mechanisms of absorption and distribution of inorganic and organic mercury. Animal studies suggest that antioxidants may be useful for decreasing the toxicity of

mercury. Additional work studying the effectiveness of prophylactic administration of vitamin E (or other antioxidants) and of proper diet are needed. Improved chelation and drug therapies for treating acute and chronic mercury poisonings are greatly needed.

Children's Susceptibility. The systemic health effects from different forms of mercury and exposure routes have been fairly well characterized (EPA 1997; Sue 1994; WHO 1990). There is generally sufficient information on the symptoms to resolve the form and route of exposure when children are exposed to high levels of mercury. There is less information to assist the physician or public health official in recognizing the symptoms that might arise from lower level exposure to multiple forms of mercury (e.g., dental amalgam and fish) and multiple pathways (inhalation and ingestion). Whether concurrent exposures would result in a different presentation of symptoms would be important information in determining the best therapeutic treatment. Some health effects categories are not well defined (e.g., immune responses). Earlier identification of immunotoxicity is of concern for children because of the progressive nature of hypersensitization to environmental pollutants, and the burden that a compromised immune system can place on a person's long-term health.

There are not presently adequate measures for neurologic development. Delayed developmental effects are of grave concern for children exposed to mercury; methods for early determination and detection of progressively worsening changes in a child's behavioral or cognitive function are needed. For the measures to be truly useful they should in some way be integrated into a more directed exposure assessment and body burden analysis and to resolve the contribution from other influences on cognitive abilities and behaviors. Other data needs related to developmental effects are discusses above under Developmental Toxicity.

Pharmacokinetics are different for children, and more data are needed to improve chelation therapies for both acute high-level poisoning and for chronic low-level exposures. This is perhaps the area that deserves the most attention because accidental poisonings continue to occur and there are virtually no therapies to ameliorate the inevitable progression of mercury intoxication. Since environmental levels of mercury are also continuing to rise, and levels in food will concurrently rise, strategies to boost the body's ability to eliminate absorbed mercury are going to become increasingly important (i.e., the alternative is to change dietary patterns, i.e., eat less fish, and the risk/benefits of doing that are already being hotly debated).

There appears to be adequate information on the metabolism of mercury, and there are no special metabolites or metabolic pathways that are unique to children and require further evaluation.

The mechanism of mercury toxicity is still largely unknown. It is not known whether there are unique mechanisms of action for the toxic effects in children that would require special consideration for treatment modalities, but at present it appears that target site is determined more by the pharmacokinetics (i.e., which tissues end up with the highest levels) than by a specific mechanism of action (e.g., a receptor binding-process initiating type of mechanism).

The results of a number of accidental food poisonings indicate that children are more vulnerable, and this vulnerability may be a function of easier access of mercury to the systemic circulation and brain, or it may be because disruption of cell growth and organization is more critical for children in developmental stages of growth. More data are needed to determine if the vulnerability of children is due to less plasticity to insult of analogous target tissue in adults, or because target tissues actually receive more toxic agent.

There are not adequate biomarkers of exposure nor adequate access to biomarkers of exposure. Hair, urine, and blood levels are gross measures of body burden and do not provide the essential information about levels of mercury at target tissues. Research is needed into better (preferably noninvasive) monitoring tools. Research is also needed on how to make monitoring tests readily and inexpensively available to the general public. Mercury is one of the top ten most hazardous substances, and its levels are increasing in the environment. There is considerable anxiety present in the general population about potential mercury toxicity from dental amalgam, but this occurs in the absence of good information on actual body burdens. The general public and health officials would benefit from readily available ways for individuals to measure personal and family member mercury body burdens.

The interactions of immediate interest are those that either affect absorption from the gastrointestinal tract or that prevent or reduce mercury toxicity. No information was identified to indicate that mercury interacts differently with iron or zinc, for example, in a child's body then it would in an adult, although the difference in children's physiology and morphology may result in a different response to that interaction. Except for the latter, which is again a toxicokinetic question, chemical interactions do not appear to be a data need.

There is a data need to develop better chelation therapies, better ways to prevent absorption of mercury into the body of children, and better ways to interfere with the mechanism of action, especially for damage to the nervous system. The current literature continues to grow with case histories of poisonings where

supportive therapy and passive observation of a progressively deteriorating health status are the best that can be done.

No information was found that parental exposure to mercury results in heritable defects or deficits in germ cell function that would be translated to the offspring. There is considerable information on the transfer of mercury from the mother to the developing child, both during the prenatal period via the placenta and during postnatal nursing; both inorganic mercury and organic mercury pass from mother to child. This is an area of active research primarily to characterize the dose, duration, and form of mercury to which the child is being exposed. Further work in this area is needed.

Child health data needs related to exposure are discussed in Section 5.8.1, Data Needs: Exposure of Children.

2.11.3 Ongoing Studies

Ongoing studies regarding mercury's health effects and mechanisms of action were reported in the Federal Research In Progress (FEDRIP 1998) database. Table 2-14 lists these studies.

3. CHEMICAL AND PHYSICAL INFORMATION

3.1 CHEMICAL IDENTITY

Information regarding the chemical identity of mercury compounds is located in Table 3-1.

3.2 PHYSICAL AND CHEMICAL PROPERTIES

Information regarding the physical and chemical properties of mercury compounds is located in Table 3-2. Mercuric acetate has been included as an organic form of mercury. However, the bonds of the salt are not covalent and, in aqueous solution, the mercury behaves like an inorganic form.

4. PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL

4.1 PRODUCTION

Mercury is a naturally occurring element that is usually found as mercuric sulfide (cinnabar), an insoluble, stable compound. It occurs in the earth's crust at levels averaging 0.5 ppm, but the actual concentration varies considerably depending on location (Merck 1989; Sidle 1993). Mercury is mined using both open pit (10% of production) and underground mining techniques (90%) (Drake 1981).

Mercury ores are processed inexpensively to produce metallic mercury. Due to the low boiling point of elemental mercury, mercury can be refined by heating the ore and condensing the vapor to form metallic mercury. This method is 95% efficient and yields mercury that is 99.9% pure. The methods used to refine mercury ores are uncomplicated. Smaller refineries use simple firing and condensing equipment, while larger operations use continuous rotary kilns or mechanically feeding and discharging multiple-hearth furnaces (Carrico 1985).

Table 4-1 lists the facilities in each state that manufacture or process mercury, the intended use, and the range of maximum amounts of mercury that are stored on site. There are currently 34 facilities that produce or process mercury in the United States. The data listed in Table 4-1 are derived from the Toxics Release Inventory (TRI96 1998). Since only certain types of facilities are required to report (EPA 1996d), this is not an exhaustive list.

With the closure of the McDermitt Mine in Nevada in 1990, mercury ceased to be a principal product of U.S. industry (USGS 1997). The figures for the total output of this mine have been withheld by the Bureau of Mines to avoid disclosure of company proprietary data (see Table 4-2). As of 1995, eight mines in California, Nevada, and Utah produced mercury as a by-product from gold mining operations. Metals in the gold ores are extracted with an aqueous cyanide solution, with typical mercury recoveries of between 10 and 20% (Jasinski 1993; USGS 1997). Approximately 58 metric tons of mercury were produced as a by-product from 8 mines in 1991 and 64 metric tons were produced as a by-product from 9 mines in 1992. Since then, production volumes have been withheld to avoid disclosing company proprietary data.

Although most of the world production of mercury is generated by mercury mines, most of the mercury produced in the United States comes from secondary production sources (recycling) (EPA 1997).

Secondary production of mercury includes the processing of scrapped mercury-containing products, and industrial waste and scrap (EPA 1997). As a result of the increasingly stricter regulations that have been placed on the disposal of mercury-containing products, secondary production using recycled mercury has increased from 165 metric tons in 1991 to 176 metric tons in 1992, 350 metric tons in 1993, 466 metric tons in 1994, and 534 metric tons in 1995. Mercury was recovered from various waste materials, including mercury batteries, dental amalgams, switches (including thermostats), manometers, chloralkali wastewater sludges, chemical solutions, and fluorescent light tubes. Refining of the recycled mercury was dominated by three companies: Bethlehem Apparatus Co., Hellertown, Pennsylvania; D.F. Goldsmith Co., Evanston, Illinois; and Mercury Refining Co., Albany, New York (USGS 1997).

4.2 IMPORT/EXPORT

Until 1989, the United States was a net importer of mercury. After that, market values of mercury fluctuated and consumption diminished, leading to a decreased need for imported mercury (Carrico 1985; Drake 1981). U.S. imports of mercury fell sharply between 1987 and 1990 (Jasinski 1993; Reese 1990). The import volumes decreased drastically during the period from 1987 to 1990: 636 metric tons in 1987, 329 metric tons in 1988, 131 metric tons in 1989, and 15 metric tons in 1990 (see Table 4-2). However, import figures generally have increased substantially since 1990: 56 metric tons in 1991, 92 metric tons in 1992, 40 metric tons in 1993, 129 metric tons in 1994, and 277 metric tons in 1995 (USGS 1997). The major reason for the recent escalation in mercury imports is the suspension of mercury sales from the National Defense Stockpile (NDS) in 1994, which had been the major supplier of mercury to the domestic market in recent years. The suspension was imposed by Congress after the EPA raised questions about potential problems associated with the release of mercury. Also, there was concern about the export of NDS mercury for uses banned in the United States (USGS 1997).

From 1978 to 1988, figures were unavailable for the amount of mercury exported by the United States. The U.S. export figures for mercury from 1989 to 1992 are: 221 metric tons in 1989, 311 metric tons in 1990, 786 metric tons in 1991 (Jasinski 1993; Reese 1990), 977 metric tons in 1992, 389 metric tons in 1993, 316 metric tons in 1994, and 179 metric tons in 1995 (USGS 1997) (see Table 4-2). General trends in exportation of mercury are difficult to characterize because the data are unavailable for the 11 years prior to 1989. However, the decline of exports in 1995 is largely due to the suspension of sales from the NDS (USGS 1997).

Major mercury producing countries (primary production from mining operations) in the world currently include Algeria, China, Czechoslovakia, Finland, Kyrgyzstan, Mexico, Morocco, Russia, Slovakia,

Slovenia, Spain, Turkey, and the Ukraine (USGS 1997). The world reserves of mercury are estimated to be sufficient to supply enough product for 100 years, given current production and consumption estimates (Jasinski 1993).

4.3 USE

Mercury has many applications in industry due to its unique properties, such as its fluidity, its uniform volume expansion over the entire liquid temperature range, its high surface tension, and its ability to alloy with other metals. However, domestic consumption of mercury has shown a downward trend since the early 1970s. In 1995, consumption was 463 metric tons, down 10% from 1994. The largest commercial use of mercury in the United States was for electrolytic production of chlorine and caustic soda in mercury cells, accounting for 35% of domestic consumption. Manufacture of wiring devices and switches accounted for 19%, measuring and control instruments for 9%, dental equipment and supplies used 7%, electric lighting used 7%, and other uses used 21% (EPA 1997; USGS 1997). Due to the high toxicity of mercury in most of its forms, many applications have been canceled as a result of attempts to limit the amount of exposure to mercury waste.

Electrical applications. Mercury is a critical element in alkaline batteries. In the past, excess amounts of mercury were used in batteries; however, alkaline battery manufacturers in Europe, Japan, and the United States are now reducing the mercury load from 0.1% to 0.025% of battery content. This reduction will ultimately limit the amount of mercury needed in the battery industry to below 4 metric tons per year (Cole et al. 1992; Reese 1990). Mercuric oxide has become increasingly important commercially in the production of galvanic cells with mercuric oxide anodes in combination with zinc or cadmium cathodes. The voltage for these small, button-shaped batteries remains constant during discharge. The batteries are used in hearing devices, digital watches, exposure meters, pocket calculators, and security installations (IARC 1993), but their use has been declining as non-mercury replacement battery production has increased. Some electrical lamps use mercury vapors in discharge tubes. These lamps are efficient, long-lasting, and produce more lumens per watt than most other industrial lamps (Drake 1981). Wiring and switching devices, such as thermostats and cathode tubes, use mercury because of its predictable contact resistance, thermal conductivity, and quiet operation (Carrico 1985; Drake 1981). In 1985, 64% of the mercury used in the United States was for electrical applications. This use declined to 29% in 1992 (IARC 1993).

Medical applications. Metallic mercury is used in dental restorations because of its ability to alloy with other metals. The World Health Organization (WHO 1991) estimated that, in industrialized countries, about 3% of the total mercury consumption is for dental amalgams. Based on 1992 dental manufacturer specifications, amalgam (at mixing) contains approximately 50% metallic mercury, 35% silver, 9% tin, 6% copper, and trace amounts of zinc. Estimates of annual mercury usage by United States dentists range from approximately 100,000 kg in the 1970s to 70,000 kg in 1995. More than 100 million fillings are replaced each year in the United States (Lorscheider et al. 1995). Until 30 years ago, mercury compounds were used extensively in pharmaceuticals. Mercury salts were components of antiseptics (e.g., merthiolate, mercurochrome), diuretics, skin lightening creams, and laxatives (calomel). Organic mercury compounds were employed in antisyphilitic drugs and some laxatives. Phenylmercury acetate was used in contraceptive gels and foams and as a disinfectant (IARC 1993). Since then, more effective and less toxic alternatives have replaced most pharmaceutical uses of mercury. Medical equipment, such as thermometers and manometers, use metallic mercury to measure temperature and pressure (Carrico 1985).

Chemical/mining applications. Mercury is a catalyst in reactions to form polymers, such as vinyl chloride and urethane foams. The preparation of chlorine and caustic soda (NaOH) from brines also uses mercury as a catalyst. In this process, mercury is used as a moving cathode to separate sodium and chlorine (Rieber and Harris 1994). This mercury can be recycled with 95% efficiency (Drake 1981). Consumption occurs as mercury is lost in wastewater treatment, is recaptured, reprocessed, and sent to landfills (Rieber and Harris 1994). Mercuric oxide and mercuric sulfide are used as pigments in paints (Winship 1985). Gold mining operations use mercury to extract gold from ores through amalgamation (Carrico 1985).

Other applications. Phenylmercuric acetate has been used in aqueous preparations such as inks, adhesives, and caulking compounds, as a catalyst for the manufacture of certain polyurethanes, and as a fungicide in seed dressings and interior and exterior paints (IARC 1993; Reese 1990). Dimethylmercury is used to prepare mercury nuclear magnetic resonance standards (Blayney et al. 1997) and mass spectrometer mercury calibration standards (Toribara et al. 1997).

Discontinued applications. The use of phenylmercuric acetate as a fungicide in interior latex paints was banned in 1990 (Reese 1990), and its use in exterior paint was banned in 1991 (Hefflin et al. 1993). Both of these bans were prompted because of releases of mercury vapors as the paint degraded. Alkyl mercurial

compounds were used until the mid-1970s as a treatment to disinfect grain seeds. Most other agricultural applications of mercury compounds in bactericides and fungicides have been banned due to the toxicity of mercury. Mercuric nitrate was used in the production of felt hats to hydrolyze rabbit fur. The use of mercury as a wood preservative has ceased due to the use of polyurethane (Drake 1981).

4.4 DISPOSAL

Mercury is an element, and therefore its chemical structure cannot be further broken down. In its elemental form, mercury is highly toxic when inhaled. Therefore, incineration of mercury is not recommended as a disposal method. Mercury-containing waste products include waste effluents from chloralkali plants and discarded mercury-containing mechanical and electrical devices (Carrico 1985). Under current federal guidelines, mercury and its compounds are considered hazardous substances, and various regulations are in effect to control the emission of mercury into the environment (especially organic compounds) (Carrico 1985). Emissions from mercury ore processing facilities and mercury cell chloralkali plants are limited to 2.3 kg/day/facility. Emissions of mercury from the incineration or drying of wastewater sludges is limited to 3.2 kg/day/facility (EPA 1975a, 1975b). In addition, dumping wastes containing more than trace amounts of mercury is prohibited.

Recycling of mercury-containing compounds is an important method of disposal. Recycling (retorting) is a treatment for five categories of mercury wastes including: (D009) characteristic mercury; (K106) chlor-alkali waste; (P065) mercury fulminate; (P092) phenylmercuric acetate; and (U151) elemental mercury (see Table 7-1). From 1987 to 1991, annual production of mercury from old scrap averaged nearly 180 metric tons, equivalent to 16% of the average reported consumption during that period (Jasinski 1993). Virtually all mercury can be reclaimed from mercury cell chloralkali plants, electrical apparati, and control instruments when plants are dismantled or scrapped (Carrico 1985). Increased recycling would decrease the mercury load from waste sites and treatment plants. As environmental concerns increase with respect to the disposal of mercury, the recovery by recycling and industrial processes will become a more significant source of domestic supply (Carrico 1985).

Of the estimated 646,896 pounds of mercury reported in the Toxics Release Inventory (TRI) in 1991 to have been released to the environment, the largest percentage (96%, or 619,310 pounds) was transferred off-site from 51 industrial processing facilities, and another 314 pounds were transferred to publicly owned

treatment works (POTWs) (TRI91 1993) (see Section 5-2 for additional information). By comparison, in 1994, only 83,064 pounds of mercury (less than 14% of the total reported in 1991) were released to the environment; and of this amount, 81% (67,480 pounds) was transferred off-site from 29 large processing facilities (TRI94 1996) and an estimated 15 pounds of mercury were released to POTWs (TRI94 1996). Again, by comparison, in 1996, only 84,772 pounds of mercury (less than 14% of the total reported in 1991) were released to the environment and of this amount, 78% (66,573 pounds) was transferred off-site from 34 large processing facilities and an estimated 15 pounds of mercury were released to POTWs (TRI96 1998). Releases of mercury to each of these compartments—the total environment, POTWs, and the volume transferred off-site—decreased dramatically (approximately 90%) in only 5 years. The data listed in the TRI should be used with caution, because only certain types of facilities are required to report (EPA 1996d). This is not an exhaustive list. A facility is required to report information to the Toxics Release Inventory only if the facility is a general manufacturing or processing facility with 10 or more full-time employees that produces, imports, or processes 75,000 or more pounds of any TRI chemical or that uses more than 10,000 pounds of a TRI chemical in a calendar year. No additional information on trends in disposal volume or on specific methods of disposal was located.

In addition, unknown quantities of metallic mercury used in religious or ethnic ceremonies, rituals, and practices (see Sections 5.4.4, 5.6, and 5.7) may reach municipal landfill sites by being improperly disposed of in domestic garbage, or may reach POTWs by being improperly discarded into domestic toilets or sink drains (Johnson [in press]). A survey was conducted to determine the use patterns of elemental mercury in the Latin American and Caribbean communities in New York City (Johnson [in press]). In a survey of 203 adults, about 54% used elemental mercury in various religious and ethnic practices. Of these users, 64% disposed of the mercury in household garbage, 27% flushed the mercury down the toilet, and 9% disposed of the mercury outdoors. It is commonly thought that the high mercury load found in sewage and garbage in New York City comes from dental clinics; however, improper disposal of mercury by religious practitioners in the Latin American and Caribbean communities may also contribute to this load (Johnson [in press]).

5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Mercury occurs naturally as a mineral and is distributed throughout the environment by both natural and anthropogenic processes. The natural global bio-geochemical cycling of mercury is characterized by degassing of the element from soils and surface waters, followed by atmospheric transport, deposition of mercury back to land and surface water, and sorption of the compound to soil or sediment particulates. Mercury deposited on land and open water is in part revolatilized back into the atmosphere. This emission, deposition, and revolatilization creates difficulties in tracing the movement of mercury to its sources. Major anthropogenic sources of mercury releases to the environment include mining and smelting; industrial processes involving the use of mercury, including chlor-alkali production facilities; combustion of fossil fuels, primarily coal; production of cement; and medical and municipal waste incinerators and industrial/commercial boilers (EPA 1996b).

The element has three valence states and is found in the environment in the metallic form and in the form of various inorganic and organic complexes. The major features of the bio-geochemical cycle of mercury include degassing of mineral mercury from the lithosphere and hydrosphere, long-range transport in the atmosphere, wet and dry deposition to land and surface water, sorption to soil and sediment particulates, revolatilization from land and surface water, and bioaccumulation in both terrestrial and aquatic food chains.

Potential sources of general population exposure to mercury include inhalation of mercury vapors in ambient air, ingestion of drinking water and foodstuffs contaminated with mercury, and exposure to mercury through dental and medical treatments. Dietary intake is the most important source of nonoccupational exposure to mercury, with fish and other seafood products being the dominant source of mercury in the diet. Most of the mercury consumed in fish or other seafood is the highly absorbable methylmercury form. Intake of elemental mercury from dental amalgams is another major contributing source to the total mercury body burden in humans in the general population (WHO 1990, 1991).

Because the two major sources of mercury body burden include dietary intake and intake from dental amalgams, mercury is present at low concentrations in a variety of human tissues. Mercury has been detected in blood, urine, human milk, and hair in individuals in the general population. Inhalation of

mercury vapor in workplace atmospheres is the main route of occupational exposure to the compound. The most recent estimate (1983–1986) indicates that about 152,000 people, including over 50,000 women, are potentially exposed to mercury in workplace environments in the United States (RTECS 1998). Occupational exposure to mercury is highest in industries processing or using the element (e.g., chloralkali workers and individuals involved in the manufacturing of industrial instruments, thermometers, and fluorescent lights). Dentists and dental staff, house painters, chemists involved in the synthesis or analysis of environmental samples containing mercury, and individuals involved in disposal or recycling of mercury-contaminated wastes are also at risk of exposure.

Members of the general public with potentially high exposures include individuals who live in proximity to former mercury mining or production sites, secondary production (recycling) facilities, municipal or medical incinerators, or coal-fired power plants. Other populations at risk of exposure include recreational and subsistence fishers who routinely consume meals of fish that may be contaminated; subsistence hunters who routinely consume the meat and organ tissues of marine mammals or other feral wildlife species; individuals with a large number of dental amalgams; pregnant women and nursing mothers (including their developing fetuses and breast-fed infants) who are exposed to mercury from dietary, medical, or occupational sources, or from mercury spills; individuals who use consumer products containing mercury (e.g., traditional or herbal remedies, or cosmetics, including skin lightening creams); and individuals living or working in buildings where mercury-containing latex paints were used, or where intentional (religious or ethnic use) or unintentional mercury spills have occurred.

Mercury (elemental) has been identified in 714 of the 1,467 hazardous waste sites on the NPL (HazDat 1998). The frequency of these sites can be seen in Figure 5-1. Of these sites, 705 are located in the contiguous United States, 6 are located in the Commonwealth of Puerto Rico (not shown), 2 are located in the U.S. Virgin Islands (not shown), and 1 is located in Guam (not shown). Mercuric acetate, mercuric chloride, mercurous chloride, and dimethylmercury have been identified in 2, 3, 1, and 2 sites, respectively, of the 1,467 hazardous waste sites on the NPL (HazDat 1998). The frequency of these sites can be seen in Figures 5-2 through 5-5. All of these latter sites are located in the contiguous United States.

5.2 RELEASES TO THE ENVIRONMENT

Mercury is released to the environment by both natural processes (e.g., volcanic activity and weathering of mercury-containing rocks) and anthropogenic sources. Anthropogenic releases are primarily to the

atmosphere. According to the Toxic Chemical Release Inventory (TRI), in 1996, a total of 84,772 pounds of mercury were released to the environment (air, water, soil, underground injection, and off-site transfer) from 31 large processing facilities (TRI96 1998). Table 5-1 lists the amounts released from these facilities. The amounts of mercury released to the various environmental compartments in 1996, 1994, and 1991 are also compared in Table 5-2. It is noteworthy that the total environmental releases of mercury have decreased by about 90% from 1991 to 1996 from those production and processing facilities that are required to report their releases to TRI. The individual quantities of mercury released to land, publicly owned treatment works (POTWs), and via off-site waste transfer have decreased most substantially since 1991 by 90%, 95%, and 89% respectively. In contrast, releases to air, water, and underground injection have fluctuated over the past few years, but overall have remained relatively unchanged or declined slightly. The data listed in the TRI should be used with caution because only certain types of facilities are required to report (EPA 1996f). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the Toxics Release Inventory only if they employ 10 or more full-time employees; if their facility is classified under Standard Industrial Classification (SIC) codes 20 through 39; and if their facility produces, imports, or processes 25,000 or more pounds of any TRI chemical or otherwise uses more than 10,000 pounds of a TRI chemical in a calendar year (EPA 1996f). Nationwide mercury emissions from a variety of emission sources are discussed in detail in Sections 5.2.1 through 5.2.3.

5.2.1 Air

Mercury is a naturally occurring metal that is ubiquitous in the environment. Mercury is released to environmental media by both natural processes and anthropogenic sources. Mercury ore is found in all classes of rocks, including limestone, calcareous shales, sandstone, serpentine, chert, andesite, basalt, and rhyolite. The normal concentration of mercury in igneous and sedimentary rocks and minerals appears to be 10–50 ng/g (ppb) (Andersson 1979); however, the mineral cinnabar (mercuric sulfide) contains 86.2% mercury (Stokinger 1981). Currently, the average mercury level in the atmosphere is about 3 to 6 times higher than the estimated level in the preindustrial atmosphere (Mason et al. 1995). Results of several studies suggest increases in anthropogenic mercury emissions over time. Zillioux et al. (1993) used peat cores to estimate that present day deposition of mercury is 2 to 3 times greater than preindustrial levels. Lindqvist (1991c) estimated that sediment concentrations in Swedish lakes are 5 times higher than background levels from precolonial times. Travis and Blaylock (1992) reported that mercury levels in tree

rings, as well as in soil and sediment cores, suggest that a four- to five-fold increase in mercury levels in air has occurred since the beginning of the industrial revolution.

A degree of uncertainty exists with respect to estimates of the relative contributions of natural and anthropogenic sources of mercury emissions to the environment reported in the scientific literature. Nriagu and Pacnya (1988) estimated anthropogenic emissions to be more than half of the total global emissions of 6,000 tons/year. Nriagu (1989) estimated mercury emissions from natural sources to be 2,500 tons/year. In contrast, WHO (1990, 1991) reported that the major source of atmospheric mercury is global degassing of mineral mercury from the lithosphere and hydrosphere at an estimated rate of 2,700–6,000 metric tons/year, which is approximately 1.3 to 3 times the rate of release from anthropogenic sources. Lindqvist (1991b) estimated world anthropogenic emissions at 4,500 tons with an additional 3,000 tons attributed to natural sources. Most recently, Pirrone et al. (1996) estimated world emissions of mercury at 2,200 metric tons/year and concluded that natural sources, industrial sources, and the recycling of anthropogenic mercury each contribute about one-third of the current mercury burden in the global atmosphere. A major source of the uncertainty is that emissions from terrestrial and marine systems include a “recycled” anthropogenic source component (WHO 1990).

Recent estimates of anthropogenic releases of mercury to the atmosphere range from 2,000–4,500 metric tons/year, mostly from the mining and smelting of mercury and other metal sulfide ores. An estimated 10,000 metric tons of mercury are mined each year, although there is considerable year-to-year variation (WHO 1990). Other anthropogenic sources include: industrial processes involving the use of mercury, including chloralkali manufacturing facilities; combustion of fossil fuels, primarily coal; production of cement; and medical and municipal waste incineration and commercial/ industrial boilers (Bache et al. 1991; EPA 1987f, 1996b; Lindberg 1984; Lindqvist 1991b; Nriagu and Pacyna 1988; WHO 1990, 1991). Stein et al. (1996) estimated that approximately 80% of the anthropogenic sources of mercury are emissions of elemental mercury to the air, primarily from fossil fuel combustion, mining, smelting, and from solid waste incineration. Another 15% of the anthropogenic emissions occur via direct application of fertilizers and fungicides and municipal solid waste (e.g., batteries and thermometers) to the land. Recently, Carpi et al. (1998) studied the contamination of sludge-amended soil with inorganic and methyl-mercury and the subsequent emission of this mercury contamination into the atmosphere. These authors reported the routine application of municipal sewage sludge to crop land significantly increased the concentration of both total mercury and methylmercury in surface soil from 80 to 6,1000 μ/kg (ppb) and 0.3 to 8.3 μ/kg (ppb), respectively. Both inorganic and methylmercury were transported from the

sludge/soil matrix to the environment by emission to the atmosphere. An additional 5% of mercury emissions occur via direct discharge of industrial effluent to bodies of water. Mercury emissions from coal-fired power plants are almost exclusively in the vapor phase (98%) (Germani and Zoller 1988). Brown et al. (1993) reported that 79–87% of mercury contained in coal was released with the flue gas at coal-fired power plants. These authors monitored emissions from plants using sub-bituminous C (low sulfur), lignite (medium sulfur), and bituminous (both low- and high-sulfur) coals. Anthropogenic emissions, mainly from combustion of fossil fuels, account for about 25% of mercury emissions to the atmosphere (WHO 1990). These mercury emissions eventually may be deposited on the surrounding soil, although soil concentrations have not been correlated with distance or direction from such plants (Sato and Sada 1992). Other potential emission sources include copper and zinc smelting operations, paint applications, waste oil combustion (EPA 1987f), geothermal energy plants (Baldi 1988), crematories (Nieschmidt and Kim 1997; WHO 1991), and incineration of agricultural wastes (Mariani et al. 1992). The incineration of medical waste has been found to release up to 12.3 mg/m^3 of mercury (Glasser et al. 1991). Medical wastes may release approximately 110 mercury mg/kg of uncontrolled emissions from medical waste incinerators, compared with 25.5 mercury mg/kg general municipal waste, indicating that medical equipment may be a significant source of atmospheric mercury. The use of scrubbers on the incinerators may remove up to 51% of the mercury emissions (Walker and Cooper 1992). Other potential emission sources of mercury emissions to the air include slag from metal production, fires at waste disposal sites, and diffuse emissions from other anthropogenic sources, such as dentistry and industrial activities. The anthropogenic mercury contributions are greater in the northern hemisphere than in the southern hemisphere, and are greatest in heavily industrialized areas.

Balogh and Liang (1995) conducted a 9-week sampling and analysis program to determine the fate of mercury entering a large municipal wastewater treatment plant. Mercury removal in primary treatment averaged 79%; and the average removal across the entire plant was 96%. Mercury loading on the secondary treatment (activated sludge) process was elevated to near plant influent levels due to recycling of the spent scrubber water from the sewage sludge incinerator control equipment. This internal recycling of the spent incinerator scrubber water resulted in elevated mercury loadings to the incinerator and reduced the mercury control efficiency to near zero. Measurements indicated that publicly owned treatment works (POTWs) can remove mercury from wastewater very effectively; however, approximately 95% of the mercury entering the plant was ultimately discharged to the atmosphere via sludge incineration emissions.

Bullock (1997) used the Regional Lagrangian Model of Air Pollution (RELMAP) to simulate the emission, transport, chemical transformation, and wet and dry deposition of elemental mercury gas, divalent mercury gas, and particulate mercury from various point and area source types to develop an atmospheric mercury emissions inventory by anthropogenic source type. The results of the RELMAP model are shown in Table 5-3. On a percentage basis, various combustion processes (medical waste incinerators, municipal waste incinerators, electric utility power production [fossil fuel burning] and non-utility power and heat generation) account for 83% of all anthropogenic emissions in the United States. Overall, of the emissions produced, 41% were associated with elemental mercury vapor (Hg^{o}), 41% with the mercuric form (Hg^{2+}), and 18% was mercury associated with particulates.

A more detailed estimate of national mercury emission rates for various categories of sources is shown in Table 5-4. As shown in this table, point sources of anthropogenic mercury emissions appear to represent the greatest contribution of mercury releases, with combustion sources representing 85% of all emissions.

According to the most recent Toxics Release Inventory (Table 5-1), in 1996, the estimated releases of 17,097 pounds of mercury to the air from 31 large processing facilities accounted for about 20% of annual environmental releases for this element (TRI96 1998). This is slightly more (13%) than the estimated 13,885 pounds that were released to the air in 1994 (TRI94 1996), but 35% less than the 21,288 pounds released to the air in 1991 (Table 5-2). The TRI data listed in Tables 5-1 and 5-2 should be used with some caution, since only certain types of facilities are required to report (EPA 1996f). This is not an exhaustive list.

Mercury has been identified in air samples collected at 25 of the 714 NPL hazardous waste sites where it has been detected in at least one environmental medium (HazDat 1998).

5.2.2 Water

Natural weathering of mercury-bearing minerals in igneous rocks is estimated to directly release about 800 metric tons of mercury per year to surface waters of the earth (Gavis and Ferguson 1972). Atmospheric deposition of elemental mercury from both natural and anthropogenic sources has been identified as an indirect source of mercury to surface waters (WHO 1991). Mercury associated with soils can be directly washed into surface waters during rain events. Surface runoff is an important mechanism for transporting mercury from soil into surface waters, particularly for soils with high humic content (Meili

1991). Mercury may also be released to surface waters in effluents from a number of industrial processes, including chloralkali production, mining operations and ore processing, metallurgy and electroplating, chemical manufacturing, ink manufacturing, pulp and paper mills, leather tanning, pharmaceutical production, and textile manufacture (Dean et al. 1972; EPA 1971c). Discharges from a regional wastewater treatment facility on the St. Louis River that received primarily municipal wastes contained 0.364 µg/L (ppb) of mercury, resulting in concentrations in the adjacent sediment of up to 5.07 µg/g (ppm) (Glass et al. 1990). Industrial effluents from a chemical manufacturing plant on the NPL (Stauffer Chemical's LeMoyne, Alabama site) contained more than 10 ppm of mercury; these effluents had contaminated an adjacent swamp and watershed with mercury concentrations in the sediments ranging from 4.3 to 316 ppm (Hayes and Rodenbeck 1992). Effluent monitoring data collected under the National Pollutant Discharge Elimination System (NPDES) Program were used to estimate pollutant loadings from effluent discharges to the San Francisco Bay Estuary between 1984 and 1987 (Davis et al. 1992). Of the 1,030 samples of industrial effluents monitored entering the San Francisco Estuary during this period, 39% were found to contain mercury (Davis et al. 1992). Although these authors did not specify the limits of detection for mercury and did not provide quantitative information on the concentrations detected, they did indicate that measurements for most of the priority pollutants including mercury were at or below the detection limit. This precluded quantitative assessment of spatial and temporal trends in calculating loadings to the estuary for all but four metals (Davis et al. 1992).

According to the most recent Toxics Release Inventory, in 1996, the estimated releases of 541 pounds of mercury to water from 31 large processing facilities accounted for about 0.64% of total environmental releases for this element (TRI96 1998). An addition 15 pounds of mercury were released indirectly to POTWs, and some of this volume ultimately may have been released to surface waters. This is approximately 215 pounds more mercury than was released to water directly or indirectly via POTWs in 1994 (TRI94 1996), but 445 pounds less than that released to water either directly (144 pounds) or indirectly via POTWs (301 pounds) in 1991 (TRI91 1993). The TRI data listed in Tables 5-1 and 5-2 should be used with some caution, since only certain types of facilities are required to report (EPA 1996f). This is not an exhaustive list.

Mercury has been identified in surface water, groundwater, and leachate samples collected at 197, 395, and 58 sites, respectively, of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.2.3 Soil

Atmospheric deposition of mercury from both natural and anthropogenic sources has been identified as an indirect source of mercury to soil and sediments (Sato and Sada 1992; WHO 1990, 1991). Mercury is released to cultivated soils through the direct application of inorganic and organic fertilizers (e.g., sewage sludge and compost), lime, and fungicides containing mercury (Andersson 1979). Recent interest in community recycling of sewage sludge and yard compost may result in increased releases of mercury from these wastes. Sewage sludge contained approximately 20 times more mercury than yard compost (2.90 ppm versus 0.15 ppm) (Lisk et al. 1992a); municipal solid waste contained the highest concentration (3.95 ppm) (Lisk et al. 1992b). Recently, Carpi et al. (1998) studied the contamination of sludge-amended soil with inorganic and methylmercury and the emission of this mercury contamination into the atmosphere. These authors reported the routine application of municipal sewage sludge to crop land significantly increased the concentration of both total mercury and methylmercury in surface soil from 80 to 6,1000 µg/kg (ppb) and 0.3–8.3 µg/kg (ppb), respectively. Both the inorganic and methylmercury were transported from the sludge/soil matrix to the environment by emission to the atmosphere.

Additional anthropogenic releases of mercury to soil are expected as a result of the disposal of industrial and domestic solid waste products (e.g., thermometers, electrical switches, and batteries) to landfills (see Table 5-5). Another source of mercury releases to soil is the disposal of municipal incinerator ash in landfills (Mumma et al. 1990). In 1987, nationwide concentrations of mercury present in the ash from municipal waste incineration ranged from 0.03 to 25 ppm (Mumma et al. 1990). Such releases may exhibit a seasonal variability. For example, fly ash collected prior to Christmas contained significantly less mercury (6.5 ppm) than ash collected after Christmas (45–58 ppm), possibly as a result of the increased use and disposal of batteries containing mercury in toys and other equipment during this season (Mumma et al. 1991). Emission sources include stack emissions, ashes collected at the stack, ashes from electrostatic precipitators, and in slags (Morselli et al. 1992). An analysis of mercury concentrations in soil, refuse combustibles, and bottom and fly ash from incinerators showed increasing concentrations of 0, 2, 4, and 100 mg/kg (ppm), respectively (Goldin et al. 1992).

According to the Toxics Release Inventory, in 1996, the estimated releases of 537 pounds of mercury to land from 31 large processing facilities accounted for about 0.63% of the total 1996 environmental releases for this element (TRI96 1998). In addition, an estimated 9 pounds of mercury (<0.01% of total environmental releases) were released via underground injection (see Table 5-1). This is approximately

57% of the mercury that was released to soil in 1994 (TRI94 1996) and is only 10% of the mercury released to soil in 1991 (see Table 5-2). The TRI data listed in Tables 5-1 and 5-2 should be used with some caution, since only certain types of facilities are required to report (EPA 1996f). This is not an exhaustive list.

Mercury has been identified in soil and sediment samples collected at 350 and 208 sites, respectively, of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.3 ENVIRONMENTAL FATE

The natural global bio-geochemical cycling of mercury is characterized by degassing of the element from soils and surface waters, followed by atmospheric transport, deposition of mercury back to land and surface waters, and sorption of the compound to soil or sediment particulates. Mercury deposited on land and open water is in part revolatilized back into the atmosphere. This emission, deposition, and revolatilization creates difficulties in tracing the movement of mercury to its sources (WHO 1990). Particulate-bound mercury can be converted to insoluble mercury sulfide and precipitated or bioconverted into more volatile or soluble forms that re-enter the atmosphere or are bioaccumulated in aquatic and terrestrial food chains (EPA 1984b).

5.3.1 Transport and Partitioning

Mercury has three valence states. The specific state and form in which the compound is found in an environmental medium is dependent upon a number of factors, including the redox potential and pH of the medium. The most reduced form is metallic or elemental mercury, which is a liquid at ambient temperatures, but readily vaporizes. Over 95% of the mercury found in the atmosphere is gaseous mercury (Hg^0), the form involved in long-range (global) transport of the element. Residence time in the atmosphere has been estimated to range from 6 days (Andren and Nriagu 1979) to 2 years (EPA 1984b). Approximately 5% of atmospheric mercury is associated with particulates, which have a shorter atmospheric residence time, are removed by dry or wet deposition, and may show a regional or local distribution pattern (Nater and Grigal 1992). Atmospheric inputs may be more significant in areas where other sources of contamination, such as contaminated rivers, are less important or nonexistent (Kelly et al. 1991). Although local sources are important, a 72-hour travel time trajectory for mercury indicates that some mercury found in rain may originate from sources up to 2,500 km (1,550 miles) away (Glass et al.

1991). Over the last 140 years, the atmospheric mercury concentrations have increased by a factor of 3.7, or approximately 2% per year (Swain et al. 1992).

Metallic mercury released in vapor form to the atmosphere can be transported long distances before it is converted to other forms of mercury, and wet and dry deposition processes return it to land and water surfaces. Dry deposition may account for approximately 70% of the total atmospheric deposition of mercury during the summer, although on an annual basis, wet and dry deposition may be of equal importance (Lindberg et al. 1991). Up to 22% of the annual input of mercury to Lake Erie is from dry deposition of mercury-containing atmospheric particles or from precipitation (Kelly et al. 1991). Wet deposition is the primary method of removal of mercury from the atmosphere (approximately 66%) (Fitzgerald et al. 1991; Lindqvist 1991c) and may account for virtually all of the mercury content in remote lakes that do not receive inputs from other sources (e.g., industrial effluents) (Hurley et al. 1991; Swain et al. 1992). Most inert mercury (Hg^{+2}) in precipitation is bound to aerosol particulates, which are relatively immobile when deposited on soil or water (Meili et al. 1991). Mercury is also present in the atmosphere to a limited extent in unidentified soluble forms associated with particulate matter. In addition to wet and dry deposition processes, mercury may also be removed from the atmosphere by sorption of the vapor form to soil or water surfaces (EPA 1984b).

In soils and surface waters, mercury can exist in the mercuric (Hg^{+2}) and mercurous (Hg^{+1}) states as a number of complex ions with varying water solubilities. Mercuric mercury, present as complexes and chelates with ligands, is probably the predominant form of mercury present in surface waters. The transport and partitioning of mercury in surface waters and soils is influenced by the particular form of the compound. More than 97% of the dissolved gaseous mercury found in water consists of elemental mercury (Vandal et al. 1991). Volatile forms (e.g., metallic mercury and dimethylmercury) are expected to evaporate to the atmosphere, whereas solid forms partition to particulates in the soil or water column and are transported downward in the water column to the sediments (Hurley et al. 1991). Vaporization of mercury from soils may be controlled by temperature, with emissions from contaminated soils being greater in warmer weather when soil microbial reduction of Hg^{+2} to the more volatile elemental mercury is greatest (Lindberg et al. 1991). Vapor-phase mercury volatilized from surface waters has been measured (Schroeder and Fanaki 1988); however, the dominant process controlling the distribution of mercury compounds in the environment appears to be the sorption of nonvolatile forms to soil and sediment particulates, with little resuspension from the sediments back into the water column (Bryan and Langston 1992). Cossa et al. (1988) found that 70% of the dissolved mercury in St. Lawrence River water was

associated with organic matter. The authors reported that the removal mechanism was flocculation of organic mercury colloids in freshwater. Methylmercury and other mercury fractions are strongly bound to organic matter in water and may be transported in runoff water from contaminated lakes to other surface waters and soils (Lee and Iverfeldt 1991). Small amounts (2–4 ng/L [ppt]) of mercury are able to move from contaminated groundwater into overlying lakes, with concentrations reaching a maximum near the sediment/water interface; however, since most of the mercury in the groundwater is derived from atmospheric sources, this low value indicates that most of the mercury deposited on soil (92–96% of the 10.3 $\mu g/m^2$/year of mercury deposited) is absorbed to the soil and does not leach down into the groundwater (Krabbenhoft and Babiarz 1992).

The sorption process has been found to be related to the organic matter content of the soil or sediment. Mercury is strongly sorbed to humic materials and sesquioxides in soil at a pH higher than 4 (Blume and Brummer 1991) and to the surface layer of peat (Lodenius and Autio 1989). Mercury has been shown to volatilize from the surface of more acidic soils (i.e., soil pH of less than 3.0) (Warren and Dudas 1992). Adsorption of mercury in soil is decreased with increasing pH and/or chloride ion concentrations (Schuster 1991). Mercury is sorbed to soil with high iron and aluminum content up to a maximum loading capacity of 15 g/kg (15,000 ppm) (Ahmad and Qureshi 1989). Inorganic mercury sorbed to particulate material is not readily desorbed. Thus, freshwater and marine sediments are important repositories for inorganic forms of the element, and leaching is a relatively insignificant transport process in soils. However, surface runoff is an important mechanism for moving mercury from soil to water, particularly for soils with high humic content (Meili 1991). Mobilization of sorbed mercury from particulates can occur through chemical or biological reduction to elemental mercury and bioconversion to volatile organic forms (Andersson 1979; Callahan et al. 1979; EPA 1984b). Metallic mercury may move through the top 3–4 cm of dry soil at atmospheric pressure; however, it is unlikely that further penetration would occur (Eichholz et al. 1988).

The volatilization and leaching of various forms of mercury (elemental, mercuric sulfide, mercuric oxide, and mercurous oxide) from soils or wastes was examined using the headspace method for volatilization and the Resource and Conservation Recovery Act (RCRA) leaching protocols for leaching through soil to determine if the leachates exceeded the RCRA limit of 200 µg/L (ppb) (Willett et al. 1992). With the exception of mercuric sulfide, the other forms of mercury increased in concentrations in the headspace vapor and in the leachate as the soil concentrations increased, although the elemental mercury concentrations never exceeded the RCRA limit, indicating that it was relatively unleachable. Mercuric sulfide also did not exceed the background level for the leachate and was consistently less than

0.001 mg/m^3 for the vapor concentrations, indicating that it was also nonleachable and did not readily volatilize. This study also showed that concentrations of mercury in leachate could not be correlated with the concentration of mercury in the soil or in the headspace vapors (Willett et al. 1992). Mercuric sulfide has been found to strongly adsorb to soil, and even with weathering, any mercury released from the mercuric sulfide is readsorbed by the soil (Harsh and Doner 1981).

The most common organic form of mercury, methylmercury, is soluble, mobile, and quickly enters the aquatic food chain. This form of mercury is accumulated to a greater extent in biological tissue than are inorganic forms of mercury (Riisgard and Hansen 1990). Methylmercury in surface waters is rapidly accumulated by aquatic organisms; concentrations in carnivorous fish (e.g., pike, shark, and swordfish) at the top of both freshwater and marine food chains are biomagnified on the order of 10,000–100,000 times the concentrations found in ambient waters (Callahan et al. 1979; EPA 1984b; WHO 1990, 1991). The range in experimentally determined bioconcentration factor (BCF) values is shown in Table 5-6. The bioaccumulation potential for methylmercury in fish is influenced by the pH of the water, with a greater bioaccumulation seen in waters with lower pH (Ponce and Bloom 1991). Mercury concentrations in fish have also been negatively correlated with other water quality factors, such as alkalinity and dissolved oxygen content (Wren 1992).

The biomagnification of methylmercury has been demonstrated by the elevated levels found in piscivorous fish compared with fish at lower levels of the food chain (Jackson 1991; Kohler et al. 1990; Porcella 1994; Watras and Bloom 1992). Biomagnification factors for methylmercury in the food webs of Lake Ontario were lowest for the transfer of methylmercury from mysids to amphipods (1.1), plankton to amphipods (1.8), and plankton to mysids (2.4); were intermediate for the transfer from mysids to fish (5.1) and amphipods to fish (6.5); and were highest for the transfer from plankton to fish (10.4) (Evans et al. 1991). (The biomagnification of methylmercury from water through several trophic levels is compared to the biomagnification of inorganic mercury in Table 5-7.) Watras and Bloom (1992) reported that biomagnification of methylmercury in Little Rock Lake seems to be a result of two processes: the higher affinity of inorganic mercury in lower trophic level organisms and the high affinity of methylmercury in fish. Fish appear to accumulate methylmercury from both food sources and the water column. However, Hall et al. (1997) found that food was the predominant source of mercury uptake in fish. The biological concentration factor (BCF) of methylmercury in fish in Little Rock Lake was three million (Porcella 1994). Mason et al. (1995) also compared bioaccumulation of inorganic mercury and methylmercury. These authors showed that passive uptake of the mercury complexes ($HgCl_2$ and CH_3HgCl) results in high

concentrations of both the inorganic and methylated mercury in phytoplankton. However, differences in partitioning within phytoplankton cells between inorganic mercury (which is principally membrane-bound) and methylmercury (which accumulated in the cytoplasm) lead to a greater assimilation of methylmercury during zooplankton grazing.

Most of the discrimination between inorganic and methylmercury thus occurs during trophic transfer, while the major enrichment factor is between water and the phytoplankton. This also has been reported for the diatom *Thalassiosura weissflogii* in a marine food chain (Mason et al. 1996). Methylmercury was accumulated in the cell cytoplasm, and its assimilation by copepods was 4 times more efficient than the assimilation of inorganic mercury. Bioaccumulation has been demonstrated for predator fish in both freshwater and marine systems and in marine mammals (see Section 5.4.4). Bioaccumulation of methylmercury in aquatic food chains is of interest, because it is generally the most important source of nonoccupational human exposure to this compound (EPA 1984b; WHO 1990, 1991).

Aquatic macrophytes have been found to bioconcentrate methylmercury in almost direct proportion to the mercury concentration in the water (Ribeyre et al. 1991). Mortimer (1985) reported bioconcentration factors (BCFs) for several species of submerged aquatic plants exposed to inorganic mercury in laboratory aquaria of 3,300, 1.3, 0.9, and 1.3 for *Utricularia*, *Ceratophyllum*, *Najas*, and *Nitella*, respectively. The concentrations factor used by this author was based on $\mu g\ g^{-1}$ dry weight in the plant/$\mu g\ mL^{-1}$ water day^{-1}.

The potential for bioaccumulation in terrestrial food chains is demonstrated by the uptake of mercury by the edible mushroom *Pleurotus ostreatus*, grown on compost containing mercury at concentrations of up to 0.2 mg/kg (ppm). The bioaccumulation factors reported ranged from 65 to 140, indicating that there are potential risks to human health if these mushrooms are eaten in large quantities (Bressa et al. 1988). Elevated concentrations of mercury in 149 samples of mushrooms representing 11 different species were reported by Kalcac et al. (1991). These authors collected mushrooms within 6 km of a lead smelter in Czechoslovakia in operation since 1786. Mercury was accumulated by *Lepista nuda* and *Lepiota rhacodes* at 11.9 mg/kg (ppm) and 6.5 mg/kg (ppm) (dry weight), respectively. The mean concentration of other species ranged from 0.3 to 2.4 mg/kg (ppm). Concentrations of mercury in most of the mushroom species collected in that location were higher than in mushrooms collected in other parts of the country.

Data from higher plants indicate that virtually no mercury is taken up from the soil into the shoots of plants such as peas, although mercury concentrations in the roots may be significantly elevated and reflect the

mercury concentrations of the surrounding soil (Lindqvist 1991e). In a study by Granato et al. (1995), municipal solid waste sludge mercury concentrations from the Metropolitan Water Reclamation District of Greater Chicago were found to range from 1.1 to 8.5 mg/kg (ppm), with a mean concentration of 3.3 mg/kg (ppm). From 1971 to 1995, sludge applications were made to a Fulton County, Illinois sludge utilization site. About 80–100% of the mercury applied to the soils in sewage sludge since 1971 still resided in the top 15 cm of soil. These authors reported that sewage sludge applications did not increase plant tissue mercury concentrations in corn or wheat raised on the sludge utilization site.

Earthworms, *Lumbricus sp*., bioaccumulate mercury under laboratory and field conditions in amounts which are dependent on soil concentrations and exposure duration (Cocking et al. 1994). Maximum mercury tissue concentrations in laboratory cultures were only 20% of the 10–14.8 µg/g (ppm) (dry weight) observed in individual worms collected from contaminated soils (21 µg/g) on the South River flood plain at Waynesboro, Virginia. Bioconcentration occurred under field conditions in uncontaminated control soil (0.2 µg Hg/g); however, total tissue mercury concentrations (0.4–0.8 µg/g dry weight) were only 1–5% of those for earthworms collected on contaminated soils. Uptake by the earthworms appeared to be enhanced in slightly acidic soils (pH 5.9–6.0) in laboratory cultures. Soil and earthworm tissue mercury contents were positively correlated under both field and laboratory conditions. Predation of earthworms contaminated with mercury could pass the contamination to such predators as moles and ground feeding birds, such as robins (Cocking et al. 1994).

5.3.2 Transformation and Degradation

Mercury is transformed in the environment by biotic and abiotic oxidation and reduction, bioconversion of inorganic and organic forms, and photolysis of organomercurials. Inorganic mercury can be methylated by microorganisms indigenous to soils, fresh water, and salt water. This process is mediated by various microbial populations under both aerobic and anaerobic conditions. The most probable mechanism for this reaction involves the nonenzymatic methylation of mercuric mercury ions by methylcobalamine compounds produced as a result of bacterial synthesis. Mercury forms stable complexes with organic compounds. Monoalkyl mercury compounds (e.g., methylmercuric chloride) are relatively soluble; however, the solubility of methylmercury is decreased with increasing dissolved organic carbon content, indicating that it is bound by organic matter in water (Miskimmin 1991). Dialkyl mercury compounds (e.g., dimethyl-mercury) are relatively insoluble (Callahan et al. 1979; EPA 1984b). Dimethylmercury is volatile, although it makes up less than 3% of the dissolved gaseous mercury found in water (Andersson et al. 1990;

Vandal et al. 1991). The major pathways for transformation of mercury and various mercury compounds in air, water, and soil are shown in Figure 5-6.

5.3.2.1 Air

The primary form of atmospheric mercury, metallic mercury vapor (Hg^0), is oxidized by ozone to other forms (e.g., Hg^{+2}) and is removed from the atmosphere by precipitation (Brosset and Lord 1991). The oxidation/reduction of mercury with dissolved ozone, hydrogen peroxide, hypochlorite entities, or organoperoxy compounds or radicals may also occur in the atmosphere (Schroeder et al. 1991). The overall residence time of elemental mercury in the atmosphere has been estimated to be 6 days to 2 years, although in clouds, a fast oxidation reaction on the order of hours may occur between elemental mercury and ozone. Some mercury compounds, such as mercuric sulfide, are quite stable in the atmosphere as a result of their binding to particles in the aerosol phase (Lindqvist 1991b). Other mercury compounds, such as mercuric hydroxide ($Hg[OH]^2$), which may be found in the aqueous phase of the atmosphere (e.g., rain), are rapidly reduced to monovalent mercury in sunlight (Munthe and McElroy 1992). The main atmospheric transformation process for organomercurials appears to be photolysis (EPA 1984b; Johnson and Bramen 1974; Williston 1968).

5.3.2.2 Water

The most important transformation process in the environmental fate of mercury in surface waters is biotransformation. Photolysis of organomercurials may also occur in surface waters, but the significance of this process in relation to biotransformation is not clear (Callahan et al. 1979).

Any form of mercury entering surface waters can be microbially converted to methylmercuric ions, given favorable conditions. Sulfur-reducing bacteria are responsible for most of the mercury methylation in the environment (Gilmour and Henry 1991), with anaerobic conditions favoring their activity (Regnell and Tunlid 1991). Yeasts, such as *Candida albicans* and *Saccharomyces cerevisiae*, whose growth is favored by low pH conditions, are able to methylate mercury and are also able to reduce ionic mercury to elemental mercury (Yannai et al. 1991). Methyl cobalamine compounds produced by bacterial synthesis appear to be involved in the nonenzymatic methylation of inorganic mercury ions (Regnell and Tunlid 1991). The rate of methylmercury formation by this process is largely determined by the concentration of methyl cobalamine compounds, inorganic mercuric ions, and the oxygen concentration of the water, with the rate

increasing as the conditions become anaerobic. Volatile elemental mercury may be formed through the demethylation of methylmercury or the reduction of inorganic mercury, with anaerobic conditions again favoring the demethylation of the methylmercury (Barkay et al. 1989; Callahan et al. 1979; Regnell and Tunlid 1991). Increased dissolved organic carbon levels reduce methylation of mercury in the water column (Gilmour and Henry 1991), possibly as a result of the binding of free mercury ions to the dissolved organic carbon at low pH, thus reducing their availability for methylation, or the dissolved organic carbon may inhibit the methylating bacteria (Miskimmin et al. 1992). Alternatively, low pH favors the methylation of mercury in the water column, particularly in acid deposition lakes, while inhibiting its demethylation (Gilmour and Henry 1991). It has also been shown that the methylation rate is not affected by addition of sulfate in softwater lakes (Kerry et al. 1991).

At a pH of 4–9 and a normal sulfide concentration, mercury will form mercuric sulfide. This compound is relatively insoluble in aqueous solution (11×10^{-17} ppb), and therefore it will precipitate out and remove mercury ions from the water, reducing the availability of mercury to fish. Under acidic conditions, however, the activity of the sulfide ion decreases, thus inhibiting the formation of mercuric sulfide and favoring the formation of methylmercury (Bjornberg et al. 1988). Low pH and high mercury sediment concentrations favor the formation of methylmercury, which has greater bioavailability potential for aquatic organisms than inorganic mercury compounds. Methylmercury may be ingested by aquatic organisms lower in the food chain, such as yellow perch, which in turn are consumed by piscivorous fish higher on food chain (Cope et al. 1990; Wiener et al. 1990). Mercury cycling occurs in freshwater lakes, with the concentrations and speciation of the mercury being dependent on limnological features and water stratification. Surface waters may be saturated with volatile elemental mercury, whereas sediments are the primary source of the mercury in surface waters. During the summer months, surface concentrations of methyl and elemental mercury decline as a result of evaporation, although they remain relatively constant in deeper waters (Bloom and Effler 1990).

Abiotic reduction of inorganic mercury to metallic mercury in aqueous systems can also occur, particularly in the presence of soluble humic substances (i.e., acidic waters containing humic and fulvic acids). This reduction process is enhanced by light, occurs under both aerobic and anaerobic conditions, and is inhibited by competition from chloride ions (Allard and Arsenie 1991).

5.3.2.3 Sediment and Soil

Mercury compounds in soils may undergo the same chemical and biological transformations described for surface waters. Mercuric mercury usually forms various complexes with chloride and hydroxide ions in soils; the specific complexes formed depend on the pH, salt content, and composition of the soil solution. Formation and degradation of organic mercurials in soils appear to be mediated by the same types of microbial processes occurring in surface waters and may also occur through abiotic processes (Andersson 1979). Elevated levels of chloride ions reduce methylation of mercury in river sediments, sludge, and soil (Olson et al. 1991), although increased levels of organic carbon and sulfate ions increase methylation in sediments (Gilmour and Henry 1991). In freshwater and estuarine ecosystems, the presence of chloride ions (0.02 M) may accelerate the release of mercury from sediments (Wang et al. 1991).

In the late 1950s, unknown quantities of mercuric nitrate and elemental mercury were released into East Fork Poplar Creek from a government facility in Oak Ridge, Tennessee. Total mercury concentrations in the flood plain soil along the creek ranged from 0.5 to 3,000 ppm (Revis et al. 1989). An estimated 170,000 pounds of that mercury remained in floodplain soil of the creek (DOE 1994). The form of that mercury has been reported to be primarily mercuric sulfide (85–88%), with 6–9% present as elemental mercury (Revis et al. 1989, 1990). A very small amount was detected in the form of methylmercury (less than 0.02%). The reported presence of the mercuric sulfide suggests that the predominant biological reaction in soil for mercury is the reduction of Hg^{+2} to mercuric sulfide by sulfate-reducing bacteria under anaerobic conditions (Revis et al. 1989, 1990). Mercuric sulfide has very limited water solubility (4.5×10^{-24} mol/L), and thus, in the absence of other solvents, is likely to have limited mobility in soil. Aerobic microorganisms can solubilize Hg^{+2} from mercuric sulfide by oxidizing the sulfide through sulfite to sulfate, with the Hg^{+2} being reduced to elemental mercury (Wood 1974). However, examination of the weathering of mercuric sulfide indicated that mercuric sulfide does not undergo significant weathering when bound to riverwash soil with a pH of 6.8, although degradation may be increased in the presence of chloride and iron (Harsh and Doner 1981).

Mercury, frequently present in mine tailings, was toxic to bacteria isolated from a marsh treatment system used to treat municipal waste waters. The minimum concentration that inhibited the bacteria (as determined by intracellular ATP levels) was approximately 0.07±0.15 mg/L (ppm) (Desjardins et al. 1988).

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to mercury and various mercury compounds depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of mercury in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of detection of current analytical methods even for determining total mercury. In reviewing data on mercury levels monitored or estimated in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable. The analytical methods available for monitoring mercury and various inorganic and organic mercury compounds in a variety of environmental media are discussed in Chapter 6.

5.4.1 Air

Indoor air mercury concentrations were determined in 37 houses in Ohio that had been painted with latex paint (Beusterien et al. 1991). Of the 37 homes studied, 21 homes had been painted with interior latex paint containing mercury a median of 86 days earlier, while the 16 control homes had not been recently painted with mercury-containing latex paints. Paint samples from the exposed homes contained a median concentration of 210 mg/L (ppm) (range, 120–610 mg/L). The median air mercury concentration (0.3 μg/m^3) was found to be significantly higher ($p<0.0001$) in the exposed homes (range, not detectable to 1.5 μg/m^3) than in the unexposed homes (range, not detectable to 0.3 μg/m^3). Among the exposed homes, there were 7 in which paint containing <200 mg/L had been applied. In these homes, the median air mercury concentration was 0.2 μg/m^3 (range, not detectable to 1 μg/m^3). Six exposed homes had air mercury concentrations >0.5 μg/m^3. The authors reported that elemental mercury was the form of mercury released to the air and that potentially hazardous mercury exposure could occur in homes recently painted with paint containing <200 mg Hg/L (Beusterien et al. 1991). In an indoor exposure study of families of workers at a chloralkali plant in Charleston, Tennessee, mercury levels in the air of the workers' homes averaged 0.92 μg/m^3 (ATSDR 1990).

Ambient air concentrations of mercury have been reported to average approximately 10–20 ng/m^3, with higher concentrations in industrialized areas (EPA 1980a). In 1990, metallic mercury concentrations in the gas and aerosol phases of the atmosphere in Sweden were 2–6 ng/m^3 and 0.01–0.1 ng/m^3, respectively (Brosset and Lord 1991). Higher levels (10–15 μg/m^3) have been detected near point emission sources, such as mercury mines, refineries, and agricultural fields treated with mercury fungicides. Atmospheric

concentrations of mercury over lakes in Wisconsin averaged 2.0 ng/m^3 (Wiener et al. 1990) and ranged from 6.3 ng/m^3 to 16.0 ng/m^3 above the water surface of the mercury-contaminated Wabigoon River in Ontario (Schroeder and Fanaki 1988). Mean vapor concentrations of mercury in air over a forested watershed (Walker Branch Watershed) in Tennessee were 5.5 ng/m^3 in 1988–1989, while particle-associated aerosol mercury concentrations were determined to be 0.03 ng/m^3, or approximately 0.5% of the total atmospheric mercury (Lindberg et al. 1991). Lindberg et al. (1994) measured mercury vapor at concentrations of 2–6 ng/m^3 and particulate mercury at 0.002–0.06 ng/m^3 at Walker Branch Watershed, Tennessee, from August 1991 to April 1992. Particulate mercury concentrations are greater in precipitation than in ambient air. In the St. Louis River estuary, mercury levels in precipitation averaged 22 ng/L (ppt), although ambient air levels averaged 3 ng/m^3 (Glass et al. 1990).

Total gaseous mercury was measured (1992–1993) as part of the Florida Atmospheric Mercury Study (FAMS) (Gill et al. 1995). Average total gaseous mercury concentrations for 3- to 6-day integrated samples ranged from 1.43 to 3.11 ng/m^3 (mean, 1.64 ng/m^3). In the same study, Dvonch et al. (1995) reported that the mean concentrations of total gaseous mercury measured at two inland Florida sites were significantly higher (3.3 and 2.8 ng/m^3) than measurements at an Atlantic coastal site (1.8 ng/m^3). The mean concentrations of particle phase mercury collected at the inland sites (51 and 49 pg/m^3) were 50% higher than those at the coastal site (34 pg/m^3). The mean mercury concentration in rain samples was 44 ng/L (ppt) (range, 14–130 ng/L). Guentzel et al. (1995) also reported results of the FAMS from 1992 to 1994. These authors found that the summer time wet season in south Florida accounted for 80–90% of the annual rainfall mercury deposition. Depositional rates in south Florida are 30 to almost 50% higher than those in central Florida. Particle phase measurements ranged from 2 to 18 pg/m^3 at all sites. Measurement of monomethylmercury in precipitation ranged from <0.005 to 0.020 ng/L (ppt).

Keeler et al. (1995) reported that particulate mercury may contribute a significant portion of the deposition of mercury to natural waters. Mercury can be associated with large particles (>2.5 μm) at concentrations similar to vapor phase mercury. Particulate phase mercury levels in rural areas of the Great Lakes and Vermont ranged from 1 to 86 pg/m^3, whereas particulate mercury levels in urban and industrial areas were in the range of 15–1,200 pg/m^3. Sweet and Vermette (1993) sampled airborne inhalable particulate matter in urban areas (southeast Chicago and East St. Louis) and at a rural site. Mean particulate phase mercury concentrations in particles (<2.5 μm and >2.5 μm) at the rural site were 0.3 ng/m^3 (range, <0.1–0.9 ng/m^3) and 0.2 ng/m^3 (range, <0.1–0.5 ng/m^3), respectively, as compared to 1.0 ng/m^3 (range, <0.1–0.7 ng/m^3) and

0.5 ng/m^3 (range, <0.1–1.5 ng/m^3), respectively, in Chicago and 0.7 ng/m^3 (range, <0.1–20 ng/m^3) and 0.5 ng/m^3 (range, <0.1–1.5 ng/m^3), respectively, in East St. Louis.

In an earlier study, Keeler et al. (1994) measured atmospheric mercury in the Great Lakes Basin. These authors reported that vapor phase mercury levels were four times higher in Chicago, Illinois, than in South Haven, Michigan, (8.7 ng/m^3 versus 2.0 ng/m^3). Furthermore, a diurnal pattern was observed in the vapor phase mercury levels measured at the Chicago site. The average concentration (ng/m^3) was 3.3 times greater for the daytime samples (8 AM to 2 PM) than for the night samples (8 PM to 8 AM), and the average concentration for the afternoon samples (2 PM to 8 PM) was 2.1 times greater than the night samples (average, 3.7 ng/m^3). Particulate phase mercury concentrations were also higher at the Chicago site than at the South Haven site (98 pg/m^3 versus 19 pg/m^3). Burke et al. (1995) reported that the concentration of mercury in vapor phase samples measured over Lake Champlain was consistent with other rural areas (mean, 2.0 ng/m^3; range, 1.2–4.2 ng/m^3), and the concentrations were consistent across all seasons. Particulate phase mercury concentrations averaged 11 pg/m^3, with the highest concentrations detected during the winter.

A monitoring program established at a facility at Oak Ridge National Laboratories found that the major sources of mercury release to the air were vaporization from soil, burning of coal for a steam plant, and fugitive exhaust from a former lithium isotope separation facility contaminated with mercury (Turner et al. 1992). When the monitoring program began in 1986, ambient air mercury vapor concentrations at the facility ranged from 0.011 to 0.108 μg/m^3. These values decreased to 0.006 to 0.071 μg/m^3 by 1990, while background levels near the facility remained at 0.006 μg/m^3. The decrease in mercury vapor concentrations occurred primarily as a result of an 80% reduction in coal burning at the steam plant; however, periods of drought and activities such as moving contaminated soil for construction were found to increase the atmospheric mercury concentrations on a transient basis (Turner et al. 1992). Turner and Bogle (1993) monitored ambient air for mercury around the same industrial complex site at Oak Ridge, Tennessee. Elemental mercury was used in large quantities at the nuclear weapons plant between 1950 and 1963 in a process similar to chloralkali production. Soil and water contamination had been found at the site. The results of weekly ambient monitoring for gaseous mercury from 1986 through 1990 showed that gaseous mercury levels were well below the National Emission Standard for Hazardous Air Pollutants (1.0 mg/m^3) with the exception of one station. Mean mercury levels at the control site ranged from 5 to 6 μg/m^3, while levels at the on-site stations were 6–11, 11–143, 68–174, 71–109, and 4–46 μg/m^3. Mean

particulate mercury levels were 0.00003 μg/m^3 at the control site, compared with mean concentrations at the on-site stations ranging from 0.00006 to 0.00024 μg/m^3 (Turner and Bogle 1993).

Mercury has been identified in air samples collected at 25 sites of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.4.2 Water

Concentrations of mercury in rainwater and fresh snow are generally below 200 ng/L (ppt) (EPA 1984b). Fitzgerald et al. (1991) measured total mercury in rainwater from May through August 1989 at Little Rock Lake, Wisconsin. The total mercury concentrations ranged from 3.2 to 15.2 ng/L (ppt). Mercury concentrations in precipitation collected in Minnesota during 1988 and 1989 averaged 18 ng/L (ppt) for an average annual mercury deposition of 15 μg/m^2 (Glass et al. 1991). Antarctic surface snow contained a mean mercury concentration of less than 1 pg/g (ppt) (Dick et al. 1990). In Ontario, Canada, mercury present in precipitation at an average concentration of 10 ng/L (ppt) accounted for more than half of the mercury inputs to surface waters compared with inputs from stream runoff, suggesting that atmospheric deposition is a significant source of mercury in surface waters (Mierle 1990). Lindberg et al. (1994) measured total mercury in rain collected at Walker Branch Watershed, Tennessee from August 1991 to April 1992. Rain concentrations of total mercury ranged from 7.57 ng/L (ppt) in February 1992 to 17.4 ng/L (ppt) in April 1992. Burke et al. (1995) reported that the average concentration of mercury in precipitation samples measured over Lake Champlain was 8.3 ng/L (ppt) for the sampling year, and the average amount of mercury deposited per precipitation event was 0.069 μg/m^2. The highest concentrations of mercury in precipitation samples occurred during spring and summer months. Guentzel et al. (1995) reported results of the Florida Atmospheric Monitoring Study from 1992 to 1994. These authors found that the summer time wet season in south Florida accounted for 80 to 90% of the annual rainfall mercury deposition. Depositional rates in south Florida are 30–50% higher than those in central Florida. Measurement of monomethylmercury in precipitation samples ranged from <0.005 to 0.020 ng/L (ppt).

The natural occurrence of mercury in the environment means that mercury is likely to occur in surface waters, even when anthropogenic sources of mercury are absent. Freshwaters without known sources of mercury contamination generally contain less than 5 ng/L (ppt) of total mercury in aerobic surface waters (Gilmour and Henry 1991). Mercury levels in water-borne particulates in the St. Louis River estuary ranged from 18 to 500 ng/L (ppt) (Glass et al. 1990). Water samples from lakes and rivers in the Ottawa,

Ontario, region of Canada had total mercury concentrations of 3.5–11.4 ng/L (ppt), with organic mercury constituting 22–37% of the total mercury (Schintu et al. 1989). Mercury was detected in water samples from Crab Orchard Lake, Illinois, at 70–281 ng/L (ppt) (Kohler et al. 1990). Total mercury concentrations in surface waters of California lakes and rivers ranged from 0.5 to 104.3 ng/L (ppt), with the dissolved particulate fraction being dominant (89%; 0.4–12 ng/L [ppt]) (Gill and Bruland 1990).

The baseline concentration of mercury in unpolluted marine waters has been estimated to be less than 2 ng/L (2 ppt) (Fowler 1990). In contrast, the New York Bight, an inshore coastal area near the industrialized areas of New York Harbor and northern New Jersey, contained dissolved mercury concentrations in the range of 10–90 ng/L (ppt) (Fowler 1990).

Near-surface groundwaters in remote areas of Wisconsin were found to contain approximately 2–4 ng/L (ppt) of mercury, of which only a maximum of 0.3 ng/L (ppt) was determined to be methylmercury, indicating that groundwater was not a source of methylmercury in the lake (Krabbenhoft and Babiarz 1992). Mercury was found at levels greater than 0.5 µg/L (ppb) in 15–30% of wells tested in some groundwater surveys (EPA 1985b). Drinking water is generally assumed to contain less than 0.025 µg/L (ppb) (EPA 1984b). A chemical monitoring study of California's public drinking water from groundwater sources was conducted by Storm (1994). This author reported that mercury was analyzed in 6,856 samples, with 225 positive detections and 27 exceedances of the maximum contaminant level (0.002 mg/L [200 ppb]). The mean mercury concentration was 6.5 ppb (median, 0.62 ppb; range, 0.21 to 300 ppb).

Mercury has been identified in surface water, groundwater, and leachate samples collected at 197, 395, and 58 sites, respectively, of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.4.3 Sediment and Soil

In a review of the mercury content of virgin and cultivated surface soils from a number of countries, it was found that the average concentrations ranged from 20 to 625 ng/g (0.020 to 0.625 ppm) (Andersson 1979). The highest concentrations were generally found in soils from urban locations and in organic, versus mineral, soils. The mercury content of most soils varies with depth, with the highest mercury concentrations generally found in the surface layers. Mercury was detected at soil concentrations ranging from 0.01 to 0.55 ppm in orchard soils in New York State (Merwin et al. 1994).

Granato et al. (1995) reported that municipal solid waste sludge mercury concentrations from the Metropolitan Water Reclamation District of Greater Chicago ranged from 1.1 to 8.5 mg/kg (ppm), with a mean concentration of 3.31 mg/kg (ppm). Sludge applications to a sludge utilization site in Fulton County, Illinois, from 1971 to 1995 significantly increased extractable soil mercury concentrations. In addition, 80–100% of the mercury applied to the soils in sewage sludge since 1971 still resided in the top 15 cm of soil.

Facemire et al. (1995) reported industrial contamination of soils and sediment in several states in the southeastern United States. The authors reported soil concentrations up to 141,000 ppm associated with contamination in northeastern Louisiana from mercury-charged manometers used to measure pressure and delivery from natural gas wells. In Tennessee, a maximum mercury concentration of 1,100 ppm (associated with previous operations of the Oak Ridge nuclear facility) was found in wetland soils adjacent to the East Fork Poplar Creek. A pharmaceutical company's effluents enriched sediments in a localized area of Puerto Rico to 88 ppm mercury (Facemire et al. 1995). Rule and Iwashchenko (1998) reported that mean soil mercury concentrations of 1.06 ppm were collected within 2 km of a former chlor-alkali plant in Saltsville, Virginia, and that these concentrations were 17 times higher than regional background soil samples (0.063 ppm). These authors further reported that soil organic content, topographic factors, wind patterns, and elevation were variables significantly related to mercury concentration as determined by regression analysis. Soil mercury levels decreasing with distance from the former plant were indicative of a point source distribution pattern. A made land soil type (Udorthent), which appears to be a by-product of the chlor-alkali manufacturing process, was found proximal to the former plant site and contained about 68 times (4.31 ppm) the regional background concentration.

The top 15 cm of sediments in Wisconsin lakes contained higher levels of mercury (0.09–0.24 μg/g [ppm]) than sediments at lower sediment levels (0.04–0.07 μg/g [ppm]). Because the lakes are not known to receive any direct deposition of mercury, it was postulated that the primary mercury source was atmospheric deposition (Rada et al. 1989). Mercury levels in surface sediments of the St. Louis River ranged from 18 to 500 ng/L (ppt) (Glass et al. 1990). Mercury was detected in sediment samples from Crab Orchard Lake in Illinois at concentrations greater than 60 μg/L (ppb) (Kohler et al. 1990). Surficial sediment samples from several sites along the Upper Connecting Channels of the Great Lakes in 1985 had mercury concentrations ranging from below the detection limit to 55.80 μg/g (ppm) (mean concentrations ranged from 0.05 to 1.61 μg/g [ppm] at four sites) (Nichols et al. 1991). Mercury concentrations were correlated with particle size fractions and organic matter content (Mudroch and Hill 1989). Surface

sediment samples from the Lake Roosevelt/Upper Columbia River in Washington State were found to contain up to 2.7 µg/g (ppm) mercury (Johnson et al. 1990). Mercury concentrations in sediments up to 28 cm in depth in lakes adjacent to coal-fired power plants near Houston, Texas ranged from 255 to 360 mg/kg (ppm) in the summer and from 190 to 279 mg/kg (ppm) in the winter (Wilson and Mitchell 1991).

Surface sediments taken from Canadian lakes receiving atmospheric input from smelters contained between 0.03 and 9.22 µg/g (ppm) mercury, with the highest values being found in lakes nearest the smelters. However, sediment concentrations were not correlated with mercury concentrations in fish from the lakes; the fish concentrations ranged from 0.003 to 0.88 µg/g (ppm), with the highest concentration found in fish from one of the least contaminated lakes (Harrison and Klaverkamp 1990).

Estuarine and coastal marine sediment samples analyzed for NOAA's National Status and Trends Program between 1984 and 1987 showed that 38 of 175 sites contained mercury concentrations in excess of 0.41 µg/g (ppm) (dry weight) (O'Connor and Ehler 1991). In addition, mercury sediment concentrations at 6 sites exceeded the NOAA ER-M concentration of 1.3 ppm (dry weight), which is the concentration determined to be equivalent to the median (50th percentile) for all sites monitored. These 6 sites included 5 sites in the Hudson River/Raritan Estuary, New York Bight, and Raritan Bay areas between New York and New Jersey (ranging from 1.6 to 3.3 ppm dry weight) and one site in the Oakland Estuary in California (2.3 ppm dry weight) (NOAA 1990). Sediments taken from coastal areas off British Columbia, Canada contained concentrations of mercury ranging from 0.05 µg/g to 0.20 µg/g (ppm), while mercury concentrations in fish from these waters were only slightly higher; bioconcentration factors ranged from less than 1 to 14 (Harding and Goyette 1989).

Mercury has been identified in soil and sediment samples collected at 350 and 208 sites, respectively, of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.4.4 Other Environmental Media

Foods. The U.S. Food and Drug Administration (FDA) conducted a Total Diet Study (April 1982 to April 1984) to determine dietary intakes of selected industrial chemicals (including mercury) from retail purchases of foods representative of the total diet of the U.S. population (Gunderson 1988). The data were collected as part of 8 food collections, termed “market baskets”, collected in regional metropolitan areas

during the 2-year study and involved individual analysis of 234 food items representing the diets of 8 different population groups. Mercury was detected in 129 adult foods; seafood, the major contributing food group, accounted for 77% (3.01 µg of the 3.9 µg of mercury) of the total mercury intake for 25–30 year old males (Gunderson 1988). Minyard and Roberts (1991) reported results of a survey conducted on food samples analyzed at 10 state food laboratories between 1988 and 1989. These laboratories conducted food regulatory programs and analyzed findings of pesticides and related chemical residues for 27,065 food samples. In 1988, these laboratories reported methylmercury residues in 13 (0.09%) of 13,980 samples, with 1 sample exceeding federal or state tolerances. Similarly, in 1989, methylmercury was detected in 25 (0.19%) of 13,085 samples, with 1 sample exceeding federal or state tolerances. A survey of 220 cans of tuna, conducted in 1991 by the FDA, found an average methylmercury content (expressed as mercury) of 0.17 ppm (range, <0.10–0.75 ppm) (Yess 1993). Levels of methylmercury were higher in solid white (0.26 ppm) and chunk white tuna (0.31 ppm) than in chunk light (0.10 ppm) or chunk tuna (0.10 ppm). Previously, the FDA had determined methylmercury concentrations in 42 samples of canned tuna between 1978 and 1990 (Yess 1993) to range from <0.01 to 0.67 ppm methylmercury (expressed as mercury), with an average concentration of 0.14 ppm. These earlier results are similar to those obtained in the 1991 survey (Yess 1993).

The use of fish meal as a food for poultry and other animals used for human consumption may result in increased mercury levels in these animals. In Germany, poultry and eggs were found to contain average mercury concentrations of 0.04 and 0.03 mg/kg (ppm), respectively. Cattle are able to demethylate mercury in the rumen and thus absorb less mercury; therefore, beef (meat) and cow's milk contained only 0.001–0.02 mg/kg (ppm) and 0.01 mg/kg (ppm) of mercury, respectively (Hapke 1991). A survey of raw foods in Germany in 1986 found that grains, potatoes, vegetables, and fruits contained average mercury concentrations of 0.005 to 0.05 mg/kg (ppm fresh weight); however, wild mushrooms contained up to 8.8 mg/kg (ppm) of mercury. Cocoa beans, tea leaves, and coffee beans contained average mercury concentrations of 0.005, 0.025, and 0.04 mg/kg (ppm), respectively. In all cases where the mercury content was high, selenium was also found in measurable, but lower, concentrations (Weigert 1991).

Pedersen et al. (1994) conducted a monitoring study to assess the levels of trace metals, including mercury, in table wine, fortified wine, beer, soft drinks, and various juices. These authors reported that in all samples tested, mercury concentrations were at or below the detection limit (6 µg/L [6 ppb]).

Fish and Shellfish. As part of the National Pesticide Monitoring Program (NPMP), the U.S. Fish and Wildlife Service collected freshwater fish during 1976–1977 from 98 monitoring stations nationwide and analyzed them for mercury and other heavy metals (May and McKinney 1981). As part of this program, duplicate composite samples of a bottom-dwelling species and several representative predatory species were collected. Bottom-dwelling species sampled included common carp, common sucker, and channel catfish or other catfish species. Predatory species sampled were rainbow, brown, brook or lake trout at cold water stations; largemouth bass or other sunfish family members, such as crappie or bluegill, at warm water stations; and walleye or other perch family members at cool water stations. May and McKinney (1981) reported that the mean concentration of mercury was 0.153 ppm (wet weight basis) in the 1972 NPMP survey and that the mean concentration declined significantly to 0.112 ppm (range, 0.01–0.84 ppm) in the 1976–1977 survey. This decline was presumably due to curtailed production, use, and emissions of mercury (Lowe et al. 1985). May and McKinney (1981) identified an arbitrary 85th percentile concentration of 0.19 ppm for mercury to identify monitoring stations having fish with higher than normal concentrations of mercury. Most of these stations were downstream of industrial sites (e.g., chloralkali operations, pulp and paper plants; or pre-1900 gold and silver mining operations), while others were located in areas with major mercury ore (cinnabar) deposits. In a follow-up NPMP study conducted from 1980–1981, Lowe et al. (1985) reported a geometric mean mercury concentration of 0.11 ppm (wet weight) (range, 0.01–1.10 ppm). These authors reported that the downward trend in mercury residues in fish reported by May and McKinney (1981) may have leveled off, since no significant difference in the geometric mean values was detected in the follow-up study conducted in 1984–1985 as part of the National Contaminant Biomonitoring Program (Lowe et al. 1985; Schmitt and Brumbaugh 1990). However, large variations in mercury uptake among the fish species sampled, as well as among size classes of fish within the same species, may mask actual trends.

From 1986 to 1989, the National Study of Chemical Residues in Fish (NSCRF) was conducted by the EPA to assess the concentrations of 60 toxic pollutants (including mercury) in the tissues of benthic and predatory gamefish nationwide (EPA 1992f). Benthic species were analyzed as whole-body samples, while game fish species were analyzed as fillet samples, and all concentrations were reported on a wet weight basis. Mercury was detected at 92% of the 374 sites surveyed nationwide at a mean concentration of 260 ng/g (0.26 ppm) (median concentration of 0.17 ppm and a maximum concentration of 1.8 ppm), and at 2% of the sites, measured mercury concentrations exceeded 1 ppm. Most of the higher mercury concentrations in fish were collected in the Northeast. Ten of the sites in the top 10th percentile for high mercury concentrations were near pulp and paper mills, four were near Superfund sites, and most of the

remaining sites were near industrial areas. However, the mercury sources could not be identified at all of these sites. Five sites were considered to represent background conditions and six U.S. Geological Survey (USGS) National Stream Quality Accounting Network (NASQAN) sites were also among the sites in the top 10th percentile (EPA 1992f).

A recent national survey conducted by the EPA solicited data on mercury concentrations in fish collected by the states as part of their fish contaminant monitoring programs (EPA 1997b). The EPA asked all states to submit mercury residue data collected from their fish sampling programs from 1990 through 1995 to assess whether there were geographic variations or trends in fish tissue concentrations of mercury. Thirty-nine states provided information on the levels of contamination in their fish. The study included the following: information on the tissue concentrations of mercury, including the number of fish sampled (by species); the mean mercury concentration; and the minimum, median, and maximum concentrations reported for each species by state. Residue information for the three most abundant species sampled in each state included such species as the largemouth and smallmouth bass; channel, flathead, and blue catfish; brown and yellow bullhead; rainbow and lake trout; carp; walleye; north pike; and white sucker. The highest mean mercury residue for an edible species was 1.4 ppm, reported by the state of Arizona; the highest maximum mercury concentrations were 7.0 ppm for bowfin in South Carolina, followed by 6.4 ppm for white sucker in Ohio and 5.7 ppm for bowfin in North Carolina. (Note: This EPA report is currently under review by the states; however, the final report should be available by December 1998).

A summary of the mean, minimum, and maximum tissue concentrations of mercury detected for two of the sampled species with the widest geographical distribution; the largemouth bass and the channel catfish are given in Tables 5-8 and 5-9. As Table 5-8 shows, the maximum mercury residues reported for the largemouth bass exceeded the FDA action level (1 ppm) in 15 of the 25 states that collected and analyzed tissue samples for this species. The highest maximum mercury concentration reported for this species was 4.36 ppm, reported by Florida. Table 5-9 shows the maximum mercury residue reported for another widely distributed species, the channel catfish. While the maximum mercury residues reported for this species are not consistently as high as those for the largemouth bass, maximum residues in channel catfish from 6 of the 20 reporting states still exceeded the FDA action level (1 ppm). The highest maximum value reported for the channel catfish was 2.57 ppm, reported by Arkansas. Consumption of large amounts of feral fish containing these high mercury residues exposes high-end fish consuming populations (those that consume >100 grams fish/day) to potentially greater risk of mercury exposure than members of the general population (see Sections 5.5 and 5.7).

Most recently, the Northeast states and Eastern Canadian provinces issued their own mercury study, including a comprehensive analysis of current mercury concentrations in a variety of fresh water sportfish species (NESCAUM 1998). This study involved a large number of fish sampling sites in each state, many of which were remote lake sites that did not receive point source discharges. Top level piscivores (i.e., predatory fish) such as walleye, chain pickerel, and large and smallmouth bass were typically found to exhibit some of the highest concentrations, with average tissue residues greater than 0.5 ppm and maximum residues exceeding 2 ppm. One largemouth bass sample was found to contain 8.94 ppm of mercury, while one smallmouth bass sampled contained 5.0 ppm. A summary of the mean and minimum–maximum (range) of mercury concentrations in 8 species of fish sampled is shown in Table 5-10. This study also identified a relationship between elevated mercury levels in fish and certain water quality parameters, including low pH, high conductivity, and elevated levels of dissolved organic carbon.

Lake trout taken from Lake Ontario between 1977 and 1988 did show a progressive decline in mercury contamination from 0.24 μg/g (ppm) in 1977 to 0.12 μg/g (ppm) in 1988 (Borgmann and Whittle 1991). Samples of zooplankton taken from an Illinois lake in 1986 contained approximately 10 ng/g (ppb) mercury; however, fish that fed on the zooplankton had whole body mercury concentrations ranging from 11.6 μg/kg (ppb) for inedible shad to 69 μg/kg (ppb) for edible largemouth bass, indicating bioaccumulation was occurring up the aquatic food chain. Older fish generally had higher mercury concentrations (Kohler et al. 1990). Mercury concentrations in crayfish taken from 13 Ontario lakes with no known mercury inputs ranged from 0.02 to 0.64 μg/g (ppm); the concentrations were positively correlated with organism weight and fish mercury concentrations (Allard and Stokes 1989). Brown trout taken from Lake Ontario contained between 0.18–0.21 μg/g (ppm) mercury in unskinned fillets and between 0.24–0.26 μg/g (ppm) mercury in skinned fillets, indicating that methylmercury is associated with the protein fraction of fish tissue (Gutenmann and Lisk 1991).

Methylmercury constitutes over 99% of the total mercury detected in fish muscle tissue, with no detection of inorganic or dimethylmercury (Grieb et al. 1990; Bloom 1992). Mercury levels were examined in aquatic organisms taken from the Calcasieu River/Lake Complex in Louisiana. The order of enrichment was as follows: shrimp (0.2 μg/g [ppm]) <mussel (0.3 μg/g [ppm]) <fish (0.4 μg/g [ppm]) = oyster (0.4 μg/g [ppm]) <zooplankton (1.4 μg/g [ppm]) (Ramelow et al. 1989). Average mercury concentrations for aquatic organisms collected from the Wabigoon/English/Winnipeg River system in Canada were as follows: 0.06–2.2 μg/g (ppm) for crayfish, 0.01–0.55 μg/g (ppm) for perch, and 0.04–1.2 μg/g (ppm) for pike. Methylmercury concentrations were found to increase with distance from the pollutant source,

possibly as a result of the increased bioavailability of organic mercury produced by aquatic micro-organisms, whereas inorganic mercury was the predominant form at the source (Parks et al. 1991).

Typical mercury concentrations in large carnivorous freshwater fish (e.g., pike) and large marine fish (e.g., swordfish, shark, and tuna) have been found to exceed 1 µg/g (ppm) (EPA 1984b; Fairey et al. 1997; FDA 1998; Hellou et al. 1992; Hueter et al. 1995), with mercury content again being positively correlated with the age of the fish (Gutenmann et al. 1992; Hueter et al. 1995). Methylmercury concentrations in muscle tissue of 9 species of sharks were analyzed from 4 locations off Florida (Hueter et al. 1995). Muscle tissue methylmercury concentration averaged 0.88 µg/g (ppm) (wet weight) and ranged from 0.06 to 2.87 µg/g (ppm), with 33.1% of the samples exceeding the FDA action level (1 ppm). A positive correlation between methylmercury and shark body length (size) also was found, such that sharks larger than 200 cm in total length contained methylmercury concentrations >1 ppm. Sharks collected off the southern and southwestern coastal areas contained significantly higher concentrations than those caught in the northeast coastal region (Cape Canaveral and north).

Methylmercury concentrations were highest in the Caribbean reef shark (*Carcharhinus perezi*). The two most abundant shark species in the U.S. East Coast commercial shark fishery, sandbar *(C. plumbeus*) and blacktip (*C. limbatus*) sharks, are of special concern with respect to human health. Although the mean concentration of methylmercury in the sandbar shark (0.77 µg/g) was below the average for all sharks, sandbar shark tissues contained up to 2.87 ppm methylmercury, and 20.9% of the samples exceeded the FDA action level of 1 ppm. A total of 71.4% of the blacktip shark samples (mean, 1.3 µg/g) exceeded the FDA action level. The authors suggest that continued monitoring of methylmercury concentrations in various sharks species in the commercial marketplace is warranted. In a recent study of sportfish collected in San Francisco Bay, Fairey et al. (1997) reported that the highest concentrations of mercury were detected in leopard shark muscle tissue (1.26 ppm). Bluefin tuna caught in the Northwest Atlantic Ocean in 1990 contained mercury at a mean muscle concentration of 3.41 µg/g (ppm) dry weight (Hellou et al. 1992).

As part of the National Oceanic and Atmospheric Administration (NOAA) Status and Trends Program conducted from 1984 to 1987, mercury concentrations were analyzed in four marine bivalve species in U.S. coastal waters (NOAA 1987). Mercury concentrations in bivalve tissues ranged from 0.01 to 0.48 µg/g (ppm) dry weight in oysters (*Crassostrea virginica*), 0.28 to 0.41 µg/g (ppm) in the Hawaiian oyster (*Ostrea sandwichensis*), 0.05 to 0.47 µg/g (ppm) in the blue mussel (*Mytilus edulis*), and 0.04 to

0.26 µg/g (ppm) in the California mussel (*Mytilus californianus*). Oysters (*Crassostrea virginica*) collected around the Gulf of Mexico between 1986 and 1989 had mercury concentrations ranging from <0.01 to 0.72 µg/g [ppm] (mean, 0.127 µg/g [ppm]) (Presley et al. 1990). Oysters taken from the Mississippi Sound in 1986 generally did not contain mercury at levels exceeding the detection limit (0.02 µg/g [ppm]), although two samples had detectable mercury levels of 0.66 and 6.6 µg/g [ppm] (Lytle and Lytle 1990).

Mercury has been detected in fish samples collected at 56 of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

Marine mammals. Mercury concentrations have been analyzed in various tissues (i.e., muscle, liver, kidneys) from several species of marine mammals, including beluga whales, narwhal, white-toothed dolphins, pilot whales, ringed seals, harp seals, and walruses in the western and eastern Canadian Arctic (Wagemann et al. 1995). The mean mercury concentration (µg/g [ppm] dry weight) in liver tissue was highest in pilot whales (78 ppm), harp seals (36 ppm), Eastern Arctic ringed seals (29 ppm), narwhal (25 ppm), and Eastern Arctic beluga (22 ppm), with lesser amounts in Arctic walrus (5 ppm) and dolphins (4 ppm). Of the three tissues analyzed, mercury was most concentrated in the liver, with successively lower concentrations in the kidney and muscle tissue. This pattern prevails in most marine mammals. The concentration of total mercury is greater by a factor of 3 in the liver than in the kidney, but can be significantly higher in some species (see Table 5-11). Mean tissue residues in ringed seals from the western Arctic had significantly higher concentrations of mercury than those from the eastern Arctic. The authors reported higher mercury levels in sediment (68–243 ng/g [ppb] dry weight) and water (11–29 ng/L [ppt]) from the western Arctic, as compared to sediment (40–60 ng/g [ppb] dry weight) and water (3.7 ng/L [ppb]) from the eastern Arctic. These differences in sediment and water mercury levels may be responsible for some of the observed differences in mercury tissue concentrations in the seals.

Mercury tissue concentrations were detected in 17 adult and 8 fetal pilot whales from two stranding episodes off Cape Cod, Massachusetts (Meador et al. 1993). Total mercury occurred in high concentrations in both the liver and kidney, and liver concentrations were significantly correlated with the animal's length. Methylmercury, as a percentage of total mercury, varied inversely with total mercury, indicating that demethylation was occurring. Mean adult mercury concentrations in µg/g (ppm) dry weight in liver and kidneys were 176 ppm (range, 1.9–626 ppm dry weight) and 27.5 ppm (range, 6.8–49.7 ppm dry weight), respectively. Mean fetal mercury concentrations in µg/g (ppm) dry weight in liver and kidneys

were 2.3 ppm (range, 0.9–5.4 ppm dry weight) and 1.9 ppm (range, 0.6–3.9 ppm dry weight), respectively. The mean methylmercury concentration in µg/g (ppm) dry weight in adult liver tissue was 8 ppm (range, 5.6–10 ppm). Aguilar and Borrell (1995) studied mercury tissue levels (1970 to 1988) in harbor porpoises in the eastern North Atlantic. These authors reported that in most tissues of harbor porpoises, the mercury was virtually all in the form of methylmercury; however, the fraction of organic mercury in the liver was much lower than in the rest of the body tissues. These authors found that for a given tissue, the concentrations detected were extremely variable between localities and years. Mercury concentrations in harbor porpoises ranged from 0.62 to 70 ppm in liver and from 0.66 to 22 ppm in muscle. The mean mercury concentration in liver for the eastern harbor porpoise population was 11.2 ppm. Mercury tissue levels progressively increased with the age of the animal; no significant differences were found between the sexes (Aguilar and Borrell 1995).

Plants. Although data on mercury distribution among freshwater vascular plant parts is lacking for unpolluted systems, Mortimer (1985) reported that total mercury in the roots of five species of freshwater vascular plants in the polluted Ottawa River was 10–40% higher than in the shoots. Speciation may be important in determining the patterns of mercury uptake, translocation, and excretion in macrophytes. Shoots of *Elodea densa* more readily accumulated methylmercury than inorganic mercury, and also excreted more inorganic mercury than methylmercury (Czuba and Mortimer 1980). Significant translocation of inorganic mercury from shoots to roots occurred in *E. densa* (Czuba and Mortimer 1980). In this species, methyl- and inorganic mercury moved in opposite directions, with methylmercury moving towards the young shoot apex, and inorganic mercury moving towards lower (older) parts of the shoot (Czuba and Mortimer 1980). Dolar et al. (1971) noted the same methylmercury pattern in the water milfoil (*Myriophyllum spicatum*). Using solution culture experiments, these authors showed that mercury accumulation was greater when plants were exposed to inorganic mercury ($HgCl_2$) than organic methylmercury (CH_3HgCl) and that mercury accumulation from the nutrient solution was rapid and approached maximum values in 2 hours. Organomercury compounds (methylmercury chloride, phenylmercuric acetate, phenylmercuric chloride, and phenylmercuric hydroxide) were more available than inorganic compounds (HgF_2 and $HgCl_{2)}$ from lake sediments. The various organomercury and inorganic mercury compounds were added to sediment at concentrations of 0, 46, 230, and 460 ppm prior to rooting water milfoil. After 20 days, concentration of mercury in the plant tissues exposure to 46, 230, and 460 ppm of the inorganic mercury compounds in the sediment ranged from 1.71 to 4.01, 4.81–6.03, and 6.61–10.2, respectively. In contrast, the concentrations of mercury in plant tissues exposed to 46, 230, and 460 ppm of the organic mercury compounds in the sediment ranged from 2.40 to 7.15 ppm, 36–84.5 ppm, and 114.6–243.1 ppm,

respectively. The control plants (no mercury compounds added to the sediments) contained 0.3 ppm mercury. It is clear from this experiment that organomercury compounds may accumulate significantly in the above-ground parts of some macrophytes. Mortimer (1985) found that although *E. densa* shoots had lower total mercury contents than roots, with 32% of the mercury in the shoots in the form of methylmercury, compared to only 10% in the roots.

Grasses sampled downwind of a municipal waste incinerator contained up to 0.20 µg/g (ppm) of mercury, with concentrations decreasing with increasing distance from the facility (Bache et al. 1991). Background mercury levels in vegetation were usually below 0.1 µg/g (ppm) dry weight (Lindqvist 1991e); however, mushrooms collected 1 km from a lead smelter in Czechoslovakia contained between 0.3 and 12 mg/kg (ppm) dry weight (Kalac et al. 1991).

Consumer and Medicinal Products. Various consumer and medicinal products contain mercury or mercury compounds (i.e., skin lightening creams and soaps, herbal remedies, laxatives, tattooing dyes, fingerpaints, artists paints, and make-up paints) (Barr et al. 1973; Dyall-Smith and Scurry 1990; Lauwerys et al. 1987; Rastogi 1992; Wendroff 1990).

Barr et al. (1973) reported elevated mercury levels in the blood of women using skin lightening creams, although the mercury compound and concentrations in the skin cream were not determined. More recently, Dyall-Smith and Scurry (1990) reported that one skin lightening cosmetic cream contained 17.5% mercuric ammonium chloride. Lauwerys et al. (1987) reported a case of mercury poisoning in a 3-month-old infant whose mother frequently used a skin lightening cream and soap containing inorganic mercury during her pregnancy and during the 1-month lactation period following birth. However, the mercury concentration and specific mercury compound in the cream and soap were not determined. Al-Saleh and Al-Doush (1997) analyzed the inorganic mercury content of 38 skin lightening creams in Saudi Arabian markets. The creams were manufactured in a variety countries, including India and Pakistan, other Arab countries, Thailand, Taiwan, Indonesia, England and Germany. Almost 50% of the creams tested exceeded the tolerance limit of 1 ppm. The mean concentration of mercury in the 38 creams was 994 ppm, with a range of 0–5,650 ppm. It is not known whether any of these products are available in the United States.

Metallic mercury was also the source of two cases of mercury poisoning caused by the dermal application of an over-the-counter anti-lice product (Bourgeois et al. 1986). The more severely poisoned individual applied 30 g of ointment containing 9 g of metallic mercury (300,000 ppm) to his entire body. Wands et al.

(1974) also reported the deaths of two individuals due to the excessive use of a laxative preparation containing mercurous chloride (calomel).

Metallic mercury has been used by Mexican American and Asian populations in traditional remedies for chronic stomach disorders (Espinoza et al. 1995; 1996; Geffner and Sandler 1980; Trotter 1985). Most recently, Perharic et al. (1994) reported cases of poisonings resulting from exposure to traditional remedies and food supplements reported to the National Poisons Unit in London, England. From 1989 to 1991, elemental mercury was implicated in several poisonings following exposure to traditional Asian medicines. In one case, the mercury concentration in the medicinal product taken orally was 540 mg/g (540,000 ppm). The mercury was in its elemental or metallic form. Espinoza et al. (1995, 1996) reported that while examining imported Chinese herbal balls for the presence of products from endangered species, the authors detected potentially toxic levels of arsenic and mercury in certain herbal ball preparations. Herbal balls are aromatic, malleable, earth-toned, roughly spherical, hand-rolled mixtures primarily composed of herbs and honey that are used to make medicinal teas. These herbal balls are used as a self-medication for a wide variety of conditions, including fever, rheumatism, apoplexy, and cataracts. Herbal balls similar to those analyzed are readily available in specialty markets throughout the United States. Mercury (probably mercury sulfide) was detected in 8 of the 9 herbal balls tested. The recommended adult dose for the herbal balls is two per day. Ingesting two herbal balls could theoretically provide a dose of up to 1,200 mg of mercury.

Samudralwar and Garg (1996) conducted trace metal analysis on a variety of plants used in Indian herbal remedies and other medicinal preparations. These authors reported mercury concentrations of 139, 180, 27, 12.5, 11.7, and <10 ppb for Bowen's kale, Neem leaves, Gulvei leaves, Kanher bark, Vekhand root, and orange peel, respectively.

Hoet and Lison (1997) reported on an unusual non-occupational source of mercury exposure that resulted in a woman that used prescription nasal drops that contained 300 mg/L (ppm) borate phenylmercury. These authors reported that the woman, who had used the nasal drops over a long period of time, had high urinary levels of mercury (82 µg/g), but that blood levels were not abnormal (5.5 µg/L).

Mercuric sulfide, or cinnabar, was reported to be used in tattooing dyes to produce a red pigmentation (Bagley et al. 1987; Biro and Klein 1967). An analysis of finger paints and make-up paints manufactured in Europe showed that they all contained less than 1 ppm mercury (Rastogi 1992). Rastogi and Pritzi (1996)

conducted another study to assess the migration of several toxic metals from crayons, watercolor paints, and water-based paints. Migration of mercury from the art materials was determined by scraping flakes of the products into dichloromethane for 2 hours at 54E C. The degreased material was then placed in an aqueous HCl solution, shaken, and centrifuged. The supernatant was then filtered through a 0.45 µ membrane filter and was analyzed. These authors reported that the migration of mercury from these art supplies was 0.24–5.98 ppm for red, 0.26–3.63 ppm for blue, 0.20–4.79 ppm for yellow, 0.22–5.68 ppm for green, and 0.17–3.63 ppm for white paint. Migration of mercury from the product occurred in 57% of the samples tested. The migration limit set by European Standard EN71-3 for mercury is 60 ppm. This value was not exceeded in any of the art supplies tested. The authors, however, believe that children might be exposed not only to mercury, but to several other metals that also co-migrated from the paints.

Cigarettes. In a study conducted in West Germany, Pesch et al. (1992) analyzed mercury concentrations in 50 brands of cigarettes manufactured in 2 Western and 6 Eastern European countries. These authors reported that in 1987, the average mercury concentration detected in cigarettes was 0.098 µg/g (ppm) (dry weight) (range, 0.06 to 0.14 ppm dry weight). In 1991, the mean mercury concentrations for cigarettes were 0.034 µg/g (ppm) dry weight (range, 0.007–0.092 ppm dry weight) for Eastern Europe and 0.015 µg/g (ppm) dry weight (range, 0.006–0.037 ppm dry weight) for Western European countries. The authors attributed the decline in mercury content of cigarettes to environmental protection measures instituted in the intervening years (Pesch et al. 1992).

Religious and Ethnic Rituals, Ceremonies, and Practices. While some of medicinal and pharmaceutical uses of mercury compounds have been replaced in recent years, individuals in some ethnic or religious groups may still use mercury in various religious or ethnic rituals, practices, and ceremonies that can expose them to elevated mercury concentrations in room air. Metallic mercury has been used in Latin American and Caribbean communities as part of certain religious practices (e.g., Voodoo, Santeria, and Espiritismo), predominantly in domestic settings (Wendroff 1990). This use of mercury can contaminate a dwelling or automobile if the mercury is not completely removed from flooring, carpeting, and woodwork in an appropriate manner. Metallic mercury (sometimes under the name *azogue)* currently is sold in shops called botanicas which stock medicinal plants, traditional medicines, incense, candles, and perfumes. Botanicas typically dispense mercury in gelatin capsules or sometimes in small glass vials. Some religious practices involve sprinkling metallic mercury on the floor of the dwelling or of a car, mixing metallic mercury with soap and water to wash the floor, or placing it in an open container to rid the house of evil spirits. Other practices involve carrying a small amount of mercury in a vial on the person, or mixing

mercury in bath water or perfumed soaps, devotional candles, ammonia or camphor. Any of these practices can liberate mercury vapor into the room air, exposing the occupants to elevated levels of mercury vapors (ATSDR 1997; Wendroff 1990, 1991). In addition to the individuals that intentionally use mercury in their dwellings, the opportunity exists for nonusers to be inadvertently exposed when they visit the dwelling, or purchase or rent dwellings in which the former tenants used mercury for religious purposes. The issuance of cautionary notices and information by health departments to members of these user populations is appropriate.

5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Potential sources of general population exposure to mercury include inhalation of elemental mercury vapors in ambient air, ingestion of drinking water and foodstuffs contaminated with elemental mercury or various mercury compounds (i.e., methylmercury), and exposures to elemental mercury and various mercury compounds through dental and medical treatments (NIOSH 1973). EPA (1984b) reported that dietary intake is the most important source of nonoccupational human exposure to mercury, with fish and fish products being the dominant sources of methylmercury in the diet. This is consistent with an international study of heavy metals detected in foodstuffs from 12 different countries (Toro et al. 1994). These authors found that mercury concentrations of 0.15 mg/kg (ppm) for fish and shellfish were approximately 10–100 times greater than for the other foods tested, including cereals, potatoes, vegetables, fruits, meat, poultry, eggs, milk, and milk products. Another author also estimated mean mercury concentrations to be 100 times greater for fish than for foods other than fish ((0.4 µg/g vs. 0.004 µg/g [ppm]) (Fishbein 1991). Recent animal and human studies, however, have also shown that the uptake, distribution, and rate of excretion of elemental mercury from dental amalgams are also major contributing factors to mercury body burden in humans (Björkman et al. 1997; Lorscheider et al. 1995).

A summary of contributing sources of mercury to the body burden of humans is presented in Table 5-12. Because of the variability in fish consumption habits among U.S. consumers and the variability in the concentrations of methylmercury detected in various fish and shellfish species, exposures for individual members of the general population are difficult to measure. Similarly, because of the variability in the number of amalgam fillings in individual members of the general population and the high retention rate for elemental mercury, a wide range of potential exposures to elemental mercury can be shown for persons with dental amalgams. Dental amalgams, however, may represent the largest single non-occupational contributing source to total body burden of some mercury in people with large numbers of amalgam fillings.

Dietary Sources of Mercury. Galal-Gorchev (1993) analyzed dietary intakes of mercury from 14 countries, including the United States, between 1980 and 1988. This author reported that the contribution of fish to the total intake of mercury varied from a low of 20% in Belgium and the Netherlands to 35% in France, the United Kingdom, and the United States. The highest contribution of fish to mercury intake (85%) was reported for Finland. The author further pointed out (based on information from the Netherlands on levels of mercury contamination in a variety of foods) that although mercury was found at higher concentrations in fish (0.1 mg/kg [ppm]) than in other foods (0.01 mg/kg or less), higher consumption of cereals and meats render the contributions of these food groups to the total mercury intake about the same as that from fish. Therefore, the general assumption that fish is the main contributor to the intake of mercury may, at times, not be justified because of dietary habits of a given population (Galal-Gorchev 1993).

The FDA's Total Diet Study (April 1982–April 1984) estimated an average daily intake of mercury (total) based on measured levels and assumed trace amounts in foods to be representative of the "total diet" of the U.S. population (Gunderson 1988). Estimated daily exposures for mercury were 0.49 µg/day for infants ages 6–11 months, 1.3 µg/d for 2-year-old children, 2.9 µg/day for females ages 25–30, and 3.9 µg/day for males 25–30 years of age. Expressed on a per body weight basis, the intake for all age groups, except 2-year-old children, was approximately 50 ng/kg/day (Clarkson 1990; Gunderson 1988). For 2-year-old children, the intake was estimated to be approximately 100 ng/kg/day (assuming 50% of the fish intake was due to fish caught locally). More recently, MacIntosh et al. (1996) calculated average daily dietary exposure to mercury and 10 other contaminants for approximately 120,000 U.S. adults by combining data on annual diet, as measured by a food frequency questionnaire, with contaminant residue data for table-ready foods that were collected as part of the annual FDA Total Diet Study (1986–1991). The estimated mean dietary exposure (µg/day) for 78,882 adult females and 38,075 adult males in 1990 was 8.2 µg/day (range, 0.37–203.5 µg/day) for females and 8.6 µg/day (range, 0.22–165.7 µg/day) for males. Assuming a body weight of 65 kg for women and 70 kg for men, the daily intakes of mercury would be 126 ng/kg/day (range, 5.7–3,131 ng/kg/day) for women and 123 ng/kg/day (range, 3.1–2,367 ng/kg/day) for men respectively. These authors found that the coefficient of variation was 44% for mercury, indicating that the exposures to this chemical estimated for a given individual may be accurate to within approximately a factor of 2. Lack of data about the actual amount of food consumed accounted for 95% of the total uncertainty for mercury. Individual food items contributing most to the uncertainty of mercury measurements were canned tuna and other fish (MacIntosh et al. 1996).

The FDA currently has advice for consumers posted on the Internet that recommends that pregnant women and women of childbearing age, who may become pregnant, limit their consumption of shark and swordfish to no more that one meal per month (FDA 1998). This advice is given because methylmercury levels are much higher in these fish species than in the more commonly consumed species. Dietary practices immediately before pregnancy could also have a direct bearing on fetal exposure, particularly during pregnancy. The FDA states that nursing mothers who follow this advice, do not expose their infants to increased health risks from methylmercury (FDA 1998). The FDA further advises that persons other than pregnant women and women of child-bearing age limit their regular consumption of shark and swordfish (which typically contain methylmercury at approximately 1 ppm) to about 7 ounces per week (about one serving) to stay below the recommended maximum daily intake for methylmercury. For fish species with methylmercury levels averaging 0.5 ppm, regular consumption should be limited to 14 ounces (about 2 servings) per week. A summary of mercury concentrations in the top 10 types of fish consumed by the general U.S. population is presented in Table 5-13. There is a wide degree of variability in the amount of fish consumed in the diet by various subpopulations within the United States. Various ethnic groups, as well as recreational and subsistence fishers often eat larger amounts of fish than the general population and may routinely fish the same waterbodies (EPA 1995k). If these waterbodies are contaminated, these populations may consume a larger dose of mercury by virtue of the fact that they consume larger amounts of fish (from >30 g/day for recreational fishers to >100 g/day for subsistence fishers) with higher concentrations of mercury in their tissues than individuals in the general population that tend to consume smaller amounts (6.5 g/day) of supermarket-purchased fish that come from a variety of sources. Table 5-14 provides a summary of the amount of fish consumed daily by the general population, as compared to recreational and subsistence fishers, including some Native American tribal groups. Those individuals that consume greater than 100 g of fish per day are considered high-end consumers; they consume more than 10 times the amount of fish estimated to be consumed by members of the general population (6.5 g/day) (EPA 1995k).

Table 5-15 provides an summary of the estimated total number of persons in the U.S. population (excluding Alaska and Hawaii), the total female population of reproductive age (ages 15–44 years), and the total population of children (<15 years). Based on the percentage of people that reported eating fish during a 3-day dietary survey conducted from 1989 to 1991 as part of the Continuing Survey of Food Intake by Individuals (CSFII), the number of persons estimated to consume fish can be calculated. Using this method, more than 76 million people in the U.S. population eat fish; of these, more than 17 million females of reproductive age (15–44 years old) consume fish, and more than 13 million children (<15 years of age) eat

fish. In addition, estimates of the total number of persons in the high-end fish consumer group (subsistence fishers) have been calculated, as were estimates of the total number of adult women of reproductive age (15 to 44 years old) and children (<15 years old) in the high-end consumer group, i.e., those potentially at greatest risk of exposure (EPA 1996e). With respect to fish consumers, more than 3.8 million are high-end consumers (>100 grams of fish/day), and of these, it is estimated that more than 887,000 are women of reproductive age (15–44 years), and 665,000 are children (<15 years old). It was also estimated that of the fish consuming females of reproductive age, more than 84,000 are pregnant in any given year.

Fish is generally considered an excellent source of protein in the diet and the health benefit of fish consumption, including the reduction in the incidence of coronary heart disease, is well recognized (Salonen et al. 1995). However, Salonen et al. (1995) studied 1,833 eastern Finnish men ages 42–60 and related high dietary intake of freshwater fish containing mercury residues, as well as elevated hair content and urinary excretion of mercury, to a risk of acute myocardial infarction and death from coronary heart disease and cardiovascular disease. Men with the highest tertile of hair mercury had a 2-fold age-specific risk and a 2.9-fold adjusted risk of acute myocardial infarction and cardiovascular death, compared to men with lower mercury hair levels. Egeland and Middaugh (1997) and Clarkson et al. (1998) contend that the Seychelles population is a more appropriate sentinel population for fish consumers in the United States because: (1) the major source of methylmercury is from open ocean fish; (2) the mercury concentrations in hair are 10–20 times the average found in the United States; and (3) because the Seychellois consistently consume about 12 fish meals per week. These authors feels that the potential adverse effects of methylmercury in fish would be detected in the Seychelles Island population, long before such effects are observed in the United States. The Finnish study (Salonen et al. 1995), however, suggests that freshwater fish, low in selenium and omega-3 polyunsaturated fatty acids, may not protect against cardiovascular risks from methylmercury. The human health benefit/cost tradeoff between fish consumption and mercury exposure varies by species and mercury dose.

Dental Amalgams. Recent animal and human studies have also identified the uptake, distribution, and rate of excretion of elemental mercury from dental amalgams as another significant contributing source to mercury body burden in humans (Björkman et al. 1997; Lorscheider et al. 1995). A summary of contributing sources of mercury to the human body burden is presented in Table 5-12. Because of the wide range of potential exposures and the high retention rate for elemental mercury, dental amalgams potentially represent the largest single contributing source of mercury exposure in some individuals with large numbers (>8) of amalgam fillings.

Dental amalgams may contain 43–54% elemental mercury (DHHS 1993). A single amalgam filling with an average surface area of 0.4 cm^2 has been estimated to release as much as 15 µg mercury/day, primarily through mechanical wear and evaporation, but also through dissolution into saliva (Lorscheider et al. 1995). The rate of release is influenced by chewing, bruxism (grinding of teeth) food consumption, tooth brushing, and the intake of hot beverages (Weiner and Nylander 1995). For the average individual with eight occlusal amalgam fillings, 120 µg of mercury could be released daily into the mouth, and a portion of that swallowed or inhaled (Lorscheider et al. 1995). Experimental results regarding estimated daily dose of inhaled mercury vapor released from dental amalgam restorations are few and contradictory (Berglund 1990). More recently, Björkman et al. (1997) reported that approximately 80% of inhaled mercury from dental amalgams is absorbed (Björkman et al. 1997). Various laboratories have estimated the average daily absorption of amalgam mercury ranging from 1 to 27 µg, with levels for some individuals being as high as 100 µg/day (Björkman et al. 1997; Lorscheider et al. 1995; Weiner and Nylander 1995). Estimates of mean daily elemental mercury uptake from dental amalgams from these and earlier studies are summarized in Table 5-16. A report from the Committee to Coordinate Environmental Health and Related Programs (CCEHRP) of the Department of Health and Human Services determined that "measurement of mercury in blood among subjects with and without amalgam restorations . . . and subjects before and after amalgams were removed . . . provide the best estimates of daily intake from amalgam dental restorations. These values are in the range of 1–5 µg/day" (DHHS 1993). Another source indicates that the consensus average estimate is 10 µg amalgam Hg/day (range, 3–17 µg/day) (WHO 1991). However, Halbach (1994) examined the data from 14 independent studies and concluded that the probable mercury dose from amalgam is less than 10 µg/day. Most recently, Richardson (1995) computed a release rate per filled tooth surface as 0.73 µg/day-surface, with a standard deviation of 0.3 µg/day-surface and a “stimulation magnification factor” of 5.3, based on a weighted average enhancement of mercury vapor concentration following chewing, eating, or tooth brushing reported in three amalgam studies.

By comparison to the estimated daily absorbance of mercury from dental amalgams (range, 3–17 µg), the estimated daily absorbance from all forms of mercury from fish and seafood is 2.31 µg and from other foods, air, and water is 0.3 µg (WHO 1991). These other sources taken together only total 2.61 µg/day, in comparison to estimates of 3–17 µg/day for dental amalgams. Assuming a person has large numbers of amalgams, this source may account for 17 µg/day out of a total absorbance of 19.61 µg/day, or 87% of the absorbed mercury. In contrast, in individuals with only a few amalgams, mercury from this source may account for only 3 µg mercury/day out of a total absorbance of 5.61 µg/day, or 53% of absorbed mercury. Halbach et al. (1994) concluded that the sum of the mercury uptake from dental amalgam and dietary

uptake is still below the dose corresponding to the acceptable daily intake (ADI) of mercury. The ADI of 40 µg total mercury, 30 µg of which are allowed for methylmercury, results in a total dose of approximately 30 µg after accounting for absorption (Halbach 1994; WHO 1976). WHO (1990) estimates a daily absorption of 2.61 µg from background exposure for persons without amalgam exposure.

In a recent study by Schweinsberg (1994), the author monitored mercury in blood, urine, and hair of subjects with amalgam fillings, in subjects who consumed fish, and in mercury-exposed workers. With respect to hair concentrations, the author reported a mean mercury level in hair of 560 µg/kg (ppb), 940 µg/kg, and 1,600 µg/kg in subjects that consumed the following mean amounts of fish per month: 120 g/month (range, 0–<400 g fish/month); 600 g/month (range, 400–<1,000 g/month); and 1,900 g/month (>1,000 g/month), respectively. Mercury concentrations in whole blood (µg/L) were 0.2–0.4 µg/L for individuals with no fish consumption and no dental amalgams, 1.047±0.797 µg/L for persons with no fish consumption and >6 dental amalgams, 2.56±2.123 µg/L for persons with fish consumption >990 g/month and no dental amalgams, and 2.852±2.363 µg/l for persons with fish consumption >990 g/month and >6 dental amalgams. Mercury concentrations in the urine of occupationally exposed thermometer factory workers were higher, by a factor of 100, than in the group with amalgam fillings. The author concluded that both amalgam fillings and the consumption of fish burden individuals with mercury in approximately the same order of magnitude.

In a more recent study of lactating women, Oskarsson et al. (1996) assessed the total and inorganic mercury content in breast milk and blood in relation to fish consumption and amalgam fillings. The total mercury concentrations (mean±standard deviation) in breast milk, blood, and hair samples collected 6 weeks after delivery from 30 Swedish women were 0.6±0.4 ng/g (ppb), 2.3±1.0 ng/g, and 0.28±0.16 µg/g, respectively. In milk, an average of 51% of total mercury was in the inorganic form, whereas in blood an average of only 26% was in the inorganic form. Total and inorganic mercury levels in blood and milk were correlated with the number of amalgam fillings. The concentrations of total mercury and organic mercury in blood and total mercury in hair were correlated with the estimated recent exposure to methylmercury via consumption of fish. There was no significant difference between the milk levels of mercury in any form and the estimated methylmercury intake. A significant correlation was found, however, between the levels of total mercury in blood and in milk, with milk levels being an average of 27% of the blood levels. There was also an association between inorganic mercury in blood and in milk; the average level of inorganic mercury in milk was 55% of the level of inorganic mercury in blood. No significant correlations were found between the levels of any form of mercury in milk and the levels of organic mercury in blood. The results indicated that

there was an efficient transfer of inorganic mercury from blood to milk and that, in the study population, mercury from amalgam fillings was the main source of mercury in breast milk. Exposure of the infant to mercury in breast milk was calculated to range up to 0.3 µg/kg/day, of which approximately one half was inorganic mercury. This exposure corresponds to approximately one-half the tolerable daily intake for adults recommended by the World Health Organization (WHO). The authors concluded that efforts should be made to decrease mercury burden in women of reproductive age.

Blood. (EPA 1996d). Because methylmercury freely distributes throughout the body, blood is a good indicator medium for estimating methylmercury exposure. However, because an individual's intake may fluctuate, blood levels may not reflect mercury intake over time (Sherlock and Quinn 1988; Sherlock et al. 1982). Recent reference values for total mercury levels in blood of non-exposed individuals in the general U.S. population are very limited. The mean concentration of mercury in whole blood based on a review of existing data from other countries, is 8 µg/L (ppb) (WHO 1990). Certain groups with high fish consumption may attain blood methylmercury levels of 200 µg/L (ppb), which is associated with a low (5%) risk of neurological damage to adults (WHO 1990).

Urine. Urine is a common indicator used to assess occupational mercury exposure (EPA 1996d). Urinary mercury is thought to indicate most closely the mercury levels present in the kidneys (Clarkson et al. 1988b). But while urinary mercury has been widely used to estimate occupational exposures, reference values for urinary mercury levels in non-exposed individuals in the general U.S. population are very limited. The mean concentration of urinary mercury, based on a review of existing data from other countries, is about 4 µg/L (ppb) (WHO 1990, 1991). For assessment of long-term inorganic mercury exposure, biological monitoring of the urinary mercury is normally used (Skare 1995). Several authors have related elevated urinary mercury levels to dental amalgams in individuals in the general population (Barregard et al. 1995; Skare 1995) and in dentists and dental personnel receiving occupational exposures (Akesson et al. 1991; Chien et al. 1996; WHO 1991).

Breast Milk. Recent reference values for mercury levels in breast milk in non-exposed individuals in the general U.S. population are very limited. The mean concentration of mercury in breast milk, based on a review of existing data from other countries, is 8 µg/L (ppb) (WHO 1990, 1991). Mean concentrations of mercury in breast milk samples from the United States and other countries are summarized in Table 5-17. Pitkin et al. (1976) reported a mean total mercury concentration of 0.93±0.23 ppb in a midwestern community in the United States. This mean value is only about one-third the mean value reported for Inuit

women living in interior (3.2±0.8 ppb) or urban areas (3.3±0.5 ppb) of Alaska and less than one-seventh the mean value for coastal Alaskan Inuit women (7.6±2.7 ppb) known to consume seal meat and oil, as well as marine fish (Galster 1976). The latter breast milk total mercury level is comparable to the median (2.45 ppb) and maximum (8.7 ppb) values reported for women in the Faroe Islands that consume large amounts of fish and pilot whale meat (Grandjean et al. 1995a).

Levels of total mercury in breast milk have been monitored in several foreign countries over the past three decades. A mean breast milk mercury concentration of 3.6±2.2 ppb (range, non-detected to 9.8 ppb) was reported for an urban population in Tokyo, Japan (Fujita and Takabatake 1977). In a study of urban women residing in Madrid, Spain, the mean breast milk mercury concentration was 9.5±5.5 ppb (range, 0.9–19 ppb) (Baluja et al. 1982). These authors did not provide any information (i.e., whether females were fish consumers, the number of dental amalgams they had, or their occupations) that would explain the relatively high mercury levels. Skerfving (1988) reported mercury concentrations ranged from 0.2 to 6.3 ppb in breast milk of Swedish women that consumed fish; however, this author did not provide specific information on the fish consumption rate or the number of dental amalgams of the study population. Most recently, Oskarsson et al. (1996) reported a mean total breast milk concentration of 0.6±0.4 ppb (range, 0.1–2.0 ppb) for a group of Swedish women that consumed freshwater fish and had an average of 12 amalgam fillings. This was a smaller range in mercury concentrations than that reported by Skerfving (1988).

All of these general population breast milk mercury concentrations are in sharp contrast to those reported for samples collected from women in Minamata, Japan, where industrial effluents containing methylmercury caused widespread contamination of local seafood. Breast milk total mercury concentrations were on the order of 63 ppb in individuals who lived in the vicinity of Minamata, Japan and had consumed highly mercury-contaminated fish (Fujita and Takabatake 1977). Similarly, in Iraq, where consumption of bread made from seed grain treated with methylmercury as a fungicide caused a similar mercury poisoning outbreak, breast milk concentrations as high as 200 ppb were reported (Bakir et al. 1973). Breast milk containing total mercury levels of >4 ppb would exceed the safe level (2 μg methylmercury/day for an average 5-kg infant) (Wolff 1983). It is important to emphasize, however, that in general, the beneficial effects associated with breast feeding seem to override or at least compensate for any neurotoxic effects on milestone development that could be due to the presence of contaminants, such as mercury, in human milk (Egeland et al. 1997).

Hair. Scalp hair is another primary indicator used to assess methylmercury exposure, because the methylmercury is incorporated into the hair at the hair follicle in proportion to its content in the blood (EPA 1996d). The typical hair-to-blood ratio in humans has been estimated to be about 250:1 expressed as µg Hg/g hair to mg Hg/L blood, but some difficulties in measurements, inter-individual variation in body burden, differences in hair growth rates, and variations in fresh and saltwater fish intake have led to varying estimates (Birke et al. 1972; Skerfving 1974). Once incorporated into the hair strand, the methylmercury is stable and gives a longitudinal history of blood methylmercury levels (WHO 1990). Care must be exercised to ensure that the analysis of methylmercury levels in hair are not confounded by adsorption of mercury vapors or inorganic mercury onto the hair (Francis et al. 1982)

Recent reference values for mercury levels in hair from non-exposed individuals in the general U.S. population are very limited. A summary of mercury concentrations in hair from residents (adults, men, women, and children) of several U.S. communities is presented in Table 5-18. Most of the these studies, however, with the exception of Fleming et al. (1995) were conducted from 7 to 20 years ago. For populations studied in the United States, the range in mean hair concentrations was 0.47–3.8 ppm for adults (maximum value of 15.6 ppm) and 0.46–0.77 ppm for children (maximum value of 11.3 ppm). The mean concentration of mercury in hair based on a review of existing data from other countries is 2 µg/g (ppm) (WHO 1990), and the WHO advisory maximum tolerable level for hair is 6 ppm.

The concentration of total mercury in hair in the general population of Japan was determined by Nakagawa (1995). This author sampled hair from 365 healthy volunteers in Tokyo and the surrounding area from June 1992 to June 1993. The mean concentration of mercury in hair was higher in males (2.98 ppm, 81 individuals sampled) than in females (2.02 ppm, 284 individuals sampled). In both males and females, the mercury concentration in hair increased with age up to the mid-30s, then gradually declined. The authors also looked at dietary preferences and found the mean hair levels in males and females were highest in individuals that had a preference for fish (4.0 and 2.7 ppm, respectively), followed by those with a preference for fish and meat (2.88 and 2.00 ppm, respectively), a preference for meat (2.38 ppm and 1.96 ppm, respectively), and was lowest in those individuals that preferred a predominantly vegetarian diet (2.27 and 1.31 ppm, respectively). In an earlier study, the mercury content in human hair was studied in Japanese couples, with husbands having significantly higher mercury concentrations (4.01 ppm) than wives (1.99 ppm), possibly as a result of greater fish consumption among the men (Chen et al. 1990). This same pattern is also apparent for all but one of the U.S. populations (San Diego, California) studied by Airey (1983b). It is noteworthy that some of the highest mercury concentrations in hair measured in women

(15.2 ppm) were from Nome, Alaska where the population consumes large amounts of fish and marine mammals (Lasora and Citterman 1991) and from Florida (15.6 ppm), where measurements were made only in adults that consumed wildlife from the Everglades area, a region where high mercury levels in wildlife have been reported (Fleming et al. 1995). Most recently, Davidson et al. (1998) reported the results of the Seychelles Child Development Study at 66 months (5.5 years) post-parturition. These researchers reported that there were no adverse neurodevelopmental outcomes observed in mother-child pairs, with mean maternal and mean child hair total mercury concentrations of 6.8 ppm and 6.5 ppm, respectively, in the Seychelles Island study.

Oral Tissues. Mercury concentrations as high as 380 µg/g (ppm) have been found in oral tissues in contact with amalgam fillings. In individuals with more than six amalgam fillings, a mean value of 2.3 µg/g (ppm) was found in tissue without direct contact with amalgam fillings (Björkman et al. 1997). In some European countries, health authorities recommend that sensitive or susceptible individuals in higher risk groups (i.e., pregnant women and individuals with kidney disease) avoid treatment with dental amalgam (Björkman et al. 1997).

Occupational Exposure. Workplace environments presenting the largest potential sources of occupational exposure to mercury include chloralkali production facilities, cinnabar mining and processing operations, and industrial facilities involved in the manufacture and/or use of instruments containing liquid mercury (Stokinger 1981). According to NIOSH (1973), the principal route of occupational exposure to mercury is vapor phase inhalation from workplace atmospheres. Studies by Barregard et al. (1992) and by Langworth et al. (1992b) revealed increased total mercury levels in blood and urine of exposed chloralkali workers. These results are summarized in Table 5-19. Personal air sampling of workers in a mercury recycling plant in Germany showed mercury levels ranging from 115 to 454 $\mu g/m^3$ (Schaller et al. 1991).

Human tissues that are routinely monitored as evidence of exposure to mercury are urine, blood, and hair. Urine is most frequently monitored as an indicator of human body burden following chronic exposure to mercury vapor, particularly in occupational settings; approximately 95% of all urine samples contain less than 20 µg /L (ppb) (EPA 1984b). A comparison of mercury content in the urine of Swedish workers exposed to high levels of mercury, dentists, occupationally unexposed workers, and unexposed workers without dental amalgams gave values of 15, 1.7, 0.8, and 0.3 µmol/mol creatinine, respectively (corresponding mercury plasma levels were 35, 9.4, 5.3, and 2.8 nmol/L [7.19, 1.89, 1.06, and 0.56 ppt], respectively) (Molin et al. 1991). Blood and urine monitoring may be useful for groups of workers subject

to chronic exposure to mercury, but the relative contribution of recent exposures to mercury levels in these media, in comparison to releases of mercury stored in tissues as a result of earlier exposures, is not well understood (EPA 1984b) (see Section 2.5).

Mercury exposure also may result from the transport of mercury to a workers' home on contaminated clothing and shoes (ATSDR 1990; Hudson et al. 1987; Zirschky 1990). Increased exposure to mercury has been reported in children of workers who are occupationally exposed (Hudson et al. 1987). The population of children at highest risk are those whose parents work in facilities that use mercury, but where no protective uniforms or footgear are used. The mercury is thought to be transferred to the workers' homes in their clothing and shoes. While prevention of employee-transported contamination to their homes is preferred, cleaning the homes of workers occupationally exposed to mercury is also effective in reducing exposure for family members (Zirschky 1990). In an exposure study of families of workers at a chloralkali plant in Charleston, Tennessee, mercury levels in the air of the workers' homes averaged 0.92 $\mu g/m^3$ (ATSDR 1990).

The use of fluorescent tube compactors by industrial facilities may also expose those operating the compactors and workers in adjacent areas to increased levels of mercury vapor if proper filters, scrubbing devices, and ventilation are not used (Kirschner et al. 1988).

Dentists and other dental professionals may have greater exposure to mercury as a result of preparing and applying dental amalgams (Ayyadurai and Krishnashamy 1988; Skare et al. 1990). Nylander et al. (1989) sampled pituitary gland tissue from autopsies of 8 dental staff and 27 control individuals in Sweden. These authors reported median mercury concentrations of 815 µg/kg (ppb) wet weight (range, 135–4,040 µg/kg) in pituitary tissue of dental staff (7 dentists and 1 dental assistant), as compared to a median of 23 µg/kg (wet weight) in 27 individuals from the general population. None of the dental staff had been working immediately prior to their deaths, and in several cases, more than a decade had passed since the cessation of their clinical work. The number of amalgams did not correlate to pituitary gland concentrations in the controls. However, if two of the controls with the highest mercury concentrations were excluded (there was some evidence that these individuals had received occupational exposures), then the correlation was significant ($p<0.01$). In another study, Nylander and Weiner (1991) also reported high mercury concentrations in the thyroid and pituitary glands, with a median of 1.1 µmol/kg (221 ppb) wet weight (range, 0.7–28 µmol/kg [140–5,617 ppb]) in the pituitary. the median mercury concentration in the pituitary of the controls was 0.11 µmol/kg (22 ppb) (range, 0.03–5.83 µmol/kg [6–1,170 ppb]).

Naleway et al. (1991) reported results of a screening study conducted in 1985 and 1986 by the American Dental Association to analyze urinary mercury concentrations in dentists and identify those individuals with elevated urinary mercury levels. In 1985, 1,042 U.S. dentists were screened, and a mean urinary mercury level of 5.8 µg/L (ppb) (maximum 84 µg/L) was reported. In 1986, 772 dentists screened had a mean urinary level of 7.6 µg/L (ppb) (maximum 115 µg/L). Their mean urinary mercury levels were substantially lower than pooled data (mean, 14.2 µg/L) from dentists participating in the screening program from 1975–1983 (Naleway et al. 1985). The authors noted a substantial decline, particularly during the last 5 years (1982–1986), which was attributed to better mercury hygiene and the reduced use of amalgam restorations. This study also evaluated responses from a questionnaire survey of 480 dentists. The results indicated that those dentists reporting skin contact with mercury amalgam had mean urinary mercury levels of 10.4 µg/L (ppb), compared to 6.3 µg/L (ppb) in dentists reporting no skin contact; this difference was found to be statistically significant. Similarly, the mean urinary mercury level in dentists reporting mercury spills in the office was 7.8 µg/L (ppb), compared to 6.0 µg/L (ppb) for those reporting no mercury spills. Again, the difference was significant. Additionally, the number of hours practiced per week was found to weakly correlate with urinary mercury concentrations (Naleway et al. 1991).

Painters are another group that may be occupationally exposed to mercury vapors from volatilization of mercury during application of paint containing phenylmercuric acetate. Hefflin et al. (1993) studied the extent of mercury exposure from the application of exterior latex paints. These authors compared the air and urinary mercury concentrations of 13 professional male painters with those of 29 men having other occupations (nonpainters). The painters applied 2 brands of exterior latex paint that contained mercury; the median concentration was 570 mg/L (ppm). The median air mercury concentration was higher for painters (1.0 $\mu g/m^3$; range, non-detectable to 4 $\mu g/m^3$) than for nonpainters (non-detected; range, not detected to 3 $\mu g/m^3$). The median urinary mercury concentration was nearly twice as high for painters (9.7 µg/L [ppb]; range, 5.9–20.4 µg/L) as for nonpainters (5.0 µg/L [ppb]); range, 2.6–11.6 µg/L [ppb]) (p=0.0001). The normal range of urinary mercury is <20 µg/L (ppb) (EPA 1984b). Among the professional painters, urinary mercury concentrations increased with the percentage of time spent applying the exterior paint. Tichenor and Guo (1991) also studied the amount of mercury emitted from latex paints containing mercury compounds. The concentrations of mercury in the 5 types of paint tested ranged from 93 ppm to 1,060 ppm. These authors also reported that from 12 to 57% of the mercury in the paint was emitted upon application as elemental mercury, with the highest emission rate within the first few hours after paint application.

Commercial artists and crafts people are another group that is also at risk of mercury exposure from a variety of professional arts and crafts materials and techniques (Grabo 1997). This author reported that mercury was a hazard to commercial artists using mercury-based pigments in airbrush painting, brush paintings, and in pastels via pigment in chalk dusts. The author concluded that occupational health professions should be aware of toxic nature of the materials used by artists, whether they are employed in industry, self-employed, or are hobbyists.

Chemists are another group at risk of occupational exposure as a result of activities involving the synthesis of mercury compounds or the analysis of environmental or biological samples containing mercury residues. Methylmercury compounds are still used in laboratory-based research, and so the possibility of occupational exposure remains. Junghans (1983) reviewed the toxicity of methylmercury compounds associated with occupational exposures attributable to laboratory use. Most recently, a poisoning incident was reported from a single acute exposure to dimethylmercury (Blayney et al. 1997). The analytical chemist involved was exposed to approximately 0.1–0.5 mL of dimethylmercury spilled on disposable platex gloves during a transfer procedure in a fume hood, while preparing a mercury nuclear magnetic resonance standard. Blood analyses 5 months after the exposure incident revealed a whole blood mercury concentration of 4,000 µg/L (ppb), which is 80 times the usual toxic threshold (50 µg/L) and 400 times the normal mercury blood range (<10 µg/L) (Blayney et al. 1997). These authors caution that highly resistant laminate gloves should be worn under a pair of long-cuffed unsupported neoprene, nitrile, or similar heavy duty gloves rather than latex or polyvinyl chloride (PVC) gloves. Another group of analytical chemists (Toribara et al. 1997) reported that during the calibration of a mass spectrometer, an operator used a pipette with a plastic tip to transfer dimethylmercury into a Pyrex glass vial equipped with a crimp top for a Teflon-lined silicone stopper in a fume hood. After transfer, the plastic tip was disposed of in a nearby wastebasket and, in a short time, the instrument (which can detect nanogram quantities of mercury) showed measurable quantities in the workplace air around the instrument and operator. Toribara et al. (1997) also cites three other historic incidents where laboratory staff and non-laboratory staff (secretaries) working in proximity to a dimethylmercury spill were poisoned. These authors caution colleagues about the hazards involved in shipping dimethylmercury, if the packaging and container is physically damaged during transport.

The National Occupational Exposure Survey (NOES) conducted by NIOSH from 1980 to 1983 estimated that 67,551 workers, including 21,153 women in 2,877 workplaces were potentially exposed to mercury in the workplace (NIOSH 1984b). Most of these workers were employed in the health services, business services, special trade contractor, and chemical and allied products industries as chemical technicians,

science technicians, registered nurses, and machine operators. These estimates were derived from observations of the actual use of mercury (97% of total estimate) and the use of trade-name products known to contain mercury (3%). It is unknown how many of the potentially exposed workers were actually exposed. Data from the NOES conducted by NIOSH from 1983 to 1986 was broken out by exposure to a variety of mercury compounds (RTECS 1998). Estimates of the total numbers of all workers and women workers potentially exposed are presented in Table 5-20. A total of 151,947 workers were potentially exposed to mercury or various mercury compounds; 33% (50,468) of these workers were women. Table 5-21 summarizes the calculated mercury absorption from air at various occupational exposure guideline concentrations.

5.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans and briefly considers potential pre-conception exposure to germ cells. Differences from adults in susceptibility to hazardous substances are discussed in Section 2.6, Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, and breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults. The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor; they put things in their mouths; they may ingest inappropriate things such as dirt or paint chips; they spend more time outdoors. Children also are closer to the ground, and they do not have the judgement of adults in avoiding hazards (NRC 1993).

Significant health risks, including numerous neuropathological and neurobehavioral effects, are associated with prenatal exposure to methylmercury (Zelikoff et al. 1995). Fetuses and breast-fed infants may be exposed to higher than background concentrations of mercury via maternal consumption of large amounts of fish or marine mammals contaminated with mercury, via maternal exposure to mercury through dental amalgams, via maternal use of consumer products containing mercury or various mercury compounds, and via occupational exposure of the mother (Zelikoff et al. 1995). Fetuses can be exposed to mercury via exposures of their mothers either before or during pregnancy; nursing infants can be exposed via consumption of contaminated breast milk from mothers exposed via medical, domestic, or occupational

exposures (see Section 5.7). Children can be exposed to various forms of mercury in a variety of ways, including playing with unsecured elemental mercury, inhalation of mercury vapors via the religious or ethnic practices of their parents or unintentional spills of elemental mercury, oral ingestion of herbal or ethnic remedies or mercury-containing consumer products, consumption of methylmercury-contaminated fish and wildlife, and dermal or oral exposure to contaminated soils and sediments.

Mercury concentrations have been measured in cord blood in one study in the United States with levels that suggest prenatal exposure. Pitkin et al. (1976) measured concentrations of total mercury in cord blood samples from 100 maternal cord blood pairs from a population in rural Iowa. The mean cord blood total mercury concentration was 1.24 ppb, while the mean of the paired maternal blood samples was 1.01 ppb. More recently, Wheatley and Paradis (1995a, 1995b) reported on the analysis of 2,405 cord blood samples collected from Canadian aboriginal peoples over the last 20 years. Of these cord blood samples, 523 (21.8%) were found to have total mercury levels greater than 20 ppb, with the highest cord blood sample containing 224 ppb. These latter samples were from populations that routinely consumed fish and marine mammal tissues. Grandjean et al. (1997b) measured cord blood samples from 894 Faroe Islands children whose mothers consumed large amounts of fish and pilot whale meat. The methylmercury exposure in the Faroe Island population is mainly from eating pilot whale meat. The geometric mean concentration of total mercury in these cord blood samples was 22.9 ppb.

Concentrations of mercury have also been measured in breast milk from several populations in the United States as well as other countries (see Table 5-17). Breast milk concentrations have been reported for two U.S. populations; one in rural Iowa (Pitkin et al. 1976) and the other from Alaska (Galster 1976). Pitkin et al. (1976) reported a total mean mercury concentration in breast milk of 0.9±0.23 ppb (range, 0.8–1.6 ppb). The mean total mercury concentrations in the Alaskan populations were 3.3± 0.5 ppb for the urban population, 3.2±0.8 ppb for the interior population, and 7.6±2.7 ppb for the coastal population that consumed fish and marine mammals.

Total mercury concentrations in breast milk from other countries and exposure scenarios were 3.6±2.2 ppb for an urban population in Tokyo, Japan (Fujita and Takabatake 1977), 0.6±0.4 ppb for Swedish women that were fish consumers with 12 dental amalgams (Oskarsson et al. 1996), 0.2–6.3 ppb (range) for Swedish women that consumed fish (Skerfving 1988), and 9.5±5.5 ppb for an urban population of women in Madrid, Spain (Baluja et al. 1982) (Table 5-17). Some of the highest levels were reported in fish eaters, and about 20% of the total mercury content of the milk was methylmercury. The median and maximum mercury

concentrations in breast milk from women in the Faroe Islands, a population that consumes large quantities of fish and marine mammal tissue, were 2.45 and 8.7 ppb, respectively (Grandjean et al. 1995a). Breast milk mercury concentrations reported by these authors were significantly associated with mercury concentrations in cord blood and with the frequency of pilot whale dinners during pregnancy. These are relatively low values in contrast to the values reported in Minamata, Japan, for women who ate contaminated seafood in the Minamata episode, which resulted in total mercury concentrations in breast milk of 63 ppb (Fujita and Takabatake 1977), and in Iraq, where consumption of homemade bread prepared from methylmercury-contaminated wheat occurred, resulted in breast milk concentrations of up to 200 ppb (about 60%) methylmercury (Amin-Zaki et al. 1976; Bakir et al. 1973).

Children can be exposed to mercury by many of the same pathways as adults as discussed in Sections 5.4.4., 5.5, and 5.7. Children can receive mercury exposures from oral or dermal contact with mercury-contaminated soils and sediments or mercury-contaminated objects. Exposure analysis of individuals living near an abandoned mercury-contaminated industrial site suggested that children were exposed primarily via soil ingestion (Nublein et al. 1995). Little experimental information on the bioavailability of mercury via oral or dermal exposure was found relative to mercury or mercury compounds sorbed to contaminated soils and sediments (De Rosa et al. 1996). Paustenbach et al. (1997) noted that, due to the presence of mercury at a number of major contaminated sites in the United States, the bioavailability of inorganic mercury following ingestion has emerged as an important public health issue. Although precise estimates are not available, *in vivo* and *in vitro* estimates of the bioavailability of different inorganic mercury species in different matrices suggest that the bioavailability of these mercury species in soil is likely to be significantly less (on the order of 3 to 10 fold), than the bioavailability of mercuric chloride, the mercury species used to derive the toxicity criteria for inorganic mercury (Paustenbach et al. 1997). These authors suggest that site specific estimates of bioavailability be conducted of various mercury compounds because bioavailability can vary significantly with soil type, soil aging, the presence of co-contaminants and other factors. Canady et al. (1997) concluded that the “100% bioavailability assumption” for mercury-contaminated soils is excessively conservative. These authors note that various mercury compounds have distinctly different bioavailability. For example, mercuric chloride has been reported to be approximately 20–25% bioavailable in adult animals (Nielsen and Andersen 1990; Schoof and Nielsen 1997). Methylmercury is thought to be nearly completely absorbed (Aberg et al. 1969; Miettinen et al. 1971; Rice 1989a, 1989b). Mercuric nitrate was reported to be only 15% bioavailable in humans (Rahola et al. 1973) and elemental mercury is thought to be very poorly absorbed, although experimental evidence is lacking for the latter. Recently, Barnett et al. (1997) reported that analysis of mercury contaminated soil from the flood

plain of East Fork Poplar Creek in Oak Ridge, Tennessee, revealed the presence of submicron, crystalline mercuric sulfide (HgS) in the form of metacinnabar. The HgS formed in place after the deposition and burial of mercury-contaminated soils. The formation of HgS is significant for remediation efforts at the site because the toxicity, leachability, and volatility of mercury in soils are dependent on the solid phase speciation. Because local hydrogeochemical conditions are not unique, the formation of HgS at this site has implications to other environments and contaminated sites as well.

Children may be exposed to mercury vapors when they play with metallic mercury. Metallic mercury is a heavy, shiny, silver liquid and when spilled, forms little balls or beads which fascinate children. Children come in contact with metallic mercury when they trespass in abandoned warehouses, closed factories, or hazardous waste sites (ATSDR 1997; George et al. 1996). Children also have taken metallic mercury from school chemistry and physics laboratories and abandoned warehouses (ATSDR 1997). Broken thermometers and other mercury-containing instruments or equipment (fluorescent light bulbs, barometers, blood pressure measurement equipment, and light switches) used in the home and in some children's sneakers that light up are other sources of metallic mercury. Muhlendahl (1990) reported a case of chronic mercury intoxication in three children who were exposed to vapors from a broken thermometer. The maximum urinary concentrations reported by this author (8 months after the broken thermometer incident) were 250.5 µg/L for a 33-month-old girl, 266.3 µg/L for a 20-month-old girl, and 137.4 ppm for the 7-year-old brother 2 days after each patient received chelation therapy with DMPS (2,3-dimercaptopropan-1-sulphonate). Sometimes children find containers of metallic mercury which were disposed of improperly (ATSDR 1997), or adults intentionally or unintentionally bring home metallic mercury from work (Ehrenberg et al. 1991; Wendroff 1990). Metallic mercury evaporates to a greater extent as the air temperature increases; when it is not stored in a closed container, children may be exposed to mercury vapors (ATSDR 1997; Wendroff 1991).

Metallic mercury is traditionally used in some religious rituals or remedies, including religions such as Santeria (a Cuban-based religion that worships both African deities and Catholic saints), voodoo (a Haitian-based set of beliefs and secret rites), Palo Mayombe (a secret form of ancestor worship practiced mainly in the Caribbean), or Espiritismo (a spiritual belief system native to Puerto Rico) (Wendroff 1990). If these rituals or spiritual remedies containing mercury are used in the home, children may be exposed and the house may be contaminated with mercury (ATSDR 1997; Johnson [in press]; Wendroff 1990, 1991; Zayas and Ozuah 1996). Metallic mercury is sold under the name "azogue" (pronounced ah-SEW-gay) in stores (sometimes called botanicas) which specialize in religious items and ethnic remedies (Johnson [in press];

Wendroff 1990; Zayas and Ozuah 1996). Azogue may be recommended by family members, spiritualists, card readers, and santeros. Typically, azogue is carried on one's person in a sealed pouch, or it is ritually sprinkled in the home or car. Some store owners suggest mixing azogue in bath water or perfume. Some people place azogue in devotional candles. Because metallic mercury evaporates into the air, there is a potential health risk from exposure to mercury vapors in a room where the mercury is sprinkled or spilled onto the floor, put in candles, or where open containers of metallic mercury are present (ATSDR 1997; Wendroff 1990, 1991). Young children spend a lot of time crawling on the floor and carpeting, so they may be subject to a higher risk of exposure, especially when mercury is sprinkled on the floors or carpets.

Very small amounts of metallic mercury (i.e., a few drops) may raise air concentrations of mercury to levels that could be harmful to health (ATSDR 1997). Metallic mercury and its vapors are extremely difficult to remove from clothes, furniture, carpet, floors, walls, and other such items. The mercury contamination can remain for months or years, and may pose a significant health risk for people continually exposed (ATSDR 1997; Johnson [in press]; Wendroff 1990, 1991).

Another potential source of children's exposure to metallic mercury is breakage or improper disposal of a variety of household products, including thermostats, fluorescent light bulbs, barometers, glass thermometers, and some blood pressure machines that contain metallic mercury (ATSDR 1997). These devices do not pose a health threat when the mercury is properly contained within the device. Should the mercury be released, however, the potential for mercury vapors to contaminate the air increases. The appropriate method for cleaning up a spill of a small amount of mercury is to clean it up manually, *without using a vacuum cleaner*, which can cause the mercury to evaporate more rapidly into the air, creating a greater risk of exposure (ATSDR 1997; Schwartz et al. 1992; Votaw and Zey 1991). Votaw and Zey 1991 reported mean mercury concentrations in air samples collected in a dental office were 8.5 μ/m^3 when a vacuum cleaner was not in use and concentrations rose to 69 μ/m^3 when a vacuum cleaner was in use. Special techniques are often needed to prevent mercury vapor from being generated in the cleanup process (Votaw and Zey 1991). The first consideration is to remove children from the area of the spill. The beads of metallic mercury should be cleaned up by carefully rolling them onto a sheet of paper or by drawing them up into an eye dropper. After the mercury has been collected, it should be put in a plastic bag or airtight container. The piece of paper or eye dropper used to remove the mercury should also be bagged and disposed of properly, according to guidance provided by the local health department. After the mercury has been removed, the room should be ventilated to the outside and closed off to the rest of the house. Electric fans should be used for a minimum of one hour to speed the ventilation process. If larger quantities of

metallic mercury are found in a container, make sure the container is airtight and call the local health department for disposal instructions. If the container of mercury is open without a lid, a piece of plastic wrap can be used to seal the container. If the larger amount is spilled, leave the area immediately and contact the local health department or fire department. Members of the general public should seek professional guidance on proper disposal procedures of mercury (ATSDR 1997).

Metallic mercury vapors are very toxic and are virtually odorless. Inhalation of mercury-laden dust, vapor, or mist should be avoided. Metallic mercury not should not come in contact with eyes, skin, or clothing. If children are exposed directly to metallic mercury, the contaminated body area should be thoroughly washed, and contaminated clothing should be removed and disposed of in a sealed plastic bag (ATSDR 1997). ATSDR and EPA recommend very strongly against the use of any uncontained metallic (liquid) mercury in homes, automobiles, day care centers, schools, offices, and other public buildings. If a child has metallic mercury on his or her clothing, skin, or hair, the fire department should be advised and the child should be properly decontaminated (ATSDR 1997).

Some Chinese herbal remedies for stomach disorders contain mercury (probably as mercury sulfide). If these herbal remedies are made into teas and are given to children, they increase the risk of harmful effects (Espinoza et al. 1995, 1996). Some remedies are in the form of herbal balls, which are aromatic, malleable, earth-toned, roughly spherical, hand-rolled mixtures of primarily herbs and honey. These herbal balls are used as a self-medication for a wide variety of conditions, including fever, rheumatism, apoplexy, and cataracts. Herbal balls similar to those analyzed by Espinoza et al. (1995, 1996) are readily available in specialty markets throughout the United States. Ingesting two herbal balls (the recommended adult dose per day) could theoretically provide a dose of up to 1,200 mg mercury; even if the mercury is in the form of mercuric sulfide, a relatively less bioavailable form, there is an increased risk of mercury entering the body. If a pregnant woman or nursing mother uses mercury-containing herbal remedies, she may also pass the mercury to her unborn child or nursing infant via breast milk. Herbal remedies that contain mercury should be stored so that children can not reach them to prevent accidental poisoning.

Consumers should check the ingredients of any prescription or non-prescription medicine. Hoet and Lison (1997) recently reported an unusual non-occupational source of mercury exposure in a woman who used prescription nasal drops over a long period of time that contained 300 mg/L (ppm) borate phenylmercury. Prescription medicines that contain mercury should be stored out of children’s reach to avoid accidental poisoning.

Children may be exposed to mercury during play at home or in school when using art supplies that contain colors from mercury compounds. Rastogi and Pritzi (1996) reported the migration of several toxic metals including mercury from crayons and artist watercolor paints (see Section 5.4). Migration of mercury from these art supply products occurred in 57% of the samples tested. The authors believe that children might be exposed not only to mercury, but to several other metals that can migrate from the paints. Grabo (1997) also reported that artists may be exposed to mercury because it is a main component in airbrush and brush painting pigments as well as a component of pastel chalks. Artist supplies that contain mercury should be stored out of children's reach to avoid accidental poisoning.

Infants and developing fetuses may be exposed to methylmercury if their mothers consume certain methylmercury-contaminated fish, shellfish, or wildlife species from contaminated waters prior to their pregnancy, during their pregnancy, or while nursing. Older children also may be exposed to methylmercury by eating contaminated fish and wildlife species. Certain states, Native American tribes, and U.S. Territories have issued fish and wildlife advisories for mercury in fresh water, estuarine, and saltwater fish and in freshwater turtles (see Section 5.7).

In a study of lactating women, Oskarsson et al. (1996) assessed the total and inorganic mercury content in breast milk and blood in relation to fish consumption and amalgam fillings (see Section 5.5). In breast milk samples collected 6 weeks after delivery, about half of the total mercury was inorganic and half was methylmercury, whereas in blood samples only 26% was inorganic and 74% was methylmercury. Exposure of the infant to mercury from breast milk was calculated to range up to 0.3 μg/kg/day, of which approximately one-half was inorganic mercury. This exposure corresponds to approximately one-half the tolerable daily intake of total mercury for adults recommended by WHO. The authors concluded that efforts should be made to decrease total mercury burden in women of reproductive age (Oskarsson et al. 1996).

Two-year-old children seem to be different in their weight-adjusted intake of methylmercury as shown by the results of the FDA Total Diet Study. Expressed on a per weight basis, methylmercury intake for all age groups except 2-year-old children was approximately 50 ng/kg/day (Clarkson 1990; Gunderson 1988). For 2-year-old children, the intake was estimated to be approximately 100 ng/kg/day (assuming 50% of the fish intake was due to fish caught locally) or about twice as much methylmercury intake per body weight as for other age groups. For additional details, see Section 5.5, General Population and Occupational Exposure.

Parental exposure can result in subsequent exposure to the developing child or embryo. Anttila and Sallmen (1995) report some epidemiologic data suggesting that paternal exposure to mercury is associated with an increase in spontaneous abortions. These authors also report that maternal exposure to mercury has not been has not been associated with an increased risk of abortion. Lauwerys et al. (1987) reported a case of mercury poisoning in a 3-month-old infant whose mother frequently used a skin lightening cream and soap containing inorganic mercury during pregnancy and the 1-month lactation period following birth. Prenatal and early postnatal exposure of infants to mercury from maternal use of these products is a source of particular concern (Lauwerys et al. 1987).

Data from the National Occupational Exposure Survey (NOES) conducted by NIOSH from 1983 to 1986, provides information on exposure to a variety of mercury compounds, with estimates of the total numbers of workers and the total number of female workers potentially exposed. As presented in Table 5-19, an estimated 50,468 women (33% of workers) were potentially exposed to mercury and various mercury compounds in occupational settings during 1983–1986 (RTECS 1998). More current estimates are not available for the number of women occupationally exposed to mercury in the United States or the percentage of women of reproductive age that may become pregnant or may breast-feed their infants while continuing to work in these occupational settings.

Mercury exposure also may result from the transport of mercury to a workers' home on contaminated clothing and shoes (ATSDR 1990; Hudson et al. 1987; Zirschky 1990). Increased exposure to mercury has been reported in children of workers who are occupationally exposed to the compound (Hudson et al. 1987). Hudson et al. 1987 investigated the exposure to mercury of children of workers in a thermometer manufacturing plant. These investigators reported that the median mercury concentrations in the homes was 0.25 $\mu g/m^3$ (range, 0.02–10 $\mu g/m^3$), and the levels of mercury in the urine of the children averaged 25 µg/L (ppb), about five times higher than that reported for the controls. While measurements of clothing contamination were not made, the authors noted that elevated mercury concentrations were found in places where work clothes were located and in some washing machines. The children at the highest risk are those whose parents work in facilities that use mercury, but where no protective uniforms or footgear are used. The mercury from these settings is thought to be transferred to the workers' homes on their clothing and shoes. Danzinger and Possick (1973) reported that mercury particles became embedded in the clothing of workers at a scientific glassware plant, especially in knitted fabrics. In an exposure study of families of workers at a chloralkali plant in Charleston, Tennessee, mercury levels in the air of the workers' homes averaged 0.92 $\mu g/m^3$ (ATSDR 1990). Although protective clothing was used, work gloves, clothes, and

boots which were soaked with mercury were taken home, exposing family members. Cases of mine workers' homes being contaminated have also been reported, although the authors did not address the impact of this contamination on the health of the family members (West and Lim 1968). Although prevention of this kind of employee transport of mercury to homes is preferred, cleaning homes of workers occupationally exposed to mercury can be effective in reducing exposure for family members (Zirschky 1990).

5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to individuals who are occupationally exposed to mercury (Section 5.5), there are several groups within the general population with potentially high exposures (i.e., higher than background levels) to metallic mercury and various mercury compounds. Historically, populations that have been exposed to higher-than-normal background levels of mercury in the air, water, soil, and/or food have included populations near industrial discharges (e.g., Minamata and Niigata, Japan) and those who inadvertently consumed methylmercury-contaminated food (e.g., grain in Iraq) (WHO 1990, 1991). People living in proximity to former mercury production facilities or mines, secondary mercury production (recycling) facilities, chloralkali facilities, municipal and medical waste incinerators, other mercury-disposal or recycling facilities, or the 714 current or former NPL hazardous waste sites where mercury has been detected (HazDat 1998) are at risk of receiving potentially higher-than-normal background levels of exposure.

Populations with potentially high exposure include recreational and subsistence fishers and hunters, Native American populations who routinely consume larger amounts of locally caught fish than the general population or who consume marine mammals in their diet. Other populations with potential for higher than average exposures are individuals with large numbers of dental amalgams, those who use various consumer products containing mercury (i.e., skin lightening creams and soaps, ethnic remedies, or fingerpaints and make-up paints containing mercury or mercury compounds), and those living or working in buildings recently painted with mercury-containing latex paints or buildings where mercury has been intentionally or unintentionally spilled.

Individuals Living Near Mercury Production, Use, and Disposal Sites. Individuals in the general population living in the vicinity of former primary production or mining sites or current secondary production sites, chloralkali plants, pulp and paper mills, coal-fired power plants, facilities where mercury is released (e.g., municipal waste or medical waste incinerators or other waste disposal facilities), or hazardous

waste sites may be exposed to mercury through several exposure pathways, including inhalation, dermal, and oral exposures. For example, numerous studies have reported increased levels of mercury in air, water, soil, plants, and fish in areas surrounding industrial facilities involved in production or use of mercury (Harnly et al. 1997; Lodenius and Tulisalo 1984; Shaw et al. 1986; Yamaguchi et al. 1971). Significant concentrations of mercury have been detected in sewer overflows and urban runoff (Murphy and Carleo 1977). Thus, general population exposure to mercury may be higher in both industrial and urban areas. Mercury has been detected in various environmental media (air, surface water, groundwater, soil, sediment, and fish and wildlife samples) collected at some of the 714 NPL sites where it has been detected in some environmental media (HazDat 1998). Populations living near hazardous waste sites may be at risk for exposure to high levels of mercury as a result of mercury contamination of surface waters, groundwater, soils, or fish. However, the available data are insufficient to allow for the characterization of the sizes of these populations or the intake levels of mercury to which they are exposed. In 1996, however, De Rosa et al. (1996) reported than in terms of populations at risk, an estimated 41 million people in the United States live within a 4-mile radius of at least one of the 1,134 NPL sites, and 3,300 people live within a 1-mile radius of an NPL site. These authors also reported that metallic mercury was ranked third on the top 10 priority list of hazardous substances found at these NPL sites.

Adults may receive higher mercury exposures from dermal contact if they work with mercury-contaminated soils. Mercury has been detected in soil and sediment at 350 and 208 sites, respectively, of the 714 NPL sites where it has been detected in some environmental media (HazDat 1998). No experimental information on dermal exposure related to the bioavailability of mercury or mercury compounds sorbed to soils was found. However, Hursh et al. (1989) conducted a study to determine the role of dermal exposure in the uptake of mercury vapor from air. These authors estimated that during an 8-hour day, a person would absorb through the skin only 2.6% of the mercury vapor retained by the lungs exposed to the same atmosphere. These authors also noted that half of the dermal uptake is lost through normal shedding of the stratum corneum. Therefore, dermal uptake of mercury adsorbed to soil is likely to be minor compared to other exposure pathways. Recent information from Harnly et al. (1997) showed that urine mercury levels in a Native American population living near an inactive mercury mine in Clear Lake, California were comparable to background levels, indicating that soil and dust exposures were not substantially elevated in the resident population near the inactive site. However, the mean blood methylmercury level in residents of this same community that consumed fish from Clear Lake was 15.6±8.8 μg/L (ppb), which was more than 7 times higher than the mean blood level in individuals that did not consume fish from the lake (2 ppb).

In addition, adults may receive potentially higher oral exposures from ingestion of mercury-contaminated soils from their unwashed hands while working in mercury-contaminated areas. Bioavailability is an integral factor in the estimation of the internal dose (or dose at the target tissue) of the chemical. Like dermal absorption, gastrointestinal absorption of various forms of mercury is highly variable (see Section 2.3.1). The more lipid soluble organic mercury compounds (e.g., methylmercury) are almost completely absorbed, while the extremely insoluble metallic mercury is poorly absorbed through the gut. The bioavailability of mercury from soil is likely to vary, since mercury binds tightly to soil, especially to soils with high organic content. Therefore, the mercury soil concentration alone may not be indicative of the potential for human health hazard from contaminated soils, and site-specific evaluation of the bioavailability of the various forms of mercury at the site is essential. However, unless toxicokinetic studies that use soil samples from the specific site are available, it is difficult to speculate on how much mercury will be bioavailable at any particular site. Adults may also receive higher doses from routine consumption of mercury-contaminated home grown fruits and vegetables (Nublein et al. 1995), and from consumption of fish from local waters receiving runoff or leachate from a waste site. Harnly et al. (1997) studied the impact of inorganic mercury in soil and dust and organic mercury in fish on a Native American population living near an inactive mercury mine near Clear Lake, California. These authors reported average methylmercury blood levels of 15.6±7 μg/L (ppb) in individuals that consumed fish from Clear Lake, which was higher than blood levels reported for individuals that did not consume fish (2 ppb). A significant correlation of methylmercury blood levels and fish consumption was observed. Mercury has been detected in fish collected at 56 of the 714 NPL sites where it has been detected in some environmental media (HazDat 1998). Adults may also receive higher mercury exposures from routine consumption of mercury-contaminated groundwater if this is the primary drinking water supply. Mercury has been detected in groundwater samples collected at 395 of the 714 NPL sites where mercury has been detected in some environmental media (HazDat 1998).

Individuals living near municipal and medical waste incinerators, power plants fired by fossil fuels (particularly coal fired plants), or hazardous waste sites may inhale vapors or particulates contaminated with mercury from ambient outdoor air. Lipfert et al. (1996) evaluated the health risks of methylmercury from burning coal using a Monte Carlo model to simulate a “baseline” and a “worst case” scenario in which a population of 5,000 fish eaters in the upper midwestern United States derived the freshwater fish portion of their diet from local waters near a large, hypothetical coal-fired power plant. The population was characterized by distributions of body mass, half-life of methylmercury, and the ratios of blood to body burden and hair to blood methylmercury. Each person’s diet consisted of varying amounts of tuna fish,

freshwater sportfish, and marine fish and shellfish, the methylmercury content of which were characterized by national distribution statistics, as were the consumption rates for marine fish. The consumption rates for freshwater fish were specific to the region. The fish portion size was linked to body mass by a variable correlation. Each meal was assumed to be an independent sample, so that as metabolic equilibrium was approached, each person's body burden of methylmercury tended to approach the value corresponding to the mean methylmercury intake for the population. Predictions of methylmercury levels in hair by this model compared well with an observed distribution in 1,437 women. Two neurological end points were examined: adult paresthesia as related to methylmercury body burden and congenital neurological effects as associated with average concentrations of methylmercury in maternal hair during pregnancy. In the baseline exposure scenario, the source of the mercury in fish was background atmospheric deposition. In the worst-case scenario, local mercury deposition and concentrations in fish were roughly doubled to represent additional deposition from the hypothetical power plant. For both scenarios, the 99th percentile of methylmercury body burden was more than an order of magnitude below the lowest level at which increased transient paresthesia in adults was experienced in an acute methylmercury poisoning incident in Iraq. The authors concluded that neurological risks to adults from methylmercury resulting from atmospheric deposition are negligible. Based on three epidemiological studies of congenital neurological risks, they found that fetal effects appeared to be more critical, and that there is a smaller margin of safety for pregnant consumers of freshwater sportfish. However, there is still a considerable margin of safety, and uncertainties in the relationships between maternal hair mercury and actual fetal exposures may have overstated the fetal risk (Lipfert et al. 1996).

Recreational and Subsistence Fishers. Methylmercury concentrations in sport fish can be at least an order of magnitude higher than in commercial fish purchased in a supermarket (see Section 5.4.4). Therefore, recreational and subsistence fishers, including some Native American peoples who consume locally caught fish from mercury-contaminated waterbodies or consume long-lived predatory oceanic species such as shark and swordfish, can be exposed to higher mercury concentrations than individuals who consume similar amounts of commercially marketed fish from a variety of sources (Ebert et al. 1996; EPA 1995k). The exposure to mercury will also be higher among people who regularly eat fish and other seafood products, compared to those who only occasionally or never eat fish or other seafood products. This increased exposure has been demonstrated by blood mercury levels several times higher in people who regularly eat fish, compared to those who occasionally or never eat fish (Buzina et al. 1989; Cappon and Smith 1982; Oskarsson et al. 1996; Phelps et al. 1980; Svensson et al. 1995). In addition, the consumption of certain species of fish (e.g., shark and swordfish) is likely to contribute disproportionately to the observed

methylmercury body burden. Because mercury is associated primarily with muscle tissue in the body of a fish, rather than with fatty deposits, trimming and skinning of mercury-contaminated fish does not reduce the mercury content of the fillet portion, as is the case for PCBs, dioxins, and other organochlorine pesticides (Armbuster et al. 1988; Gutenmann and Lisk 1991).

Several recent studies have documented higher fish consumption rates among subsistence fishers, some of which are Native American populations. In 1990, there were an estimated 1,959,234 Native Americans in the United States, including 1,878,285 American Indians, 57,152 Eskimos, and 23,797 Aleuts (Paisano 1998). Approximately 218,320 Native Americans were living on ten reservations and tribal lands, and these people accounted for half of all Native Americans living on reservations. Therefore, approximately 440,000 Native Americans live on reservations. The median family income in 1990 for Native Americans was $21,750, about 65% of the $35,225 median income of all U.S. families. In addition 27% of all Native Americans are living in poverty, compared with 10% of the general population. In a study of 11 Alaskan communities, Nobmann et al. (1992) reported an average daily fish consumption rate of 109 g/day. This average consumption rate for subsistence fishers is more than 16.8 times the mean fish consumption rate of 6.5 g/day estimated for the general population (EPA 1995k). A recent study of fish consumption patterns among the Umatilla, Nez Perce, Yakama, and Warm Springs tribes of the Columbia River Basin in Washington and Oregon (CRITFC 1994) found that adults in these tribes consume an average of 59 g/day and that the 95th percentile of fishers consume 170 g/day of fish. The mean consumption rate for the four tribes is more than nine times the mean fish consumption rate estimated for the general population (EPA 1995k). Furthermore, the consumption rate for Native American children (5 years and younger) from these four tribes was 20 g/day (a rate over 3 times that for adults in the general population) (see Section 5.6).

In order to reduce methylmercury exposure from consumption of mercury-contaminated fish and shellfish, consumption advisories are issued by states recommending that individuals restrict their consumption of specific fish and shellfish species from certain waterbodies where mercury concentrations in fish and shellfish tissues exceed the human health level of concern. This level of concern is set by individual state agencies, but several states use the FDA action level of 1 ppm to issue advisories recommending no consumption or restricting consumption of contaminated fish and shellfish from certain waterbody types (e.g., lakes and/or rivers). The FDA value was designed to protect consumers from the health risks associated with consumption of fish and shellfish that are shipped in interstate commerce and that are purchased in commercial markets. The FDA action level was not intended to be used as a criterion for the

protection of high-end fish consumers who routinely and repeatedly consume large quantities of fish from local bodies of water.

To address this concern, the EPA Office of Water issued guidance to states on sampling and analysis procedures to use in assessing the health risks from consuming locally caught fish and shellfish. The risk assessment method proposed by EPA was designed to assist states in developing fish consumption advisories for recreational and subsistence fishers, including pregnant women, nursing mothers, and children in these high-end consumption populations (EPA 1995k). Recreational and subsistence fishers consume larger quantities of fish and shellfish than the general population and frequently fish the same waterbodies routinely. Because of this, these populations are at greater risk of exposure to mercury and other chemical contaminants, if the waters they fish are contaminated. The EPA's Office of Water advises states to use a screening value of 0.6 ppm mercury (wet weight) in fillets for the general population as a criterion to evaluate their fishable waters (EPA 1995k). Currently, 1,782 advisories restricting the consumption of mercury-contaminated fish and shellfish are in effect in 41 states and one U.S. Territory (American Samoa) (EPA 1998b). The number of mercury advisories currently in effect in each state is shown in Figure 5-7. It should be noted that mercury is the chemical pollutant responsible in part for over 77% of the fish advisories issued in the United States (EPA 1998a). It is important to note that 11 states (Connecticut, Indiana, Maine, Massachusetts, Michigan, Missouri, New Hampshire, New Jersey, North Carolina, Ohio, and Vermont) currently have state-wide mercury advisories recommending that residents restrict consumption of locally caught freshwater fish. In addition, 5 states (Alabama, Florida, Louisiana, Massachusetts, and Texas) have issued statewide coastal mercury advisories for specific marine fish and shellfish species. In two states (Arizona and Minnesota), wildlife advisories recommending that residents restrict their consumption of freshwater turtles have been issued.

Subsistence Hunters. Native American populations, such as the Inuit of Alaska and other subsistence hunters (particularly those living in high latitude areas of the United States), may be exposed to mercury in wild game (e.g., seals, narwhal, walrus, and other game species or marine mammals). Mercury has been detected in liver, kidney, and muscle tissues of pilot whales, harp seals, narwhal, and walrus (Meador et al. 1993; Wagemann et al. 1995). Mean total mercury concentrations and methylmercury concentrations were highest in pilot whale liver tissue: 176 ppm (dry weight) and 8 ppm (dry weight), respectively. In fish, almost all of the mercury (>95%) body burden is methylmercury (Bloom 1992), but in marine mammals, the percentage of inorganic mercury is much higher, at least in liver tissue. For example, in Alaskan beluga whales, mean methylmercury levels were 0.788 ppm (μg/g wet weight), but mean total mercury levels were

28 ppm (wet weight), in liver tissue (Becker et al. 1995). Similarly, in Alaskan ringed seal, mean methylmercury levels were 0.410 ppm (wet weight) and mean total mercury levels were 1.970 ppm (wet weight) in liver tissue. However, no information was available for methylmercury levels in muscle tissue from Alaskan mammals. An older report by Smith and Armstrong (1975) also examined total mercury and methylmercury levels in marine mammal livers eaten by native Inuit in the Northwest Territory of Canada. Smith and Armstrong (1975) reported total mercury concentrations of 143 and 26.2 ppm (wet weight) and mean methylmercury levels of 0.300 and 0.120 ppm (wet weight) in liver tissue of bearded seals sampled in 1973 and 1974, respectively. Smith and Armstrong (1975) also reported total mercury concentrations of 27.5 ppm (wet weight) (maximum, 184 ppm), and 0.72 ppm in liver and muscle tissue, respectively, and mean methylmercury levels of 0.96 and 0.83 ppm in liver and muscle tissue, respectively, of ringed seals sampled near Victoria Island in Canada's Northwest Territory. These authors also reported a mean total mercury concentration of 143 ppm and a mean methylmercury concentration of 0.30 ppm in liver tissue of bearded seals. The mean total mercury concentration in the muscle tissue of the bearded seals was 0.53 ppm (no methylmercury concentrations in muscle tissue were available for this species).

In Greenland, the percentage of total mercury that was methylmercury in seal muscle tissue was 57–86%; however, the concentration of total mercury was very low. Mercury concentrations in the blood of mothers and infants in Greenland were closely correlated with the amount of marine mammal meat the mothers consumed. Mercury concentrations in the blood of mothers eating primarily imported food ranged from 11.0 to 32.7 μg/L (ppb) and concentrations in the blood of their children ranged from 15.0 to 51.4 μg/L (ppb). In contrast, mercury concentrations in the blood of mothers who consumed primarily a local diet heavy in marine animals ranged from 16.4 to 44.6 μg/L (ppb) and concentrations in the blood of their children ranged from 27.5 to 140.0 μg/L (ppb) (Hansen 1991).

Native American populations that depend heavily on marine mammals are considered to be at higher risk than the general population. Wheatley and Paradis (1995a, 1995b) reported blood mercury levels in native peoples from 514 communities across Canada. Of these individuals, 23% had methylmercury blood levels >20 μg/L (the WHO assessment level), while 1.6% of these individuals had blood levels >100 μg/L (the WHO benchmark for at-risk populations). Native American populations in the western Arctic (Alaska) may be at similar risk as a result of their consumption of marine mammals, although no recent information on methylmercury concentrations in blood, hair or urine for these populations was located. In Alaskan Inuit women that consume marine mammal tissue, Galster (1976) reported higher total mercury levels in breast

milk of women living in coastal areas (7.6±2.7 ppb) than in breast milk of Inuit women living in the interior (3.2±0.8 ppb) or in urban areas (3.3±0.5 ppb). In addition, mercury red blood cell concentrations were also higher in Inuit women living in coastal areas (33.5±5.1 ppb), as compared to those living in the interior (22.6±3.0 ppb) or in urban areas (8.9±0.9 ppb). Higher mercury levels in coastal populations were attributed to higher consumption of seal meat and oil and marine fish (Galster 1976). By analogy to the Canadian populations of native peoples (Wheatley and Paradis 1995a, 1995b), it is anticipated that methylmercury concentrations in these tissues are likely to be higher among individuals who consume large quantities of marine mammal species with high concentrations of methylmercury (as well as inorganic mercury) in their tissues than among members of the general population. In a study of subsistence economies in the State of Alaska, Wolfe and Walker (1987) reported that total annual per capita harvest of wild game species (including land mammals, marine mammals, and fish) ranged from 10 to 1,498 pounds (median harvest of 252 pounds), compared to 222 pounds of meat, fish, and poultry (combined) consumed each year per individual in the western United States. The wild game harvest in 84% of the 98 Alaskan subsistence communities surveyed was at least half or greater than the 222 pounds consumed in the western United States. Because hunters often share wild game they harvest with other family members, the amount harvested may not represent the actual amount consumed (Egeland et al. 1998). The average daily per capita consumption was estimated to be 0.67 pounds of fish and 0.23 pounds of land mammals based on all 98 communities, and 0.2 pounds of marine mammals based on the 41 coastal communities surveyed. Marine mammals consumed in these communities included seal, walrus, and whales. Subsistence hunters and their families are a population at potentially higher risk of mercury exposure, if the wild game species they consume are contaminated with high concentrations of inorganic and methylmercury. Although the existence of larger amounts of mercury in subsistence diets does give cause for concern, the available Alaskan data do not support the conclusion that current exposures are a serious problem for Alaskan subsistence hunters (Egeland et al. 1998).

Individuals with Large Numbers of Dental Amalgams. Individuals with dental amalgams have greater exposure to elemental mercury than members of the general population that do not have dental amalgams. Richardson (1995) computed a release rate per filled tooth surface of 0.73 μg/day-surface, with a standard deviation of 0.3 μg/day-surface and a “stimulation magnification factor” of 5.3, based on a weighed average enhancement of mercury vapor concentration following chewing, eating, or tooth brushing

reported in three amalgam studies. Patterson et al. (1985) measured elemental mercury in exhaled breath, and levels of mercury ranging from 0.0001 to 62 ng/L (ppb) (mean, 0.0082 µg/L [ppb]) were detected in 167 persons with dental restorations, compared to 0.000008–0.0001 µg/L (ppb) (mean, 0.00006 µg/L [ppb]) in 5 persons with no amalgams; however, these values were measured after the people had brushed their teeth. Jokstad et al. 1992 reported that mercury urine concentrations increased with increasing number of amalgams. Individuals with 36 to 39 dental amalgams had mercury urine levels of 6 ppb compared to 1.2 ppb in individuals without amalgams. Mercury concentrations in whole blood were also higher in persons who ate no fish, but had >6 dental amalgam fillings (mean, 1.047±0.797 µg/L [ppb] as compared to persons who did not eat fish and had no dental amalgams (0.2±0.4 µg/L [ppb]) (Schweinberg 1994). Individuals who have large numbers of dental amalgams installed or replaced at one time are likely to exhibit transient elevated blood and urine mercury levels (PHS 1995).

Individuals Exposed to Consumer Products and Medicinal Products Containing Mercury. Individual who use various consumer products containing mercury (i.e., medicinal herbal remedies, skin lightening creams and soaps, laxatives, tattoo dyes, fingerpaints, and make-up paints) are also exposed to higher mercury levels than the general population (Barr et al. 1973; Dyall-Smith and Scurry 1990; Espinoza et al. 1995; Geffner and Sandler 1980; Lauwerys et al. 1987; Rastogi 1992; Wendroff 1990). Metallic mercury has been used by Mexican American and Asian populations in traditional remedies for a variety of medical conditions, including chronic stomach disorders. Several papers have been published related to the use of metallic mercury as a folk remedy (ATSDR 1992, 1997; Department of Health 1997; Geffner and Sandler 1980; Hartman 1995; Johnson [in press]; Trotter 1985; Wendroff 1990, 1991; Zayas and Ozuah 1996). Some Mexican-Americans believe that disorders of the alimentary tract may be caused by a bolus of food adhering to the stomach wall, a condition known as *empacho*. Geffner and Sandler (1980) reported cases of two young patients with acute gastroenteritis who received traditional remedies of oral administration of metallic mercury, presumably to dislodge the bolus. Both patients were successfully treated and released from the hospital after 2 and 10 days of treatment, respectively. Trotter (1985) reported that metallic mercury known as *azogue* is in common use in New Mexico and the bordering areas for treating this gastrointestinal condition, *empacho*. Metallic mercury was also implicated in two cases of mercury poisoning caused by the dermal application of an over-the-counter antilice product (Bourgeois et al. 1986). Wands et al. (1974) reported the deaths of two individuals due to the excessive use of a laxative

preparation containing mercurous chloride (calomel). Espinoza et al. (1995) reported that while examining imported Chinese herbal balls for the presence of products from endangered species, the authors detected potentially toxic levels of mercury and arsenic in certain herbal ball preparations. Herbal balls are aromatic, malleable, earth-toned, roughly spherical, hand-rolled mixtures of primarily herbs and honey. These herbal balls are used as a self-medication for a wide variety of conditions, including fever, rheumatism, apoplexy, and cataracts. Herbal balls similar to those analyzed are readily available in specialty markets throughout the United States. Mercury (probably mercury sulfide) was detected in 8 of the 9 herbal balls tested. The recommended adult dose for the herbal balls is two per day. Ingesting two herbal balls could theoretically provide a dose of up to 1,200 mg of mercury. Perharic et al. (1994) reported poisonings resulting from exposure to traditional remedies and food supplements reported to the National Poisons Unit in London, England. From 1989 to 1991, metallic mercury was implicated in several poisonings following exposure to Asian medicines. The issuance of informational notices by health departments cautioning members of these subpopulation about the toxic properties of mercury may be appropriate.

Mercuric sulfide, or cinnabar, was reported to be used in tattooing dyes to produce a red pigmentation (Bagley et al. 1987; Biro and Klein 1967). An analysis of finger paints and make-up paints manufactured in Europe showed that they all contained less than 1 ppm mercury (Rastogi 1992). The author did not discuss whether these products are available in the United States. While some of medicinal and pharmaceutical uses of mercury compounds have been replaced in recent years, individuals in some ethnic or religious groups may still use mercury in various traditional remedies, ceremonies, and rituals.

Individuals that Use Mercury in Religious Ceremonies and/or Ethnic Practices or Live in Dwellings where Intentional or Unintentional Elemental Mercury Spills have Occurred.

Metallic mercury has been used in Latin American and Caribbean communities as part of certain religious practices (e.g., Voodoo, Santeria, and Espiritismo) predominantly in domestic settings (Wendroff 1990). Metallic mercury is sold in shops called botanicas (sometimes under the name *azogue)* which stock medicinal plants, magical medicines, incense, candles, and perfumes. Botanicas typically dispense mercury in gelatin capsules or, sometimes, in small glass vials. Some practices involve sprinkling metallic mercury on the floor of the dwelling or of a car, mixing elemental mercury with soap and water to wash the floor, or placing it in an open container to rid the house of evil spirits. Other practices involve carrying a small amount of mercury in a vial on the person or mixing mercury in bath water or perfumed soaps, devotional

candles, ammonia, or camphor. Any of these practices can liberate mercury vapor into the room air exposing the occupants to unnecessarily elevated levels of mercury vapors (ATSDR 1997; Wendroff 1990, 1991). The issuance of cautionary notices by health departments to members of these user populations may be appropriate. While some medicinal and pharmaceutical uses of mercury compounds have been replaced in recent years, individuals in some religious and ethnic groups may still use mercury in various rituals. This use of mercury can contaminate the dwelling if the mercury is not removed from flooring, carpeting, and woodwork in an appropriate manner.

Individuals Living in Homes Where Mercury-containing Latex Paints Have Been Used. Prior to 1991, phenylmercuric compounds were used as biocides in 25–30% of interior and exterior latex paints; however, this use of mercury was voluntarily discontinued for interior paint in 1990 and for exterior paint in 1991 (Hefflin et al. 1993; Reese 1990). This use of phenylmercury resulted in the exposure of house painters and residents to elemental mercury vapors in homes where interior or exterior latex paint was applied. The concentration of mercury in interior paints was less than 200 ppm; however, the atmospheric concentrations of elemental mercury vapor were found to be as high as 200 $\mu g/m^3$ less than 6 hours after painting, 10 $\mu g/m^3$ at 24 hours, and 6 $\mu g/m^3$ after 1 month. Although the use of mercury biocides in latex paint has been discontinued, it is possible that people who use old latex paint in their homes will be exposed to mercury for a considerable time (Blondell and Knott 1993). Furthermore, although phenylmercury use in exterior latex paints was discontinued in 1991, paint companies were allowed to continue to produce and sell paint containing phenylmercury until the existing stocks of phenylmercury were exhausted. Paint produced after 1990 containing phenylmercury must be so labeled. Exterior latex paints may have contained phenylmercury at concentrations of up to 1,500 ppm, and their use has been shown to result in elevated mercury levels in painters (see Section 5.5) (Hefflin et al. 1993). However, each year many homeowners (66%) repaint their own homes, rather than employing professional painters; therefore, these individuals may also be exposed (Hefflin et al. 1993). In addition, consumers can mistakenly use exterior paints indoors, which may produce higher exposures to mercury than when the paints are used outdoors. Blondell and Knott (1993) estimated that approximately 13 million people could be exposed to mercury through painting, assuming the interior of houses were painted once every 5 years, that 78% of the interior paint used is latex, and that one-third of the interior latex paint contained mercury. These authors emphasize that key populations at risk include the painters, residents in the painted homes and children living in those homes.

5.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of mercury is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of mercury.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

5.8.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of metallic mercury and its inorganic and organic compounds have been well characterized to permit estimation of their environmental fate (Lewis 1993; Merck 1989; NFPA 1994; Osol 1980; Spencer and Voigt 1968; Verschueren 1983; Weast 1988; Weiss 1986). Most values are available for the log K_{ow}, log K_{oc}, Henry's law constant, vapor pressure, and solubility in water. Experimental data exist that allow characterization of the environmental fate of metallic mercury and inorganic and organic mercury compounds in a variety of environmental media.

Production, Import/Export, Use, Release, and Disposal. Information on mercury production, import/export, and use are well documented (Blayney et al. 1997; Drake 1981; EPA 1997a; Hefflin et al. 1993; IARC 1993; Jasinski 1993; Reese 1990; Reiber and Harris 1994; Toribara et al. 1997; USGS 1997).

Information on disposal methods and recycling of mercury and mercury containing wastes are available (Carrico 1985; DOI 1989; Jasinski 1993; TRI96 1998).

One area that requires additional study is the use of elemental mercury by members of specific religious or ethnic groups in their ceremonies, rituals, and practices so an assessment of the magnitude of these activities can be made. In addition, information on how mercury is used in these ceremonies and rituals, as well as the methods of mercury disposal used, would be helpful in assessing the potential pathways for human exposure and environmental releases.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI), which contains this information for 1996, became available in May 1998. This database will be updated yearly and should provide a list of industrial production facilities and emissions.

Environmental Fate. Mercury released to the atmosphere may be transported long distances before being removed by wet or dry deposition. Residence time in the atmosphere has been estimated to range from 60–90 days to 0.3–2 years (EPA 1984b; Glass et al. 1991). Volatile forms of mercury released in water or soil can enter the atmosphere, but most mercury is adsorbed to soil and sediment (EPA 1984b; Meili et al. 1991). Sorbed mercury may be reduced to elemental mercury or bioconverted to volatile organic forms (EPA 1984b). The major transport and transformation processes involved in the environmental fate of mercury have been fairly well defined; the most important fate process for human exposure, bioaccumulation of methylmercury in aquatic food chains is also well defined (Callahan et al. 1979; EPA 1984b; Stein et al. 1996). Additional information on mercury transport and flux in waterbodies would be helpful.

Bioavailability from Environmental Media. Metallic mercury vapors in the air are readily absorbed through the lungs following inhalation exposure, while inorganic and organic mercury compounds are poorly absorbed via this route (Berlin et al. 1969). Gastrointestinal (GI) absorption of methylmercury is nearly complete, while GI absorption of inorganic mercury is low (typically <10%) (Clarkson 1989; Friberg and Nordberg 1973). Metallic mercury vapor can be absorbed following dermal exposure; however, dermal absorption of the vapor accounts for a much smaller percentage (2.6% of the total absorbed through the lungs) than absorption through the inhalation route (Hursh et al. 1989). Inorganic mercury salts and organomercury compounds can also be dermally absorbed to some extent (Blayney et al. 1997; Junghaus

1983; Schamberg et al. 1918; Toribara et al. 1997). Data are needed regarding the bioavailability of elemental, inorganic, and organic mercury forms from contaminated surface water, groundwater, soil, or plant material. Data are also needed regarding the bioavailability of mercuric chloride in air because of the possibility of inhalation of volatilized mercuric chloride near emission sources. Additional data on the bioavailability of elemental mercury, inorganic mercury compounds, and organic mercury compounds (specifically, methylmercury) in soil would also be useful in assessing the risks from dermal and oral exposures at mining, industrial, or hazardous waste sites.

Food Chain Bioaccumulation. Mercury is known to bioconcentrate in aquatic organisms and biomagnify in aquatic food chains (ASTER 1997; EPA 1984b; Jackson 1991; Kohler et al. 1990; Mason et al. 1995, 1996; Porcella 1994; Watras and Bloom 1992). While bioconcentration in the aquatic food chain is well studied, little is known about the bioaccumulation potential for terrestrial food chains, although it appears to be smaller than in aquatic systems (Lindqvist 1991a). Additional information on the potential for terrestrial food chain biomagnification would be useful in light of the binding of mercury to organic matter in soils and sediment. Information on foliar uptake of mercury and of plant/mercury chemistry is needed to determine whether plants convert elemental or divalent mercury into other forms of mercury that are more readily bioaccumulated and whether plants are able to emit these different forms to the air. Additional information is also needed to improve biotransfer factors for mercury from soil to plants to animals.

Exposure Levels in Environmental Media. Environmental monitoring data are available for mercury in ambient air, surface water, groundwater, drinking water, soils, sediments, and foodstuffs (EPA 1984b, 1985; Glass et al. 1990; Lindqvist 1994); however, additional monitoring data on mercury levels in all environmental media, particularly drinking water, would be helpful in determining current exposure levels. Estimates of human intake from inhalation of ambient air and ingestion of contaminated foods and drinking water are available (Burger et al. 1992), although the estimates may be based on specific intake scenarios (e.g., information is most extensive for fish and other seafood products). Better estimates of fish consumption rates for high-end consumers (subsistence fishers) and recreational fishers is needed, as is information on fish-specific consumption rates by these populations. Additional information on the levels of mercury in foods other than fish and seafood would be very useful in determining total dietary intakes. Additional research is needed to characterize mercury exposures via consumption of marine mammal species. Available data indicate that the ratio of methylmercury to total mercury varies within tissues, and that only a small portion of mercury is methylated in the marine mammal's liver. Also, other trace metal constituents of marine mammal tissues such as selenium, cadmium, and other metals may interact with and

influence the bioavailability of mercury. Additional studies are needed in order to understand why the relatively high concentrations of mercury measured in marine mammal tissues do not appear to result in elevation of hair mercury levels among Alaskan natives that consume marine mammal tissues.

Reliable monitoring data for the levels of mercury in contaminated media at hazardous waste sites are needed so that the information obtained on levels of mercury in the environment can be used in combination with the known body burden of mercury to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Exposure Levels in Humans. Mercury has been measured in human blood, hair, breast milk, urine, feces, and saliva (Bakir et al. 1973; EPA 1984b; Fujita and Takabatake 1977; Galster 1976; Oskarsson et al. 1996; Pitkin et al. 1976; Wheatley and Paradis 1995a, 1995b; WHO 1990). However, current information on mercury levels in blood, hair, breast milk, and urine of members of the general U.S. population are almost entirely lacking. Data are needed for the general population that measure the levels of mercury in blood, hair, breast milk, and urine derived from dietary exposures (such as fish consumption) versus mercury derived from dental amalgams in order to obtain additional information about the importance of each of these exposure pathways to resulting mercury body burden. Additional information on mercury levels in urine of persons with varying numbers of amalgam surfaces as well as in persons that have had amalgam fillings removed or replaced would be useful in evaluating mercury exposure form this source. Data are available for some Native American populations (Galster 1976) and several foreign populations that consume large amounts of locally caught fish and wildlife (Airey 1983b; Fleming et al. 1995; Lasora and Citterman 1991). The most common method of assessing human exposure in the workplace involves the measurement of mercury in urine (Baser and Marion 1990; Bell et al. 1973; Lindstedt et al. 1979; Roels et al. 1987; Rosenman et al. 1986). Urine mercury levels have been correlated with ambient air exposure levels, particularly to mercury vapor. A longitudinal epidemiological study that tracks individual exposure levels to metallic mercury vapors in occupational settings (chloralkali industry workers, fluorescent lightbulb manufacturers, or other mercury utilizing industries) on a daily basis and associated these exposure levels with weekly urine and blood samples for a period of 1–2 years is needed. Neurobehavioral testing should also be conducted of these workers at 6-month intervals. Workers new to these industries would make the best subjects since they could provide pre-exposure blood and urine levels as a point of reference. Information is available on populations living near former production sites or hazardous waste sites (Harnly et al. 1997; Nublein et al. 1995; Reif et al. 1993; Shaw et al. 1986). Additional information on the biological monitoring of populations living in the vicinity of hazardous waste

sites would be helpful in estimating exposure of these populations to mercury compounds. This information is useful for assessing the need to conduct health studies on these populations.

Exposures of Children. Children are exposed to mercury by a variety of exposure pathways depending on their age. The most important pathways appear to be via inhalation of metallic mercury vapors, intake of inorganic mercury associated with dental amalgams in children up to 18 years old, and ingestion of methylmercury in foods primarily fish and shellfish. These are the same important pathways of exposure for adults as well. Infants can also be exposed to mercury from mother's milk. More data are needed on the levels of mercury exposure in nursing women from inhalation of metallic mercury in occupational or domestic situations, including religious and ethnic uses (ATSDR 1997; Johnson [in press]; Wendroff 1990, 1991; Zayas and Ozuah 1996); from use of commercial or hobby arts and crafts (Grabo 1997; Rastogi and Pritzi 1996); from mercury-containing herbal remedies, cosmetics, and prescription drugs (Al-Saleh and Al-Doush 1997; Barr et al. 1973; Dyall-Smith and Scurry 1990; Espinoza 1995, 1996; Lauwerys et al. 1987; Perharic et al. 1994); and from consumption of mercury-contaminated fish and wildlife, including marine mammals (CRITFC 1994; Egeland et al. 1998; Oskarsson et al. 1996). Exposure and body burden studies especially related to consumption of freshwater fish in the U.S. populations are needed to determine exposure levels, particularly in the children of recreational and subsistence fishers. Individual members of freshwater sport fish species in the Northeastern United States have been found to have tissue concentrations as high as 8.94 ppm mercury, while some species have mean tissue concentrations as high as 0.77 ppm (NESCAUM 1998). Exposure and body burden studies are also needed in Alaskan populations of subsistence hunters that consume large amounts of marine mammal tissues. Existing data on levels of mercury in breast milk in Alaskan women (Galster 1976) are dated and may not reflect either current levels of mercury contamination in fish and wildlife or dietary habits of Inuit or other subsistence fishing/hunting populations.

A unique exposure pathway that has received little research attention is the exposure to children from religious and ethnic uses in homes and cars or in remedies containing metallic mercury (ATSDR 1997; Johnson [in press]; Wendroff 1990, 1991). In some religious practices of Latin American or Caribbean origin, there are traditional rituals or remedies that involve mercury. These include intentional sprinkling of liquid elemental mercury on the floor, burning candles made with mercury, using mercury in baths, adding it to perfume, or wearing small containers of mercury around the neck for good luck. There is an urgent need to obtain information on the levels of exposure from these practices to determine if children or adults are at risk. Mercury vapor concentrations may be much higher after use during the winter months when the heat is

turned on and the windows are closed, so data that reflect a variety of possible exposure scenarios are also needed.

Results of the Total Diet Study conducted by the FDA suggest that two-year-old children differ in their weight-adjusted intake of mercury, based on the assumption that 50% of the fish consumed were locally caught species (Clarkson 1990; Gunderson 1988). Additional information on weight adjusted intakes would be helpful for the general population, and particularly in determining the health risks for young children in Native American populations. Children in these populations may consume relatively large quantities of locally caught fish as part of their traditional ceremonial practices (CRITFC 1994) or may consume large quantities of marine mammal tissues (blubber, muscle, and organ meats) if they are in subsistence fishing or hunting populations.

One childhood-specific means of decreasing exposure scenarios for children is through better education of school age children and their parents on the health risks particularly of metallic mercury exposure from accidental spillage, intentional uses, or from improper industrial exposures.

Exposure Registries. New York State has instituted a Heavy Metals Registry that monitors occupational exposure to heavy metals, including mercury. Cases are reported when mercury exposure is equal to or exceeds 50 µg/L (ppb) in blood or 20 µg/L (ppb) in urine. Between 1982 and 1986, 1,000 cases of mercury exposure were reported and linked to 47 companies. Most exposures (494 cases) occurred in workers in the alkali and chlorine industry, where mercury is used as a cathode because exposure occurs when the cells are opened; the median blood mercury concentration was 76 µg/L (ppb) (maximum concentration 916 µg/L [ppb]). The second most frequent exposure category (213 cases) was the manufacture of industrial instruments, such as the manual assembly and fabrication of thermometers; median blood mercury concentration was 145 µg/L (ppb) and the maximum concentration was 889 µg/L (ppb) (Baser and Marion 1990).

This substance is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to this substance.

5.8.2 Ongoing Studies

A search of Federal Research in Progress (FEDRIP 1998) identified numerous research studies that are currently being conducted that may fill some of the data needs discussed in Section 5.8.1. Ongoing studies and long-term research concerning occupational or general population exposures to mercury and studies that address the issue of the religious and ethnic uses of elemental mercury are presented in Table 5-22.

6. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting, and/or measuring, and/or monitoring mercury, its metabolites, and other biomarkers of exposure to and effects of mercury. The intent is not to provide an exhaustive list of analytical methods. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used for environmental samples are the methods approved by federal agencies and organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that modify previously used methods to obtain lower detection limits, and/or to improve accuracy and precision.

The analysis of metals in biological and environmental samples is complicated by the different organic and inorganic forms of the metal that may be present. For mercury, this complication is usually overcome by reducing all the mercury in the sample to its elemental state prior to analysis; this solution is not appropriate when information about the individual mercury species is desired. Mercury has an additional problem of being relatively volatile and, therefore, easily lost during sample preparation and analysis. In spite of these complications, several methods have been developed for determining trace amounts of mercury in biological and environmental samples, even in complex media. Careful attention must be paid to inadvertent contamination of the sample with mercury, especially when determining trace concentrations. Labware (glass or Teflon) should be thoroughly cleaned and acid-leached before being used for trace-level analysis. It has been shown that final soaking of laboratory ware, particularly Teflon, in hot (70 EC) 1% HCL removes any traces of oxidizing compounds (e.g., chlorine) that may subsequently destroy methylmercury in solution (Horvat 1996). Appropriate method blanks must be included.

Attention must be paid also to sample preservation to avoid perturbing the distribution of mercury compounds in the sample (Horvat 1996). The preservation of aqueous samples is often accomplished using acidification. However, suspended matter must be removed prior to acidification and dimethylmercury and Hg(0) have to be removed or else conversion of these species into methylmercury and mercury(II) can occur (Horvat 1996). For solid matrices, the preservation method of choice is freezing (Bloom 1993). Freezing preserves all major mercury species indefinitely, although coagulation will occur for sediments thus making it difficult to obtain representative subsamples of the sediment for analysis. For most metals, such storage

issues would be solved by drying the samples first, but for mercury, especially methylmercury, there is a risk of losses from volatilization. Tissue samples may be freeze-dried without loss of methylmercury. Repeated freezing and thawing of wet, biological samples can also cause loss of methylmercury (Horvat and Byrne 1992) but such degradations are dependant on the matrix.

Numerous standard or certified reference materials exist for verifying the reliability of new or modified methods, especially for total mercury; standard reference materials for individual organomercury species can be more difficult to obtain. The existing methods for determining mercury in biological and environmental matrices are described more fully in the following sections.

6.1 BIOLOGICAL MATERIALS

Many researchers have attempted to determine mercury levels in the blood, urine, tissues, and hair of humans and animals. Most methods have used atomic absorption spectrometry (AAS), atomic fluorescence spectrometry (AFS), or neutron activation analysis (NAA). In addition, methods based on mass spectrometry (MS), spectrophotometry, and anodic stripping voltametry (ASV) have also been tested. Of the available methods, cold vapor (CV) AAS is the most widely used. In most methods, mercury in the sample is reduced to the elemental state. Some methods require predigestion of the sample prior to reduction. At all phases of sample preparation and analysis, the possibility of contamination from mercury found naturally in the environment must be considered. Rigorous standards to prevent mercury contamination must be followed. Table 6-1 presents details of selected methods used to determine mercury in biological samples. Methods have been developed for the analysis of mercury in breath samples. These are based on AAS with either flameless (NIOSH 1994) or cold vapor release of the sample to the detection chamber (Rathje et al. 1974). Flameless AAS is the NIOSH-recommended method of determining levels of mercury in expired air (NIOSH 1994). No other current methods for analyzing breath were located.

In recent years, increasing attention has been paid to human exposure to mercury via dental amalgams (Skare 1995). Exposure results from elemental mercury vapor released from amalgams that is either inhaled directly or swallowed after dissolution in saliva. A Jerome 511 Gold Film Mercury Vapor Analyzer (Arizona Instrument Corp., Jerome, AZ) has been used to measure mercury vapor released from amalgam during routine dental procedures (Engle et al. 1992) or at other times to establish baseline exposure data (Halbach 1995). Accuracy and precision data were not reported. Although the detection limit for this method was not reported, mercury concentrations at μg concentrations are detectable. A similar instrument

(Jerome 431X Mercury Vapor Analyzer) was used by Chien et al. (1996) to measure elemental mercury vapor released from dental amalgams in the oral cavity and was reported to have a sensitivity of 0.003 mg/m^3. Absorbed mercury can be measured using blood and urine measurements as described below.

CVAAS is the primary method that is used to determine mercury in blood and serum (Friese et al. 1990; Ngim et al. 1988; Vermeir et al. 1988, 1989; Vesterberg 1991). Using CVAAS, concentrations in the sub- to low-ppb can be reliably measured. Both direct reduction of sample (Friese et al. 1990; Ngim et al. 1988) and predigestion followed by reduction (Oskarsson et al. 1996; Vermeir et al. 1988, 1989) produced good accuracy and precision. However, with predigestion techniques, best results were obtained on samples that were heated in a closed teflon container in a microwave oven and preconcentrated on gold-coated sand (Vermeir et al. 1989). A complimentary method to CVAAS for total mercury determination in blood is electrothermal atomic absorption (ETAAS) (Emteborg et al. 1992). Recoveries are excellent and sensitivity is 2 µg/dm^3. GC/microwave-induced plasma atomic emission detection (MPD) can also be used to measure both organic and inorganic mercury in blood samples (Bulska et al. 1992). Sensitivity is in the sub-ppb range, and recovery is excellent (100%).

Methylmercury and inorganic mercury were extracted from human whole blood samples, as their diethyldithiocarbamate complexes, into toluene and butylated them by using a Grignard Reagent (Bulska et al. 1992). The mercury species were then detected by a microwave-induced plasma atomic emission spectrometric system (GC/MPD). The absolute detection limit was calculated to be 1 pg of mercury in either the methylmercury or inorganic mercury form. This corresponds to a detection limit of about 0.4 µg/L. The method is reproducible. Methods for inorganic mercury and organic mercury (mostly methylmercury) have been reported for blood, urine, hair, and breast milk (Akagi et al. 1995; Bergdahl et al. 1995; Oskarsson et al. 1996). Total mercury is typically determined using CVAAS after complete conversion of all mercury to the volatile elemental form using harsh (nitric acid/perchloric acid, bromate/bromide) digestions followed by reduction of ionic mercury to the elemental form. Inorganic mercury can be determined after milder digestions (HCl, sulfuric acid) and reduction. The organic form is determined by the difference between total and inorganic. Sub-ng/g (ppb) detection limits are routine. Methylmercury is also determined using GC with electron capture detection (ECD) (Akagi et al. 1995).

There is evidence to suggest that urinary mercury levels are good measures of exposure to inorganic mercury in the environment (Ikingura and Akagi 1996). The primary method used to analyze urine for mercury is CVAAS (Akagi et al. 1995; Friese et al. 1990; Ngim et al. 1988; Oskarsson et al. 1996; Ping

and Dasgupta 1989, 1990; Vesterberg 1991). Methods using AFS (Corns et al. 1994; Vermeir et al. 1991a, 1991b), ASV (Liu et al. 1990), and isotope-dilution spark source (IDSS) MS have also been developed. CVAAS is sensitive (low-ppt), reliable (recovery is >76% and precision is generally <10% relative standard deviation [RSD]), and may be used on either digested or undigested samples (Friese et al. 1990; Ngim et al. 1988; Ping and Dasgupta 1989, 1990). Improved sensitivity (sub-ppt), accuracy (>90% recovery), and precision (7% RSD or better) were obtained with AFS when samples were digested in a closed container in a microwave (Vermeir et al. 1991a, 1991b). Good results have also been achieved with ASV (Liu et al. 1990) and IDSSMS (Moody and Paulsen 1988). The precision of these methods is especially high (<5% RSD), and recoveries with ASV are >90%. Both these methods require predigestion of the sample. As an alternative to CVAAS, total mercury determination in blood and urine can be performed by inductively coupled plasma-atomic emission spectroscopy (ICP-AES) or ICP-mass spectrometry (Buneaux et al. 1992; Kalamegham and Ash 1992). These methods are sensitive, with detection limits in the sub-ppb range. Recoveries (>90%) and precision (<17% coefficient of variation [CV]) are good.

AAS-based methods and NAA have been used to measure mercury in tissues. The AAS methods differ in the way the sample is released for detection. CVAAS is the best-defined of the AAS techniques. Mercury concentrations in the sub- to low-ppb have been reliably determined in tissue samples (Friese et al. 1990; Vermeir et al. 1988, 1989). Best results were obtained when the sample was digested in a closed container in a microwave oven, and the vaporized mercury was preconcentrated on gold-coated sand (Vermeir et al. 1989). Flameless AAS, which uses an electric furnace to atomize the mercury, has yielded high recoveries, but no data are available on the sensitivity or precision of the technique (Ichinose and Miyazawa 1989). Separative column atomizer AAS (SCA-AAS) introduces the mercury to the detector by running the sample through a heat-activated charcoal column (Yanagisawa et al. 1989). Little sample preparation is required, but high background interference is a problem with this method. Good results were reported for tissue samples with sub-ppm mercury concentrations (from control rats), but decreased accuracy and precision occurred in samples containing higher levels (from dosed rats). AFS offers a good alternative to CVAAS. Sensitivity was in the sub-ppt range, and recovery and precision were excellent (Vermeir et al. 1991a, 1991b). In addition, sample preparation is relatively simple and rapid. NAA permits determination of mercury in tissue samples at the sub- to low-ppb level, but erratic accuracy and precision make the method less reliable (Taskaev et al. 1988; Zhuang et al. 1989). An extraction method using zinc diethyldithiocarbamate produced good results with NAA (Zhuang et al. 1989). GC equipped with a flame ionization detector (FID) has also been used to detect methylmercury in tissues at ng levels (Baldi and Filippelli 1991). Recovery and precision data were not reported.

Studies have indicated that the mercury concentration in the hair correlates well with dietary mercury exposure (Inasmasu et al. 1986; Wilhelm and Idel 1996). Methylmercury is the primary dietary mercury contaminant and is present in large amounts in seafood (Ikingura and Akagi 1996). Most of the mercury measured in hair is methylmercury; hair is a good matrix for assessing exposure to methylmercury (Wilhelm and Idel 1996). Hair analysis has been conducted using CVAAS, AFS, and NAA (Grandjean et al. 1992; Ngim et al. 1988; Pineau et al. 1990; Suo et al. 1992; Suzuki et al. 1992; Taskaev et al. 1988; Vermeir et al. 1991a, 1991b; Zhuang et al. 1989). Segmental hair analysis is commonly used as a means of determining an historical record of exposure or uptake of mercury (Grandjean et al. 1992; Suzuki et al. 1992). The method involves cutting the hair strands into smaller segments, usually 1 cm each, and analyzing each segment separately. Detection limits for hair using CVAAS were not reported but are expected to be similar to those for tissue (sub- to low-ppb). The sensitivity of NAA is similar to that of CVAAS, but variable recoveries and precision make NAA less reliable. Good results were reported for one NAA method (Zhuang et al. 1989). Results from studies using AFS suggest this method may be the most sensitive and reliable technique (Suo et al. 1992; Vermeir et al. 1991a, 1991b). A detection limit in the sub-ppt range was obtained, and precision and accuracy were both excellent.

An X-ray fluorescence (XRF) technique has been used to measure mercury in the wrist and temporal areas of dentists exposed to various heavy metals in the work place (Bloch and Shapiro 1986). This technique allows simultaneous evaluation of the tissue burden of a number of different metals. Bone levels may be more closely related to long-term exposure than levels in blood, urine, and hair. The detection limit for XRF is in the low ppm.

A method for detecting methylmercury in biological samples by its enzymatic conversion to methane is an alternative biological technique for methylmercury or other organomercurial analyses (Baldi and Filippelli 1991). *Pseudomonas putida* strain FB1, a broad spectrum mercury-resistant strain, is able to enzymatically convert methylmercury to Hg^0 and methane either in whole cell or in cell-free extracts. GC/FID was used to determine methane produced by the biological derivatization of methylmercury. The detection limit was 15 ng of methylmercury extracted from 1 g of biological tissue. The coefficient of variation was 1.9%. Chemical interferences are negligible in the enzymatic determination of methyl-mercury. The specificity of this determination places the method among the most reliable ones. Recovery was not reported.

6.2 ENVIRONMENTAL SAMPLES

Mercury levels have been determined in numerous environmental matrices, including air, water (surface water, drinking water, groundwater, sea water, and industrial effluents), soils and sediments, fish and shellfish, foods, pharmaceuticals, and pesticides. The sample preparation varies with the complexity of the matrix, but most complex samples require decomposition of the matrix and reduction of the mercury to its elemental form. As described Section 6.1 for biological samples, special sample preparation methods need to be employed if inorganic and organic mercury are to be determined separately, or if the individual species of the organic mercury fraction are to be determined. More detailed information on selected methods in various environmental samples is given in Table 6-2.

Both CVAAS and CVAFS have been used to monitor air and suspended particulates in air for mercury (Baeyens and Leermakers 1989; Bloom and Fitzgerald 1988; Friese et al. 1990; NIOSH 1994; Paudyn and Van Loon 1986; Sengar et al. 1990; Stockwell et al. 1991; Temmerman et al. 1990). Both methods are sensitive, accurate, and precise, although slightly greater sensitivity was reported with AFS (low ppt) than with AAS (mid ppt); AFS is becoming a more common method of analysis (Horvat 1996). When AAS or AFS was combined with gas chromatography (GC), the different mercury species (inorganic mercury, dimethylmercury, diethylmercury, and methylmercury chloride) present in the air could be separated (Bloom and Fitzgerald 1988; Paudyn and Van Loon 1986). A colorimetric method, based on the formation of a colored complex formed in the presence of mercury, has been used as a quick and simple field test that can detect mercury present at the mid-ppb level (Cherian and Gupta 1990).

Numerous methods, including CVAAS, ASV, inductively coupled plasma (ICP) MS, ICP atomic emission spectrometry (AES), microwave-induced plasma (MIP) AES, NAA, GC/AFS, high-performance liquid chromatography (HPLC)/UV, HPLC/ECD, and spectrophotometry, have been used to determine mercury levels in aqueous media. Mercury has been measured in drinking water, surface water, groundwater, snow, waste water effluents, and sea water. Of the available methods, CVAAS is the method of choice (Baxter and Frech 1989, 1990; Birnie 1988; Eaton et al. 1995; Goto et al. 1988; Lee et al. 1989; Mateo et al. 1988; Munaf et al. 1991; Paudyn and Van Loon 1986; Ping and Dasgupta 1989; Robinson and Schuman 1989; Schintu et al. 1989; Shkinev et al. 1989) and the method recommended by EPA and AOAC (AOAC 1984; Beckert et al. 1990; EPA 1994f, 1994g). This method is very sensitive for mercury in water (sub- to low-ppt) and has been proven to be reliable. Water samples generally do not require digestion, but mercury in the samples is usually reduced to the elemental state and preconcentrated prior to analysis. When combined

with GC, CVAAS has been used to separate and determine individual mercury species in aqueous samples (Paudyn and Van Loon 1986). Spectrophotometry has often been used to determine mercury in aqueous matrices (Abbas et al. 1989; Ajmal et al. 1989; Eaton et al. 1995; Raman and Shinde 1990; Singh et al. 1989). Sample preparation methods vary and have included separation by thin-layer chromatography (TLC) (Ajmal et al. 1989) or column chromatography (Yan et al. 1989), selective extraction (Abbas et al. 1989), and ligand formation (Raman and Shinde 1990; Singh et al. 1989). While recoveries were good, spectrophotometry is not as sensitive a technique as CVAAS. Tests of additional methods, including ASV (Liu et al. 1990), ICP/MS (Haraldsson et al. 1989), NAA (Itawi et al. 1990), AES-based techniques (Kitagawa and Nishimoto 1989; Mahanti 1990; Nakahara et al. 1988), HPLC-based techniques (Evans and McKee 1988; Shofstahl and Hardy 1990), and graphite-furnace (GF) AAS (LeBihan and Cabon 1990) indicate that these methods may also be useful for determining mercury in water samples. One of the most promising methods is GC/AFS, which has the advantages of increased sensitivity and precision compared to CVAAS and can also be used to isolate individual mercury species (Bloom 1989). A colorimetric assay has also been developed that is useful for rapid preliminary screening of field samples (Cherian and Gupta 1990).

CVAAS is the most commonly used technique for determining the mercury concentration of sediments, soils, and sludge (Bandyopadhyay and Das 1989; Beckert et al. 1990; EPA 1994g; Van Delft and Vos 1988). As with other matrices, it is sensitive, reliable, and requires little sample preparation beyond digestion of the matrix and reduction of the mercury to its elemental form. It is the method recommended by EPA for solid matrices (Beckert et al. 1990; EPA 1994g). A method based on CVAFS that uses flow injection analysis with on-line microwave digestion for the determination of total mercury has been described recently (Morales-Rubio et al. 1995). Good sensitivity (90 ppt) and precision (4% RSD) was demonstrated. Gas chromatography in conjunction with atomic emission detection (GC/AED) has been used to determine organomercury species in soils and sediments (Liu et al. 1994). Direct current ASV (DCASV) has been tested for use in determining mercury levels in river sediment (Lexa and Stulik 1989). The accuracy and sensitivity of this method are good, but it is less precise than CVAAS. A field method using XRF has been developed to monitor soil contamination (Grupp et al. 1989). This method is rapid and portable, but its high detection limit (low-ppm) makes it useful only for on-site screening.

Methods have been developed for the determination of mercury in fish, shellfish, foods, food sources, and pharmaceuticals. AAS, usually with cold vapor generation (CVAAS), is one of the primary methods used to measure mercury in these complex matrices (Carrillo et al. 1986; Friese et al. 1990; Landi et al. 1990;

Navarro et al. 1992; Odukoya 1990; Vermeir et al. 1988, 1989), because of its sensitivity and reliability. Although the sensitivity (sub- to low-ppb), accuracy, and precision are not as good as with less complex gaseous and aqueous media, it is still one of the best methods available for analysis of mercury in any matrix. Flameless AAS without cold vapor generation has also produced good results when used to determine ppb levels of mercury in wine (Cacho and Castells 1989) and fish (Filippelli 1987); it is also one of the methods recommended by AOAC for fish and food (AOAC 1984). When combined with high resolution GC (HRGC), the individual organic mercury species in fish could be determined (Jiang et al. 1989). Sub-ppt levels of mercury in powdered milk and oyster tissue were reliably determined using AFS (Vermeir et al. 1991a, 1991b). NAA was used to measure mercury levels in copepod homogenate and tomato leaves, but the sensitivity (mid- to low-ppb) and reliability were not as good as that of CVAAS or AFS (Taskaev et al. 1988; Zhuang et al. 1989). Several other methods, including IDSSMS (Moody and Paulsen 1988), HPLC/ICP/MS (Bushee 1988), square-wave voltametry (ASV) (Mannino et al. 1990), ASV (Golimowski and Gustavsson 1983), MIP/AES (Natajaran 1988), GC/ECD (Ahmed et al. 1988; AOAC 1984), and spectrophotometry (Agrawal and Desai 1985; Marquez et al. 1988) have also been used to analyze fish, plant material, and pharmaceuticals for mercury. HPLC/ICP/MS has the additional advantage of permitting separation and quantitation of individual mercury species (Bushee 1988). An AOAC-recommended colorimetric method is available for screening food samples (AOAC 1984).

Several other environmental matrices have been analyzed for mercury content. These include coal fly ash (Horvat and Lupsina 1991; Lexa and Stulik 1989), coal dust (Wankhade and Garg 1989), minerals (Bichler 1991), pesticides (Sharma and Singh 1989), gasoline (Costanzo and Barry 1988), and oily waste (Campbell and Kanert 1992). The methods used include CVAAS, DCASV, NAA, spectrophotometry, and GC/alternating current plasma detection (ACPD). The data on each method for each matrix were insufficient for making comparisons.

6.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of mercury is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of mercury.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. There are reliable methods for detecting and quantifying elemental mercury in human breath, blood, urine, milk, tissues, hair, and bones. The method of choice is CVAAS (Akagi et al. 1995; Friese et al. 1990; Pineau et al. 1990; Ping and Dasgupta 1989, 1990; Rathje et al. 1974; Vermeir et al. 1988, 1989; Vesterberg 1991). Other methods that have produced good results include ETAAS (Emteborg et al. 1992), AFS (Corns et al. 1994; Vermeir et al. 1991a, 1991b; Suo et al. 1992), flameless AAS (NIOSH 1994), IDSSMS (Moody and Paulsen 1988), XRF (Bloch and Shapiro 1986), NAA (Fung et al. 1995; Zhuang et al. 1989), GC/MPD (Bulska et al. 1992), ICP-AES (Buneaux et al. 1992), and ICP-MS (Kalamegham and Ash 1992). Using these methods, mercury levels at μg to pg concentrations are detectable. This makes them useful for measuring background and higher levels (Ikingura and Agaki 1996). Many of the methods can also distinguish between organic and inorganic mercury. No further methods for analysis of elemental mercury in biological fluids and tissues are needed. Additional research will be needed to validate the determination of individual mercury species (i.e., methylmercury, phenyl mercury, mercury acetate, etc.) in matrices determined to be important. Methods exist for the separation and detection of these species, but few standard reference materials exist for comparative studies.

Biochemical indicators of possible renal dysfunction (increased urinary NAG levels, and elevated porphyrins) have been associated with increased urinary levels of mercury (Rosenman et al. 1986; Wada et al. 1969; Woods 1996). Functional indicators of adverse neurological effects (reduced nerve conduction velocity, prolonged nerve latency, increased tremor frequency, increased reaction time, reduced hand-eye coordination, and performance on memory and verbal intelligence tests) have also been correlated with increased urinary levels of mercury (Levine et al. 1982; Piikivi et al. 1984; Smith et al. 1970, 1983; Verberk et al. 1986; Vroom and Greer 1972; Williamson et al. 1982). Decreased nerve conduction velocity has been correlated with increased tissue levels of mercury (Shapiro et al. 1982). These biomarkers are not specific for mercury and may be induced by exposure to other metals and chemicals or to disease conditions. Other

nonspecific indicators of possible mercury exposure (insomnia, emotional instability, paresthesia, and abnormal EEG) that have been observed in exposed individuals cannot be quantified, but an increased incidence in specific populations may be correlated with increased urinary levels of mercury in the population (Davis et al. 1974; Jaffe et al. 1983; McFarland and Reigel 1978). The existing analytical methods that have been discussed for exposure can reliably measure the levels in blood, urine, and tissue at which these effects occur. Standard methods exist to measure the effects that can be quantified. No further methods need to be developed.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. There are analytical methods to detect and measure elemental and organic mercury in air, water, sediment, soil, sludge, foods, plant materials, and other environmental matrices. The methods used include CVAAS (the most commonly used and recommended method) (AOAC 1984; Baxter and Frech 1989; Eaton et al. 1995; EPA 1994f, 1994g; Munaf et al. 1991; Navarro et al. 1992; Paudyn and Van Loon 1986; Ping and Dasgupta 1989), AFS (Bloom 1989; Bloom and Fitzgerald 1988; Morales-Rubio et al. 1995; Vermeir et al. 1991a, 1991b), IDSSMS (Moody and Paulsen 1988), flameless AAS (Cacho and Castells 1989; Filippelli 1987; NIOSH 1994), and several other methods. Several of the methods have been proven reliable and are sensitive enough to measure background levels. Methods also exist to determine individual mercury species (Bloom and Fitzgerald 1988; Liu et al. 1994; Paudyn and Van Loon 1986). No further methods are needed for mercury analysis in environmental samples. Additional work would be required to validate methods for individual organomercury species in particular matrices.

6.3.2 Ongoing Studies

Ongoing studies concerning the detection and measurement of mercury in biological or environmental samples identified through a search of Federal Research in Progress (FEDRIP 1998) are shown in Table 6-3.

7. REGULATIONS AND ADVISORIES

The international, national, and state regulations and guidelines regarding mercury and mercury compounds in air, water, and other media are summarized in Table 7-1. Unless otherwise indicated, the listings in the table refer to mercury.

An MRL of 0.0002 mg/m^3 has been derived for chronic-duration inhalation exposure (365 days or more) to metallic mercury vapor in a group of 26 mercury-exposed workers from three industries exposed to low levels of mercury for an average of 15.3 years (range, 1–41 years) (Fawer et al. 1983).

An MRL of 0.007 mg mercury/kg/day has been derived for acute-duration oral exposure (14 days or less) to inorganic mercury based on a NOAEL of 0.93 mg mercury/kg for renal effects (increased absolute and relative kidney weights) in rats exposed to gavage doses of mercuric chloride for 14 days (NTP 1993).

An MRL of 0.002 mg mercury/kg/day has been derived for intermediate-duration (15–364 days) oral exposure to inorganic mercury based on a NOAEL of 0.23 mg mercury/kg for renal effects (increased absolute and relative kidney weights) in rats (Dieter et al. 1992; NTP 1993).

An MRL of 0.0003 mg mercury/kg/day has been derived for chronic-duration (365 days or more) oral exposure to methylmercury, based on neurodevelopmental outcomes in a study by Davidson et al. (1998) of children exposed *in utero* to methylmercury from maternal fish ingestion.

EPA has derived an oral RfD of 8×10^{-5} mg/kg/day (0.08 µg/kg/day) for phenylmercuric acetate as mercury (IRIS 1997). The RfD is based on a LOAEL of 0.5 ppm mercury or 0.042 mg/kg/day phenyl mercuric acetate for detectable kidney damage in female rats after 2 years (Fitzhugh et al. 1950). EPA has derived an oral RfD of 3×10^{-4} mg/kg/day (0.3 µg/kg/day) for mercuric chloride. The RfD is based on LOAELs of 0.226, 0.317, and 0.633 mg/kg/day of mercuric chloride. Although no one study was found adequate for deriving an oral RfD, EPA's mercury workgroup derived an oral RfD of high confidence using the weight of evidence from three studies (Andres 1984; Bernaudin et al.; Druet et al. 1978) which used Brown-Norway rats, and an intensive review and discussion of the entire inorganic mercury data base (IRIS 1997). EPA has derived an oral RfD of 1×10^{-4} mg/kg/day (0.1 µg/kg/day) for methylmercury based on developmental neurological abnormalities in human infants (IRIS 1997). EPA has not derived an RfD value for elemental mercury. The EPA inhalation reference concentration (RfC) for elemental mercury is $3x10^{-4}$ mg/m^3

(0.3 µg/m^3). The RfC is based on a LOAEL of 0.025 ppm for human occupational exposure studies. Critical effects seen during these studies included hand tremors, increases in memory disturbances, and slight subjective and objective evidence of autonomic dysfunction (IRIS 1997). No RfC was reported for other mercury compounds.

The American Conference of Governmental Industrial Hygienists (ACGIH) and the EPA have determined that inorganic forms of mercury, including metallic mercury, are not classifiable as to their human carcinogenicity. These agencies have assigned mercury and its inorganic compounds the weight-of-evidence classifications of A4 and D, respectively (ACGIH 1996; IRIS 1997). Mercuric chloride and methylmercury have been assigned EPA's weight-of evidence classification of C, which indicates that they are possible human carcinogens (IRIS 1997).

OSHA requires employers of workers who could be occupationally exposed to mercury to institute engineering controls and work practices which ensure that during any part of the workday, mercury concentrations do not exceed the ceiling value of 1 mg/10 m^3 (0.1 mg/m^3) (OSHA 1974).

Mercuric cyanide, mercuric nitrate, mercuric sulfate, mercuric thiocyanate, mercurous nitrate, mercury, and mercury fulminate have been designated as hazardous substances pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980 (EPA 1995i). Mercuric acetate, mercuric chloride, and mercuric oxide are mercury compounds that have been individually designated as extremely hazardous substances under Section 313 of Title III of the Superfund Amendments and Reauthorization Act (SARA) of 1986 (EPA 1995j). Phenylmercury acetate is consider both a hazardous substance and an extremely hazardous substances. The statutory sources designating mercury and regulated mercury compounds as CERCLA hazardous substances are section 307(a) of the Clean Water Act (CWA), section 112 of the Clean Air Act (CAA), and section 3001 of the Resource Conservation and Recovery Act (RCRA) (EPA 1995i). The owner and operator of facilities using these substance on their sites are required to immediately report releases to any environmental media, if the amount released exceeds the established "reportable quantity" (EPA 1995i). The statutory and final reportable quantities for mercury and regulated mercury compounds as established by Section 102 of CERCLA are given in Table 7-1 (EPA 1995i). Although mercury compounds are listed generically as CERCLA hazardous substances no reportable quantity has been established for them as a broad class (EPA 1995i). Title III of SARA is also known as "The Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986." As chemicals subject to the emergency planning and release reporting requirements of EPCRA, owners and operators of

facilities that have mercuric acetate, mercuric chloride, and mercuric oxide on their sites in amounts exceeding the “threshold planning quantity” established for these substances must develop a program that addresses implementing emergency response plans and for notifying the public of accidental releases (EPA 1987a, 1995j). When extremely hazardous substances are formulated as a solids they are subject to either of two threshold planning quantities (EPA 1995j). If the solid exits in powdered form and has a particle size less than 100 microns, it is subject to the lower number. If the solid does not meet this criteria, it is subject to the higher number. The threshold planning quantities for mercuric acetate, mercuric chloride, mercuric oxide, and phenylmercury acetate are given in Table 7-1. It is important to note that reportable quantities for these compounds are the same as their threshold planning quantities.

The EPA regulates mercury under the Clean Air Act (CAA) and has designated it as a hazardous air pollutant (HAP). Emission standards for release of mercury to the atmosphere have been promulgated for mercury cell chloralkali plants, mercury ore processing facilities, major stationary sources, and municipal waste combustors (EPA 1975a, 1975b, 1995a, 1996b).

In accordance with the authority of the Safe Drinking Water Act (SDWA), EPA has established a safe drinking water standard for mercury at 2 µg/L (FSTRAC 1995). Under the Clean Water Act (CWA) EPA provides criterion concentrations for mercury as a priority toxic pollutant (EPA 1992).

Mercury is regulated as a “priority pollutant” in accordance with the Clean Water Act (CWA). The CWA establishes the basic structure for regulating the discharge of pollutants to waterways and is designed to ensure that all waters are sufficiently clean to protect public health and/or the environment. However, if waters and their sediments become contaminated from sources such as atmospheric deposition and discharges from industrial, municipal, or agricultural operations, toxic substances could concentrate in the tissue of fish and wildlife.

Advisories have been developed and issued to warn people about the health risks of consuming methylmercury-contaminated fish, shellfish, or wildlife and provide guidance as to the amount of fish or wildlife that can be safely consumed by each group (adults, pregnant women, nursing mothers, and young children). Each state, Native American tribe, or U.S. Territory establishes its own criteria for issuing fish and wildlife advisories. A fish or wildlife advisory will specify which waters (lake, rivers, estuaries, or coastal areas) or hunting areas have restrictions. The advisory provides information on the species and size range of the fish or wildlife of concern. The advisory may completely ban eating fish, shellfish, or

freshwater turtles, or it may recommend consumption limits (numbers of fish meals per specified time period) considered to be safe to eat. For example, an advisory may recommend that a person eat a certain type of fish no more than once a month. Advisories may specify the tissues of the fish or wildlife that can be safely eaten or proper preparation and cooking practices to help decrease exposure to methylmercury. The fish or wildlife advisory is typically more restrictive to protect pregnant women, nursing mothers, and young children. To reduce children's exposure to methylmercury, state advisory recommendations for fish consumption limits (meals per week or meals per month) should be strictly observed. Published information in the form of brochures on fish and wildlife advisories is available from State Public Health Departments, Natural Resources Departments, or Fish and Game Departments. Signs may be posted in certain fishing and hunting areas frequently used by recreational fishers and hunters to warn them about specific contamination problems (EPA 1995 Fish Sampling analysis and Guidance Document).

Currently, 1,782 advisories are in effect in 41 states and one U.S. Territory (American Samoa) restricting the consumption of mercury-contaminated fish, shellfish, or wildlife (freshwater turtles) (EPA 1998a). Methylmercury is the chemical pollutant responsible, in part, for over 77% of fish advisories issued in the United States (EPA 1998b). Eleven states (Connecticut, Indiana, Maine, Massachusetts, Michigan, Missouri, New Hampshire, New Jersey, North Carolina, Ohio, and Vermont) currently have state-wide mercury advisories recommending that all residents restrict consumption of locally caught freshwater fish. In addition, 5 states (Alabama, Florida, Louisiana, Massachusetts, and Texas) have issued statewide coastal mercury advisories for specific marine fish or shellfish species. In two states (Arizona and Minnesota), wildlife advisories have been issued recommending that residents restrict their consumption of freshwater turtles (EPA 1998a, 1998b).

The FDA currently has advice for consumers (posted on the Internet) recommending that pregnant women, and women of childbearing age who may become pregnant, limit their consumption of shark and swordfish to no more that one meal per month (FDA 1998). Methylmercury levels are much higher in these fish species than in the more commonly consumed species. The FDA advisory covers women of childbearing age who might become pregnant because dietary practices immediately before the pregnancy may have a direct bearing on fetal exposure during pregnancy. The FDA states that nursing mothers who follow this advice, will not expose their infants to increased health risks from methylmercury (FDA 1998). The FDA consumer advice hotline telephone number is **1-800-332-4010** and the FDA Web site is **www.FDA.gov**.

7. REGULATIONS AND ADVISORIES

The Food and Drug Administration (FDA) regulates the use of mercury compounds in the cosmetics industry. The FDA regulations on the use of mercury compounds in cosmetics state that "because of the known hazards of mercury, its questionable efficacy as a skin-bleaching agent, and the availability of effective and less toxic non-mercurial preservatives, there is no justification for the use of mercury in skin-bleaching preparations or its use as a preservative in cosmetics, with the exception of eye-area cosmetics" (FDA 1974). The use of mercury compounds as cosmetic ingredients has primarily been limited to their use as preservatives in eye area cosmetics for which no other effective and safe non-mercurial preservative is available. In other preparations they must contain no more than trace amounts of mercury that are unavoidable under the conditions of good manufacturing practices (FDA 1974). The mercurial concentration in these other preparations must measure less than 1 ppm or 0.0001% mercury metal (FDA 1974).

The FDA has also established an action level of 1 ppm for methylmercury in fish (FDA 1994, 1996). Because of reports that swordfish, shark and other large predatory fish may contain methylmercury levels which exceed the FDA 1 ppm limit, the agency’s advice to consumers warns pregnant women and women of childbearing age to limit their consumption of shark and swordfish to no more than one meal a month (FDA 1996). For others, the agency recommends that regular consumption of fish species with methylmercury levels around 1 ppm be limited to approximately 7 ounces per week; for fish with levels averaging 0.5 ppm, the limit is about 14 ounces per week (FDA 1996). The consumption advice is considered unnecessary for the top 10 species of fish that make up approximately 80% of the seafood market (FDA 1996). Canned tuna, shrimp, pollock, salmon, cod, catfish, clams, flatfish, crabs, and scallops are the top 10 species of fish consumed (FDA 1996). Since methylmercury levels in these species are usually less than 0.2 ppm and because few people eat more than the suggested weekly limit of 2.2 pounds (1 kilogram) for this contamination level, consumption limits are considered unnecessary (FDA 1996).

On May 28, 1998, the Consumer Product Safety Commission (CPSC) issued a guidance statement recommending that manufacturers of liquid-filled consumer products eliminate the use of hazardous chemicals in the liquid portion of their products (CPSC 1998). The guidance statement was issued as an effort to reduce the risk of exposing young children to hazardous chemicals contained in the liquid. The hazardous chemicals found in the liquid include mercury, ethylene glycol, diethylene glycol, methanol, methylene chloride, petroleum distillates, toluene, and xylene. Children’s products identified by the Commission as containing these hazardous chemicals include rolling balls, maze toys, bubble watches, and necklaces. Paperweights, keychains, liquid timers, and pens were household items identified as containing

mercury or other hazardous chemicals (CPSC 1998). In addition to the recommendation that manufacturers eliminate the use of hazardous chemicals in these products, the Commission also recommends that importers, distributors, and retailers who purchase a liquid-filled product for resale, obtain from the manufacturer assurances that their product does not contain hazardous liquid chemicals. Although the guidance is not a rule, it focuses on certain obligations authorized by the Federal Hazardous Substance Act (FHSA). Under the FHSA toys or other articles that contain an accessible and harmful amount of hazardous chemical and are intended for use by children are banned (CPSC 1998). Articles that are not intended for use by children, but create a risk of injury because they contain hazardous chemicals, require precautionary labeling under the FHSA (CPSC 1998).

In 1995, the CPSC assisted in facilitating the recall of necklaces bearing small vials or glass balls containing metal mercury (CPSC 1995). Although the vials and glass balls posed no immediate health threat, the recall noted that exposure to mercury vapor could cause long term health problems, especially for small children and pregnant women, if the vials or balls were broken (CPSC 1995).

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9. GLOSSARY

Absorption—The taking up of liquids by solids, or of gases by solids or liquids.

Acute Exposure—Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Adsorption—The adhesion in an extremely thin layer of molecules (as of gases, solutes, or liquids) to the surfaces of solid bodies or liquids with which they are in contact.

Adsorption Coefficient (K_{oc})—The ratio of the amount of a chemical adsorbed per unit weight of organic carbon in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (Kd)—The amount of a chemical adsorbed by a sediment or soil (i.e., the solid phase) divided by the amount of chemical in the solution phase, which is in equilibrium with the solid phase, at a fixed solid/solution ratio. It is generally expressed in micrograms of chemical sorbed per gram of soil or sediment.

Benchmark Dose (BMD)—is usually defined as the lower confidence limit on the dose that produces a specified magnitude of changes in a specified adverse response. For example, a BMD_{10} would be the dose at the 95% lower confidence limit on a 10% response, and the benchmark response (BMR) would be 10%. The BMD is determined by modeling the dose response curve in the region of the dose response relationship where biologically observable data are feasible.

Benchmark Dose Model—is a statistical dose-response model applied to either experimental toxicological or epidemiological data to calculate a BMD.

Bioconcentration Factor (BCF)—The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the same time or during the same period.

Biomarkers—are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility.

Cancer Effect Level (CEL)—The lowest dose of chemical in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Carcinogen—A chemical capable of inducing cancer.

Case-Control Study—A type of epidemiological study which examines the relationship between a particular outcome (disease or condition) and a variety of potential causative agents (such as toxic chemicals). In a case-controlled study, a group of people with a specified and well-defined outcome is identified and compared to a similar group of people without outcome.

Case Report—describes a single individual with a particular disease or exposure. These may suggest some potential topics for scientific research but are not actual research studies.

Case Series—describes the experience of a small number of individuals with the same disease or exposure. These may suggest potential topics for scientific research but are not actual research studies.

Ceiling Value—A concentration of a substance that should not be exceeded, even instantaneously.

Chronic Exposure—Exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Cohort Study—A type of epidemiological study of a specific group or groups of people who have had a common insult (e.g., exposure to an agent suspected of causing disease or a common disease) and are followed forward from exposure to outcome. At least one exposed group is compared to one unexposed group.

Cross-sectional Study—A type of epidemiological study of a group or groups which examines the relationship between exposure and outcome to a chemical or to chemicals at one point in time.

Data Needs—substance-specific informational needs that if met would reduce the uncertainties of human health assessment.

Developmental Toxicity—The occurrence of adverse effects on the developing organism that may result from exposure to a chemical prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Dose-Response Relationship—the quantitative relationship between the amount of exposure to a toxicant and the incidence of the adverse effects.

Embryotoxicity and Fetotoxicity—Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurs. The terms, as used here, include malformations and variations, altered growth, and in utero death.

Environmental Protection Agency (EPA) Health Advisory—An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Epidemiology—refers to the investigation of factors that determine the frequency and distribution of disease or other health-related conditions within a defined human population during a specified period.

Genotoxicity—a specific adverse effect on the genome of living cells that, upon the duplication of affected cells, can be expressed as a mutagenic, clastogenic or carcinogenic event because of specific alteration of the molecular structure of the genome.

Half-life—a measure of rate for the time required to eliminate one half of a quantity of a chemical from the body or environmental media.

Immediately Dangerous to Life or Health (IDLH)—The maximum environmental concentration of a contaminant from which one could escape within 30 minutes without any escape-impairing symptoms or irreversible health effects.

Incidence—The ratio of individuals in a population who develop a specified condition to the total number of individuals in that population who could have developed that condition in a specified time period.

Intermediate Exposure—Exposure to a chemical for a duration of 15-364 days, as specified in the Toxicological Profiles.

Immunological Effects—are functional changes in the immune response.

Immunologic Toxicity—The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

***In Vitro*—**Isolated from the living organism and artificially maintained, as in a test tube.

***In Vivo*—**Occurring within the living organism.

Lethal Concentration$_{(LO)}$ (LC_{LO})—The lowest concentration of a chemical in air which has been reported to have caused death in humans or animals.

Lethal Concentration$_{(50)}$ (LC_{50})—A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population.

Lethal Dose$_{(LO)}$ (LD_{LO})—The lowest dose of a chemical introduced by a route other than inhalation that has been reported to have caused death in humans or animals.

Lethal Dose$_{(50)}$ (LD_{50})—The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time$_{(50)}$ (LT_{50})—A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Lowest-Observed-Adverse-Effect Level (LOAEL)—The lowest exposure level of chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Lymphoreticular Effects—represent morphological effects involving lymphatic tissues such as the lymph nodes, spleen, and thymus.

Malformations—Permanent structural changes that may adversely affect survival, development, or function.

Minimal Risk Level (MRL) —An estimate of daily human exposure to a hazardous substance that is likely to be without an appreciable risk of adverse noncancer health effects over a specified route and duration of exposure.

Modifying Factor (MF)—A value (greater than zero) that is applied to the derivation of a minimal risk level (MRL) to reflect additional concerns about the database that are not covered by the uncertainty factors. The default value for a MF is 1.

Morbidity—State of being diseased; morbidity rate is the incidence or prevalence of disease in a specific population.

Mortality—Death; mortality rate is a measure of the number of deaths in a population during a specified interval of time.

Mutagen—A substance that causes mutations. A mutation is a change in the DNA sequence of a cell's DNA. Mutations can lead to birth defects, miscarriages, or cancer.

Necropsy—The gross examination of the organs and tissues of a dead body to determine the cause of death or pathological conditions.

Neurotoxicity—The occurrence of adverse effects on the nervous system following exposure to a chemical.

No-Observed-Adverse-Effect Level (NOAEL)—The dose of a chemical at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Octanol-Water Partition Coefficient (K_{ow})—The equilibrium ratio of the concentrations of a chemical in *n*-octanol and water, in dilute solution.

Odds Ratio—a means of measuring the association between an exposure (such as toxic substances and a disease or condition) which represents the best estimate of relative risk (risk as a ratio of the incidence among subjects exposed to a particular risk factor divided by the incidence among subjects who were not exposed to the risk factor). An odds ratio of greater than 1 is considered to indicate greater risk of disease in the exposed group compared to the unexposed.

Organophosphate or Organophosphorus Compound—a phosphorus containing organic compound and especially a pesticide that acts by inhibiting cholinesterase.

Permissible Exposure Limit (PEL)—An Occupational Safety and Health Administration (OSHA) allowable exposure level in workplace air averaged over an 8-hour shift of a 40 hour workweek.

Pesticide—general classification of chemicals specifically developed and produced for use in the control of agricultural and public health pests.

Pharmacokinetics—is the science of quantitatively predicting the fate (disposition) of an exogenous substance in an organism. Utilizing computational techniques, it provides the means of studying the absorption, distribution, metabolism and excretion of chemicals by the body.

Pharmacokinetic Model—is a set of equations that can be used to describe the time course of a parent chemical or metabolite in an animal system. There are two types of pharmacokinetic models: data-based and physiologically based. A data-based model divides the animal system into a series of compartments which, in general, do not represent real, identifiable anatomic regions of the body whereby the physiologically based model compartments represent real anatomic regions of the body.

Physiologically Based Pharmacodynamic (PBPD) Model—is a type of physiologically based dose-response model which quantitatively describes the relationship between target tissue dose and toxic end points. These models advance the importance of physiologically based models in that they clearly describe the biological effect (response) produced by the system following exposure to an exogenous substance.

Physiologically Based Pharmacokinetic (PBPK) Model—is comprised of a series of compartments representing organs or tissue groups with realistic weights and blood flows. These models require a variety of physiological information: tissue volumes, blood flow rates to tissues, cardiac output, alveolar ventilation rates and, possibly membrane permeabilities. The models also utilize biochemical information 4such as air/blood partition coefficients, and metabolic parameters. PBPK models are also called biologically based tissue dosimetry models.

Prevalence—The number of cases of a disease or condition in a population at one point in time.

Prospective Study--a type of cohort study in which the pertinent observations are made on events occurring after the start of the study. A group is followed over time.

q_1*—The upper-bound estimate of the low-dose slope of the dose-response curve as determined by the multistage procedure. The q_1* can be used to calculate an estimate of carcinogenic potency, the incremental excess cancer risk per unit of exposure (usually µg/L for water, mg/kg/day for food, and µg/m^3 for air).

Recommended Exposure Limit (REL)—A National Institute for Occupational Safety and Health (NIOSH) time-weighted average (TWA) concentrations for up to a 10-hour workday during a 40-hour workweek.

Reference Concentration (RfC)—An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious noncancer health effects during a lifetime. The inhalation reference concentration is for continuous inhalation exposures and is appropriately expressed in units of mg/m^3 or ppm.

Reference Dose (RfD)—An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the No-Observed-Adverse-Effect Level (NOAEL- from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to nonthreshold effects such as cancer.

Reportable Quantity (RQ)—The quantity of a hazardous substance that is considered reportable under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA). Reportable quantities are (1) 1 pound or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Section 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity—The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

Retrospective Study—A type of cohort study based on a group of persons known to have been exposed at some time in the past. Data are collected from routinely recorded events, up to the time the study is undertaken. Retrospective studies are limited to casual factors that can be ascertained from existing records and/or examining survivors of the cohort.

Risk—the possibility or chance that some adverse effect will result from a given exposure to a chemical.

Risk Factor—An aspect of personal behavior or lifestyle, an environmental exposure, or an inborn or inherited characteristic, that is associated with an increased occurrence of disease or other health-related event or condition.

Risk Ratio—The ratio of the risk among persons with specific risk factors compared to the risk among persons without risk factors. A risk ratio greater than 1 indicates greater risk of disease in the exposed group compared to the unexposed.

Short-Term Exposure Limit (STEL)—The American Conference of Governmental Industrial Hygienists (ACGIH) maximum concentration to which workers can be exposed for up to 15 min continually. No more than four excursions are allowed per day, and there must be at least 60 min between exposure periods. The daily Threshold Limit Value - Time Weighted Average (TLV-TWA) may not be exceeded.

Target Organ Toxicity—This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

Teratogen—A chemical that causes structural defects that affect the development of an organism.

Threshold Limit Value (TLV)—An American Conference of Governmental Industrial Hygienists (ACGIH) concentration of a substance to which most workers can be exposed without adverse effect. The TLV may be expressed as a Time Weighted Average (TWA), as a Short-Term Exposure Limit (STEL), or as a ceiling limit (CL).

Time-Weighted Average (TWA)—An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Toxic Dose$_{(50)}$ (TD$_{50}$)—A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Toxicokinetic—The study of the absorption, distribution and elimination of toxic compounds in the living organism.

Uncertainty Factor (UF)—A factor used in operationally deriving the Minimal Risk Level (MRL) or Reference Dose (RfD) or Reference Concentration (RfC) from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using Lowest-Observed-Adverse-Effect Level (LOAEL) data rather than No-Observed-Adverse-Effect Level (NOAEL) data. A default for each individual UF is 10; if complete certainty in data exists, a value of one can be used; however a reduced UF of three may be used on a case-by-case basis, three being the approximate logarithmic average of 10 and 1.

Xenobiotic—any chemical that is foreign to the biological system.

APPENDIX A

ATSDR MINIMAL RISK LEVEL AND WORKSHEETS

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [42 U.S.C. 9601 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 99–499], requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the U.S. Environmental Protection Agency (EPA), in order of priority, a list of hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL); prepare toxicological profiles for each substance included on the priority list of hazardous substances; and assure the initiation of a research program to fill identified data needs associated with the substances.

The toxicological profiles include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations of a hazardous substance. During the development of toxicological profiles, Minimal Risk Levels (MRLs) are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels.

MRLs are derived for hazardous substances using the no-observed-adverse-effect level/uncertainty factor approach. They are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs are derived for acute (1–14 days), intermediate (15–364 days), and chronic (365 days and longer) durations and for the oral and inhalation routes of exposure. Currently, MRLs for the dermal route of exposure are not derived because ATSDR has not yet identified a method suitable for this route of exposure. MRLs are generally based on the most sensitive chemical-induced end point considered to be of relevance to humans. Serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) are not used as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur.

MRLs are intended only to serve as a screening tool to help public health professionals decide where to look more closely. They may also be viewed as a mechanism to identify those hazardous waste sites that are not expected to cause adverse health effects. Most MRLs contain a degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, nutritionally or immunologically compromised) to the effects of hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address this uncertainty consistent with the public health principle of prevention. Although human data are preferred, MRLs often must be based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive to the effects of hazardous substance than animals and that certain persons may be particularly sensitive. Thus, the resulting MRL may be as much as a hundredfold below levels that have been shown to be nontoxic in laboratory animals.

Proposed MRLs undergo a rigorous review process: Health Effects/MRL Workgroup reviews within the Division of Toxicology, expert panel peer reviews, and agencywide MRL Workgroup reviews, with participation from other federal agencies and comments from the public. They are subject to change as new information becomes available concomitant with updating the toxicological profiles. Thus, MRLs in the most recent toxicological profiles supersede previously published levels. For additional information regarding MRLs, please contact the Division of Toxicology, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road, Mailstop E-29, Atlanta, Georgia 30333.

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Mercury (metallic, vapor)
CAS Number: 7439-97-6
Date: June 15, 2001
Profile Status: Final Draft
Route: [X] Inhalation [ ] Oral
Duration: [ ] Acute [ ] Intermediate [X] Chronic
Key to figure: 21
Species: Human

Minimal Risk Level: 0.0002 [ ] mg/kg/day [X] mg/m^3

Reference: Fawer RF, de Ribaupierre Y, Guillemin MP, et al. 1983. Measurement of hand tremor induced by industrial exposure to metallic mercury. British Journal of Industrial Medicine 40:204-208.

Experimental design. Hand tremors were measured in 26 male workers exposed to metallic mercury and 25 control males working in the same facilities, but not exposed to mercury. Workers had been exposed to mercury through the manufacture of fluorescent tubes, chloralkali, or acetaldehyde. Mercury-exposed workers had a duration of exposure of 15.3±2.6 years, blood mercury of 41.3±3.5 micromoles Hg/L, and urinary mercury of 11.3±1.2 micromoles Hg/mole of creatinine. The mean mercury level measured using personal air monitors was 0.026±0.004 mg/m^3 (3 subjects were exposed to greater than 0.05 mg/m^3). Hand tremors were measured in the subjects using an accelerometer attached to the dorsum of the hand both at rest and while holding 1,250 grams. The highest peak frequency of the acceleration was determined.

Effects noted in study and corresponding doses: The highest peak frequency of the tremor was greater in exposed men than in controls. The highest peak frequency corresponded significantly to duration of exposure and age. Comparison of tremors using an index of the entire spectrum of the tremor showed no differences between exposed men and controls at rest, but the changes observed between rest and load were higher in the exposed men. These changes correlated with the duration of exposure and biological indices of exposure (blood and mercury levels), but not with age.

Dose and end point used for MRL derivation: 0.026 mg/m^3; increased frequency of tremors.

[ ] NOAEL [X] LOAEL

Uncertainty and Modifying Factors used in MRL derivation: 30

[ ] 1 [X] 3 [ ] 10 (for use of a minimal LOAEL)
[X] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [ ] 3 [X] 10 (for human variability)

Was a conversion factor used from ppm in food or water to a mg/body weight dose?
If so explain: No.

Was a conversion used from intermittent to continuous exposure?
If so, explain: Yes. To estimate an equivalent continuous exposure concentration, the average concentration assumed for the 8 hour/day exposures was multiplied by 8/24 and 5/7 (0.026 mg/m^3 x 8/24 hours/day x 5/7 days/week = 0.0062 mg/m^3). Uncertainty factors of 10 for variability in sensitivity to mercury within the human population and 3 for use of a minimal effect LOAEL in MRL derivation were then applied to the calculated 0.0062 mg/m^3 value, yielding a chronic inhalation MRL of 0.2 $\mu g/m^3$. It should be noted that this MRL, although based upon an adult working population, is considered also to be sufficiently protective of neurodevelopmental effects in developing embryos/fetuses and children, the most sensitive subgroups for metallic mercury toxicity.

$LOAEL_{(ADJ)}$ = 0.026 mg/m^3 x (8 hr/24 hr) x (5 days/7 days)
= 0.0062 mg/m^3

MRL = $LOAEL_{(ADJ)}$ ÷ UF = 0.0062 mg/m^3 ÷ 30 = 0.0002 mg/m^3

If an inhalation study in animals, list the conversion factors used in determining human equivalent concentration (HEC): No.

Additional studies or pertinent information which lend support to this MRL: Inhaled metallic mercury is quickly absorbed through the lungs into the blood. Its biologic half-life in humans is approximately 60 days, with the half-life varying with the physiological compartment (e.g., 21 days in the head, versus 64 days in the kidneys; Cherian et al. 1978). Since the duration of exposure does influence the level of mercury in the body, the exposure level reported in the Fawer et al. (1983) occupational study was extrapolated from an 8-hour/day, 40-hour/workweek exposure to a level equivalent to a continuous 24 hour/day, 7 days/week exposure as might be encountered near a hazardous waste site containing metallic mercury.

The ability of long-term, low level exposure to metallic mercury to produce a degradation in neurological performance was also demonstrated in other studies. One such study (Ngim et al. 1992) attributed adverse neurological effects to a lower average level of exposure than did the Fawer et al. (1983) study; however, this study was not used in deriving a chronic inhalation MRL due to uncertainties concerning the study protocol, including methodological and reporting deficiencies. In the Ngim et al. (1992) study, dentists with an average of 5.5 years of exposure to low levels of metallic mercury were reported to have demonstrated impaired performance on several neurobehavioral tests. Exposure levels measured at the time of the study ranged from 0.0007 to 0.042 mg/m^3, with an average of 0.014 mg/m^3. Mean blood mercury levels among the dentists ranged from 0.6 to 57 µg/L, with a geometric mean of 9.8 µg/L. The performance of the dentists on finger tapping (motor speed measure), trail making (visual scanning measure), digit symbol (measure of visuomotor coordination and concentration), digit span, logical memory delayed recall (measure of visual memory), and Bender-Gestalt time (measures visuomotor coordination) were significantly poorer than controls. The exposed dentists also showed higher aggression than did controls. Furthermore, within the group of exposed dentists, significant differences were reported to have been observed between a subgroup with high mercury exposure compared to a subgroup with lower exposure. These exposure severity subgroups were not compared to controls, and average exposure levels for the subgroups were not reported. The design and reporting of this study limit its usefulness in deriving an MRL for metallic mercury. The exposure status of the subjects was known to the investigator during testing, mercury levels were not reported for controls, and methods used to correct for confounders (especially the common use in this population of traditional medicines containing mercury) were not reported. It was also unclear whether the results for the mercury exposure group were inordinately influenced or skewed by the individual dentists with the highest exposures and/or blood levels. These confounding factors precluded the use of the Ngim et al. (1992) study for the derivation of an MRL, but the study does provide support for both the premise that

low-dose chronic exposure to metallic mercury can result in adverse health sequelae and the chronic inhalation MRL that is based upon the Fawer et al. (1983) study of occupationally exposed individuals.

Other occupational studies further support the ability of metallic mercury to induce neurologic deficits. Several studies have reported significant effects on tremor or cognitive skills among groups exposed occupationally to comparable or slightly higher (up to 0.076 mg/m^3) levels (Ehrenberg et al. 1991; Piikivi et al. 1984; Roels et al. 1982). Difficulty with heel-to-toe gait was observed in thermometer plant workers subjected to mean personal breathing zone air concentrations of 0.076 mg/m^3 (range of 0.026–0.27 mg/m^3 (Ehrenberg et al. 1991).

Tremors have also been reported in occupationally exposed workers with urinary mercury concentrations of 50–100 µg/g creatinine, and blood levels of 10–20 µg/L (Roels et al. 1982). By comparison, blood mercury levels in the Fawer et al. (1983) study averaged 41.3 and 16.6 µmol Hg/L for the exposed and control groups, respectively. Urinary mercury levels for the exposed workers in the Fawer et al. (1983) study averaged 11.3 µmol Hg/mol creatinine (about 20 µg/g creatinine), compared with 3.4 µmol/mol creatinine in the controls. In another study (Piikivi et al. 1984), decreases in performance on tests that measured intelligence (similarities) and memory (digit span and visual reproduction) were observed in chloralkali workers exposed for an average of 16.9 years (range, 10–37 years) to low levels of mercury when compared to an age-matched control group. In this study, significant differences from controls were observed on these tests among 16 workers with blood levels ranging from 75 to 344 nmol/L and urine levels ranging from 280 (about 56 µg/L) to 663 nmol/L. Abnormal nerve conduction velocities have also been observed in chloralkali plant workers at a mean urine concentration of 450 µg/L (Levine et al.1982). These workers also experienced weakness, paresthesias, and muscle cramps. Prolongation of brainstem auditory evoked potentials was observed in workers with urinary mercury levels of 325 µg/g creatinine (Discalzi et al. 1993). Prolonged somatosensory evoked potentials were found in 28 subjects exposed to airborne mercury concentrations of 20–96 mg/m^3 (Langauer-Lewowicka and Kazibutowska 1989).

Agency Contact (Chemical Manager): John Risher

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Mercury inorganic
CAS Number: 7439-97-6
Date: June 15, 2001
Profile Status: Final Draft
Route: [ ] Inhalation [X] Oral
Duration: [X] Acute [ ] Intermediate [ ] Chronic
Key to figure: 7
Species: Rat

Minimal Risk Level: 0.007 [X] mg/kg/day [ ] mg/m^3

Reference: NTP. 1993. NTP technical report on the toxicology and carcinogenesis studies of mercuric chloride (CAS no. 7487-94-7) in F344/N rats and $B6C3F_1$ mice (gavage studies). NTP TR408.

Experimental design: Fischer 344 rats (5/sex/group) were administered 0, 0.93, 1.9, 3.7, 7.4, or 14.8 mg Hg/kg/day as mercuric chloride once daily for 14 days, excluding weekends. The mercuric chloride was administered in deionized water via gavage. Body weights were measured and a complete necropsy was performed. Organ weights were obtained for the brain, heart, kidney, liver, lung, and thymus.

Effects noted in study and corresponding doses: The relative and absolute kidney weights were significantly increased for males exposed to at least 1.9 mg Hg/kg/day and for females exposed to at least 3.7 mg Hg/kg/day. An increased incidence of renal tubular necrosis (graded minimal in severity) was observed in 3 of 5 males and 1 of 5 females at the 3.7 mg Hg/kg/day dose level. At 7.4 mg Hg/kg/day, 5/5 males and 3/5 females had minimal-to-mild effects, and at 14.8 mg Hg/kg/day all animals exhibited mild-to-moderate effects.

Dose and end point used for MRL derivation: 0.93 mg Hg/kg/day; no renal effects.

[X] NOAEL [ ] LOAEL

Uncertainty Factors used in MRL derivation: 100

[ ] 1 [ ] 3 [ ] 10 (for use of a LOAEL)
[ ] 1 [ ] 3 [X] 10 (for extrapolation from animals to humans)
[ ] 1 [ ] 3 [X] 10 (for human variability)

Was a conversion factor used from ppm in food or water to a mg/body weight dose?
If so explain: No.

Was a conversion used from intermittent to continuous exposure?
If so, explain: Yes. To estimate an equivalent continuous exposure concentration, the average concentration was multiplied by 5 days/7 days.

$NOAEL_{(ADJ)}$ = 0.93 mg/kg/day x (5 days/7 days)
= 0.66 mg/kg/day

MRL = $NOAEL_{(ADJ)}$ ÷ UF = 0.66 mg/kg/day ÷ 100 = 0.007 mg/kg/day

If an inhalation study in animals, list the conversion factors used in determining human equivalent concentration (HEC): None.

Additional studies or pertinent information which lend support to this MRL: Several other studies examining the effects of oral exposure to inorganic mercury salts have also shown renal toxicity in humans as a result of acute oral exposures. Kidney effects (i.e., heavy albuminuria, hypoalbuminemia, edema, and hypercholesterolemia) have been reported after therapeutic administration of inorganic mercury (Kazantzis et al. 1962). Acute renal failure has been observed in a number of case studies in which mercuric chloride has been ingested (Afonso and deAlvarez 1960; Murphy et al. 1979; Samuels et al. 1982). Autopsy of a 35-year-old man who ingested a lethal dose of mercuric chloride and exhibited acute renal failure showed pale and swollen kidneys (Murphy et al. 1979). A case study reported acute renal failure characterized by oliguria, proteinuria, hematuria, and granular casts in a woman who ingested 30 mg mercury/kg as mercuric chloride (Afonso and deAlvarez 1960). Another case study reported a dramatic increase in urinary protein secretion by a patient who ingested a single dose of 15.8 mg mercury/kg as mercuric chloride (assuming a body weight of 70 kg) (Pesce et al. 1977). The authors of the report surmised that the increased excretion of both albumin and β_2-microglobulin were indicative of mercury-induced tubular and glomerular pathology. Acute renal failure that persisted for 10 days was also observed in a 19-month-old child who ingested an unknown amount of powdered mercuric chloride (Samuels et al. 1982). Decreased urine was also observed in a 22-year-old who attempted suicide by ingesting approximately 20 mg mercury/kg (Chugh et al. 1978).

Agency Contact (Chemical Manager): John Risher

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical name(s): Mercury (inorganic)
CAS number(s): 7439-97-6
Date: June 15, 2001
Profile status: Final Draft
Route: [ ] Inhalation [X] Oral
Duration: [ ] Acute [X] Intermediate [ ] Chronic
Key to figure: 17
Species: Rat

Minimal Risk Level: 0.002 [X] mg/kg/day [ ] ppm

Reference: NTP. 1993. NTP technical report on the toxicology and carcinogenesis studies of mercuric chloride (CAS no. 7487-94-7) in F344/N rats and B6C3F$_1$ mice (gavage studies). NTP TR408.

Experimental design: Fischer 344 rats (10/sex/group) were administered 0, 0.23, 0.46, 0.93, 1.9, or 3.7 mg Hg/kg/day as mercuric chloride in deionized water by oral gavage once daily 5 days per week for 26 weeks. Body weights were recorded weekly. Surviving animals were sacrificed and necropsied. Organ weights were determined for the brain, heart, liver, lung, kidney, thymus, and testes. Histopathological examinations were performed.

Effects noted in study and corresponding doses: The relative and absolute kidney weights were significantly increased for dosed males and for females exposed to at least 0.46 mg/kg/day. At the two low-dose groups and the control group, minimal nephropathy was observed in nearly all the males. At 0.93 mg/kg/day level, renal tubule necrosis became more severe (moderate) and was statistically significant and remained at this severity at the higher dose groups. The female rats had a significant increased incidence at the high dose only, and severity was minimal. Nephropathy was characterized by foci of tubular regeneration, thickened tubular basement membrane, and scattered dilated tubules containing hyaline casts. Macroscopic changes included granular kidneys in dosed males. After 4 months of exposure, urinary levels of alkaline phosphatase, aspartate aminotransferase, lactate dehydrogenase, and gamma-glutamyl transferase were significantly elevated in both sexes at 3.7 mg Hg/kg/day, but at 6 months control levels had increased such that enzyme levels in males were no longer statistically significant and only levels of alkaline phosphatase and gamma-glutamyl transferase were significantly elevated in females.

Dose end point used for MRL derivation: 0.23 mg Hg/kg/day; no renal effects
[X] NOAEL [ ]LOAEL

Uncertainty and modifying factors used in MRL derivation: 100

[ ] 1 [ ] 3 [ ] 10 (for use of a LOAEL)
[ ] 1 [ ] 3 [X] 10 (for extrapolation from animals to humans)
[ ] 1 [ ] 3 [X] 10 (for human variability)

Was a conversion factor used from ppm in food or water to a mg/body weight dose?
If so explain: No conversion factor used.

Was a conversion used from intermittent to continuous exposure?

If so, explain: Yes. The dose was adjusted for a continuous exposure by multiplying the NOAEL (0.23 mg/kg/day) by a conversion factor of 5/7:

$NOAEL_{(ADJ)}$ = 0.23 mg/kg/day x (5 days/7 days)
= 0.16 mg/kg/day

MRL = $NOAEL_{(ADJ)}$ ÷ UF = 0.16 mg/kg/day ÷ 100 = 0.002 mg/kg/day

If an inhalation study in animals, list conversion factors used in determining human equivalent dose:
Not applicable.

Additional studies or pertinent information that lend support to this MRL: Renal toxicity has been observed in other intermediate-duration oral studies on rats and mice exposed to inorganic mercury (Carmignani et al. 1992; Jonker et al. 1993a; NTP 1993), as well as case reports on humans ingesting inorganic mercury for acute and chronic durations (Afonso and deAlvarez 1960; Davis et al. 1974; Kang-Yum and Oransky 1992; Nielsen et al. 1991; Pesce et al. 1977).

Agency Contact (Chemical Manager): John Risher

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Methylmercury
CAS Number: 22967-92-6
Date: June 15, 2001
Profile Status: Final Draft
Route: [ ] Inhalation [X] Oral
Duration: [ ] Acute [ ] Intermediate [X] Chronic
Key to figure: 88
Species: Human

Minimal Risk Level: 0.0003 [X] mg/kg/day [ ] mg/m^3

Reference: Davidson et al. 1998. Effects of prenatal and postnatal methylmercury exposure from fish consumption on neurodevelopment: Outcomes at 66 months of age in the Seychelles Child Development Study. JAMA 280(8):701-707.

Experimental design. This MRL is based on the results of the Seychelles Child Development Study (SCDS), a series of evaluations on a population in the Seychelles Islands. The chronic oral MRL for methylmercury is based upon the Seychelles Child Development Study (SCDS), in which over 700 mother-infant pairs have, to date, been followed and tested from parturition through 66 months of age (Davidson et al. 1998). The SCDS was conducted as a double-blind study and used maternal hair mercury as the index of fetal exposure. Enrollees were recruited by the head nurse/hospital midwife by asking the mothers if they wished to participate in the study when they arrived at the hospital for delivery. The first 779 who did not decline participation became the mothers in the study cohort. Of the initial 779 mothers enrolled in the study at parturition, 740 remained at the predetermined child testing age of 6.5 months, 738 remained in the 19-month cohort, 736 remained at 29 months, and 711 remained for the 66-month neurobehavioral and developmental examinations.

The Seychellois were chosen as a study population for a number of reasons. (1) All fish contain some level of methylmercury (Davidson et al. 1998); and the Seychellois regularly consume a large quantity and variety of ocean fish, with 12 fish meals per week representing a typical methylmercury exposure. (2) The median total mercury concentration in 350 fish sampled from 25 species consumed by the Seychellois was <1 ppm (range, 0.004–0.75 ppm), comparable to that consumed by the U.S. population; thus, the methylmercury levels in the Seychellois population are 10–20 times those in the United States, not because they consume more highly contaminated fish than do Americans, but rather because they consume more fish than the U.S. population. (3) The Seychelles represent a relatively pristine environment, with no local industry for pollution, and are situated more than 1,000 miles from any continent or large population center. (4) The population is highly literate, cooperative, and has minimal immigration and emigration. (5) The Seychellois constitute a generally healthy population, with low maternal alcohol consumption and tobacco use (<2%). (6) In the 66-month study cohort, the mean maternal hair level of total mercury during pregnancy was 6.8 ppm (range, 0.5–26.7 ppm).

Effects noted in study and corresponding doses: The results of the 66-month testing in the SCDS revealed no evidence of adverse effects attributable to chronic ingestion of low levels of methylmercury in fish (Davidson et al. 1998). In this study, developing fetuses were exposed *in utero* through maternal fish ingestion before and during pregnancy (Davidson et al. 1998). Neonates continued to be exposed to maternal mercury during breastfeeding (i.e., some mercury is secreted in breast milk), and methylmercury

exposure from the regular diet continued after the gradual post-weaning shift to a fish diet. In the 66-month study cohort, the mean maternal hair level of total mercury during pregnancy was 6.8 ppm (range, 0.5–26.7 ppm; n = 711), and the mean child hair level at the 66-month testing interval was 6.5 ppm (range, 0.9–25.8 ppm; n = 708). The 66-month test battery, which was designed to test multiple developmental domains, included as primary measures the following: (1) General Cognitive Index (GCI) of the McCarthy Scales of Children's Abilities (to estimate cognitive ability); (2) the Preschool Language Scale (PLS) total score (to measure both expressive and receptive language ability); (3) the Letter and Word Recognition and (4) Applied Problems subtests of the Woodcock-Johnson (W-J) Tests of Achievement (to measure reading and arithmetic achievement); (5) the Bender-Gestalt test (to measure visual-spatial ability); and (6) the total T score from the Child Behavior Checklist (CBCL) (to measure the child's social and adaptive behavior). Serum sampling revealed no detectable levels of PCBs (detection limit = 0.2 ng/mL).

None of the tests indicated an adverse effect of methylmercury exposure. In contrast, four of the six measures showed better scores in the highest MeHg-exposed groups, compared with lower exposure groups for both prenatal and postnatal exposure (the four test were the (1) General Cognitive Index (GCI) of the McCarthy Scales of Children's Abilities (to estimate cognitive ability); (2) the Preschool Language Scale (PLS) total score (to measure both expressive and receptive language ability); (3) the Letter and Word Recognition and (4) Applied Problems subtests of the Woodcock-Johnson (W-J) Tests of Achievement (to measure reading and arithmetic achievement). While the positive outcomes are not considered to indicate any beneficial effect of methylmercury on neurological development or behavior, they might be more appropriately attributed to the beneficial effects of omega-3 fatty acids or other constituents present in fish tissue, since the methylmercury levels in hair are known to correlate closely with fish intake. The slight decreases in the subjectively reported activity level of boys reported in the 29-month observations were not seen during the 66-month tests. The mean maternal hair level of 15.3 ppm in the group with the highest exposure in the 66-month test cohort is, therefore, considered a NOAEL for SCDS, and is used by ATSDR as the basis for derivation of a chronic oral MRL for methylmercury. A related study (Myers et al. 1997) by the same team of researchers from the University of Rochester examined the Seychellois children for attainment of the same developmental milestones reported to have been delayed in the Iraqi poisoning incident in the early 1970s (Cox et al. 1989) and found no such delays in the Seychellois children exposed in utero. Since the children had been exposed *in utero*, they represent the most sensitive subpopulation.

Sensitivity of Neurobehavioral Measures /Reliability of Tests Used in Critical Study

The neurobehavioral test battery used in the 66-month Seychelles study was designed to assess multiple developmental domains (Davidson et al. 1998). The tests were considered to be sufficiently sensitive and accurate to detect neurotoxicity in the presence of a number of confounding factors. On-site test administration reliability was assessed by an independent scorer, and mean interclass correlations for interscorer reliability were 0.96–0.97 (Davidson et al. 1998). The sample size was determined to be sufficient to detect a 5.7 point difference on any test with a mean (SD) of 100 (16) between low (0–3 ppm) and high >12 ppm) hair mercury concentration groups for a 2-sided test (A = 0.05 at 80% power).

Converting blood concentration to daily intake.

The concentration of mercury in the blood may be converted to a daily intake by using the following equation from WHO (1990):

$$C = \frac{f(d}{b(V} = \frac{A_D(A_B(d}{b(V}$$

Where:

C	=	concentration in blood
f	=	fraction of the daily intake taken up by the blood
d	=	daily dietary intake
b	=	elimination constant
A_D	=	percent of mercury intake in diet that is absorbed
A_B	=	percent of the absorbed amount that enters the blood
V	=	volume of blood in the body

Hair to Blood Concentration Ratio.

The hair:blood concentration ratio for total mercury is frequently cited as 250. However, a precise basis for this particular value is unclear. Ratios reported in the literature range from 140 to 370, a difference of more than a factor of 2.5 (see Table 2-9). Differences in the location of hair sampled (head versus chest, distance of sample from head or skin) may contribute to differences in observed ratios between studies. For example, as much as a 3-fold seasonal variation in mercury levels was observed in average hair levels for a group of individuals with moderate-to-high fish consumption rates, with yearly highs occurring in the fall and early winter (Phelps et al. 1980; Suzuki et al. 1992). Thus, it is important to obtain hair samples as close to the follicle as possible to obtain an estimate of recent blood levels. Large errors (the direction of which depends on whether samples were taken while blood levels were falling or rising) could result if hair samples are not taken close to the scalp. Several studies did not report the distance to the scalp for the hair samples taken. The high slope reported by Tsubaki (1971a) may have reflected the fact that mercury levels were declining at the time of sampling (Berglund et al. 1971), so the hair levels may reflect earlier, higher blood levels. Hair taken from different parts of the body also may yield different ratios. In 26 subjects with moderate-to-high fish consumption, axillary hair (i.e., from the armpit area) was found to contain an average of 23% less mercury than head hair (Skerfving et al. 1974).

Phelps et al. (1980) obtained multiple blood samples and sequentially analyzed lengths of hair from 339 individuals in Northwestern Ontario. The large sample size and the attention to sampling and analysis with regard to the hair:blood relationship make this study the most appropriate to use for estimating the mercury blood levels of the Seychellois women during pregnancy. The actual ratio Phelps et al. (1980) observed between the total mercury concentration in hair taken close to the scalp and simultaneous blood sampling for this group was 296. To estimate the actual ratio, the authors assumed that blood and hair samples were taken following complete cessation of methylmercury intake. They also assumed a half-life of methylmercury in blood of 52 days and a lag of 4 weeks for appearance of the relevant level in hair at the scalp. Based on these assumptions, they calculated that if the actual hair:blood ratio were 200, they would have observed a ratio of 290 (i.e., essentially equivalent to the observed value of 296). Based on these and other considerations, Phelps et al. (1980) state that the actual ratio is "probably higher than 200, but less than the observed value of 296." As the authors point out, two-thirds of the study population were sampled

during the falling phase of the seasonal variation and one-third or less in the rising phase. This fact would tend to result in a lower observed ratio; therefore, the actual average value is likely to be >200.
Phelps et al. (1980) also provide estimates assuming a 2-week lag for the appearance of the relevant level of mercury in the centimeter of hair nearest the scalp. For a 2-week lag time, an actual ratio of 250 would have resulted in an observed ratio of 301 (again, essentially identical to the observed value of 296). A study of ingestion of a large dose of mercuric chloride in one individual suggests that the lag time is longer than 2 weeks (Suzuki et al. 1992). Hair samples were taken at 41 and 95 days following ingestion of the mercuric chloride. In the 41-day hair sample, a large mercury peak occurred in the centimeter of hair closest to the scalp, with no elevation in mercury in the second centimeter of hair. Head hair grows at a rate of about 1.1 cm a month (Al-Shahristani and Shihab 1974; Cox et al. 1989). If emergence had occurred so that the elevation in mercury could be measured in the first centimeter of hair by 2 weeks after exposure, then by day 41 after exposure the peak should have moved into the second centimeter of hair, at least enough to raise the mercury level slightly in the second centimeter. Because no elevation was seen in the second centimeter of hair at 41 days, it would appear that emergence occurred at a lag of >2 weeks. In the hair sample taken at 95 days, the leading edge of the mercury peak occurred in the third centimeter of hair.

Based on the data presented in Phelps et al. (1980) and the lag time indicated in the individual studied by Suzuki et al. (1992), the actual average value is likely to be somewhere between 200 and 250. Because the data do not allow a more accurate determination of an average ratio, the value 250 is acceptable for the purpose of estimating average blood levels in the Seychellois population. Using 250 rather than a lower number results in a lower MRL. It should be noted that a wide range in hair:blood ratios has been reported for individuals in various studies: 137–342 in Soria et al. (1992), 171–270 in Phelps et al. (1980), and 137–585 in Birke et al. (1972). Therefore, this ratio (250) should not be used as the sole basis for determining levels of exposure and potential effect for individuals.

Calculation of dietary intake from blood concentration.

Fraction of mercury in diet that is absorbed (A_D). Radiolabeled methyl-mercuric nitrate was administered in water to three healthy volunteers (Aberg et al. 1969). The uptake was >95%. Miettinen et al. (1971) incubated fish liver homogenate with radiolabeled $MeHgNO_3$ to yield a methylmercury proteinate. The proteinate was then fed to fish that were killed after a week, cooked, and fed to volunteers after confirmation of the methylmercury in the fish. Mean uptake exceeded 94%. For the derivation of an MRL, an absorption factor of 0.95 is used.

Fraction of the absorbed dose that is found in the blood (A_B). The value 0.05 has been used for this parameter in the past (Berglund et al. 1971; WHO 1990). Three studies report observations of the fraction of the absorbed methylmercury dose distributed to blood volume in humans. Kershaw et al. (1980) report an average fraction of 0.059 of the absorbed dose in the total blood volume, based on a study of 5 adult male subjects who ingested methylmercury-contaminated tuna. In a group of 9 male and 6 female volunteers who had received ^{203}Hg-methylmercury in fish, approximately 10% of the total body burden was present in 1 L of blood in the first few days after exposure, dropping to approximately 5% over the first 100 days (Miettinen et al. 1971). In another study, an average value of 1.14% for the percentage of absorbed dose in 1 kg of blood was derived from subjects who consumed a known amount of methylmercury in fish over a period of 3 months (Sherlock et al. 1984). Average daily intake for the 4 groups observed in the study ranged from 43 to 233 µg/day. The authors report a dose-related effect on the estimated percentage of the absorbed dose in 1 kg of blood, with 1.26% of the absorbed dose in 1 kg of blood at an average daily intake of 43 µg/day and 1.03% of the absorbed dose in 1 kg of blood at an average daily intake of 233 µg/day. The average for all subjects in the study was 1.14%. When individual values for distribution to one kilogram of blood reported in the study are converted into the percentage of the absorbed

dose in the total blood volume (assuming that blood is 7% of body weight [Best 1961] and using body weights reported for individuals in the study), the average value for A_B for all individuals is 0.056 (0.057 using the values for percentage in 1 kg normalized for body weight as reported in the study). The average value for A_B for 6 women as reported in Sherlock et al. (1984) is 0.048 (0.047 using values normalized for body weight). The average for 14 men is 0.059 (0.061 using values normalized for body weight).

The average values for A_B for all studies ranged from 0.047 to 0.061 (the values for women and men reported in Sherlock et al. [1984]). The data suggest that the average value of A_B for women may be lower than that for men, and they further suggest that 0.05 may be appropriate for modeling intake in a group of women (Sherlock et al. 1984). Based on these studies, the best estimate of A_B based on the available data is 0.05. Use of a higher value (i.e., 0.06 instead of 0.05) for this parameter would result in a lower MR, but the sensitive populations are pregnant women and developing fetuses, making the 0.5 value more appropriate for the Seychelles study population.

Elimination constant (b). Reported clearance half-times for methylmercury from blood or hair range from 48 to 65 days (Table 2-5). The average elimination constant based on the 6 studies listed in Table 2.5 is 0.014. The average of the individual values for *b* reported for 20 volunteers ingesting from 42 to 233 µg Hg/day in fish for 3 months (Sherlock et al. 1984) is also 0.014. Use of the value 0.014 for this parameter, rather than 0.01 (as used by WHO 1990), results in a higher MRL.

Volume of blood in body (V), and body weight. Blood volume is assumed to be 7% of body weight, with an increase to about 9% during pregnancy (Best 1961). Data for the body weight of the Seychelles Islands women were not found. Assuming an average body weight of 60 kg for women, the blood volume is 4.2 L (60 kg x 0.07 L/kg).

Calculation of Exposure Dose

The concentration of mercury in hair is assumed to be 250 times the concentration in blood. Using the mean total mercury level of 15.3 ppm in maternal hair taken at parturition to represent a NOAEL in the 66-month Seychelles testing (Davidson et al. 1998), the corresponding methylmercury concentration in blood would be: 1/250 x 15.3 µg/g x 1 mg/1,000 µg x 1,000 g/L = 0.061 mg/L.

Calculation of Daily Intake from Blood Concentration

$$C = \frac{f(d}{b(V} = \frac{A_D(A_B(d}{b(V}$$

Using the above equation to relate the concentration in blood (C, in µg/L) to daily intake (d, in µg/day): where C = (percent of ingested dose absorbed through the GI tract x percent of that dose absorbed into the blood x the daily amount ingested) divided by (elimination constant x blood volume in a 60 kg female)

that is,

C = (0.95 x 0.05 x d)/(0.014 x 4.2)
C = 0.81 d
0.061 mg/L = 0.81 d
d = 0.075 mg/day

Using the assumed body weight of 60 kg for women, the estimated dose that would result in a hair level of 15.3 ppm is 0.075/60 kg = 0.0013 mg/kg/day. Therefore, the NOAEL derived from the highest exposure group (n = 95) at 66 months is 0.0013 mg/kg/day.

Dose and end point used for MRL derivation: 0.0013 mg/kg/day NOAEL

[X] NOAEL [ ] LOAEL

Uncertainty and Modifying Factors used in MRL derivation:

[ ] 1 [ ] 3 [ ] 10 (for use of a minimal LOAEL)
[ ] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [X] 3 [ ] 10 (for human pharmacokinetic and pharmacodynamic variability)
[X] 1.5 [ ] 3 [ ] 10 (Modifying factor to account for domain-specific findings in Faroe study)

Consideration of Uncertainty

The standard/traditional areas of uncertainty addressed in any duration-specific MRL are: (1) interspecies variability (i.e., cross-species extrapolation of a NOAEL or LOAEL); (2) intra-human variability (i.e., differences in susceptibility to a substance or effect within the human population); (3) use of an LOAEL for MRL derivation when an NOAEL for the critical effect is not available; and (4) extrapolation from subchronic to chronic duration. In addition, a modifying factor may also be used when special circumstances exist that may contribute to, or introduce, uncertainty into the calculated health guidance value (MRL) in an area not typically covered by the traditional uncertainty factor approach.

The NOAEL of 15.3 ppm mercury in maternal hair from Davidson et al. (1998) used as the starting point for MRL derivation was based upon an unusually large study cohort of the population considered most sensitive to the neurodevelopmental effects of methylmercury, i.e., pregnant women and their developing fetuses. The negative results of this study are strongly supported by the BMD NOAEL range of 13 to 21 ppm calculated for the New Zealand cohort of 237 mother-child pairs (Crump et al. 1998). Consequently, much of the uncertainty normally present in the MRL derivation process does not exist in the case of methylmercury. Nonetheless, in view of the nature of the most susceptible group (developing fetuses) and some questions raised in the vast human data base for this chemical, an aggregate value of 4.5 was employed.

This value (4.5) was based upon three separate components, two of which are interrelated and the other independent. For the Seychelles data, a value of 1.5 was used to address the variability in hair-to-blood ratios among women and fetuses in the U.S. population, as determined by pharmacokinetic modeling of actual data by Clewell et al. (1998); a second value of 1.5 was applied to address the remainder of any inter-individual variability (i.e., pharmacodynamics) in the U.S. population. A third, and independent, factor of 1.5 was employed to account for the possibility that the domain-specific tests, as employed extensively in the Faroe Islands, but not the Seychelles (which used primarily neurobehavioral tests of global function) might be able to detect very subtle neurological effects not tested for in the 66-month Seychelles cohort.

The World Health Organization (WHO, 1993, 1996) has defined the -kinetic and -dynamic components of intrahuman variability as being equal contributors to, and collectively constituting the total of, human variability. In order to ensure a conservative approach, these two interdependent components were added to give a composite uncertainty factor of three (i.e., 1.5 + 1.5 = 3) to account for the full range of variability attributable to mercury in the Seychelles study. A modifying factor of 1.5 was also used to account for the

possibility of domain-specific effects, as were seen in the Faroe study, being attributable to mercury. Since these effects were considered to be entirely separate or "independent" events, this modifying factor of 1.5 was multiplied by the uncertainty factor of 3.0 (for uncertainty attributable solely to the Seychelles study) to yield an aggregate uncertainty of 4.5 for chronic oral exposure to methylmercury.

While domain-specific tests from the Seychelles were reviewed at the North Carolina meeting in November 1998 and the results failed to demonstrate effects, the tests do not represent the full range of domain-specific tests that were administered in the Faroe Islands. For these reasons, and based on our consultation with our Board of Scientific Counselors about concerns for "missing" data sets (i.e., in relation to the Executive Order of children's health and the agency's efforts to protect the health of children, including the developing fetus), ATSDR determined that an additional factor of 1.5 should be used since the full range of domain-specific neuropsychological test results from the Seychelles are not yet available. When these results become available and if they fail to show domain-specific effects, this additional factor of 1.5 would no longer be needed. At that time ATSDR will re-evaluate its MRL, as well as all other relevant data, in compliance with the agency's mandates and authorities.

Therefore, in the calculation of the chronic oral MRL for methylmercury, the NOAEL of 0.0013 mg/kg/day from the 66-month study (Davidson et al. 1998) is divided by 4.5, giving a chronic oral MRL for methylmercury of 0.0003 mg/kg/day [0.0013 mg/kg/day / 4.5 (UF) = 0.0003 mg/kg/day].

<u>If an inhalation study in animals, list the conversion factors used in determining human equivalent concentration (HEC)</u>: Not applicable.

<u>Additional studies or pertinent information which lend support to this MRL</u>:

Crump et al. (1998) conducted benchmark dose (BMD) calculations and additional regression analyses of data collected in a study in which a series of scholastic and psychological tests were administered to children whose mothers had been exposed to methylmercury during pregnancy. Hair samples were collected from 10,970 new mothers in New Zealand in 1977 and 1978. High hair mercury levels were considered to be those over 6 ppm, which was the hair level predicted to result at steady state from consumption of mercury at the WHO/FAO Provisional Tolerable Weekly Intake of 0.3 mg total mercury/week and 0.2 mg methylmercury/week. By this criterion, 73 of approximately 1,000 mothers who had consumed fish more than three times/week during pregnancy were determined to have high hair mercury levels. In 1985, when the children were 6 to 7 years of age, 61 children (1 set of twins) of the 73 mothers in the high hair mercury group were located, and constituted the high exposure group, which was matched with three control groups (one with 3-6 ppm maternal hair mercury levels, one with 0-3 ppm whose mothers had been high fish consumers, and one with 0-3 ppm whose mothers had not been high fish consumers). The entire study cohort consisted of 237 children. A battery of 26 psychological and scholastic tests were administered to the children at school during the year 1985. Mothers were interviewed at the time of test administration to obtain additional data on social and environmental factors. In the high exposure group of children, one boy's mother had a hair mercury level of 86 ppm, which was more than four times higher than the next highest hair mercury level of 20 ppm. BMDs (10% response rate) calculated from five tests ranged from 32 to 73 ppm, when the 86 ppm mother's child was included. This corresponded to a BMDL range of 17 to 24 ppm. Although none of the 86 ppm child's test scores was an outlier according to the definition used in the analyses, his scores were significantly influential in the analyses. When this child was omitted from the analyses, BMDs ranged from 13 to 21, with corresponding BMDLs of 7.4 to 10 ppm.

Developing fetuses in the SCDS were exposed through maternal fish ingestion before and during pregnancy. Each child was evaluated at 19 months and again at 29 months (±2 weeks) for infant intelligence (Bayley

Scales of Infant Development [BSID] Mental and Psychomotor Scales), with a modified version of the BSID Infant Behavior Record to measure adaptive behaviors at 29 months (Davidson et al. 1995b). Testing was performed by a team of Seychellois nurses extensively trained in administration of the BSID. Maternal hair concentrations, measured in hair segments that corresponded to pregnancy, ranged from 0.5 to 26.7 ppm, with a median exposure of 5.9 ppm for the entire study group. The mean BSID Mental Scale Indices determined at both 19 and 29 months were found to be comparable to the mean performance of U.S. children. The BSID Psychomotor Scale Indices at both measurement intervals were two standard deviation units above U.S. norms, but were still consistent with previous findings of motor precocity in children reared in African countries. The study found no effect that could be attributed to mercury on the BSID scores obtained at either the 19- or 29-month measurement/testing interval. The 29-month cohort represented 94% of the 779 mother-infant pairs initially enrolled in the study, and approximately 50% of all live births in the Seychelles in 1989.

The only observation in the 29-month testing that might be attributable to prenatal mercury exposure was a slight decrease in the activity level in boys (but not girls) as determined by the Bayley Infant Behavior Record (subjective observation). Whereas this decrease was significant in males ($p = 0.0004$), it was not statistically significant in females ($p = 0.87$). When the subjective activity scores for male and female children were evaluated collectively, no statistically significant or remarkable decrease in activity was apparent outside the >12 ppm maternal hair concentration group. The affect on activity level in boys is not considered an adverse effect by the authors of the study.

Grandjean et al. (1997b, 1998) reported another epidemiological study of methylmercury exposure for a population in the Faroe Islands. Although the Faroese are a fishing culture, the major source of methylmercury exposure for this population is pilot whale meat, which is intermittently consumed as part of the cultural tradition . The initial study cohort consisted of 1,022 singleton births occurring in a 21-month window during 1986-1987. At approximately 7 years of age, neurobehavioral testing was conducted on 917 of the remaining cohort members. No abnormalities attributable to mercury were found during clinical examinations or neurophysiological testing. A neuropsychological test battery was also conducted, which included the following: Finger Tapping; Hand-Eye Coordination; reaction time on a Continuous Performance Test; Wechsler Intelligence Scale for Children - Revised Digit Spans, Similarities, and Block Designs; Bender Visual Motor Gestalt Test; Boston Naming Test; and California Verbal Learning Test (Children). Neuropsychological tests emphasized motor coordination, perceptual-motor performance, and visual acuity. Pattern reversal visual evoked potentials (VEP) with binocular full-field stimulation, brain stem auditory evoked potentials (BAEP), postural sway, and the coefficient of variation for R-R inter-peak intervals (CVRR) on the electrocardiogram were all measured. The neuropsychological testing indicated mercury-related dysfunction in the domains of language, attention, memory, and visuospatial and motor function (to a lesser extent), which the authors considered to remain after the children of women with maternal hair mercury concentrations above 10 µg/g (10 ppm) were excluded. While this study represents a significant contribution to the human database for methylmercury exposure and effects, a number of potentially influential factors not fully considered as possible covariates somewhat cloud the interpretation of the results.

These differences between the neuropsychological effects observed in the Faroe Island cohort and the absence of effects reported in the Seychelles Island cohort might result from a variety of factors. The Faroe Island children were older (7–8 years versus 5.5 in the SCDS). Some of the measurement instruments (i.e., the neuropsychological test administered) were also different. Since the first neuropsychological testing in the Faroe study was not conducted until 7 years of age, it is not known whether the observed effects might have been apparent at an earlier age. Ongoing and planned future testing of the Seychelles population will provide additional information on the progression of any observed effects. Further examination of the

Seychelles population using the neuropsychological test that showed positive results in the Faroe Islands population will also allow a more direct comparison of results.

The diet in the two studies was also considerably different. The majority of the mercury exposure to the Faroe Island population came from whale meat (estimated at about 3 ppm in muscle tissue) with a relatively small portion coming from fish. Some of the mercury in whale meat is in the form of inorganic mercury. In the Seychelles study, all of the mercury came from fish as methylmercury with concentrations of around 0.3 ppm. Whale meat blubber is widely consumed in the Faroe Islands and also contains polychlorinated biphenyls (PCBs). Grandjean et al. (1995b) estimated a daily intake of 200 µg of PCB. This value can be compared to the Tolerable Daily Intake of PCBs established by the FDA, of 60–70 µg/day for an adult. Further statistical analysis of the possible influence of PCBs on the observed study results needs to be conducted (see the discussion below on Peer Panel 1Review of Key Studies for additional comments).

The primary biomarker used to estimate mercury exposure was also different between the two studies. The Faroe Island analysis used cord blood, and the Seychelles study used maternal hair level. The use of mercury in cord blood has the advantage of being a more direct measure of exposure to the fetus, but the levels at term may not reflect exposures at earlier developmental stages. While Grandjean et al. (1997) did report maternal hair mercury levels, the mean hair level for the interquartile range of 2.6–7.7 ppm was reported only as a geometric average (4.27 ppm). In contrast, the Seychellois study reported only an arithmetic mean level for the entire study population (6.8 ppm). While both are valid measures, a direct comparison of “average” values for the two studies is not possible without further statistical analysis of both data sets.

In the case of the Faroe study, there were no data presented in the peer-reviewed publications to address variability of food/whale meat or blubber intake among the Faroe Islanders, making it difficult to evaluate the possibility of peak intake levels during critical development phases. Consumption data was reported only as <1 pilot whale meat meal/month and 1-2 fish meals per week. In contrast, the Seychelles dietary habits provide a relatively stable intake, and a high degree of correlation was found between mean hair levels in samples covering each trimester versus levels in samples for the entire pregnancy (Cernichiari et al. 1995a). Cernichiari et al. (1995b) also report a good correlation between levels of total mercury in neonatal brain and levels in the corresponding maternal hair. While the contribution of continued mercury exposure through breast feeding or post-weaning diet was not fully addressed in the Seychellois study reports (Davidson 1995, 1998), that is not considered a significant drawback with the study, since no effects on neurobahavioral/neuropsychological testing were seen at any maternal hair level. In the Faroese assessment of latent neuropsychological effects from an *in utero* exposure to mercury, however, the role of continuing postnatal exposure to mercury either from breast milk or from ingestion of methylmercury-containing foods (e.g., pilot whale meat) is less clear. Specifically, it is not known what proportion, if any, of the neuropsychological effects reported in the Faroe Island population could be attributed to seven years of postnatal exposure to methylmercury in food. The variability and magnitude of this postnatal exposure should, therefore, be further evaluated.

Peer Panel Review of Key Studies

In addition to the traditional peer review process that precedes publication in most scientific journals, the studies considered by ATSDR for use in estimating a chronic oral MRL for methylmercury underwent two stringent reviews by recognized experts in the environmental health field.

On July 20 and 21, 1998, ATSDR assembled a panel of 18 experts from the scientific and medical communities to review current issues and the relevant literature on mercury and its compounds, including

methylmercury (ATSDR 1999). Several members of each of the respective research teams that conducted the Iraqi, Seychelles, Faroe, and Madeira studies were included among the expert panelists, and provided extensive overviews of their studies. The presentations were followed by an open, wide- ranging scientific discussion of the merits and interpretations of the currently available studies. Topics of significant discussion included the relative merits of the respective study populations, exposure regimens, sensitivity of neurobehavioral measures, and determination of an uncertainty factor. While it was unanimously agreed that the Seychelles and Faroe studies were both excellent studies that provided a significant contribution to the human database for methylmercury exposure and effects, a number of factors that could have contributed to the study results, but were not considered as possible statistical covariates, were discussed. In the case of the Faroe study, the consumption of whale blubber, which is known to be contaminated with PCBs, DDT, and possibly other organochlorines, introduces a potentially significant influence on the study results. Weihe et al. (1996) reported that the PCB and DDT concentrations in blubber of pilot whales taken in Faroese waters are about 30 ppm and 20 ppm, respectively. In contrast, the Seychellois population does not eat marine mammals at all. In addition, the Faroe study did not address other possible statistical covariates, such as the dietary and nutritional status of the study population and the use of tobacco during pregnancy, further complicating the interpretation of the neuropsychological test results.

On November 18–20, 1998, a workshop on Scientific Issues Relevant to the Assessment of Health Effects from Exposure to Methylmercury was conducted in Raleigh, North Carolina. Jointly sponsored by the U.S. Department of Health and Human Services, the National Institute of Environmental Health Sciences (NIEHS), the Centers for Disease Control and Prevention (CDC), the Food and Drug Administration (FDA), the U.S. Environmental Protection Agency (EPA), the National Oceanic and Atmospheric Administration (NOAA), the Office of Science and Technology Policy (OSTP), the Office of Management and Budget (OMB), and ATSDR, the purpose of this workshop was to discuss and evaluate the major epidemiologic studies that associated methylmercury exposure and the results of an array of developmental measures in children. These studies monitored and evaluated exposed populations in Iraq, the Seychelles Islands, the Faroe Islands, and the Amazon River Basin. A number of animal studies were also considered in support of a human health risk assessment. Presentation of these studies by the research team that conducted the study was followed by an expert panel evaluation that examined each study, taking into consideration the exposure data, experimental design and statistical analysis, potential confounders and variables, and neurobehavioral endpoints evaluated. A fifth panel evaluated the results of relevant animal studies. Significant issues that were discussed included the use of umbilical cord blood mercury levels vs. hair mercury concentrations as an index of methylmercury exposure during pregnancy, the patterns of exposure, the dietary/health status of study populations, other potentially relevant exposures, other confounding influences, and the adjustments made for statistical covariates. All five panels at this workshop commended the efforts of the investigators and respective staffs of the Seychelles and Faroe studies for conducting highly sophisticated investigations under difficult conditions. However, specific findings of several of the panels raise issues that, at present, preclude the Faroe data from consideration as a starting point for MRL derivation.

In their addressal of the potential influence of concurrent PCB exposure on the Faroe results, the Confounders and Variables (Epidemiology) panel indicated that with respect to four of the pre-natal outcomes (related primarily to verbal and memory performance), when PCBs were included in the model, only one of these outcomes is specifically related to mercury exposure. Concerning this matter, the panel wrote that "... the most likely explanation is that both (mercury and PCBs)... affect these three outcomes, but their relative contributions cannot be determined given their concurrence in this population." The Neurobehavioral Endpoints Panel also looked at this issue, and noted that "PCB exposure might act as an effect modifier, increasing the susceptibility to MeHg."; however, this panel further indicated that it did not believe that the effects seen in the Faroe Islands were due to uncontrolled confounding by PCBs. A third

panel that addressed the issue of concurrent PCB exposures, the Statistics/Design Panel, noted that only 3 of 208 PCB congeners were measured in the Faroe study, and stated that it "seems likely that mercury was measured more accurately than the biologically relevant PCB exposure. Consequently even if the neurological effects seen in this study were caused entirely by PCBs, it is possible that mercury would still be more highly correlated with these effects than PCBs." The Statistics/Design Panel also said that "the best method to deal with this problem would be to study a population where exposure to PCBs is not an issue." This statement points directly to the Seychelles study as the study most appropriate for MRL derivation.

Another issue raised at Raleigh workshop concerned the taking of hair samples for determining pre-natal exposure. In the Seychelles, hair samples were collected 6 months post-partum, and segments corresponding to pregnancy were selected for analysis. In the case of the Faroese, hair samples were taken at the scalp. Regarding that, the Confounders and Variables (Epidemiology) panel stated that "Given the time it takes the Hg to be excreted into the hair, we can assume that samples collected at parturition do not cover the last 6 weeks of gestation, during which critically important neuronal proliferation and differentiation is taking place."

Regarding both the Seychelles and Faroe studies, the Neurobehavioral Endpoints Panel found "no specific neurobehavioral signature injury from MeHg" in the data from either study (Seychelles or Faroe). The same panel also noted that episodic exposure in the Faroe Islands (1–2 fish meals/week and <1 pilot whale meal/month) "may reduce the likelihood of detecting a consistent 'neurobehavioral signature injury' specific to MeHg and may account for different observations in children with the same average exposure."

Based upon the discussions at the Raleigh workshop and the individual panel findings, as well as the aforementioned Atlanta expert panel review, ATSDR has determined the Seychellois study to represent the most appropriate and reliable data base currently available for calculation of a chronic oral MRL from a population exposed only to methylmercury by a relevant route of exposure for the overall U.S. population.

[It should be emphasized that the Seychelles study and the Faroe study represent credible scientific contributions by widely respected research teams. Similarly, both studies extend our knowledge base well beyond that provided by the Iraqi study and make significant contributions to our understanding of the effects of low-level exposure to methylmercury by an exposure route and vehicle (i.e., food) relevant to U.S. populations. The continuing monitoring and evaluation of the Seychellois and Faroese populations with more comparable neurobehavioral indices should help strengthen our understanding of the effects of low level chronic methylmercury exposure and should reduce the uncertainty regarding the public health implications of exposure.]

Other epidemiology studies were also considered by the workshop panels. Lebel et al. (1997) evaluated a fish-eating populations in the Amazon River Basin with a neurofunctional test battery and clinical manifestations of nervous system dysfunction in relation to hair mercury concentrations. The villagers examined live along the Tapajos River, a tributary of the Amazon. The study population consisted of 91 adult inhabitants 15-31 years of age. Hair mercury levels were below 50 µg/g (ppm). Clinical examinations were essentially normal, although persons displaying disorganized movements on an alternating movement task and those with restricted visual fields generally had higher hair mercury levels. Near visual contrast, sensitivity, and manual dexterity (adjusted for age) were found to decrease significantly with increasing mercury levels, while a tendency for muscular fatigue and decreasing strength were observed in women. The authors suggested that dose-dependent nervous system alterations might be associated with hair mercury levels below 50 ppm. This study, however, also had a number of potentially confounding factors. The impact of parasitic and other diseases endemic to the study area is of primary

concern in the interpretation of the Lebel et al. (1997) results. In addition, the overall nutritional status of the study population was not known or reported, and the use of neuroactive drugs (from local herbs, plants, roots, or mushrooms) was not considered as a potential confounder or covariate. The previous mercury exposure history of the study cohort was also unclear. This is of particular importance because gold mining procedures that use metallic mercury have been commonly practiced along the Amazon Basin for decades. Finally, the endpoints of the Lebel et al. (1977) study evaluated adult toxicity and not effects in the developing fetus or the newborn (i.e., the most sensitive human population).

The panel also reviewed the Iraqi study. Cox et al. (1989) and WHO (1990) reported delayed onset of walking in offspring in Iraqi children whose mothers were exposed to methylmercury through the consumption of seed grain treated with methylmercury as a fungicide (Al-Mufti et al. 1976; Bakir et al. 1973; Cox et al. 1989; Marsh et al. 1981, 1987). Exposure to methylmercury from other sources (e.g., fish or meat) was probably very low or nonexistent (Al-Mufti et al. 1976). It is likely that the children were exposed both prenatally through the placenta and postnatally through the mother's milk. A maternal exposure level of 0.0012 mg/kg/day, corresponding to the hair level of 14 ppm, was estimated using a simple, one-compartment pharmacokinetic model.

Myers et al. (1997) evaluated the population of the SCDS for developmental milestones similar to those determined in Iraq. As part of this ongoing study, cohort children were evaluated at 6.5, 19, 29, and 66 months of age. At 19 months care-givers were asked at what age the child walked (n=720 out of 738) and talked (n=680). Prenatal mercury exposure was determined by atomic absorption analysis of maternal hair segments corresponding to hair growth during the pregnancy. The median mercury level in maternal hair for the cohort in this analysis was 5.8 ppm, with a range of 0.5–26.7 ppm. The mean age (in months) at walking was 10.7 (SD=1.9) for females and 10.6 (SD=2.0) for males. The mean age for talking (in months) was 10.5 (SD=2.6) for females, and 11.0 (SD=2.9) for males. After adjusting for covariates and statistical outliers, no association was found between the age at which Seychellois children walked or talked and prenatal exposure to mercury. The ages for achievement of the developmental milestones were normal for walking and talking in the Seychellois toddlers following prenatal exposure to methylmercury from a maternal fish diet. The 5.8 ppm NOAEL of this study is considerably below the one estimated from the dose-response analysis of the data for the Iraqi methylmercury poisonings (10 ppm).

Clarkson (1995) raised some interesting issues concerning whether is it reasonable to apply health effects data based on an acute exposure to methylmercury fungicide eaten in homemade bread (in the 1971–1972 Iraq incident) to fish-eating populations having chronic exposure to much lower concentrations of methylmercury. Clarkson (1995) addressed two specific issues. The first regards the body's "defense mechanisms" that serve to mitigate the potential damage from mercury. One such mechanism in the case of methylmercury involves an enterohepatic cycling process in which methylmercury from dietary sources absorbed through the intestine is carried to the liver, where substantial quantities are secreted back into the bile and returned to the intestinal tract. During the residence time in the gut, microflora break the carbon-mercury bond, converting methylmercury into inorganic mercury, which in turn is poorly absorbed and is excreted in the feces. This creates an effective detoxification pathway for low-dose dietary exposures to methylmercury, but probably not for acute, high-dose exposures, such as occurred in Iraq. Secondly, the transport of methylmercury into brain tissue is inhibited by the presence of many amino acids, including leucine, methionine, and phenylalanine. Thus, it is possible that the rising plasma concentrations of amino acids from ingestion of fish protein may serve to depress the uptake of methylmercury by the brain.

While both of these issues need further laboratory/clinical investigation, they do raise appropriate questions concerning the relevance of the relatively short-term (i.e., about six weeks), high-level contaminated grain exposure scenario encountered in Iraq to the dietary methylmercury exposure scenarios encountered in many

fish-eating populations (e.g., the Seychelles Islanders, Faroe Islanders, Peruvian villagers, and Inuit native people of Greenland). This position is supported by Cicmanec (1996), who reviewed data from the Iraqi study, as well as data from studies of fish-consuming populations in the Faroe Islands, Seychelles Islands, and Peruvian fishing villages. Cicmanec concluded that the Iraqi population does not represent a sensitive subpopulation within a perinatal group; rather, the relative lower threshold identified in that study was the result of confounders. Crump et al. (1995) reanalyzed the dose-response data from the Cox et al. (1989) report of the Iraqi incident and found the results to be potentially skewed by inadequacies in the study design and data-collection methods. Shortcomings or potentially confounding factors include: (1) the retrospective recall of developmental milestones by mothers and other family members; (2) the lack of precision in the determination of birth and other milestone dates; (3) and the possible biasing of the dose-response analysis by variation in symptom reporting and infant sex composition in the two study subcohorts. Crump et al. (1995) noted that perhaps the most serious limitation of the Iraqi study is the inability to assess the potential effects of low-level chronic-duration exposure to methylmercury, as these particular data are based on very high intake levels over a relatively brief period of time.

No increase in the frequency of neurodevelopmental abnormalities in early childhood was observed in a cohort of 131 infant-mother pairs in Mancora, Peru (Marsh et al. 1995b). The mean concentration of mercury in maternal hair was determined to be 8.3 ppm (range, 1.2–30 ppm), and the source of the mercury was believed to be from consumption of marine fish. Similarly, a study of 583 Faroe Island infants for the first 12 months after birth found no decrease in the age of attainment of sitting, creeping (crawling), and standing developmental milestones (Grandjean et al. 1995a). The age at which a child reached a particular developmental milestone was not only not found to be associated with prenatal mercury exposure, but infants that reached a milestone early were found to have significantly higher mercury concentrations in their hair at 12 months of age. It was also found that early milestone attainment was clearly associated with breast-feeding, which was in turn related to higher infant hair mercury levels. The authors (Grandjean et al. 1995a) concluded that the beneficial effects associated with breast-feeding seemed to overrule, or to compensate for, any neurotoxic effects on milestone development that could be due to the presence of contaminants (e.g., mercury) in human milk.

Additional studies have shown developmental toxicity after oral exposure of humans and animals to organic mercury compounds (Amin-Zaki et al. 1974; Bakir et al. 1973; Bornhausen et al. 1980; Cagiano et al. 1990; Elsner 1991; Engleson and Herner 1952; Fowler and Woods 1977; Guidetti et al. 1992; Harada 1978; Hughes and Annau 1976; Ilback et al. 1991; Inouye and Kajiwara 1988; Khera and Tabacova 1973; Lindstrom et al. 1991; McKeown-Eyssen et al. 1983; Nolen et al. 1972; Olson and Boush 1975; Rice 1992; Rice and Gilbert 1990; Snyder and Seelinger 1976; Stoltenburg-Didinger and Markwort 1990).

The accumulation of mercury is greater in larger fish and in fish higher in the food chain. The tendency for increased mercury concentration with increasing fish body weight is particularly noticeable in carnivorous fish species. Malm et al. (1995) analyzed mercury concentrations in 16 species of carnivorous fish from the Tapajos River basin in Brazil and hair samples from local populations who regularly ate such fish. Mercury levels in the fish averaged 0.55 ppm (range, 0.04–3.77 ppm), and the mercury levels in the hair of the affected fish-eating populations averaged approximately 25 ppm. In one population that consumed higher quantities of large carnivorous fish at the end of the local rainy season, 8 of 29 persons evaluated had hair mercury levels above 40 ppm, and one individual had a hair mercury concentration of 151 ppm. Some villages along the river can have per capita daily fish consumption rates around 200 g or more, which would greatly impact the human body burden and hair levels of mercury in such populations.

Alternative Derivations of the MRL

To ensure a health guidance value based upon the best use of the Seychelles study data (widely considered the most relevant data available), ATSDR evaluated alternate MRL derivation methods for methylmercury.

One such method was a physiologically based pharmacokinetic approach using the mean total mercury level of 6.8 ppm in maternal hair for the entire Seychellois study cohort. Using the same formula as in the previous MRL calculation,

$$C = (0.95 \times 0.05 \times d) / (0.014 \times 4.2)$$
$$C = 0.81\ d$$
$$(1/250 \times 6.8) = 0.027$$
$$0.027\ \text{mg/L} = 0.81\ d$$
$$d = 0.034\ \text{mg/day}$$
$$0.034\ \text{mg/day} / 60\ \text{kg} = 0.0006\ \text{mg/kg/day}$$

In consideration of uncertainty factors for this MRL approach, multiple factors also apply. In this case, the mean value of 6.8 ppm for the NOAEL is for the entire study cohort at 66 months (n = 711). An uncertainty factor of 1.5 was used to account for the pharmacokinetically based variability of hair-to-blood ratios (95% confidence level) in pregnant women and fetuses in the U.S. population (Clewell et al. 1998, 1999). The extremely large size of the study population (n=711), in combination with an uncertainty factor of 1.5, is considered adequate to encompass the full range of pharmacokinetic and pharmacodynamic variability within the human population. An independent modifying factor of 1.5 was also used to take into consideration the positive results of the domain-specific tests administered in the Faroe study (Grandjean et al. 1997, 1998). The uncertainty factor of 1.5, multiplied by the modifying factor of 1.5, yields a total aggregate value of 2.25. Applying the factor of 2.25 to the daily intake calculated from the 6.8 ppm NOAEL yields a chronic oral MRL value of 0.0003 mg/kg/day for methylmercury (0.0006 mg/kg/day divided by 2.25 = 0.0003 mg/kg/day).

A third approach to deriving a health guidance value is the use of bench mark dose (BMD) modeling. Clewell et al. (1998) used a benchmark dose analysis to determine a reference dose (RfD, a health guidance value used by the Environmental Protection Agency and, in some ways, the equivalent of ATSDR's chronic oral MRL). Clewell et al. (1998) used the data from the 29-month test in the Seychellois population (Davidson et al. 1995b) for their analysis (i.e., the 66-month study had not been published at the time of their benchmark dose analysis). The BMD is calculated by fitting a mathematical dose-response model to dose-response data. The bench mark dose level (BMDL) is a lower statistical confidence bound on the BMD and replaces the NOAEL in the calculation of a health guidance value. The BMD approach has been proposed as superior to the use of "average" or "grouped" exposure estimates when dose-response information is available, as is the case for the Seychelles study. Clewell et al. (1998) note that the Faroe Islands study reported by Grandjean et al. (1997b) could not be used for dose-response modeling due to inadequate reporting of the data and the confounding influence of co-exposure to PCBs.

For the 29-month Seychelles data, Clewell et al. (1998) used the 95% lower bound on the 10% benchmark dose level (BMDL), which represents a conservative estimate of the traditional NOAEL. The benchmark dose modeling over the entire range of neurological endpoints reported by Davidson et al. (1995b) yielded a lowest $BMDL_{10}$ of 21 ppm methylmercury in maternal hair. This $BMDL_{10}$ was then converted to an expected distribution of daily ingestion rates across a population of U.S. women of child-bearing age by using a Monte Carlo analysis with a physiologically based pharmacokinetic (PBPK) model of methylmercury developed by Gearhart et al. (1995). This analysis addresses the impact of interindividual

pharmacokinetic variability on the relationship between ingestion rate and hair concentration for methyl-mercury. The resulting distribution had a geometric mean value of 0.00160 mg/kg/day (S.D. 0.00133). The 1st, 5th, and 10th percentiles of that distribution were 0.00086, 0.00104, and 0.00115 mg/kg/day, respectively. Clewell et al. (1998) suggested that the 5th percentile of 0.00104 mg/kg/day provides a scientifically based, conservative basis that incorporates the pharmacokinetic variability across the U.S. population of child-bearing women and that no other uncertainty factor for interindividual variability would be needed. To the benchmark-estimated NOAEL of 21 ppm derived from the Seychelles 29-month data, Clewell et al. (1998) applied an uncertainty factor of 3 to account for data base limitations. (Note: The 66-month Seychelles data was not yet published at the time; hence the reliance on the 29-month Seychelles data for the benchmark analysis.) Consequently, Clewell et al. (1998) concluded that using a NOAEL of 7 ppm (21 ppm / 3 (UF) provides additional protection against the possibility that effects could occur at lower concentrations in some populations. Based upon this reasoning, they recommended a health guidance value (i.e., an RfD) of 0.0004 mg/kg/day. If a modifying factor of 1.5 is used to further address the domain-specific findings in the Faroe study, a final MRL of 0.3 µg/kg/day results.

The above benchmark analysis of 29-month data from the Seychelles Child Development Study strongly supports the MRL of 0.0003 mg/kg/day calculated by ATSDR in this profile. Similarly, addressing the Seychellois 66-month data from the perspective of using the mean value (15.3 ppm) of the highest exposure group in the study, a method prescribed in ATSDR's published guidance for MRL development (Chou et al. 1998), also results in an identical MRL. ATSDR therefore has high confidence that this level is protective of the health of all potentially exposed human populations.

Agency Contact (Chemical Manager): John F. Risher

APPENDIX B

USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in non-technical language. Its intended audience is the general public especially people living in the vicinity of a hazardous waste site or chemical release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the chemical.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Tables and Figures for Levels of Significant Exposure (LSE)

Tables (2-1, 2-2, and 2-3) and figures (2-1 and 2-2) are used to summarize health effects and illustrate graphically levels of exposure associated with those effects. These levels cover health effects observed at increasing dose concentrations and durations, differences in response by species, minimal risk levels (MRLs) to humans for noncancer end points, and EPA's estimated range associated with an upper- bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. Use the LSE tables and figures for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of No-Observed-Adverse- Effect Levels (NOAELs), Lowest-Observed-Adverse-Effect Levels (LOAELs), or Cancer Effect Levels (CELs).

The legends presented below demonstrate the application of these tables and figures. Representative examples of LSE Table 2-1 and Figure 2-1 are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See LSE Table 2-1

(1) Route of Exposure One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. When sufficient data exists, three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 2-1, 2-2, and 2-3, respectively). LSE figures are limited to the inhalation (LSE Figure 2-1) and oral (LSE Figure 2-2) routes. Not all substances will have data on each route of exposure and will not therefore have all five of the tables and figures.

(2) Exposure Period Three exposure periods - acute (less than 15 days), intermediate (15–364 days), and chronic (365 days or more) are presented within each relevant route of exposure. In this example, an inhalation study of intermediate exposure duration is reported. For quick reference to health effects occurring from a known length of exposure, locate the applicable exposure period within the LSE table and figure.

(3) Health Effect The major categories of health effects included in LSE tables and figures are death, systemic, immunological, neurological, developmental, reproductive, and cancer. NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer. Systemic effects are further defined in the "System" column of the LSE table (see key number 18).

(4) Key to Figure Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to derive a NOAEL and a Less Serious LOAEL (also see the 2 "18r" data points in Figure 2-1).

(5) Species The test species, whether animal or human, are identified in this column. Section 2.5, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 2.3, "Toxicokinetics," contains any available information on comparative toxicokinetics. Although NOAELs and LOAELs are species specific, the levels are extrapolated to equivalent human doses to derive an MRL.

(6) Exposure Frequency/Duration The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to 1,1,2,2-tetrachloroethane via inhalation for 6 hours per day, 5 days per week, for 3 weeks. For a more complete review of the dosing regimen refer to the appropriate sections of the text or the original reference paper, i.e., Nitschke et al. 1981.

(7) System This column further defines the systemic effects. These systems include: respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not covered in these systems. In the example of key number 18, 1 systemic effect (respiratory) was investigated.

(8) NOAEL A No-Observed-Adverse-Effect Level (NOAEL) is the highest exposure level at which no harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of 3 ppm for the respiratory system which was used to derive an intermediate exposure, inhalation MRL of 0.005 ppm (see footnote "b").

(9) LOAEL A Lowest-Observed-Adverse-Effect Level (LOAEL) is the lowest dose used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse health effects first appear and the gradation of effects with increasing dose. A brief description of the specific endpoint used to quantify the adverse effect accompanies the LOAEL. The respiratory effect reported in key number 18 (hyperplasia) is a Less serious LOAEL of 10 ppm. MRLs are not derived from Serious LOAELs.

(10) Reference The complete reference citation is given in chapter 8 of the profile.

(11) CEL A Cancer Effect Level (CEL) is the lowest exposure level associated with the onset of carcinogenesis in experimental or epidemiologic studies. CELs are always considered serious effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report doses not causing measurable cancer increases.

(12) Footnotes Explanations of abbreviations or reference notes for data in the LSE tables are found in the footnotes. Footnote "b" indicates the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

LEGEND

See Figure 2-1

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the reader quickly compare health effects according to exposure concentrations for particular exposure periods.

(13) Exposure Period The same exposure periods appear as in the LSE table. In this example, health effects observed within the intermediate and chronic exposure periods are illustrated.

(14) Health Effect These are the categories of health effects for which reliable quantitative data exists. The same health effects appear in the LSE table.

(15) Levels of Exposure concentrations or doses for each health effect in the LSE tables are graphically displayed in the LSE figures. Exposure concentration or dose is measured on the log scale "y" axis. Inhalation exposure is reported in mg/m^3 or ppm and oral exposure is reported in mg/kg/day.

(16) NOAEL In this example, 18r NOAEL is the critical endpoint for which an intermediate inhalation exposure MRL is based. As you can see from the LSE figure key, the open-circle symbol indicates to a NOAEL for the test species-rat. The key number 18 corresponds to the entry in the LSE table. The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).

(17) CEL Key number 38r is 1 of 3 studies for which Cancer Effect Levels were derived. The diamond symbol refers to a Cancer Effect Level for the test species-mouse. The number 38 corresponds to the entry in the LSE table.

(18) Estimated Upper-Bound Human Cancer Risk Levels This is the range associated with the upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are derived from the EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1*).

(19) Key to LSE Figure The Key explains the abbreviations and symbols used in the figure.

SAMPLE

1 6

TABLE 2-1. Levels of Significant Exposure to [Chemical x] – Inhalation

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	LOAEL (effect) Less serious (ppm)	LOAEL (effect) Serious (ppm)	Reference
2 6 INTERMEDIATE EXPOSURE							
	5	6	7	8	9		10
3 6 Systemic	9	9	9	9	9		9
4 6 18	Rat	13 wk 5d/wk 6hr/d	Resp	3[b]	10 (hyperplasia)		Nitschke et al. 1981
CHRONIC EXPOSURE							
					11		
Cancer					9		
38	Rat	18 mo 5d/wk 7hr/d			20	(CEL, multiple organs)	Wong et al. 1982
39	Rat	89–104 wk 5d/wk 6hr/d			10	(CEL, lung tumors, nasal tumors)	NTP 1982
40	Mouse	79–103 wk 5d/wk 6hr/d			10	(CEL, lung tumors, hemangiosarcomas)	NTP 1982

[a] The number corresponds to entries in Figure 2-1.

12 6 [b] Used to derive an intermediate inhalation Minimal Risk Level (MRL) of $5x10^{-3}$ ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).

CEL = cancer effect level; d = days(s); hr = hour(s); LOAEL = lowest-observed-adverse-effect level; mo = month(s); NOAEL = no-observed-adverse-effect level; Resp = respiratory; wk = week(s)

Chapter 2 (Section 2.5)

Relevance to Public Health

The Relevance to Public Health section provides a health effects summary based on evaluations of existing toxicologic, epidemiologic, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1. What effects are known to occur in humans?

2. What effects observed in animals are likely to be of concern to humans?

3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The section covers end points in the same order they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, dermal) and within route by effect. Human data are presented first, then animal data. Both are organized by duration (acute, intermediate, chronic). *In vitro* data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this section. If data are located in the scientific literature, a table of genotoxicity information is included.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. Minimal risk levels (MRLs) for noncancer end points (if derived) and the end points from which they were derived are indicated and discussed.

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information is available, we have derived minimal risk levels (MRLs) for inhalation and oral routes of entry at each duration of exposure (acute, intermediate, and chronic). These MRLs are not meant to support regulatory action; but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans. They should help physicians and public health officials determine the safety of a community living near a chemical emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicologic information on which the number is based. Chapter 2.5, "Relevance to Public Health," contains basic information known about the substance. Other sections such as 2.8, "Interactions with Other Substances," and 2.9, "Populations that are Unusually Susceptible" provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses for lifetime exposure (RfDs).

To derive an MRL, ATSDR generally selects the most sensitive endpoint which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential systemic, neurological, and developmental effects. If this information and reliable quantitative data on the chosen endpoint are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest NOAEL that does not exceed any adverse effect levels. When a NOAEL is not available, a lowest-observed-adverse-effect level (LOAEL) can be used to derive an MRL, and an uncertainty factor (UF) of 10 must be employed. Additional uncertainty factors of 10 must be used both for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the LSE Tables.

APPENDIX C

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH	American Conference of Governmental Industrial Hygienists
ADI	Acceptable Daily Intake
ADME	Absorption, Distribution, Metabolism, and Excretion
AFID	alkali flame ionization detector
AFOSH	Air Force Office of Safety and Health
AML	acute myeloid leukemia
AOAC	Association of Official Analytical Chemists
atm	atmosphere
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
BAT	Best Available Technology
BCF	bioconcentration factor
BEI	Biological Exposure Index
BSC	Board of Scientific Counselors
C	Centigrade
CAA	Clean Air Act
CAG	Cancer Assessment Group of the U.S. Environmental Protection Agency
CAS	Chemical Abstract Services
CDC	Centers for Disease Control and Prevention
CEL	Cancer Effect Level
CELDS	Computer-Environmental Legislative Data System
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
Ci	curie
CL	ceiling limit value
CLP	Contract Laboratory Program
cm	centimeter
CML	chronic myeloid leukemia
CNS	central nervous system
CPSC	Consumer Products Safety Commission
CWA	Clean Water Act
d	day
Derm	dermal
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DNA	deoxyribonucleic acid
DOD	Department of Defense
DOE	Department of Energy
DOL	Department of Labor
DOT	Department of Transportation
DOT/UN/ NA/IMCO	Department of Transportation/United Nations/ North America/International Maritime Dangerous Goods Code
DWEL	Drinking Water Exposure Level

ECD	electron capture detection
ECG/EKG	electrocardiogram
EEG	electroencephalogram
EEGL	Emergency Exposure Guidance Level
EPA	Environmental Protection Agency
F	Fahrenheit
F_1	first-filial generation
FAO	Food and Agricultural Organization of the United Nations
FDA	Food and Drug Administration
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FPD	flame photometric detection
fpm	feet per minute
ft	foot
FR	*Federal Register*
g	gram
GC	gas chromatography
Gd	gestational day
gen	generation
GLC	gas liquid chromatography
GPC	gel permeation chromatography
HPLC	high-performance liquid chromatography
hr	hour
HRGC	high resolution gas chromatography
HSDB	Hazardous Substance Data Bank
IDLH	Immediately Dangerous to Life and Health
IARC	International Agency for Research on Cancer
ILO	International Labor Organization
in	inch
IRIS	Integrated Risk Information System
Kd	adsorption ratio
kg	kilogram
kkg	metric ton
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{Lo}	lethal concentration, low
LC_{50}	lethal concentration, 50% kill
LD_{Lo}	lethal dose, low
LD_{50}	lethal dose, 50% kill
LT_{50}	lethal time, 50% kill
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
m	meter
MA	*trans,trans*-muconic acid
MAL	Maximum Allowable Level
mCi	millicurie
MCL	Maximum Contaminant Level

MCLG	Maximum Contaminant Level Goal
mg	milligram
min	minute
mL	milliliter
mm	millimeter
mm Hg	millimeters of mercury
mmol	millimole
mo	month
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NAAQS	National Ambient Air Quality Standard
NAS	National Academy of Science
NATICH	National Air Toxics Information Clearinghouse
NATO	North Atlantic Treaty Organization
NCE	normochromatic erythrocytes
NCI	National Cancer Institute
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
NFPA	National Fire Protection Association
ng	nanogram
NLM	National Library of Medicine
nm	nanometer
NHANES	National Health and Nutrition Examination Survey
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPD	nitrogen phosphorus detection
NPDES	National Pollutant Discharge Elimination System
NPL	National Priorities List
NR	not reported
NRC	National Research Council
NS	not specified
NSPS	New Source Performance Standards
NTIS	National Technical Information Service
NTP	National Toxicology Program
ODW	Office of Drinking Water, EPA
OERR	Office of Emergency and Remedial Response, EPA
OHM/TADS	Oil and Hazardous Materials/Technical Assistance Data System
OPP	Office of Pesticide Programs, EPA
OPPTS	Office of Prevention, Pesticides and Toxic Substances, EPA
OPPT	Office of Pollution Prevention and Toxics, EPA
OSHA	Occupational Safety and Health Administration
OSW	Office of Solid Waste, EPA
OTS	Office of Toxic Substances
OW	Office of Water
OWRS	Office of Water Regulations and Standards, EPA

PAH	Polycyclic Aromatic Hydrocarbon
PBPD	Physiologically Based Pharmacodynamic
PBPK	Physiologically Based Pharmacokinetic
PCE	polychromatic erythrocytes
PEL	permissible exposure limit
PID	photo ionization detector
pg	picogram
pmol	picomole
PHS	Public Health Service
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PSNS	Pretreatment Standards for New Sources
REL	recommended exposure level/limit
RfC	Reference Concentration
RfD	Reference Dose
RNA	ribonucleic acid
RTECS	Registry of Toxic Effects of Chemical Substances
RQ	Reportable Quantity
SARA	Superfund Amendments and Reauthorization Act
SCE	sister chromatid exchange
sec	second
SIC	Standard Industrial Classification
SIM	selected ion monitoring
SMCL	Secondary Maximum Contaminant Level
SMR	standard mortality ratio
SNARL	Suggested No Adverse Response Level
SPEGL	Short-Term Public Emergency Guidance Level
STEL	short-term exposure limit
STORET	Storage and Retrieval
TD_{50}	toxic dose, 50% specific toxic effect
TLV	threshold limit value
TOC	Total Organic Compound
TPQ	Threshold Planning Quantity
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TRI	Toxics Release Inventory
TWA	time-weighted average
U.S.	United States
UF	uncertainty factor
VOC	Volatile Organic Compound
yr	year
WHO	World Health Organization
wk	week
$>$	greater than
$\geq$	greater than or equal to
$=$	equal to

<	less than
≤	less than or equal to
%	percent
α	alpha
β	beta
γ	gamma
δ	delta
µm	micrometer
µg	microgram
q_1^*	cancer slope factor
–	negative
+	positive
(+)	weakly positive result
(–)	weakly negative result

http://www.epa.gov/mercury/exposure.htm
Last updated on Monday, September 24th, 2007.

Mercury

You are here: EPA Home Mercury Human Exposure

Human Exposure

Mercury exists in various forms, and people are exposed to each in different ways. The most common way people in the U.S. are exposed to mercury is by eating fish containing methylmercury. Other exposures may result from using or breaking products containing mercury. The health effects of these exposures are discussed in a separate section.

- Methylmercury
- How mercury enters the environment
- Moving up the food chain
- Elemental mercury
- Other mercury compounds (inorganic and organic)

People who use mercury in the workplace need to take special precautions.

Information for...

Businesses
Consumers
Health Care Providers
Parents
Schools

Basic Information

Frequent Questions
Health Effects
Fish Consumption Advisories

Methylmercury exposure

Outbreaks of methylmercury poisoning have made it clear that adults, children, and developing fetuses are at risk from dietary exposure to methylmercury. During these poisoning outbreaks some mothers with no symptoms of nervous system damage gave birth to infants with severe disabilities and it became clear that the developing nervous system of the fetus may be more vulnerable to methylmercury than is the adult nervous system. Mothers who are exposed to methylmercury and breast-feed their babies may also expose their infant children through their milk.

In 2004 EPA and FDA issued the first-ever joint consumer advice about methylmercury in fish and shellfish. This advice was for women who might become pregnant; women who are pregnant; nursing mothers; and young children. The advisory provides three recommendations for selecting and eating fish or shellfish to ensure that women and young children will receive the benefits of eating fish and shellfish and be confident that they have reduced their exposure to the harmful effects of methylmercury. EPA also hosts a web-based compilation of fish advisories issued by States, tribes, territories and local governments. Fish Consumption Advisories

Recent human biological monitoring by the Centers for Disease Control and Prevention in 1999 and 2000 (PDF) (3 pp., 42 KB, About PDF) shows that most people have blood mercury levels below a level (5.8 µg/L of whole blood) associated with possible health effects. Consumption of fish with higher methylmercury levels can lead to elevated levels of mercury in the bloodstream of unborn babies and young children and may

Terms Defined

Reference Dose (RfD): An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive

harm their developing nervous system. These disabilities have been documented in ability to use language, to process information, and in visual/motor integration. U.S. EPA's 2001 Reference Dose (RfD) for methylmercury was calculated to protect the developing nervous system. Currently, U.S. EPA uses a RfD of 0.1 µg/kg body weight/day as an exposure without recognized adverse effects. A description of EPA's Reference Dose for methylmercury may be found at http://www.epa.gov/iris/subst/0073.htm.

In U.S. EPA's Mercury Study Report to Congress (1997) EPA estimated that 7% of women of childbearing age would have blood mercury concentrations greater than those equivalent to the RfD. The estimate of 7% of women of childbearing age above the RfD was based on patterns of fish and shellfish consumption and methylmercury concentrations present in fish and shellfish. Blood mercury analyses in the 1999-2000 National Health and Nutrition Examination Survey (1999-2000 NHANES) for 16-to-49 year old women showed that approximately 8% of women in the survey had blood mercury concentrations greater than 5.8 ug/L (which is a blood mercury level equivalent to the current RfD). Based on this prevalence for the overall U.S. population of women of reproductive age and the number of U.S. births each year, it is estimated that more than 300,000 newborns each year may have increased risk of learning disabilities associated with in utero exposure to methylmercury. More recent data from the CDC support this general finding.

Nearly all methylmercury exposures in the U.S. occur through eating fish and shellfish. Microscopic organisms convert inorganic mercury into methylmercury, which accumulates up the food chain in fish, fish-eating animals, and people.

This process is explained below.

- How mercury enters the environment
- Moving up the food chain

subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data used. Generally used in EPA's noncancer health assessments.

No-Observed-Adverse-Effect Level (NOAEL): The highest exposure level at which there are no biologically significant increases in the frequency or severity of adverse effect between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered adverse or precursors of adverse effects.

Lowest-Observed-Adverse-Effect Level (LOAEL): The lowest exposure level at which there are biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group

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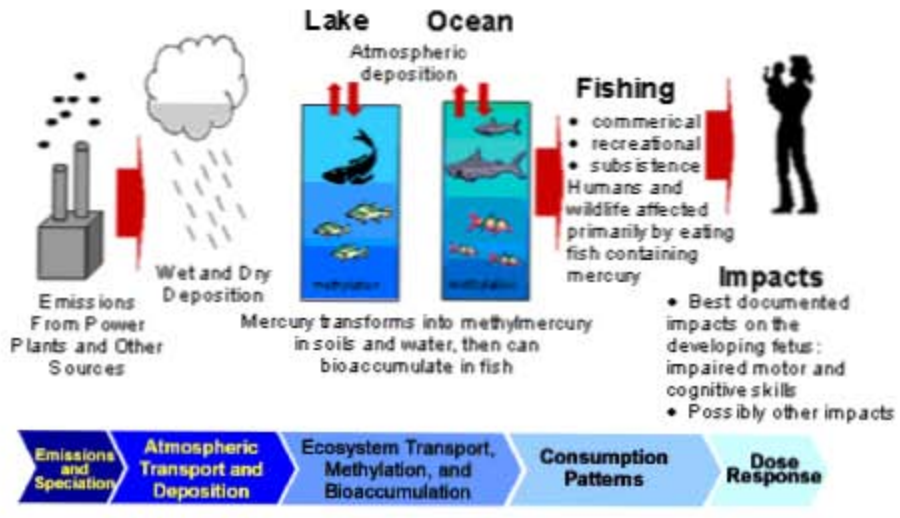
How mercury enters the environment



Mercury is emitted to the air by human activities, such as manufacturing or burning coal for fuel, and from natural sources, such as volcanos.

Typically, mercury is released into the atmosphere in one of three forms:

- elemental mercury: can travel a range of distances, may remain in the atmosphere up to one year and may travel globally before undergoing transformation
- particle-bound mercury: can fall out of the air over a range of distances
- oxidized mercury (sometimes called ionic or reactive gaseous mercury (RGM)): found predominantly in water-soluble forms, which may be deposited at a range of distances from sources depending on a variety of factors including topographic and meteorologic conditions downwind of a source.

What happens to mercury after it is emitted depends on several factors:

- the form of mercury emitted
- the location of the emission source
- how high above the landscape the mercury is released (e.g., the height of the stack)
- the surrounding terrain
- the weather.

Depending on these factors, atmospheric mercury can be transported over a range of distances before it is deposited, potentially resulting in deposition on local, regional, continental and/or global scales. Mercury that remains in the air for prolonged periods of time and travels across continents is said to be in the "global cycle."

Recent emissions estimates of annual global mercury emissions from all sources, natural and anthropogenic (human-generated), which are highly uncertain, are about 4800-8300 tons per year.

U.S. anthropogenic mercury emissions are estimated to account for roughly 3 percent of the total global emissions, and the U.S. power sector is estimated to account for about 1 percent the total global emissions. EPA has estimated that about one third of U.S. emissions are deposited within the contiguous U.S. and the remainder enters the global cycle.

Current estimates are that less than half of all mercury deposition within the U.S. comes from U.S. sources, although deposition varies by geographic location. For example, compared to the country as a whole, U.S. sources represent a greater fraction of the total deposition in

parts of the Northeast because of the direction of the prevailing winds.

Moving up the food chain

When mercury falls in rain or snow, it may flow into bodies of water like lakes and streams. When it falls out of the air as dry deposition, it may eventually be washed into those bodies by rain. Bacteria in soils and sediments convert mercury to methylmercury. In this form, it is taken up by tiny aquatic plants and animals. Fish that eat these organisms build up methylmercury in their bodies. As ever-bigger fish eat smaller ones, the methylmercury is concentrated further up the food chain. This process is called "bioaccumulation".

Methylmercury concentrations in fish depend on many factors, including mercury, the concentration in water, water pH and temperature, the amount of dissolved solids and organic matter in the water, and what organisms live in the water. Methylmercury concentrations in fish may also be affected by the presence of sulfur and other chemicals in the water. Because of these variables, and because food webs are very complex, bioaccumulation is hard to predict and can vary from one water body to another.

However, in a given water body, the highest concentrations of methylmercury are generally found in large fish that eat other fish. The concentrations of methylmercury in large fish can be over a million-fold larger than in the surrounding water. EPA discussions of estimates bioaccumulation can be found in Chapter 6 and Appendix A of the Water Quality Criterion for the Protection of Human Health: Methylmercury.

Elemental mercury exposure

When elemental mercury is spilled or a device containing mercury breaks, the exposed elemental mercury can evaporate and become an invisible, odorless toxic vapor. This is especially true in warm or poorly-ventilated rooms or spaces. Sources of potential exposure to elemental mercury are described below.

- Elemental or metallic mercury is the liquid metal used in thermometers, barometers, and thermostats and other electrical switches. Metallic mercury is often found in school laboratories as well as in thermometers, barometers, switches, thermostats, and other devices found in school science labs.

- It is not uncommon for children to break fever thermometers in their mouths. Mercury that is swallowed in such cases poses low risk comparison to the risk of breathing mercury vapor.

- There are some necklaces imported from Mexico that contain a glass pendant that contains mercury. The mercury-containing pendants can come in various shapes such

as hearts, bottles, balls, saber teeth, and chili peppers. If broken, they release metallic mercury to the environment.

Mercury is used in dentistry in dental amalgam. Dental amalgam is a direct filling material used in restoring teeth. It is made up of approximately 40-50% mercury, 25% silver and 25-35% a mixture of copper, zinc and tin. Amalgam use is declining because the incidence of dental decay is decreasing and because improved substitute materials are now available for certain applications. The Centers for Disease Control and Prevention (CDC) reports that, at present, there is scant evidence that the health of the vast majority of people with dental amalgam is compromised, nor that removing amalgam fillings has a beneficial effect on health. More information is available at:
http://www.cdc.gov/oralhealth/factsheets/amalgam.htm

Ritual Use of Mercury - Persons who use metallic mercury in ethnic folk medicine and for religious practices may be at risk of exposure to mercury. Metallic mercury is sold under the name "azogue"in stores (sometimes called botanicas), which specialize in religious items used in Esperitismo (a spiritual belief system native to Puerto Rico), Santeria (a Cuban-based religion that venerates both African deities and Catholic saints), and voodoo. The use of azogue in religious practices is recommended in some Hispanic communities by family members, spiritualists, card readers, and santeros. Typically, azogue is carried on one's person in a sealed pouch prepared by a spiritual leader or sprinkled in the home or automobile.

Exposure to other mercury compounds (inorganic and organic)

Inorganic mercury compounds take the form of mercury salts. They are generally white powders or crystals, with the exception of mercuric sulfide (cinnabar) which is red. Inorganic compounds and organic compounds (such as phenylmercury acetate and ethylmercury), have been commonly used as fungicides, antiseptics or disinfectants. They have also been used in a variety of products. Most of these uses have been discontinued, but small amounts of these compounds can still be found as preservatives in some medicines. The U.S. Food and Drug Administration maintains a list of medicines that contain mercury.

Excessive exposure to inorganic and organic mercury compounds can result from misuse or overuse of mercury-containing products, especially outdated products containing more mercury. Exposure to mercury compounds is primarily through ingestion, but can occur through other pathways. Ingested organic mercury compounds are more readily absorbed through the gastrointestinal tract than are inorganic compounds.

Methylmercury: Epidemiology Update

Kathryn R. Mahaffey, Ph.D.
U.S. Environmental Protection Agency
Washington D.C.

Fish Forum – San Diego - 2004

Reports in 2003/2004 . . .

- 1999-2000 NHANES organic blood Hg
- *Close association with fish intake in 1999-2000 NHANES examinees.*
- Confirmation of cord blood [Hg] : adult blood [Hg] in Japanese.
- *Estimate at least 300,000 newborns in US each year with in utero blood [Hg] greater than 5.8 µ/L.*

Reports in 2003/2004 (*continued*)

- Seychelles cohort update.
- *Methylmercury-associated adult neuro-psychological changes at hair [Hg] < 50 ppm.*
- Distribution of omega-3 fatty acids (EPA and DHA) in fish and shellfish vs. [Hg] in fish and shellfish.

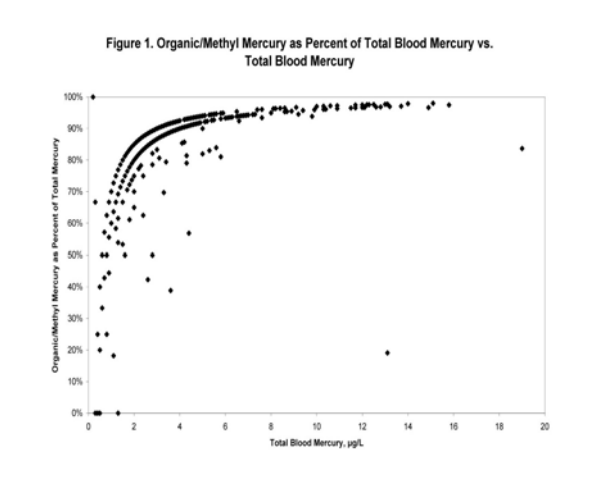
1999-2000 NHANES Blood Mercury

- Blood organic mercury (i.e., methylmercury) among 1709 women of childbearing age representative of US population.
- *Overall, 9% of women consumed fish at least once a week. Fish consumption higher among women over age 30 and among Asians and people of "Island" ethnicity.*

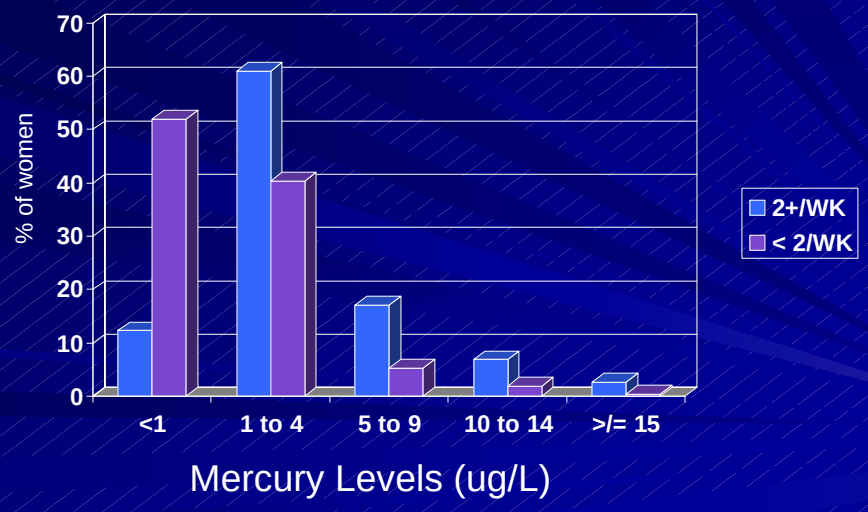
1999-2000 NHANES Blood Mercury

- Association: R = 0.5 to 0.6 between dietary total mercury and blood organic mercury (Mahaffey et al., 2003).
- Blood mercury concentrations were 7 X higher among women who reported eating 9+ fish/shellfish meals within past 30 days (i.e., 2 or more times per week) compared with women who reported no fish/shellfish consumption in the past 30 days (Mahaffey et al., 2003).

Methylmercury as a Percent of Total Blood Mercury: 1999-2000 NHANES Adult Women of Childbearing Age



Total Mercury Levels in Women, Aged 16-49 by Weekly Fish Consumption Levels



Basis for Uncertainty Factor of 10 in the Reference Dose for Methylmercury

Three-fold for toxicokinetics:

- Basis for the UF of 10:
  - Variability and uncertainty in estimating an ingested mercury dose from cord blood mercury concentration.
  - Cord:maternal ratio for blood [Hg] ranges from > 3 to less than 1. Average ~ 1.7 to 1.8. New Japanese data indicate ratio of 1.6 for cord : maternal pairs.

Three-fold for toxicodynamics and uncertainty.

Estimated Number of Newborns with *In Utero* Methylmercury Exposures >/= RfD

- Number of US births in 2000: 4,058,814 (*National Vital Statistics Reports*).
- 1 : 1 ratio of cord to maternal blood [Hg], i.e., 5.8 cord to 5.8 maternal, 7.8% of women had total blood [Hg] >/= 5.8, ~ 300,000 newborns each year > 5.8 ug/L (Mahaffey et al., 2003).
- 1.7 : 1 ratio of cord to maternal blood [Hg], i.e. 5.8 cord to ~ 3.5 maternal, 15.7% of women had total blood [Hg] >/= 3.5 ug/L, ~ 630,000 newborns each years >/= 5.8 ug/L cord blood. [Note: this estimate is preliminary in nature, and is based on recently available information about mercury in umbilical cord blood versus maternal blood. This new information was presented as part of an ongoing scientific dialogue on how best to understand mercury exposures. EPA is still reviewing these new studies and their potential implications. This recalculation does not impact or change the established Reference Dose (RfD); rather this work focuses solely on an exposure estimate.]

2003/2004 Reports on Neuropsychological Evaluations of Methylmercury Toxicity

- **Myers et al. 2003.** Seychelles cohort update (Lancet). Continued to observe no adverse effects of methylmercury exposure under the circumstances present in the Seychelles Islands.
- ***Yokoo et al. 2003.*** *Reduced function on tests of fine motor speed and dexterity and on tests of verbal memory among adult Amazonian villagers exposed to methylmercury.*
- **Beuter and Edwards, 2003**. Cree Indians. Additional studies among adults showed difficulty with accuracy and sharpness of visual fixation and pursuit in dynamic eye movements.

Emerging Question on Adult Neurotoxic Effects of Methylmercury Exposures

WHO proposed threshold for adult neurotoxicity based on 5% prevalence of paresthesias at 50 ppm hair mercury (1990).

No physiological basis to assume there are no effects at lower exposures

Dose-response at lower levels needs to be determined.

Mercury and Omega-3 Fatty Acids

- **In 2003 additional epidemiology data raised more interest in mercury as a cardiac toxin.**
- ***Omega-3 fatty acids in fish frequently cited as a health benefit of fish and shellfish intake.***
- **Key piece of information is that there are substantial species-specific differences in the distribution of mercury and of the omega-3 fatty acids.**
- ***Species high in mercury are not necessarily high in omega-3s and species high in omega-3s are not necessarily higher in mercury.***

Comparison of Mercury (ppm) and Omega-3 Fatty Acid (g/100g) in Fish Species

- **High Mercury Species**

Tilefish: 1.6 Hg, 0.17 O-3s

Shark: 1.3 Hg, 0.07 O-3s

King Mackerel: 0.97Hg, 0.18 O-3s

Swordfish: 0.95 Hg, 0.58 O-3s

- **High Omega-3 Species**

Mackerel: 0.08 Hg, 3.61 O-3s

Salmon-sockeye: 0.03 Hg, 3.00 O-3s

Herring: 0.01 Hg, 2.34 O-3s

Tuna, albacore: 0.26 Hg, 2.33 O-3s

Variation in Mercury and Omega-3 Fatty Acids in Fish and Shellfish

- **Mercury concentrations range from < 0.02 ppm Hg in shellfish such as abalone to several ppm Hg in large predatory fish.**
- ***Omega-3 fatty acids (combined EPA and DHA) range from < 0.1 gram/100 grams of fish (e.g., shark species) to > 3.5 grams/100 grams of fish (mackerel species).***
- **There is minimal association between the omega-3 fatty acid concentration in the fish species and the mercury concentration in the species.**

Upcoming Meeting

- Meeting on medical issues related to mercury exposure.
- Orlando, Florida
- April – 2004
- Sponsored by US EPA and US HHS in conjunction with multiple medical associations.

Preliminary Results from Steubenville Hg Deposition Source Apportionment Study

Briefing for Tim Oppelt
April 27, 2005

Presented by Tim Watkins, NERL
Research conducted by Matt Landis, Gary Norris, and David Olson
in collaboration with the University of Michigan

Purpose

- Provide a “Heads-Up” of the significant preliminary findings of the Steubenville study
  - Scientifically and Politically significant
- Seeking guidance on how to proceed with review and release of study results

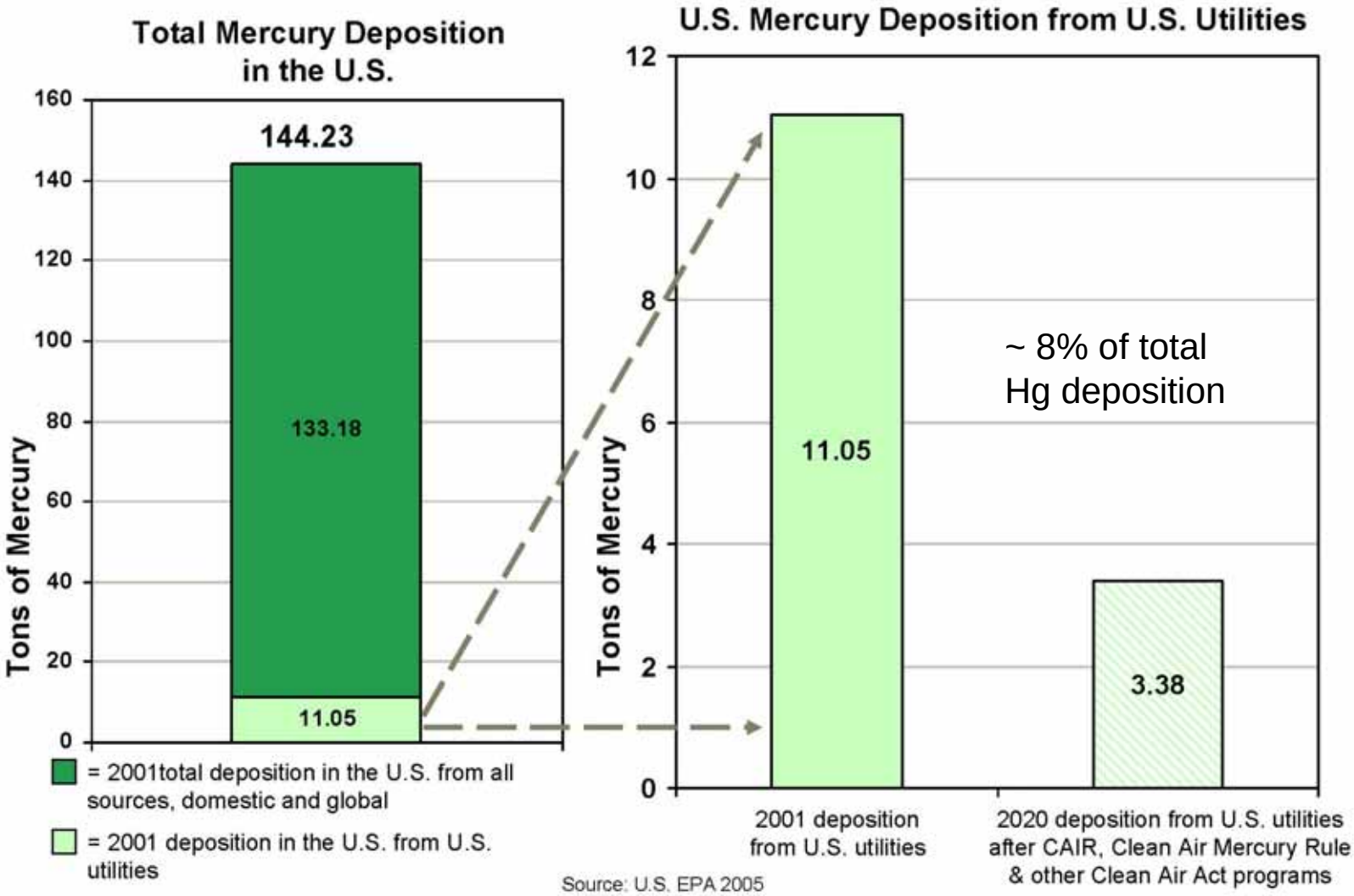
Results

- Approximately 70% of Hg wet deposition at Steubenville site is attributable to local/regional fossil fuel (coal and oil) combustion sources
  - Not entirely attributable to electric utilities
- Preliminary results
  - Additional analysis to finalize results will be completed within a month

Significance of Results

- Current models (including those used by EPA for CAMR analyses) estimate a much lower local/regional source contribution to Hg deposition, on average
  - On average, approximately 8% of domestic Hg deposition estimated to be from domestic electric utility coal combustion
- Implications for potentially vulnerable areas (i.e., “Hotspots”)
- Significant deposition decreases predicted for Steubenville area

Mercury Deposition in the U.S.

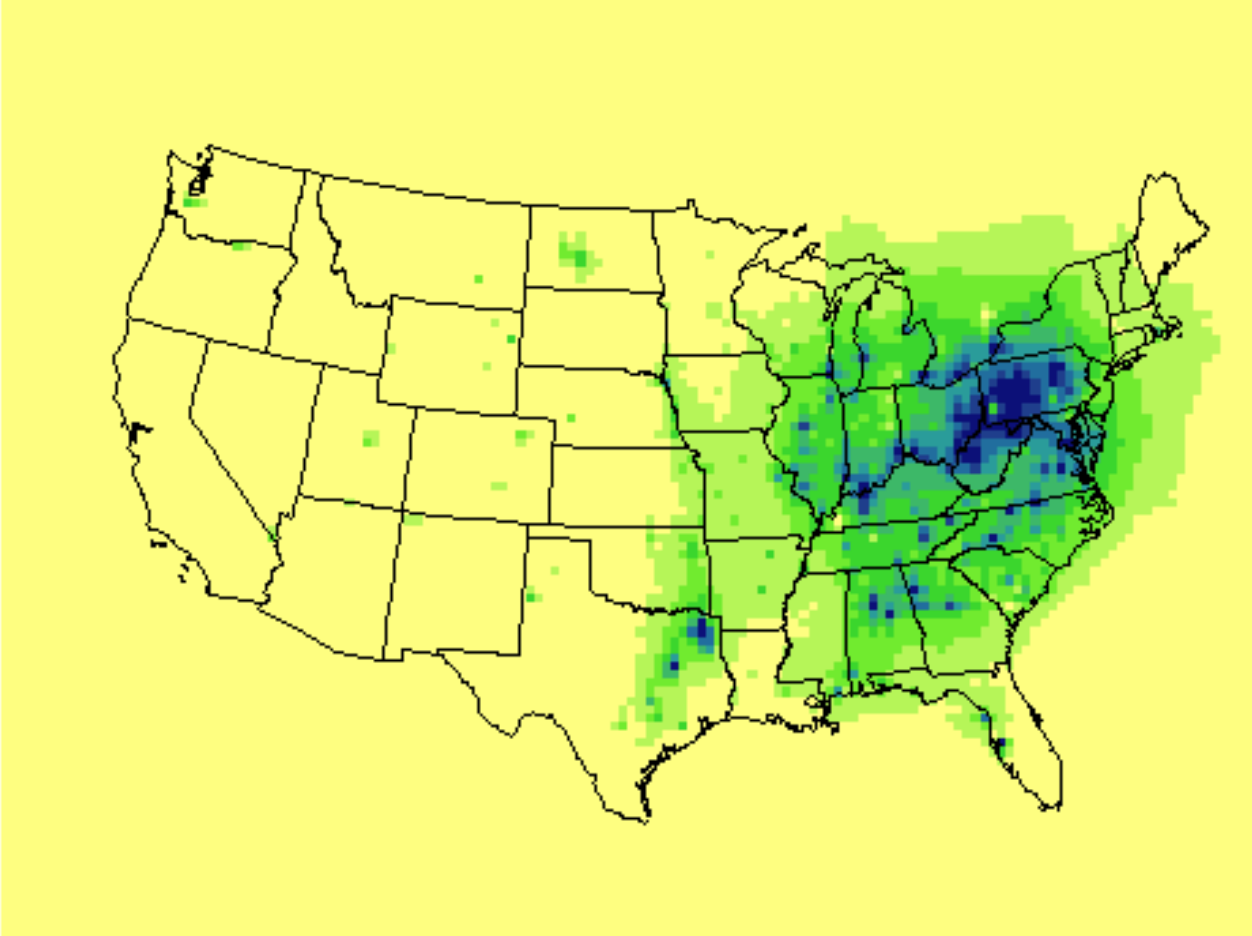


Source – US EPA Clean Air Mercury Rule website (8% note added)

Percent Change in Deposition from a 100% Reduction of Utility Emissions

Percent Change in Deposition

- 0.00 - 5.00
- 5.01 - 10.00
- 10.01 - 15.00
- 15.01 - 20.00
- 20.01 - 25.00
- 25.01 - 30.00
- 30.01 - 35.00
- 35.01 - 40.00
- 40.01 - 68.45



Explanation of Results

Are these results plausible?

- Yes
  - Steubenville site was chosen as because it was anticipated to be impacted by coal combustion
  - Results would appear to contradict EPRI claims of RGM to elemental Hg plume conversion

Note: Mercury is emitted from combustion sources in one of three species – reactive gaseous mercury (RGM), particulate mercury, and elemental mercury. RGM and particulate mercury contribute to local/regional deposition, while elemental mercury tends to be transported longer distances.

Why haven't similar results been presented previously?

- Recent convergence of newly available tools
  - New collection method
    - Precipitation-event-based sampler
  - Improved analytical method
    - Extremely low detection limits for trace elements
    - Use to measure potential co-pollutants (e.g., sulfur, selenium, vanadium)
  - State-of-the-art EPA receptor modeling tools
    - Statistical tools to identify relative source contributions
    - New releases of EPA UNMIX and Positive Matrix Factorization (PMF)
- Current mercury monitoring efforts do not collect data needed to conduct this type of analysis
  - Mercury Deposition Network (MDN) not designed for source apportionment studies
    - Weekly instead of event-based samples
    - In general, no co-located trace element measurements
- First time that state-of-the-art receptor models have been applied to mercury precipitation data

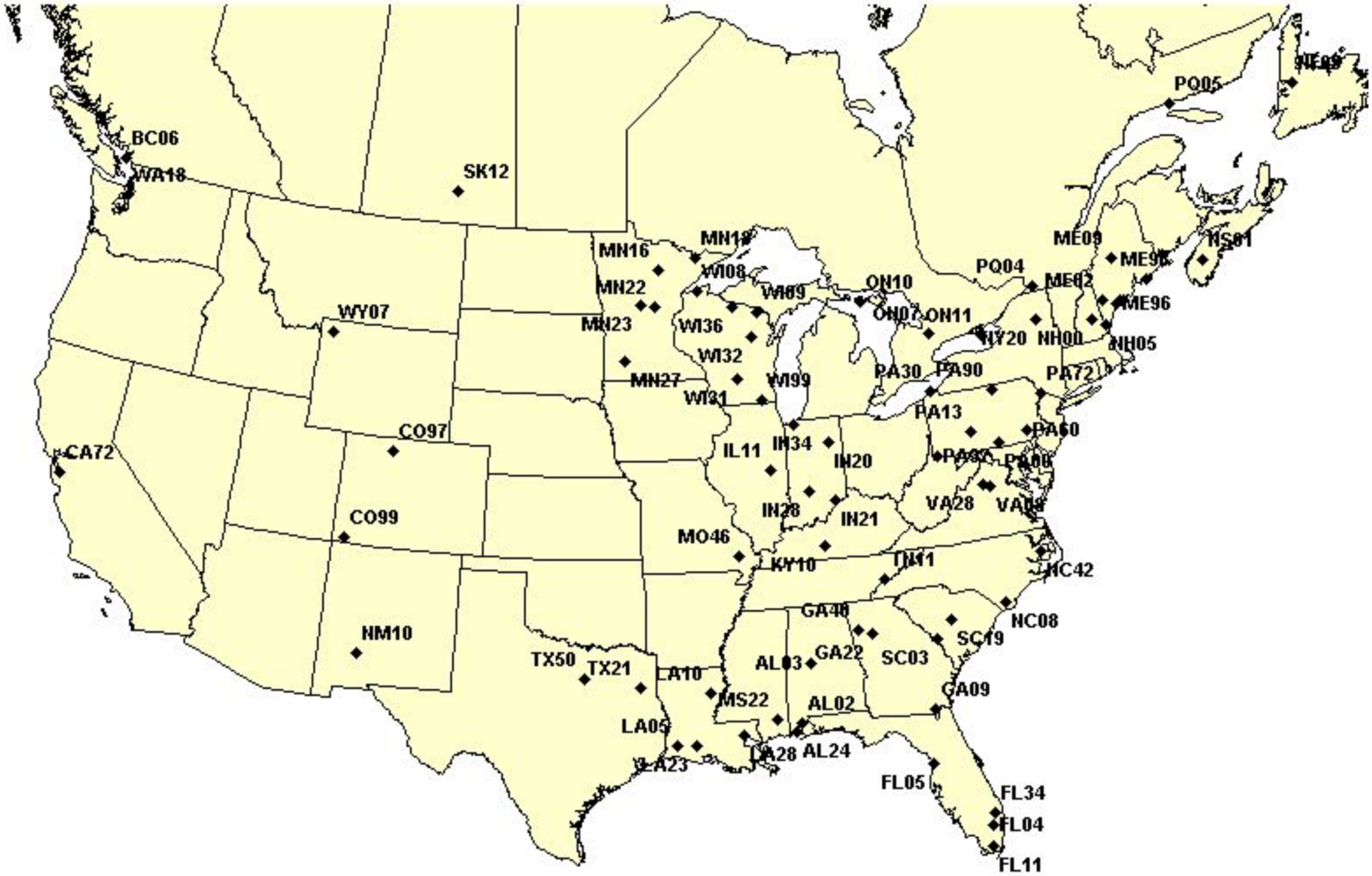
Are the results consistent with other studies?

- Consistent with studies conducted in south FL, but . . .
  - Local deposition was attributed to municipal and medical waste incinerators
  - Used more simplistic source apportionment tool

Why are estimated regional/local deposition values higher than those predicted in current air quality models?

- Lack of event-based empirical deposition data for model evaluation
- Deposition parameters in current models based and evaluated against Mercury Deposition Network (MDN) data
  - MDN sites generally located in rural areas
  - Emerging but limited empirical evidence of very high Hg concentrations/deposition in urban areas
    - Chicago, Charlotte, St. Louis, and Detroit
  - Potential underestimate of predicted deposition

National Atmospheric Deposition Program
Mercury Deposition Network



Why weren't these results available for CAMR analyses?

- Mercury MYP included 2004 APM
  - Initial efforts to run receptor models with one-year data set (2003) were not conclusive
  - APM delayed
- Completed second year of data collection (including QA/QC) in early April 2005
- Receptor models now producing conclusive results with two-year (2003 and 2004) data set

Will these results be replicated?

- Yes, because new tools are available and in use
  - New collection method
    - Developed by University of Michigan
    - Applied in Steubenville, at several other sites in Michigan, at one site in Vermont, and in Tampa, FL
  - Improved analytical method
    - University of Michigan has this capability
  - State-of-the-art receptor models
    - New EPA Receptor models are publicly available

Michigan Mercury Monitoring Sites Operated by the University of Michigan

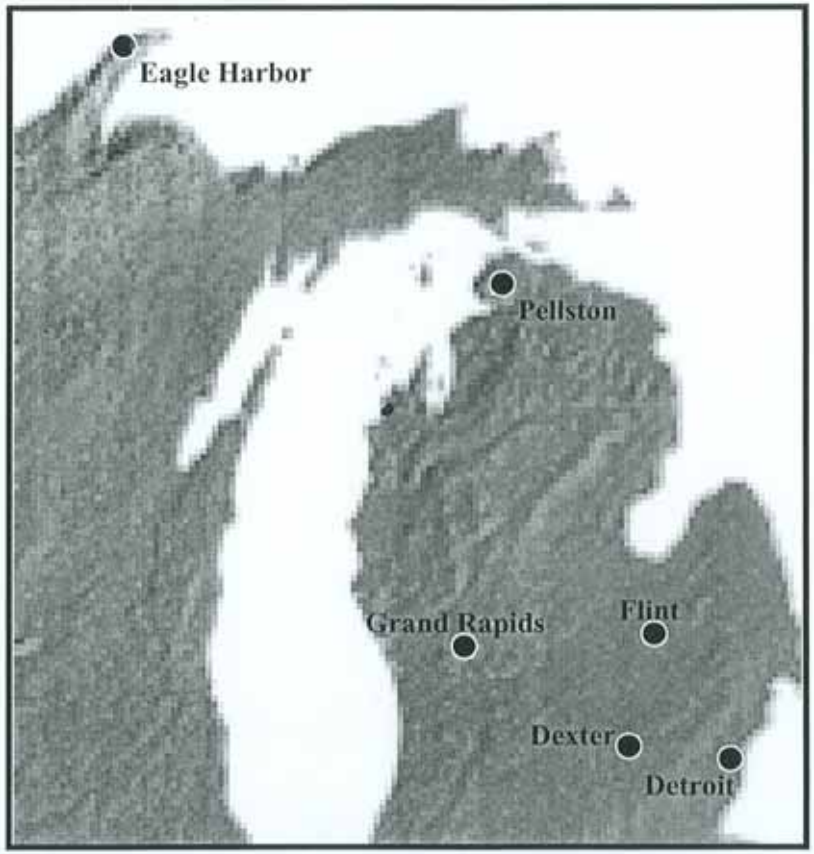


Figure 1. Location of Atmospheric Mercury Monitoring Sites in Michigan.

Planned Next Steps

- Additional data analyses
  - Complete review of receptor modeling to finalize study results
  - Refined analyses of peak events
    - Explore meteorology data
    - Identify specific source regions and/or sources contributing to deposition events in Steubenville
- Prepare materials for EPA (OAR in particular) dissemination
- Prepare manuscript for peer-reviewed journal submission
- Future work
  - Conduct similar studies in other existing locations
    - North-South gradient in Ohio Valley (data from Univ of Mich)
    - East-West gradient using sites in Vermont (data from Univ of Mich)
    - Tampa, FL
  - Extend analysis to mercury dry deposition
  - Continue monitoring in Steubenville
    - Location for Clean Air Mercury Rule (CAMR) Accountability?
  - Integrate results into air quality models (CMAQ)

Issues for Discussion

- How and when should we disseminate the results of this study within EPA (particularly to OAR)?

- Do we need to utilize additional peer-review mechanisms beyond journal submission and review?

Supplemental Information

- Steubenville Study Approach
- An Overview of source apportionment
- Additional information on new source apportionment tools
  1) New event-based collection method
  2) Improved analytical method
  3) State-of-the-art receptor models

Study Approach

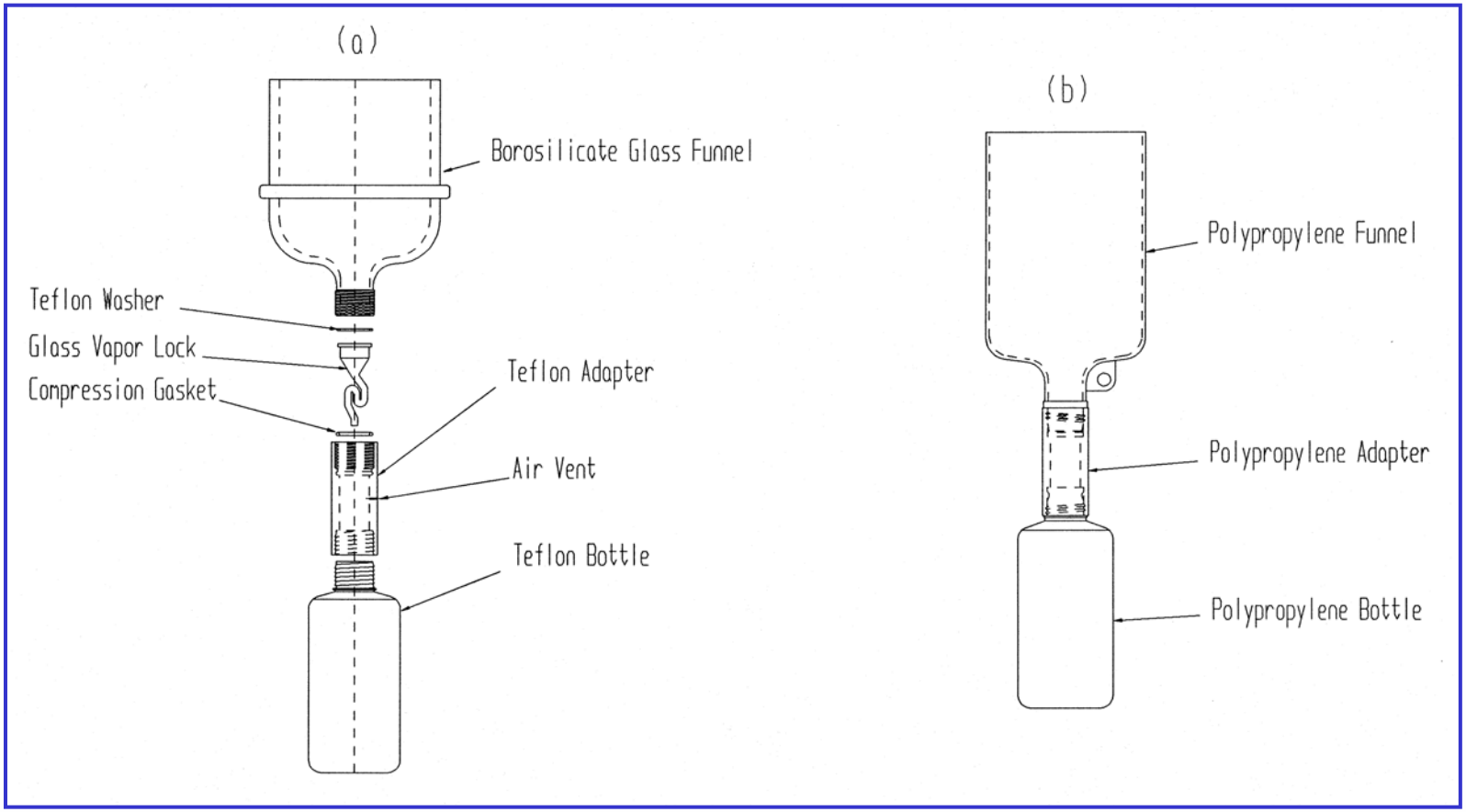
- Site location / duration
  - Steubenville, OH – anticipated to impacted by coal combustion sources
  - 4-year study
    - 2-years of data collected to date (2003 and 2004)
- Cooperative agreement with the University of Michigan
- Collecting detailed measurements
  - Precipitation event-based deposition sampling
  - Potential source co-pollutants (trace elements)
  - Meteorology
- Applying state-of-the-art receptor models
  - Latest version of EPA models – UNMIX and PMF
  - First time modeling approaches applied to Hg precipitation data

An Overview of Source Apportionment

- Relating source emissions to their quantitative impact on environmental concentrations (i.e., ambient or deposition) is referred to as source apportionment
- Two approaches to source apportionment
  - Dispersion modeling (e.g., CMAQ)
    - Combines emissions, chemistry, and meteorology information to relate sources to predicted ambient concentrations
  - Receptor modeling
    - Uses environmental measurements and statistics to identify sources or source categories impacting the receptor.
- Single-sample receptor models
  - Require source profiles or “fingerprints”
  - Example – Chemical Mass Balance (CMB)
- Multivariate receptor models
  - Require many samples (100 or more)
  - Source profile information not needed
  - Examples – UNMIX and Positive Matrix Factorization (PMF)

New Precipitation-event-based Collection Method

Wet-Only Precipitation Collection



* Landis and Keeler *Environ. Sci. Technol.*, **1997**, 31, 2610-2615

High Resolution ICP-MS Capability
(Precipitation & Aerosol Samples)

- Low Resolution
  - Li, Be, Rb, Sr, Mo, Ag, Cd, In, Sn, Sb
    Cs, Ba, La, Ce, Nd, Sm, W, Tl, Pb, U
- Medium Resolution
  - Na, Mg, Al, P, S, Ca, Sc, Ti, V, Cr
    Mn, Fe, Co, Ni, Cu, Zn
- High Resolution
  - K, As, Se
- Stable Isotope Ratios
  - Pb

Statistical Receptor Model Development

- **UNMIX**
  - Multi-linear model (Ron Henry – USC)
  - Usually requires at least 100 samples
  - Does not use data below MDL
  - Generates source profiles and uncertainties
- **Positive Matrix Factorization (PMF)**
  - Multi-linear model (Phil Hopke – Clarkson University)
  - Usually requires at least 100 samples
  - Uses data below MDL
  - Incorporates uncertainties and weights individual data points
  - Generates source profiles and uncertainties

Modeling the Fate and Transport of Atmospheric Mercury in the Chesapeake Bay Region

Dr. Mark Cohen
NOAA Air Resources Laboratory
Silver Spring, Maryland

Presentation at

NOAA Chesapeake Bay Office
May 17, 2004, Annapolis MD

- **Modeling Methodology**
- **Hg Emissions Inventory**
- **Model Evaluation**
- **Some Results for Chesapeake Bay**
- **Some Next Steps**

Modeling Methodology

Three “forms” of atmospheric mercury

Elemental Mercury: Hg(0)

- ~ 95% of total Hg in atmosphere
- *not* very water soluble
- long atmospheric lifetime (~ 0.5 - 1 yr); globally distributed

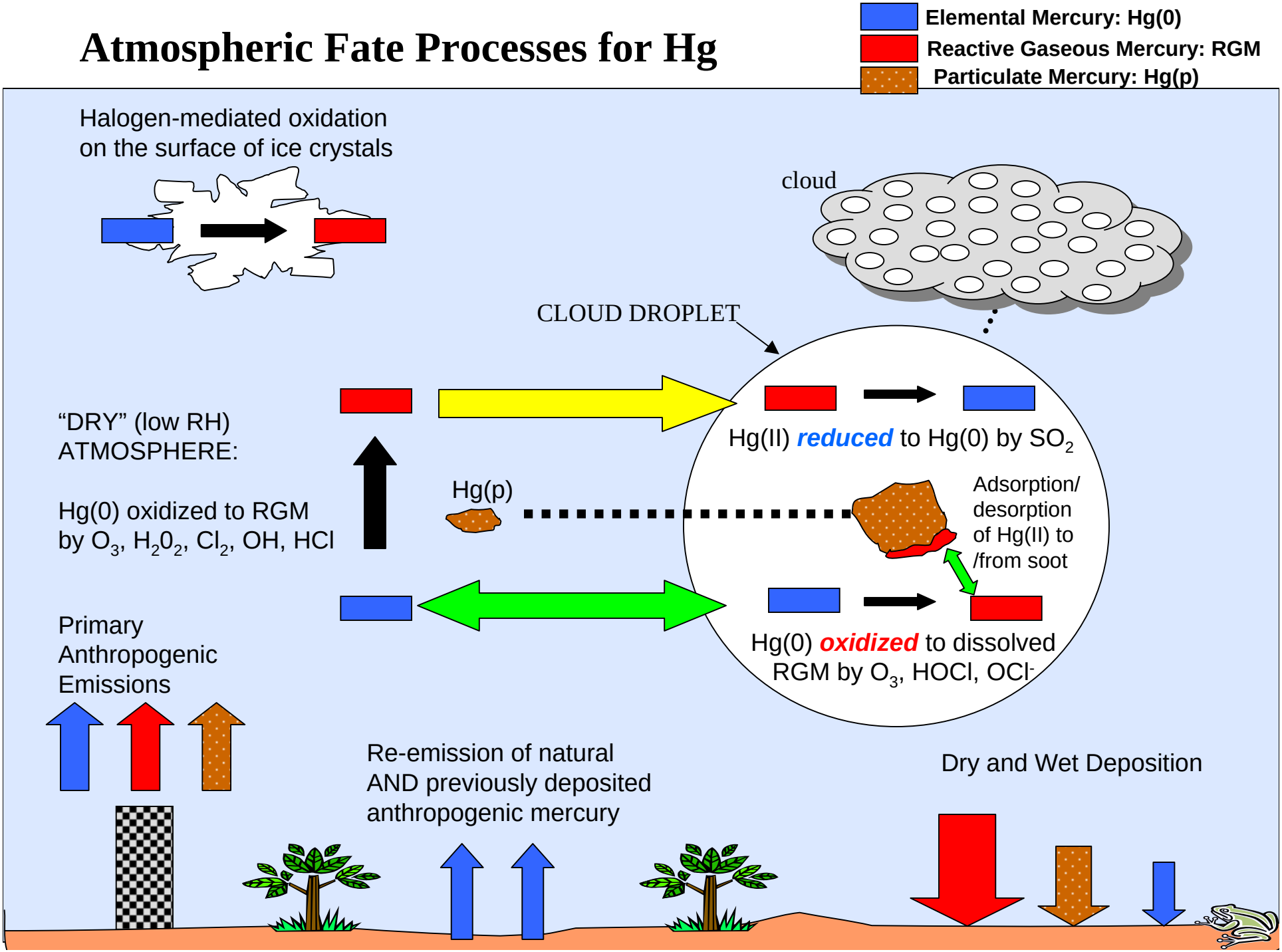
Reactive Gaseous Mercury (“RGM”)

- a few percent of total Hg in atmosphere
- oxidized mercury: Hg(II)
- HgCl2, others species?
- somewhat operationally defined by measurement method
- *very* water soluble
- short atmospheric lifetime (~ 1 week or less);
- more local and regional effects

Particulate Mercury (Hg(p)

- a few percent of total Hg in atmosphere
- not pure particles of mercury…
  (Hg compounds associated with atmospheric particulate)
- species largely unknown (in some cases, may be HgO?)
- moderate atmospheric lifetime (perhaps 1~ 2 weeks)
- local and regional effects
- bioavailability?

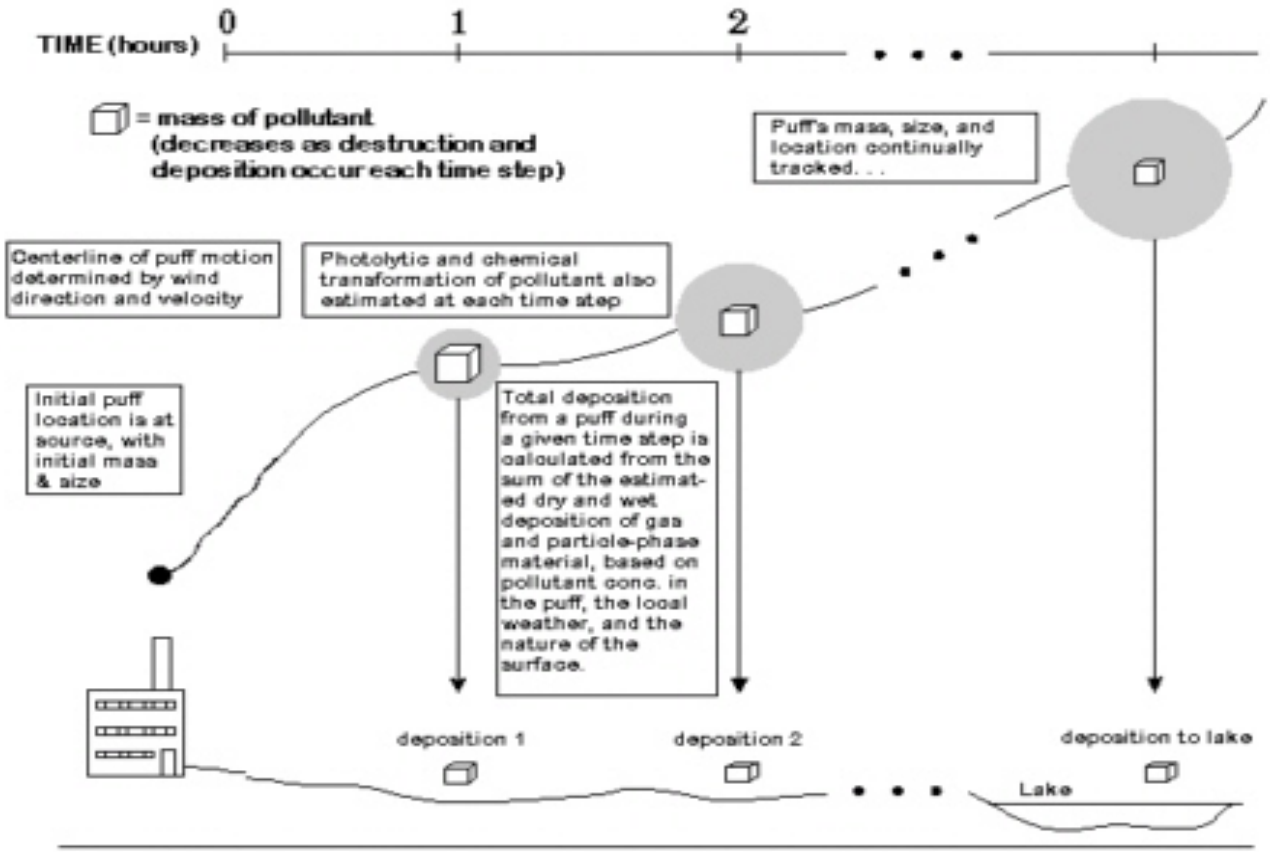
Atmospheric Fate Processes for Hg



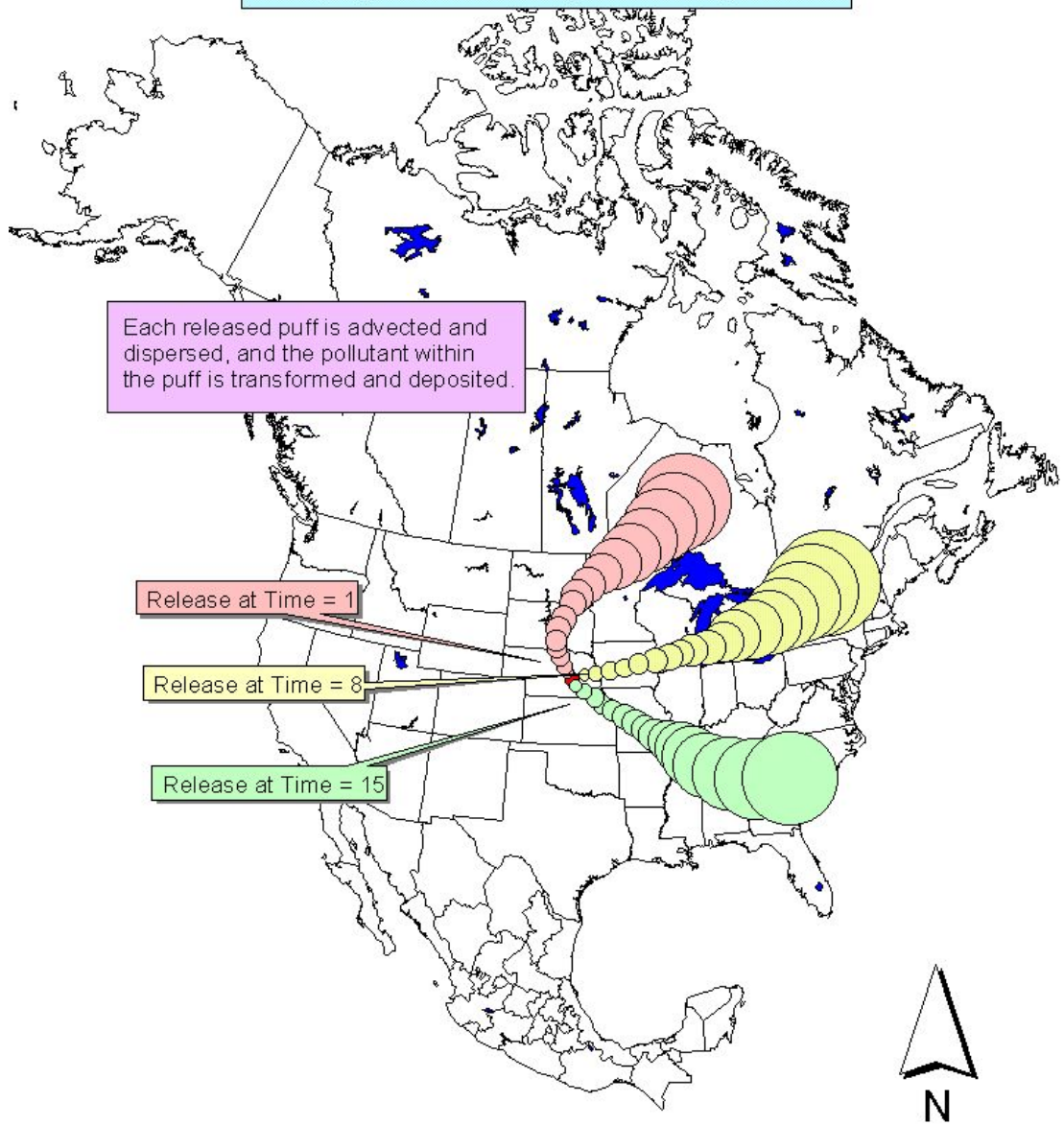
Atmospheric Chemical Reaction Scheme for Mercury

Reaction	Rate	Units	Reference
GAS PHASE REACTIONS			
$Hg^0 + O_3 \rightarrow Hg(p)$	3.0E-20	cm^3/molec-sec	Hall (1995)
$Hg^0 + HCl \rightarrow HgCl_2$	1.0E-19	cm^3/molec-sec	Hall and Bloom (1993)
$Hg^0 + H_2O_2 \rightarrow Hg(p)$	8.5E-19	cm^3/molec-sec	Tokos et al. (1998) (upper limit based on experiments)
$Hg^0 + Cl_2 \rightarrow HgCl_2$	4.0E-18	cm^3/molec-sec	Calhoun and Prestbo (2001)
$Hg^0 + OH \rightarrow Hg(p)$	8.7E-14	cm^3/molec-sec	Sommar et al. (2001)
AQUEOUS PHASE REACTIONS			
$Hg^0 + O_3 \rightarrow Hg^{+2}$	4.7E+7	$(molar\text{-}sec)^{-1}$	Munthe (1992)
$Hg^0 + OH \rightarrow Hg^{+2}$	2.0E+9	$(molar\text{-}sec)^{-1}$	Lin and Pehkonen(1997)
$HgSO_3 \rightarrow Hg^0$	$T*e^{((31.971*T)-12595.0)/T)}$ sec^{-1} [T = temperature (K)]		Van Loon et al. (2002)
$Hg(II) + HO_2 \rightarrow Hg^0$	~ 0	$(molar\text{-}sec)^{-1}$	Gardfeldt & Jonnson (2003)
$Hg^0 + HOCl \rightarrow Hg^{+2}$	2.1E+6	$(molar\text{-}sec)^{-1}$	Lin and Pehkonen(1998)
$Hg^0 + OCl^{-1} \rightarrow Hg^{+2}$	2.0E+6	$(molar\text{-}sec)^{-1}$	Lin and Pehkonen(1998)
$Hg(II) \leftrightarrow Hg(II)_{(soot)}$	9.0E+2	liters/gram; t = 1/hour	eqlbrm: Seigneur et al. (1998) rate: Bullock & Brehme (2002).
$Hg^{+2} + h \rightarrow Hg^0$	6.0E-7	$(sec)^{-1}$ (maximum)	Xiao et al. (1994); Bullock and Brehme (2002)

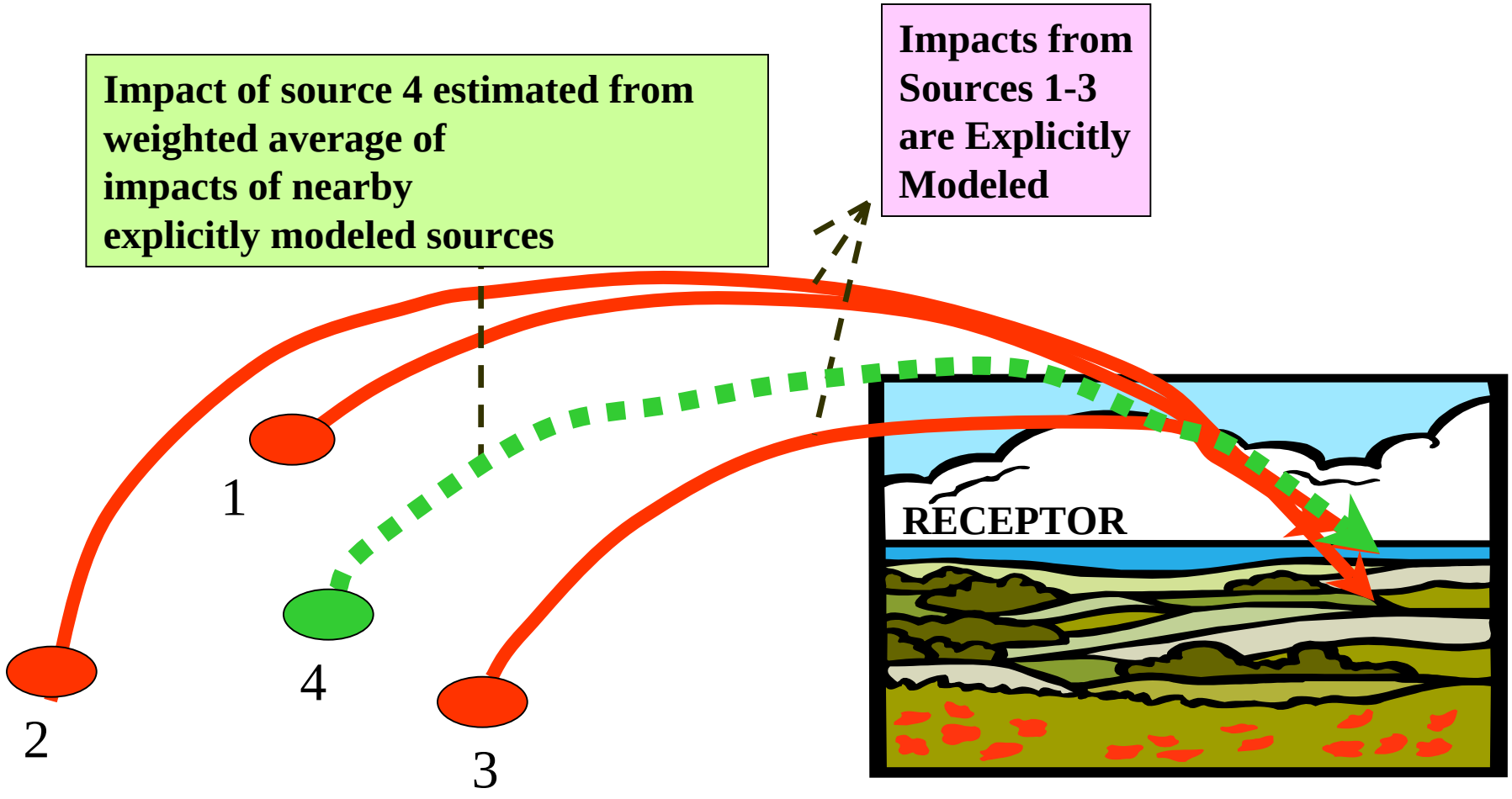
Figure 1. Lagrangian Puff Air Transport and Deposition Model



Over the entire modeling period (e.g., one year), puffs are released at periodic intervals (e.g., once every 7 hours).



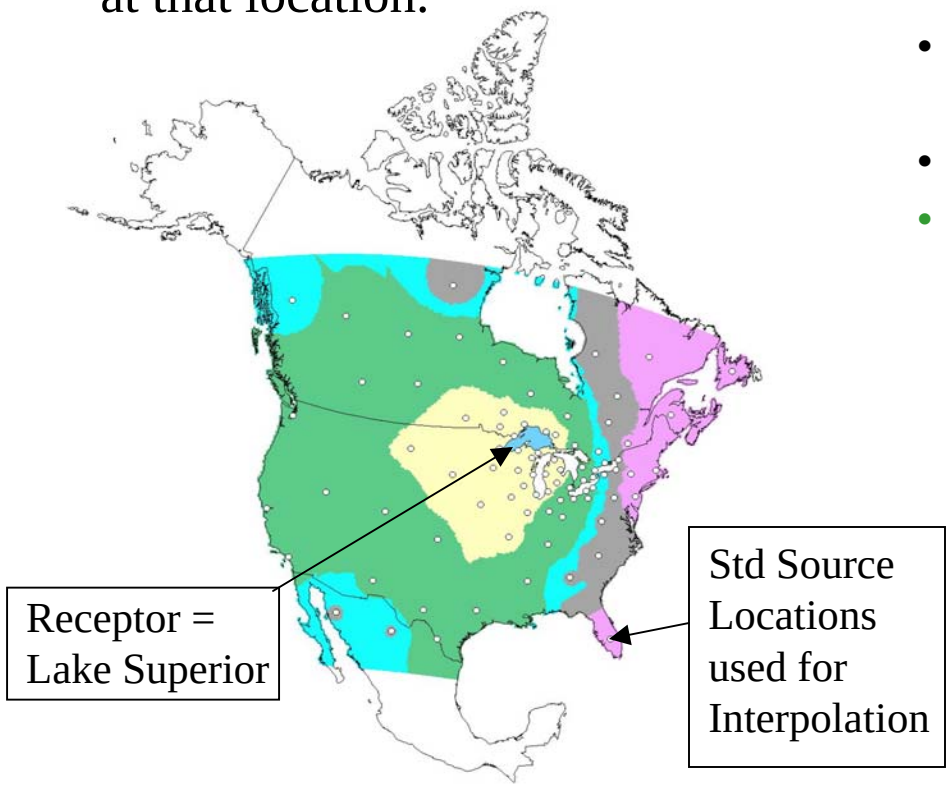
Spatial interpolation

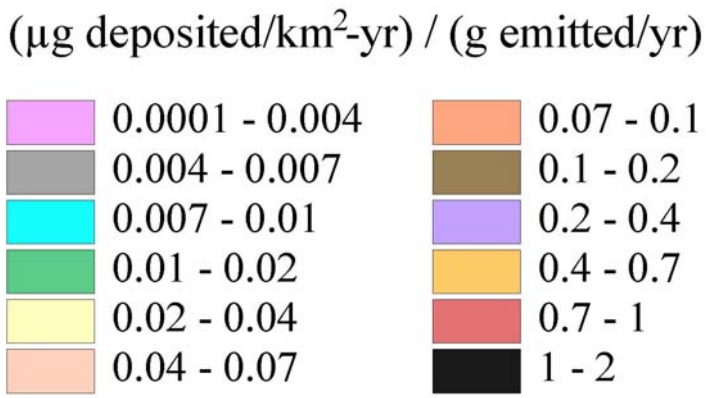


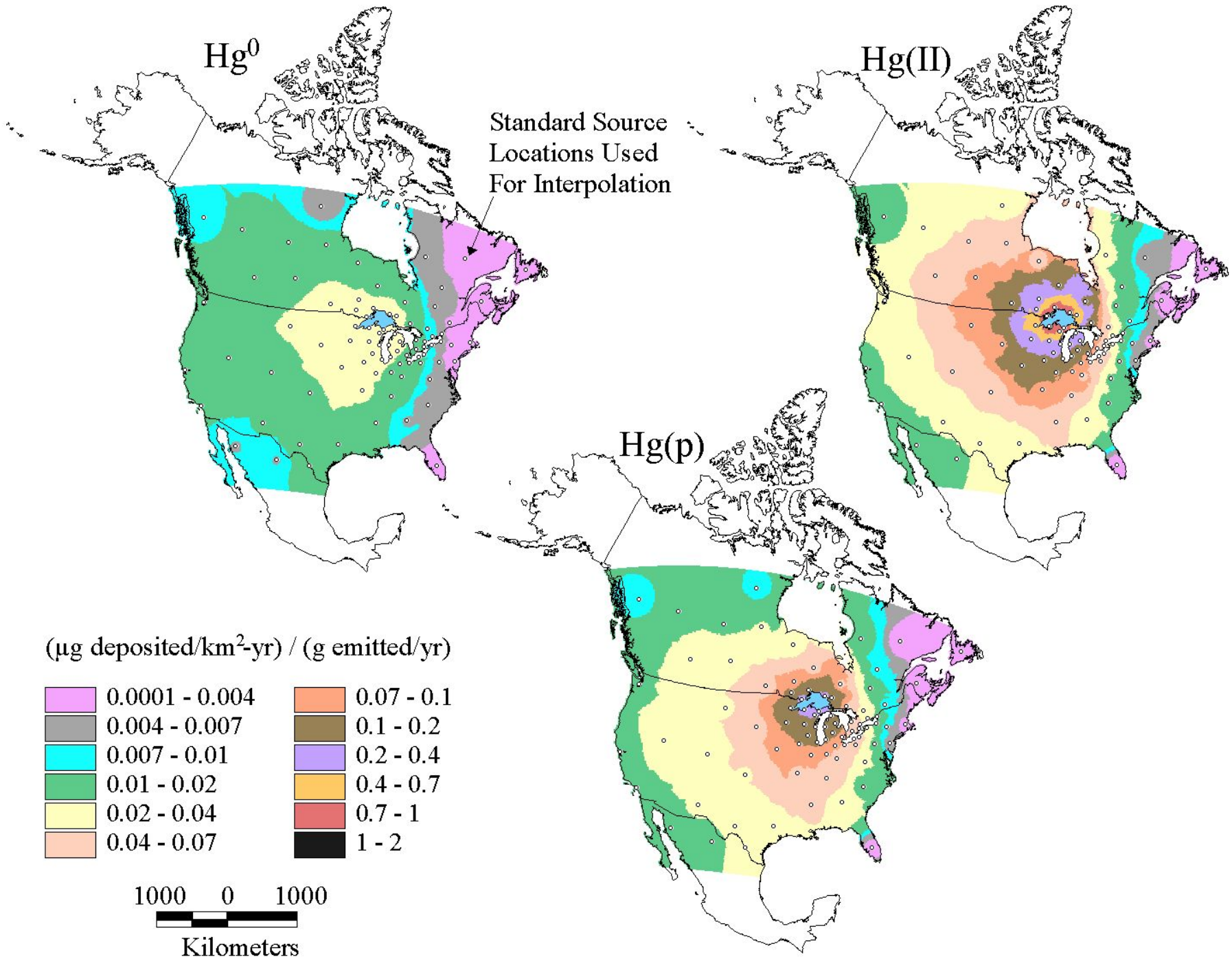
at any given location,
the ***transfer coefficient***
is defined as the amount
that would be deposited
in the given receptor
(in this case, Lake Superior)
if there were emissions
at that location.

Transfer Coefficients

- **refer to *hypothetical* emissions;**
  [are independent of actual emissions]
- **can be formulated with different units**
  [in this example: total Hg deposition flux (ug/km2-yr) / emissions (g/yr)]
- **will depend on the pollutant**
  [in this example: Hg(0)]
- **will depend on the receptor**
  [in this example: Lake Superior]
- **and the time period being modeled**
- ***[in this example: entire year 1996]***






Hg0
Hg(II)
Hg(p)
Standard Source
Locations Used
For Interpolation
(µg deposited/km2-yr) / (g emitted/yr)
0.0001 - 0.004
0.004 - 0.007
0.007 - 0.01
0.01 - 0.02
0.02 - 0.04
0.04 - 0.07
0.07 - 0.1
0.1 - 0.2
0.2 - 0.4
0.4 - 0.7
0.7 - 1
1 - 2
1000 0 1000
Kilometers

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Modeling the atmospheric transport and deposition of mercury to the Great Lakes☆

Mark Cohen,[a,*] Richard Artz,[a] Roland Draxler,[a] Paul Miller,[b] Laurier Poissant,[c] David Niemi,[d] Dominique Ratté,[d] Marc Deslauriers,[d] Roch Duval,[e] Rachelle Laurin,[e,1] Jennifer Slotnick,[f] Todd Nettesheim,[g] and John McDonald[h]

[a] NOAA Air Resources Laboratory, 1315 East West Highway R/ARL, Room 3316, Silver Spring, MD 20910, USA
[b] Commission for Environmental Cooperation, Montreal, Que., Canada
[c] Atmospheric Toxic Processes Service, Meteorological Service of Canada, Environment Canada–Quebec region, Montreal, Que., Canada
[d] Pollutant Data Branch, Environment Canada, Hull, Que., Canada
[e] Environmental Monitoring and Reporting Branch, Ontario Ministry of the Environment, Toronto, Ont., Canada
[f] University of California, Berkeley, CA, USA
[g] US EPA Great Lakes National Program Office, Chicago, IL, USA
[h] International Joint Commission, Windsor, Ont., Canada

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Abstract

A special version of the NOAA HYSPLIT_4 model has been developed and used to estimate the atmospheric fate and transport of mercury in a North American modeling domain. Spatial and chemical interpolation procedures were used to expand the modeling results and provide estimates of the contribution of each source in a 1996 anthropogenic US/Canadian emissions inventory to atmospheric mercury deposition to the Great Lakes. While there are uncertainties in the emissions inventories and ambient data suitable for model evaluation are scarce, model results were found to be reasonably consistent with wet deposition measurements in the Great Lakes region and with independent measurement-based estimates of deposition to Lake Michigan. Sources up to 2000 km from the Great Lakes contributed significant amounts of mercury through atmospheric transport and deposition. While there were significant contributions from incineration and metallurgical sources, coal combustion was generally found to be the largest contributor to atmospheric mercury deposition to the Great Lakes.
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Keywords: Mercury; Atmospheric deposition; Great Lakes; Source–receptor modeling; Emissions

Mercury contamination in the Great Lakes and many other ecosystems is increasingly being recognized as a serious environmental concern. The dominant route of human exposure to mercury is through fish consumption, and significant portions of the general population are believed to be consuming toxicologically significant levels of mercury (e.g., National Research Council, 2000). Historical discharges—e.g., from chlor-alkali production using the mercury-cell process—are believed to have caused large accumulations of mercury in sediments in Lake Erie and Lake Ontario (Marvin et al., 2003). As these discharges have been substantially reduced, atmospheric deposition is now believed to be a more significant loading pathway for these lakes. Mass balance calculations for Lake Michigan (Mason and Sullivan, 1997) and Lake Superior (Dolan et al., 1993) indicate that atmospheric deposition accounts for approximately 75% of the overall mercury loading to these lakes.

While there have been several mercury modeling efforts in North America (Bullock et al., 1998; Bullock and Brehme, 2002; Dvonch et al., 1998; Lin et al., 2001; Pai et al., 1997; Seigneur et al., 2000, 2001, 2003a, b; Shannon and Voldner, 1995; Xu et al., 2000a–c), none has developed detailed source–receptor relationships for the Great Lakes, as advocated in Annex 15 of the Great

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* Corresponding author. Fax: +301-713-0119.
E-mail address: mark.cohen@noaa.gov (M. Cohen).
[1] Current address: IPSRA Canada/The Institute of Environmental Research, Concord, Ontario, Canada.

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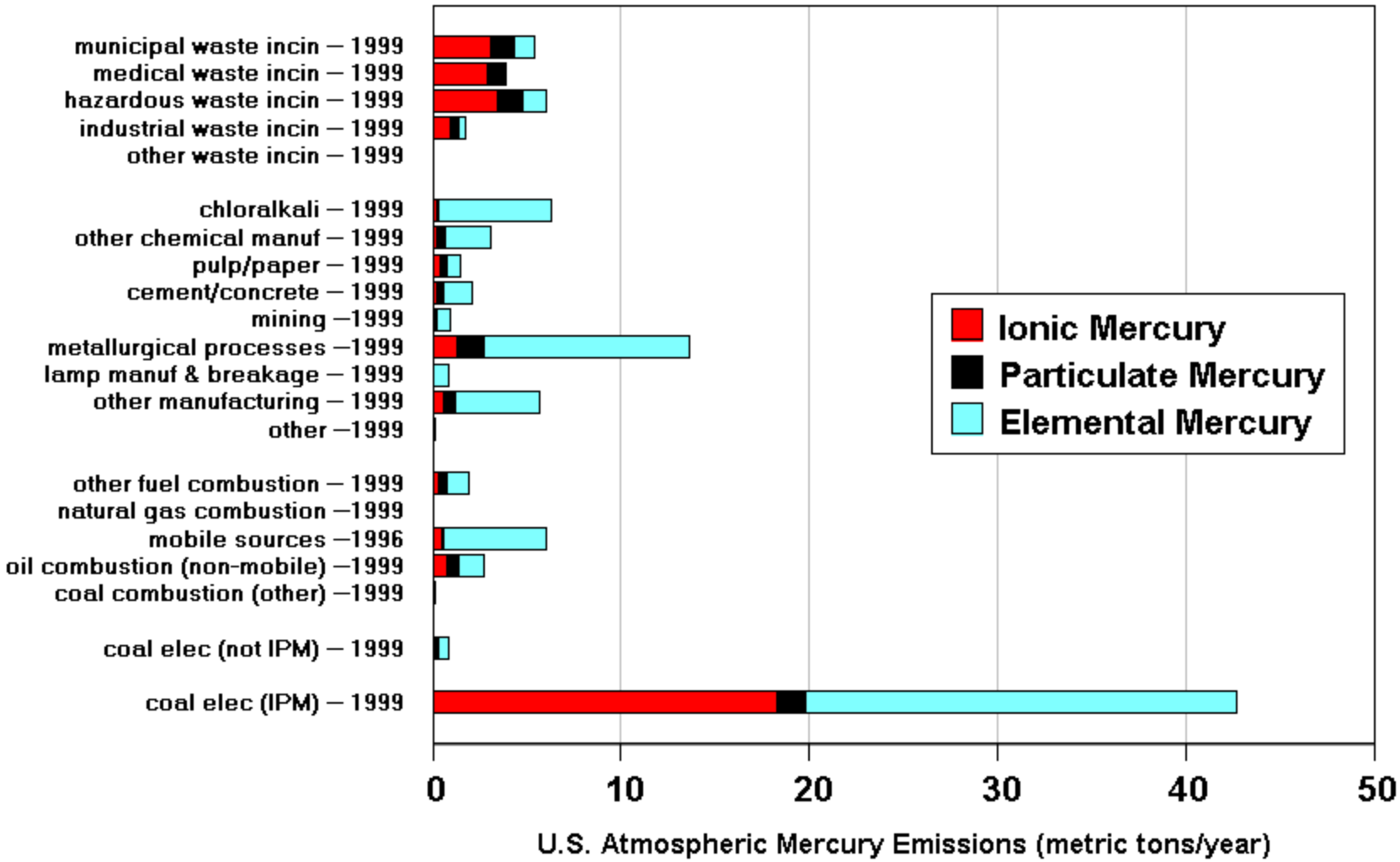
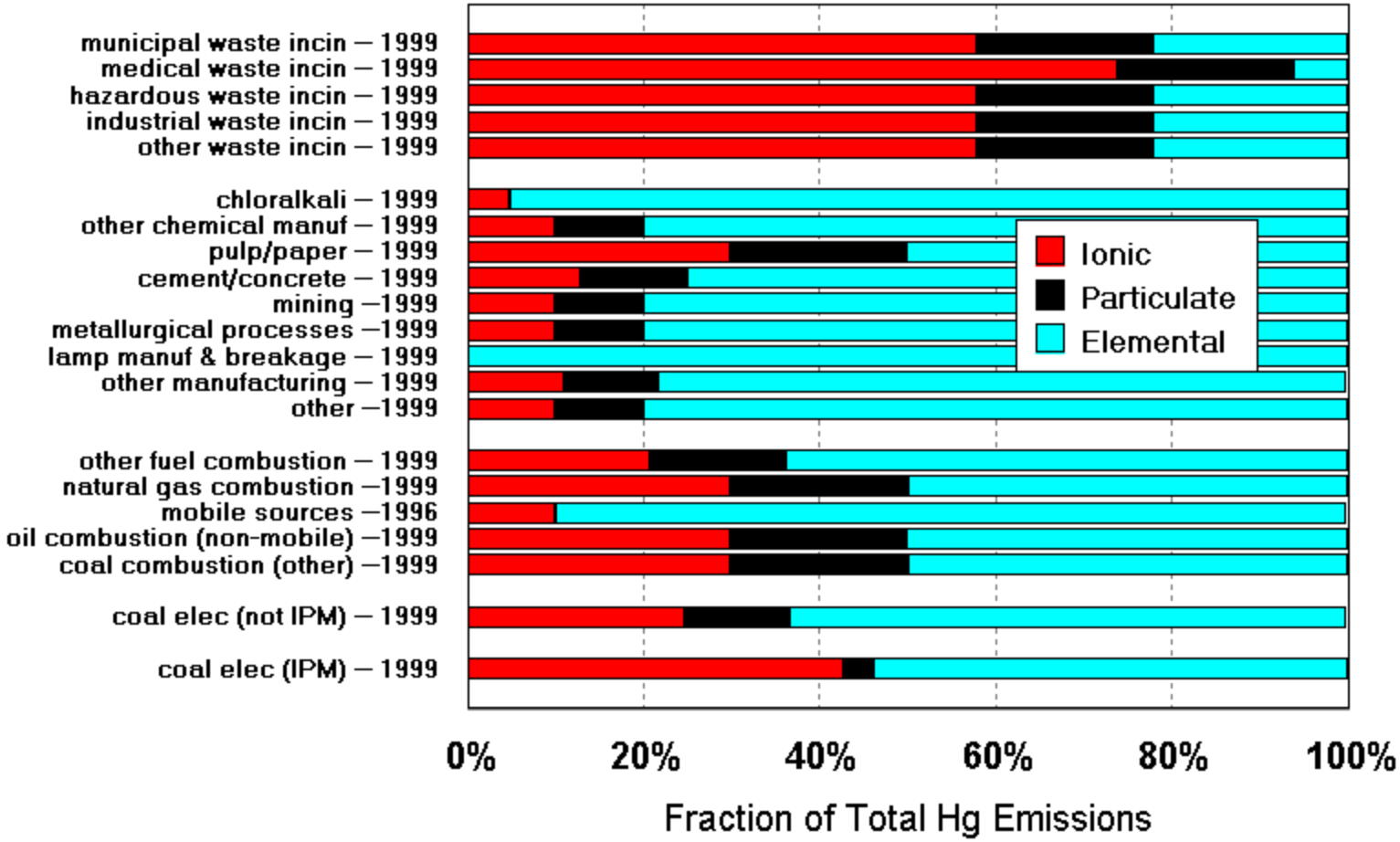
Mercury Emissions Inventory

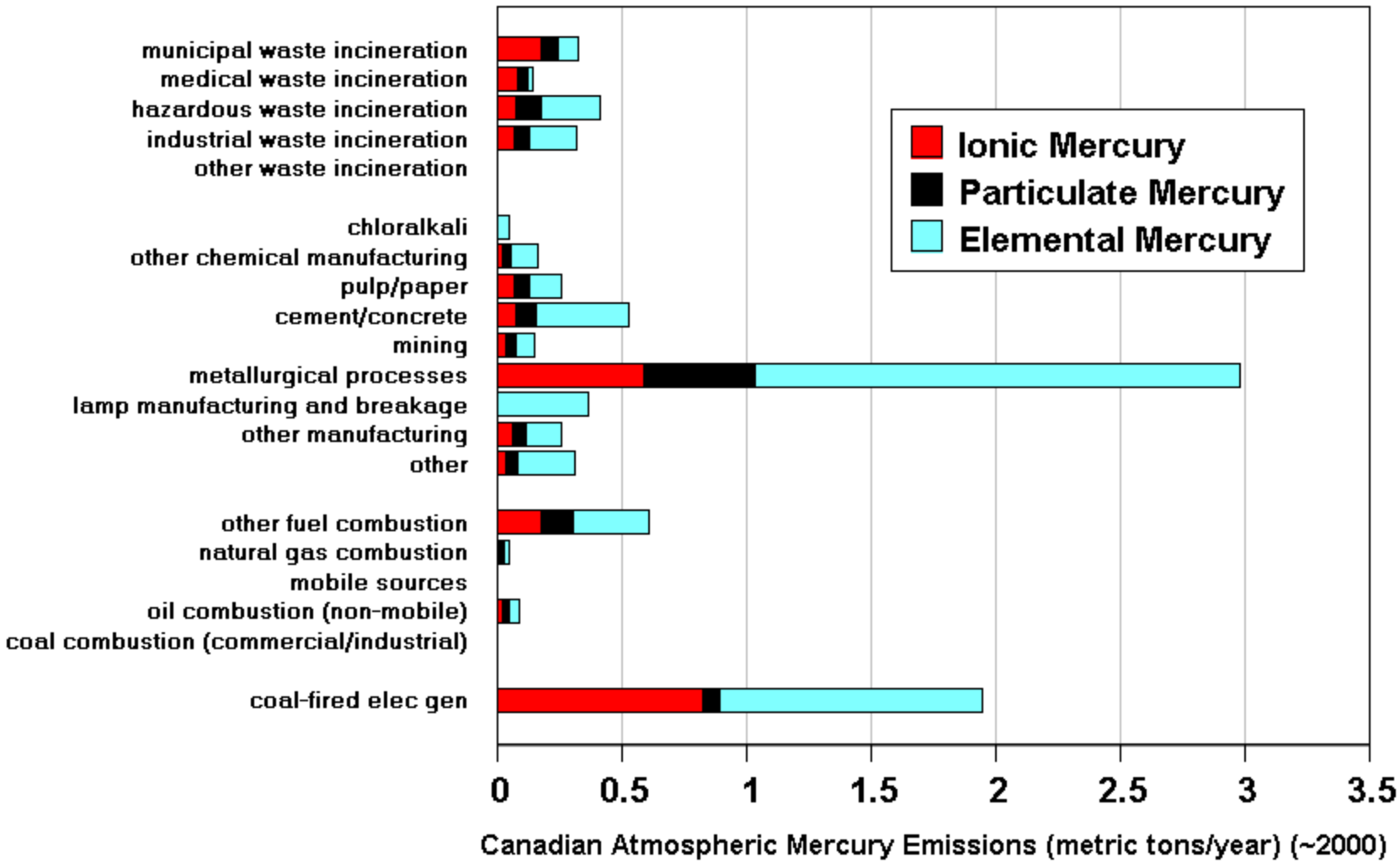
Estimated 1999 U.S. Atmospheric Anthropogenic Mercury Emissions



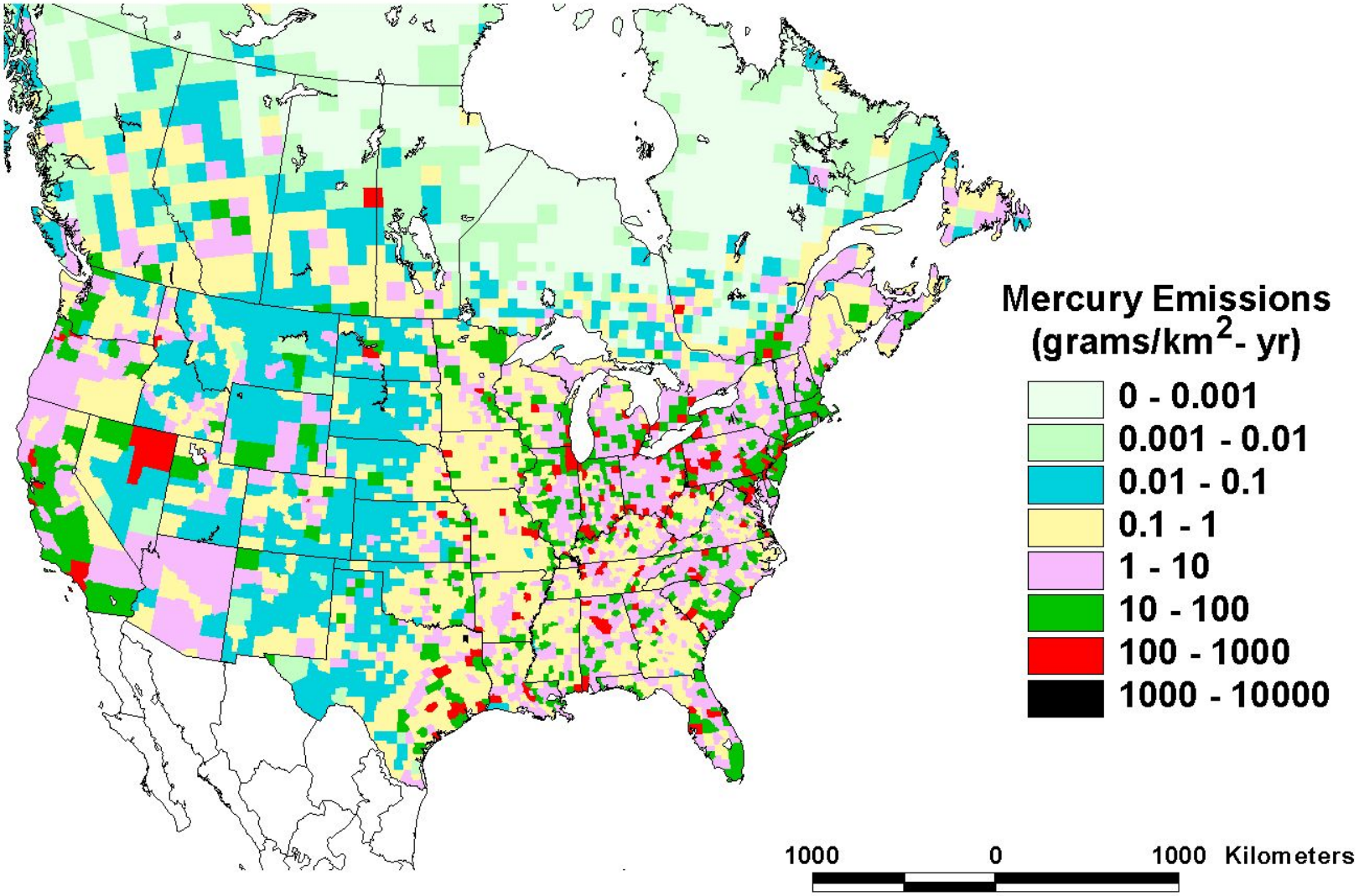
Estimated Speciation Profile for 1999 U.S. Atmospheric Anthropogenic Mercury Emissions

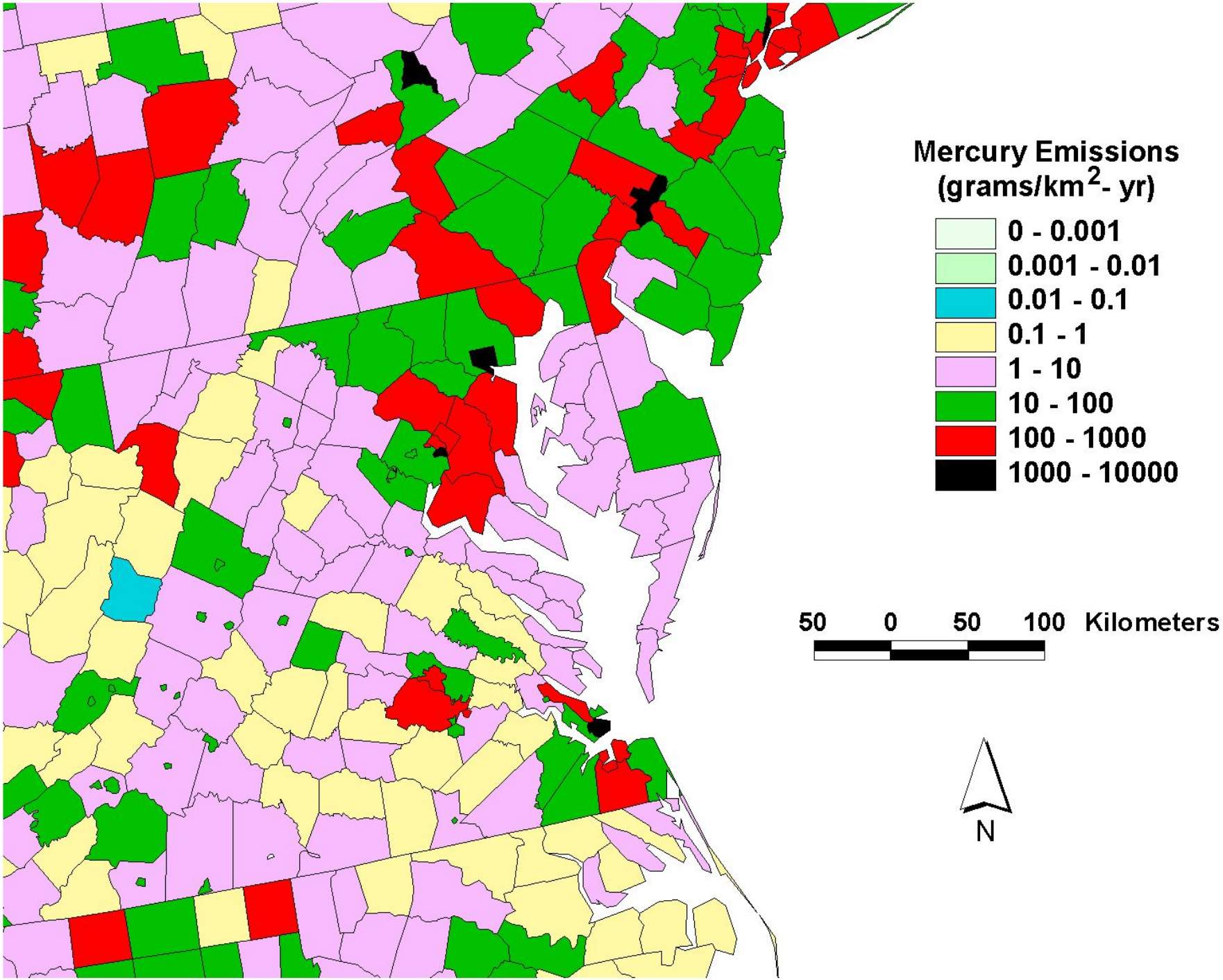


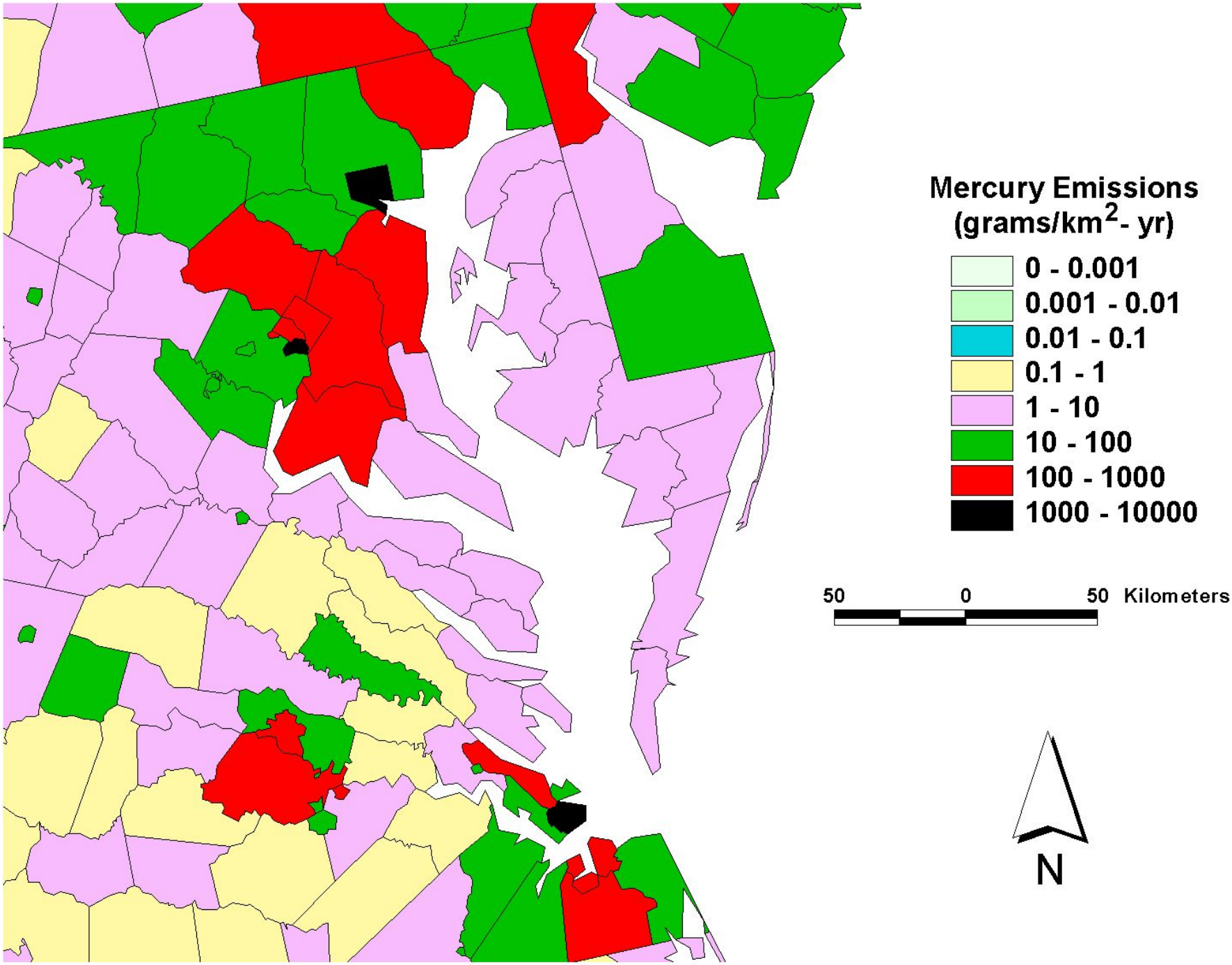
Estimated 2000 Canadian Atmospheric Anthropogenic Mercury Emissions

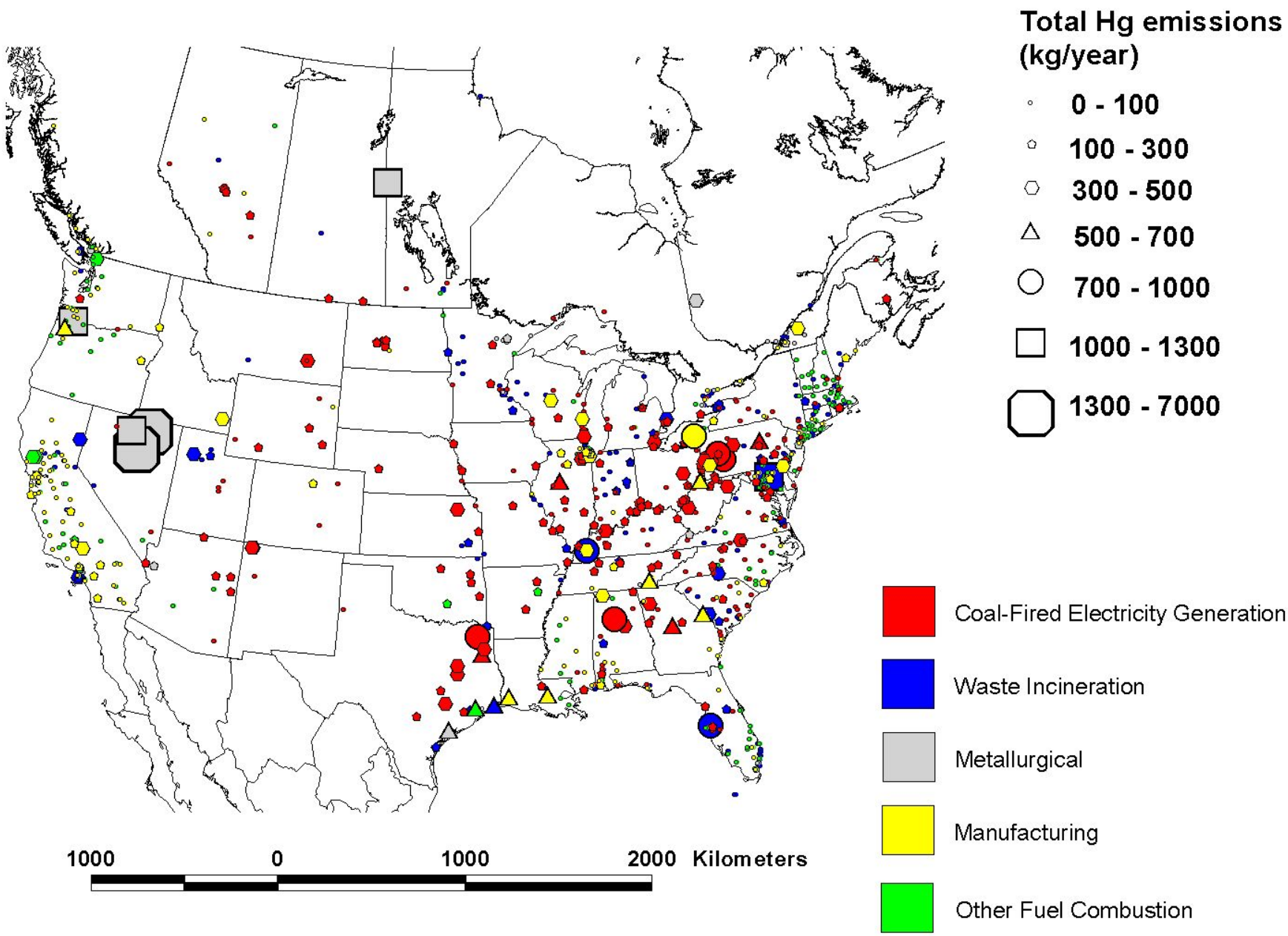


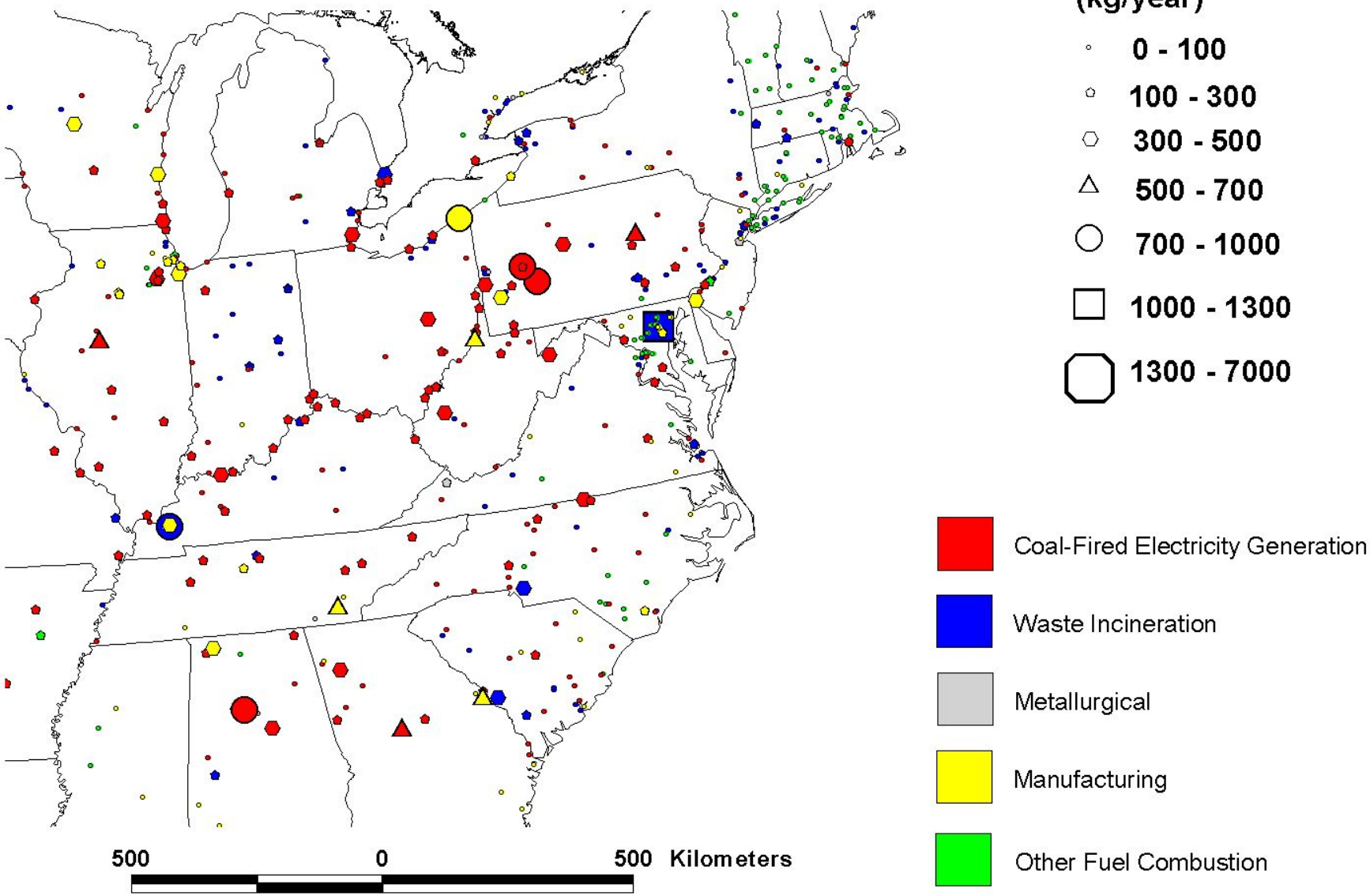
Geographic Distribution of Estimated Anthropogenic Mercury Emissions in the U.S. (1999) and Canada (2000)

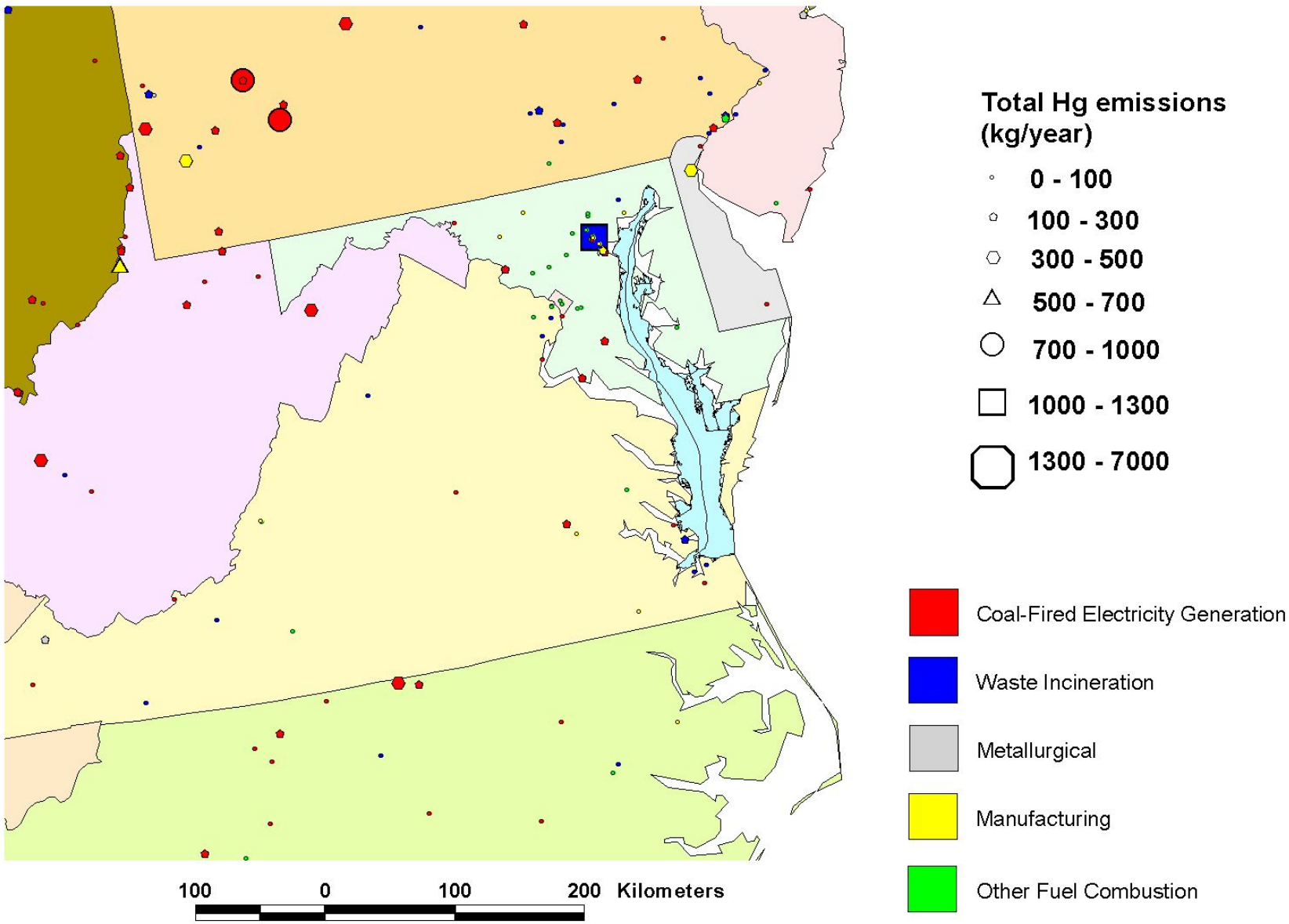


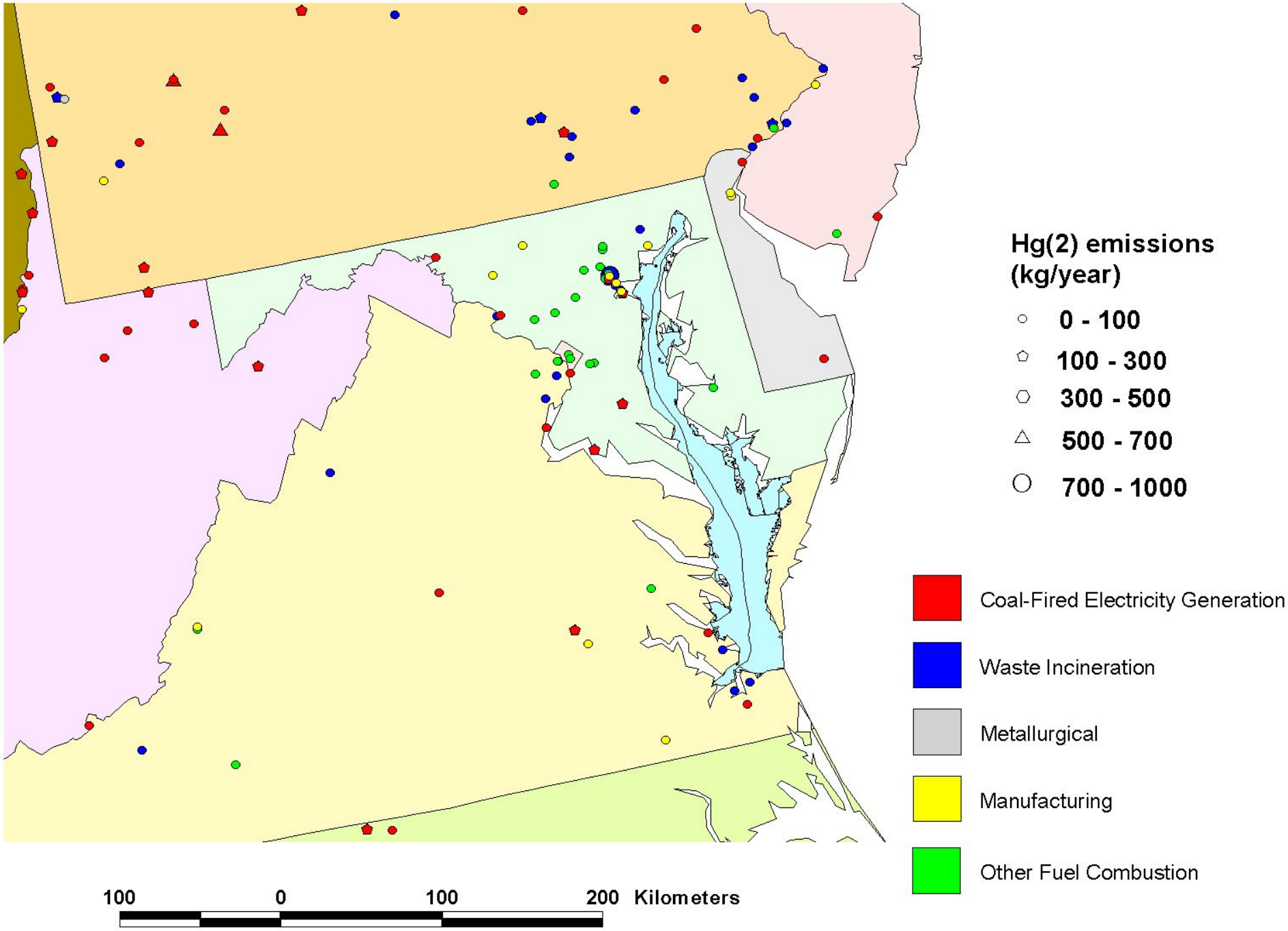

Mercury Emissions
(grams/km2- yr)
0 - 0.001
0.001 - 0.01
0.01 - 0.1
0.1 - 1
1 - 10
10 - 100
100 - 1000
1000 - 10000
50 0 50 100 Kilometers
N


Mercury Emissions
(grams/km2- yr)
0 - 0.001
0.001 - 0.01
0.01 - 0.1
0.1 - 1
1 - 10
10 - 100
100 - 1000
1000 - 10000
50
0
50 Kilometers
N

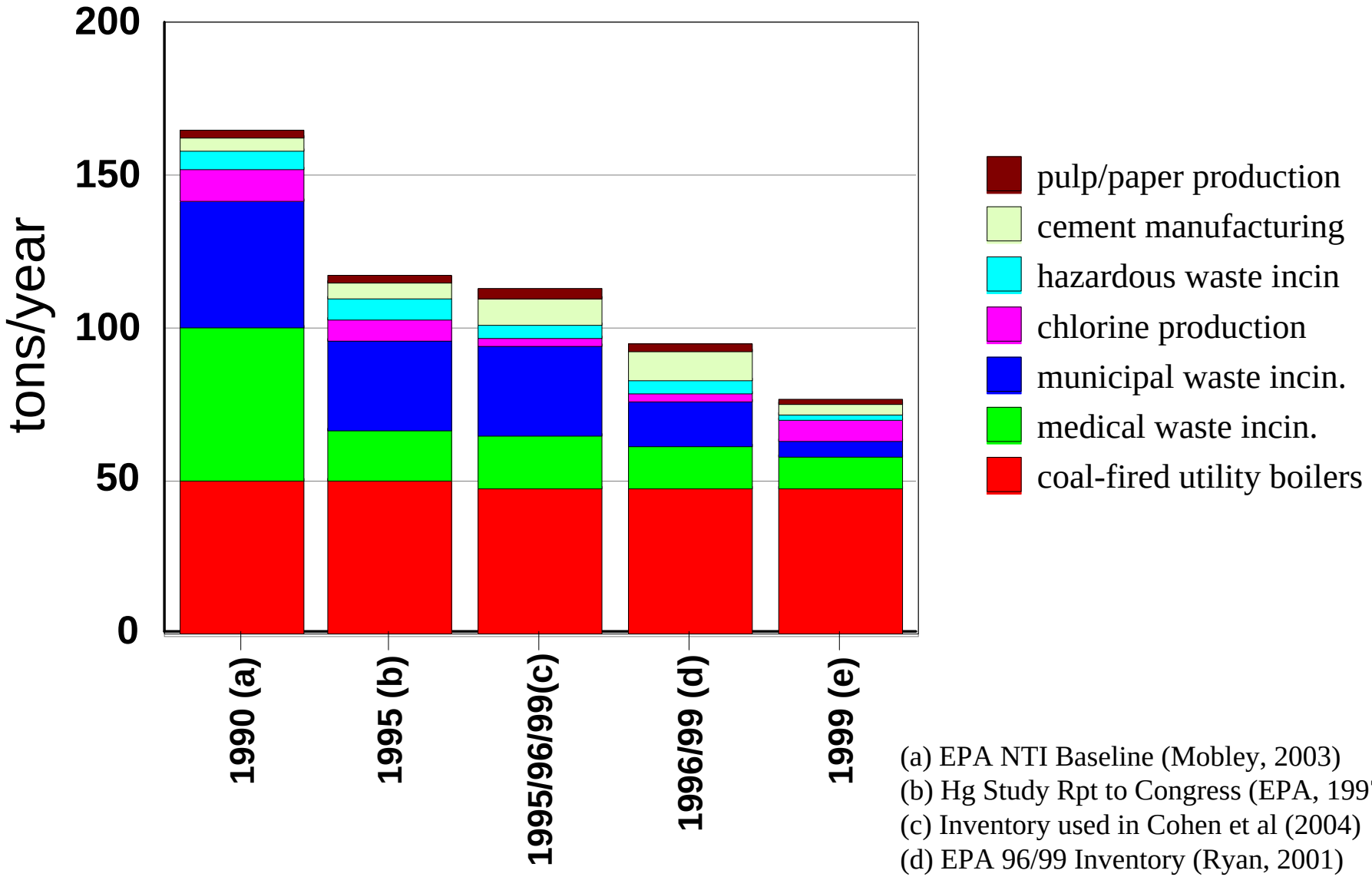

Total Hg emissions
(kg/year)
0 - 100
100 - 300
300 - 500
500 - 700
700 - 1000
1000 - 1300
1300 - 7000
Coal-Fired Electricity Generation
Waste Incineration
Metallurgical
Manufacturing
Other Fuel Combustion
1000
0
1000
2000
Kilometers


Total Hg emissions
(kg/year)
0 - 100
100 - 300
300 - 500
500 - 700
700 - 1000
1000 - 1300
1300 - 7000
Coal-Fired Electricity Generation
Waste Incineration
Metallurgical
Manufacturing
Other Fuel Combustion
500
0
500 Kilometers

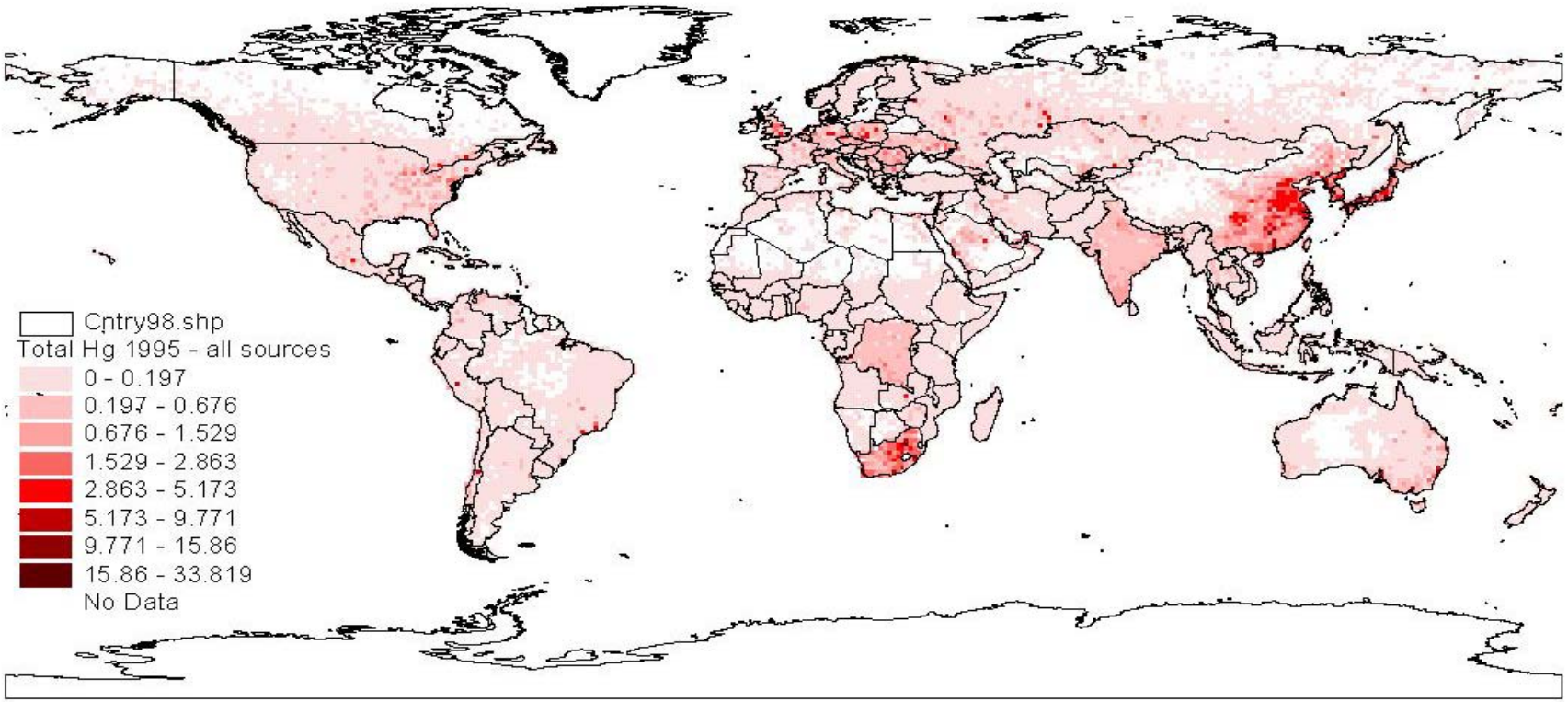

Total Hg emissions
(kg/year)
0 - 100
100 - 300
300 - 500
500 - 700
700 - 1000
1000 - 1300
1300 - 7000
Coal-Fired Electricity Generation
Waste Incineration
Metallurgical
Manufacturing
Other Fuel Combustion
100
0
100
200
Kilometers


Hg(2) emissions
(kg/year)
0 - 100
100 - 300
300 - 500
500 - 700
700 - 1000
Coal-Fired Electricity Generation
Waste Incineration
Metallurgical
Manufacturing
Other Fuel Combustion
100
0
100
200
Kilometers

Reported trends in U.S. atmospheric mercury emissions 1990-1999 (selected source categories)



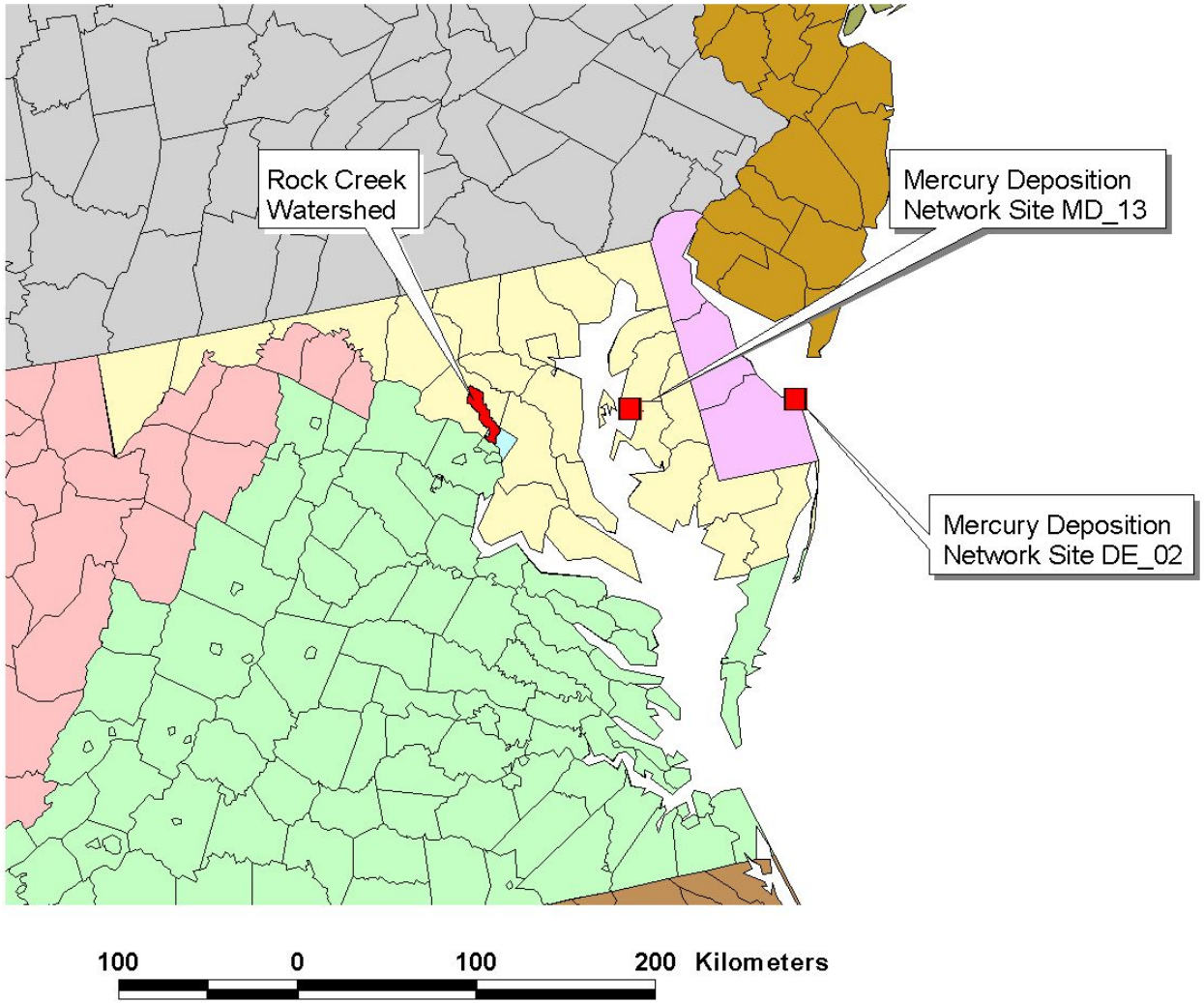
(a) EPA NTI Baseline (Mobley, 2003)
(b) Hg Study Rpt to Congress (EPA, 1997)
(c) Inventory used in Cohen et al (2004)
(d) EPA 96/99 Inventory (Ryan, 2001)
(e) EPA NEI 1999 (draft) (Mobley, 2003)



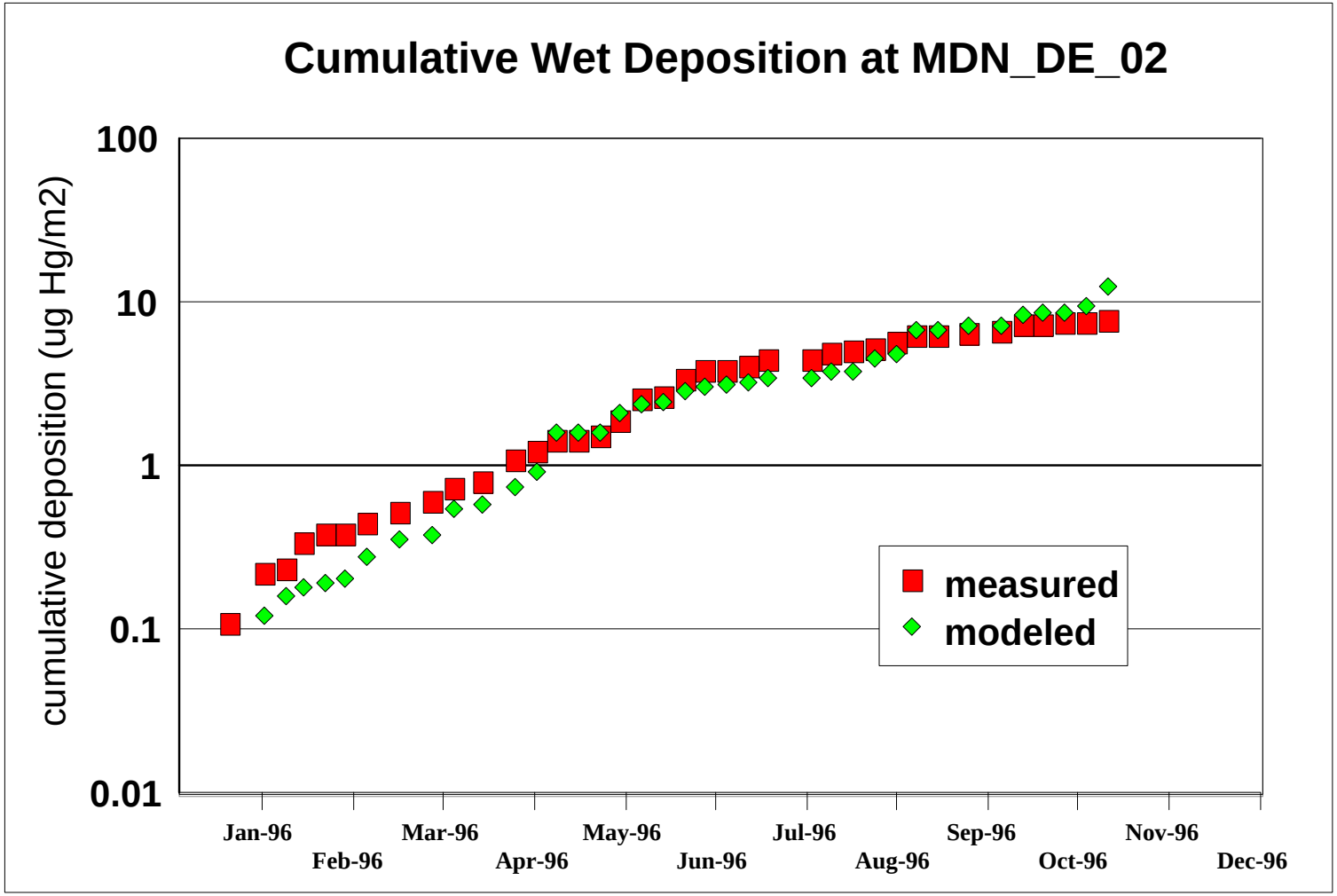
1995 Global Hg Emissions Inventory
Josef Pacyna,NILU, Norway (2001)

Model Evaluation

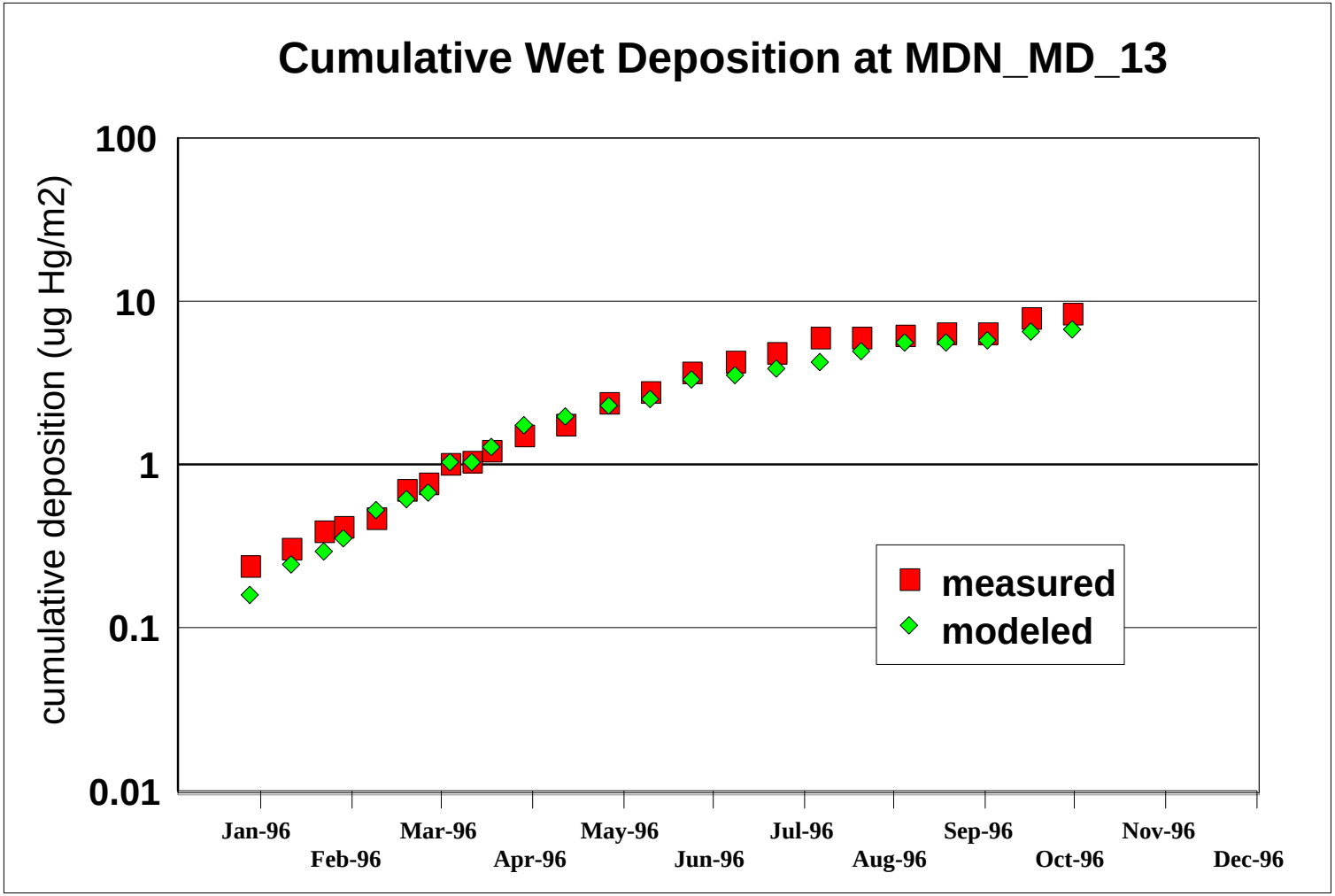
Mercury Deposition Network Sites with 1996 data in the Chesapeake Bay Region



Modeled vs. Measured Wet Deposition at Mercury Deposition Network Site DE_02 during 1996

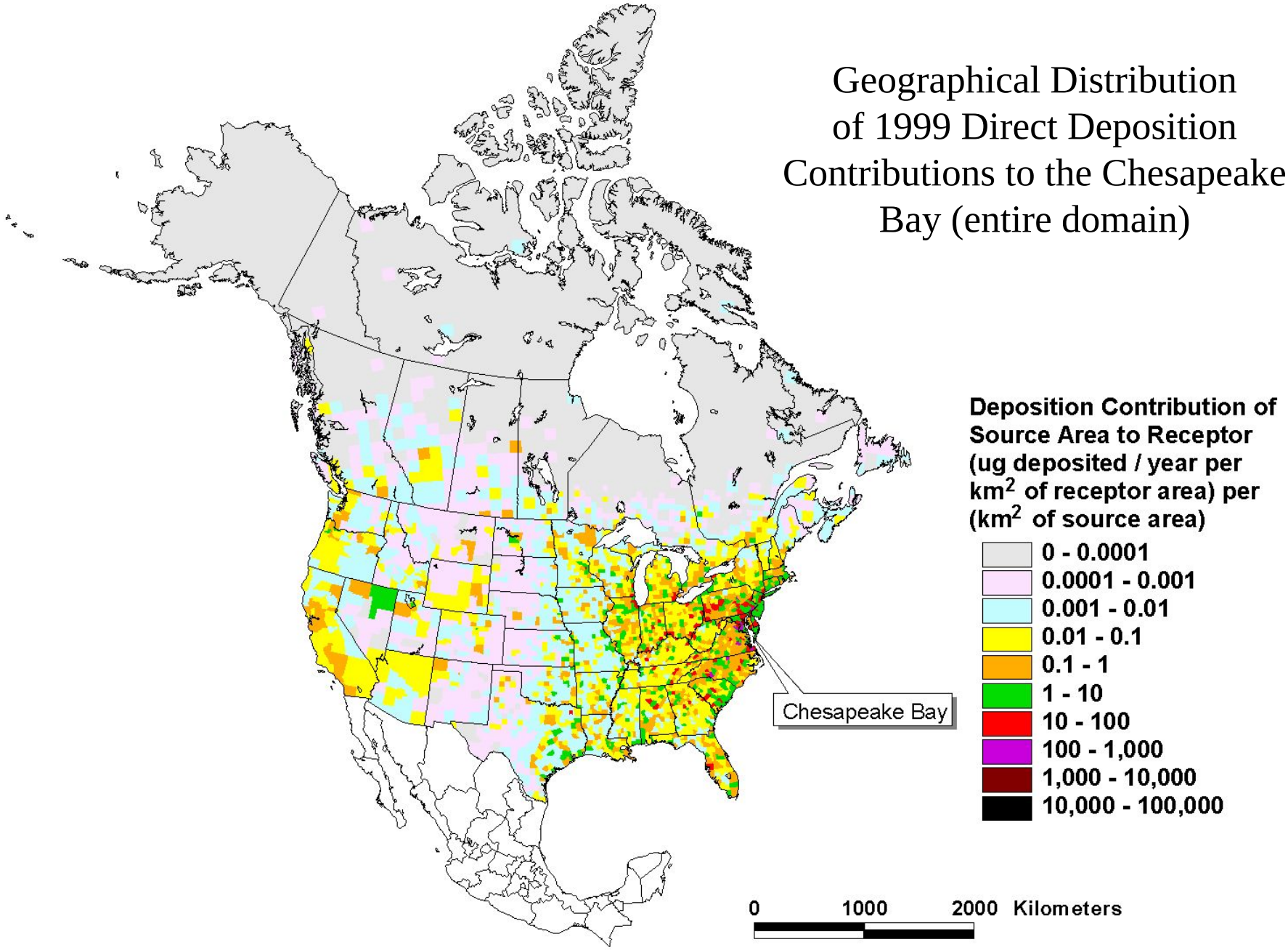


Modeled vs. Measured Wet Deposition at Mercury Deposition Network Site MD_13 during 1996

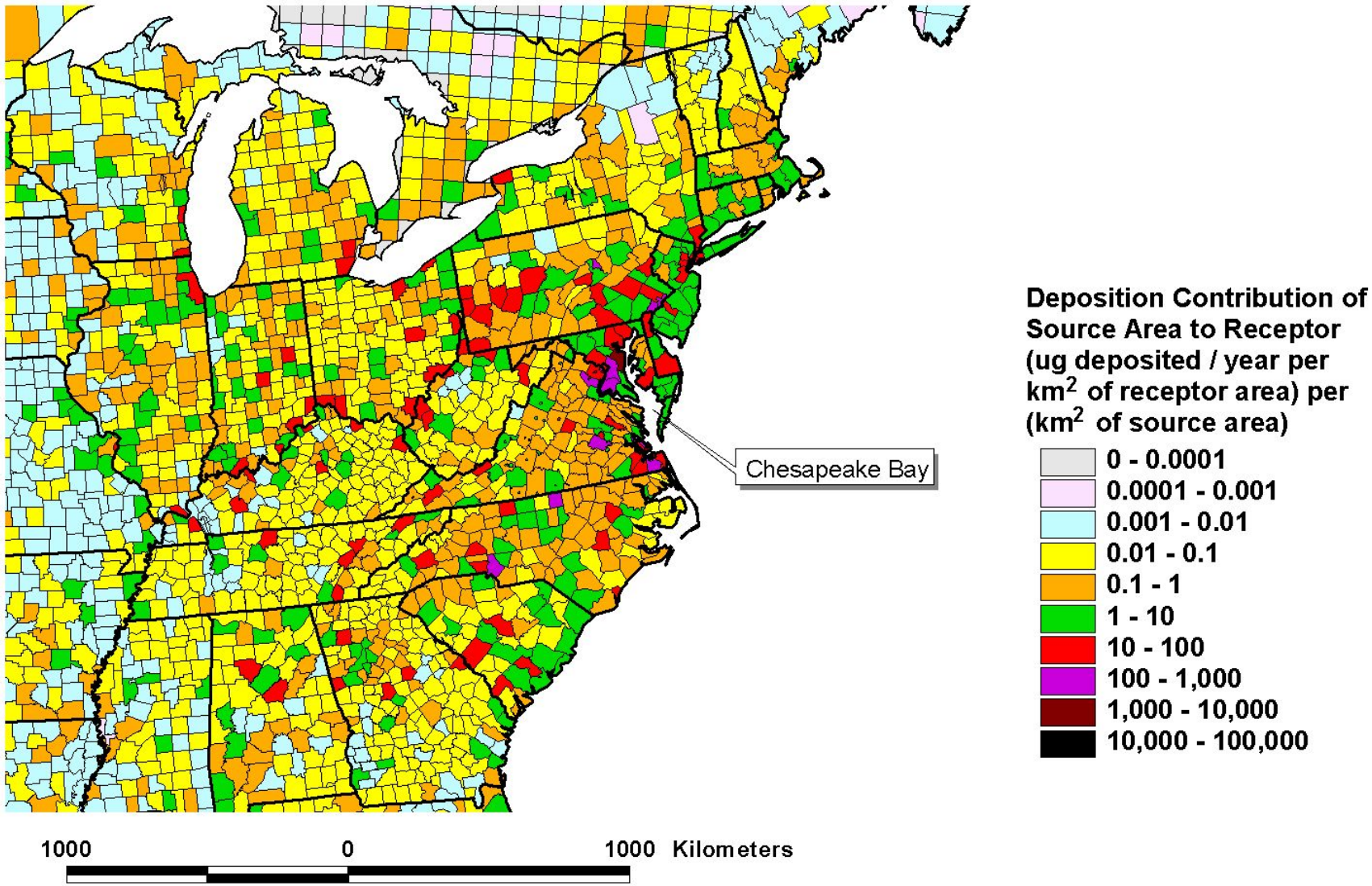


1999 Results for Chesapeake Bay

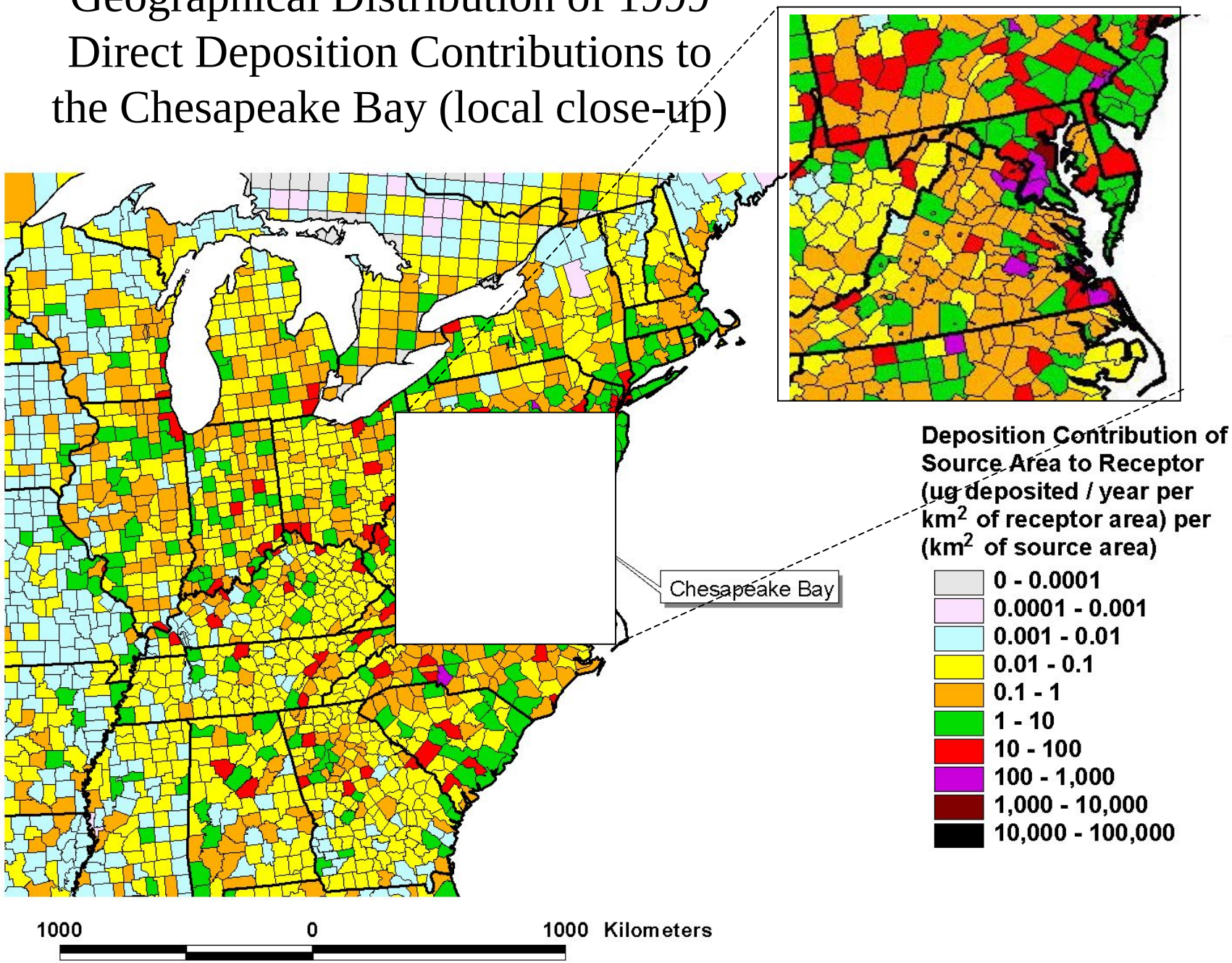
Geographical Distribution of 1999 Direct Deposition Contributions to the Chesapeake Bay (entire domain)



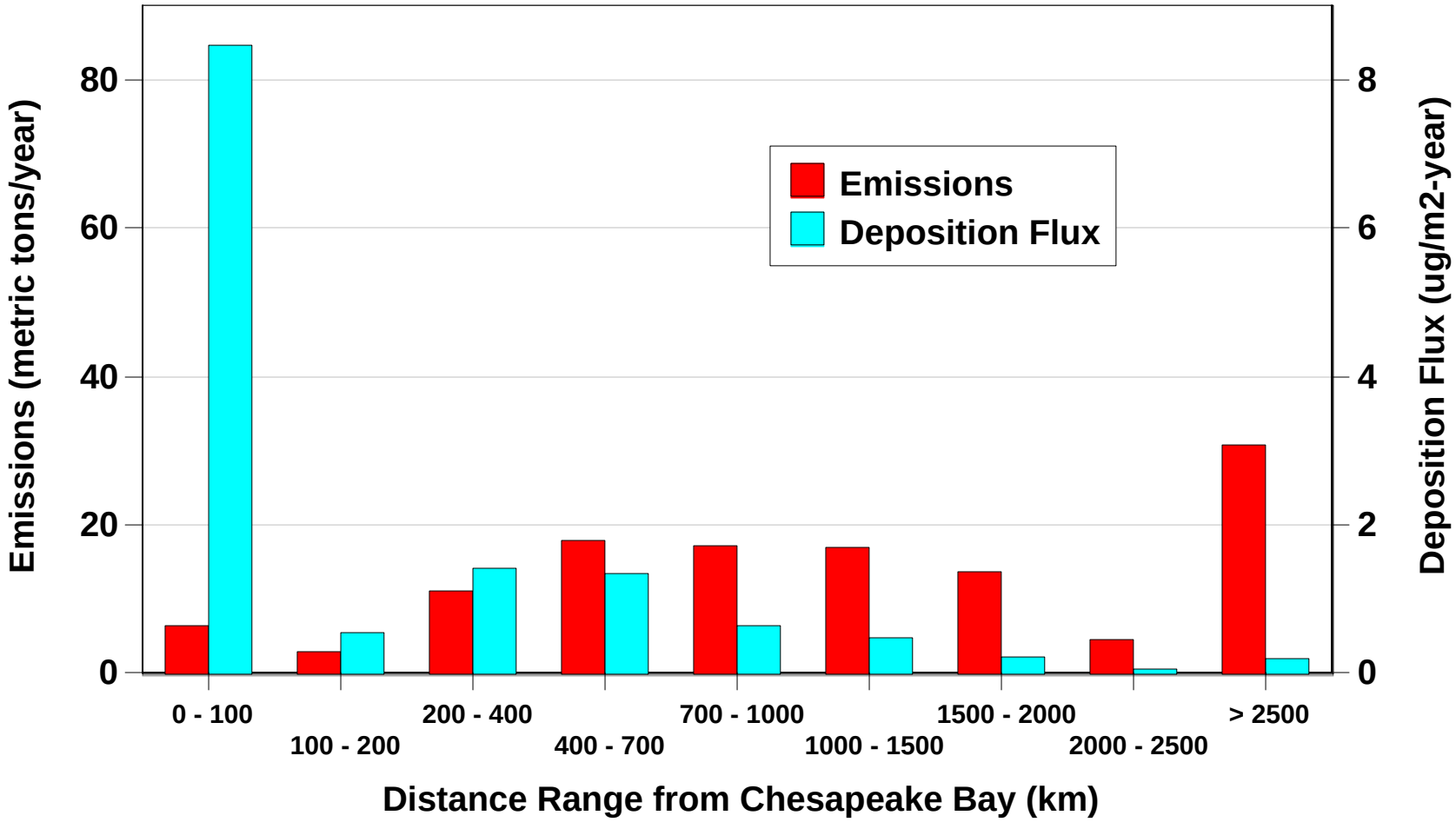
Geographical Distribution of 1999 Direct Deposition Contributions to the Chesapeake Bay (regional close-up)



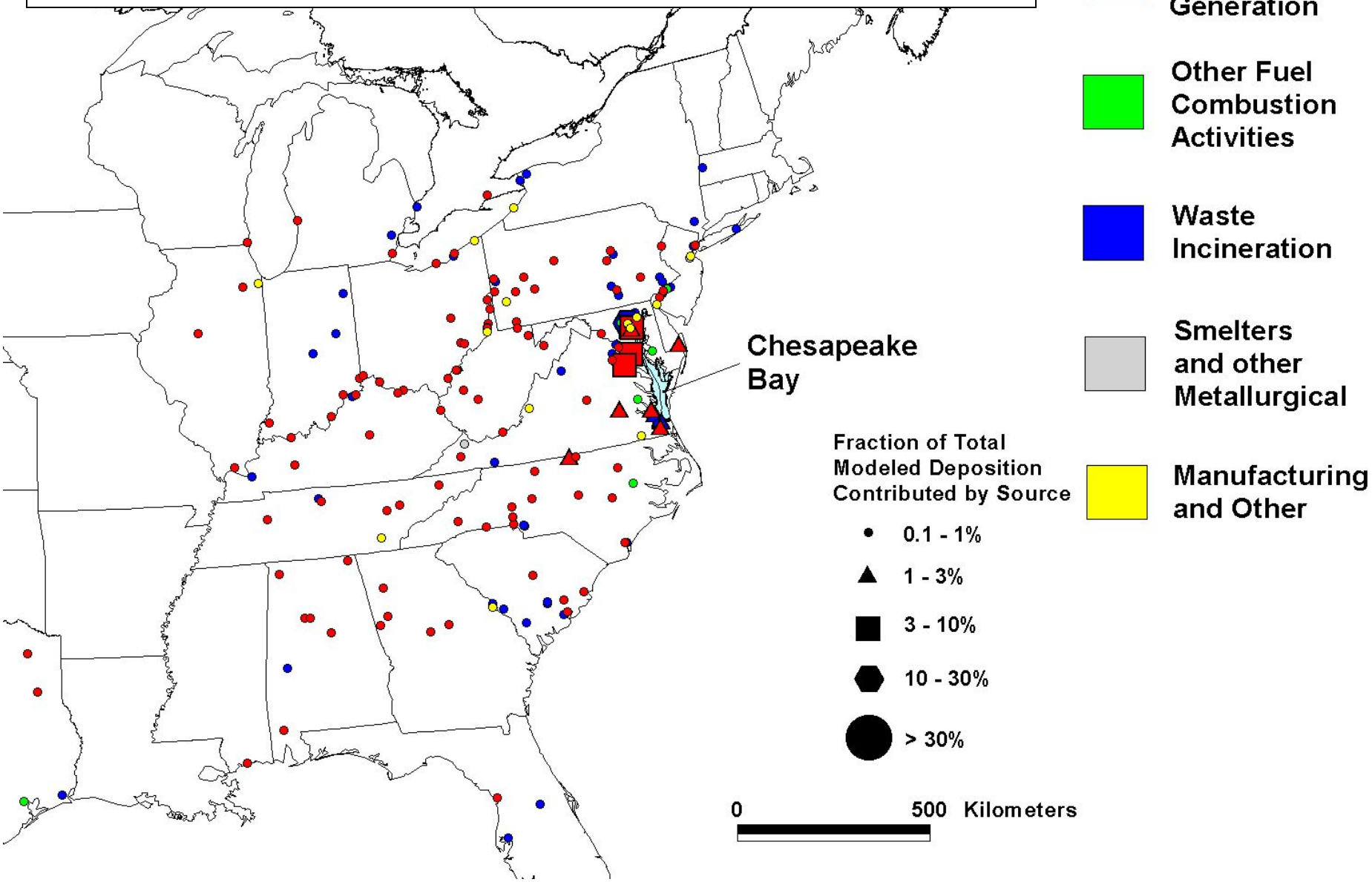
Geographical Distribution of 1999 Direct Deposition Contributions to the Chesapeake Bay (local close-up)



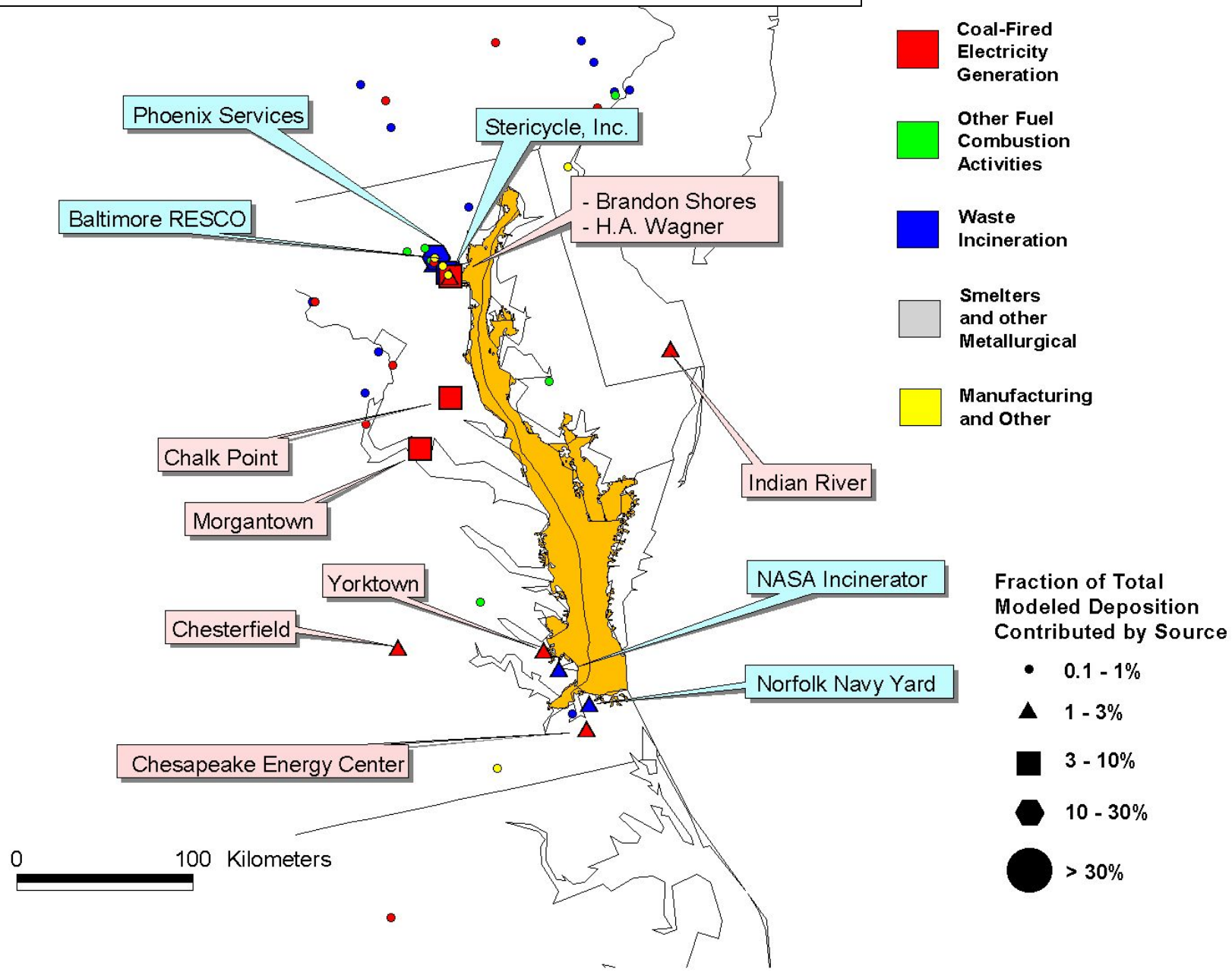
Emissions and Direct Deposition Contributions from Different Distance Ranges Away From the Chesapeake Bay



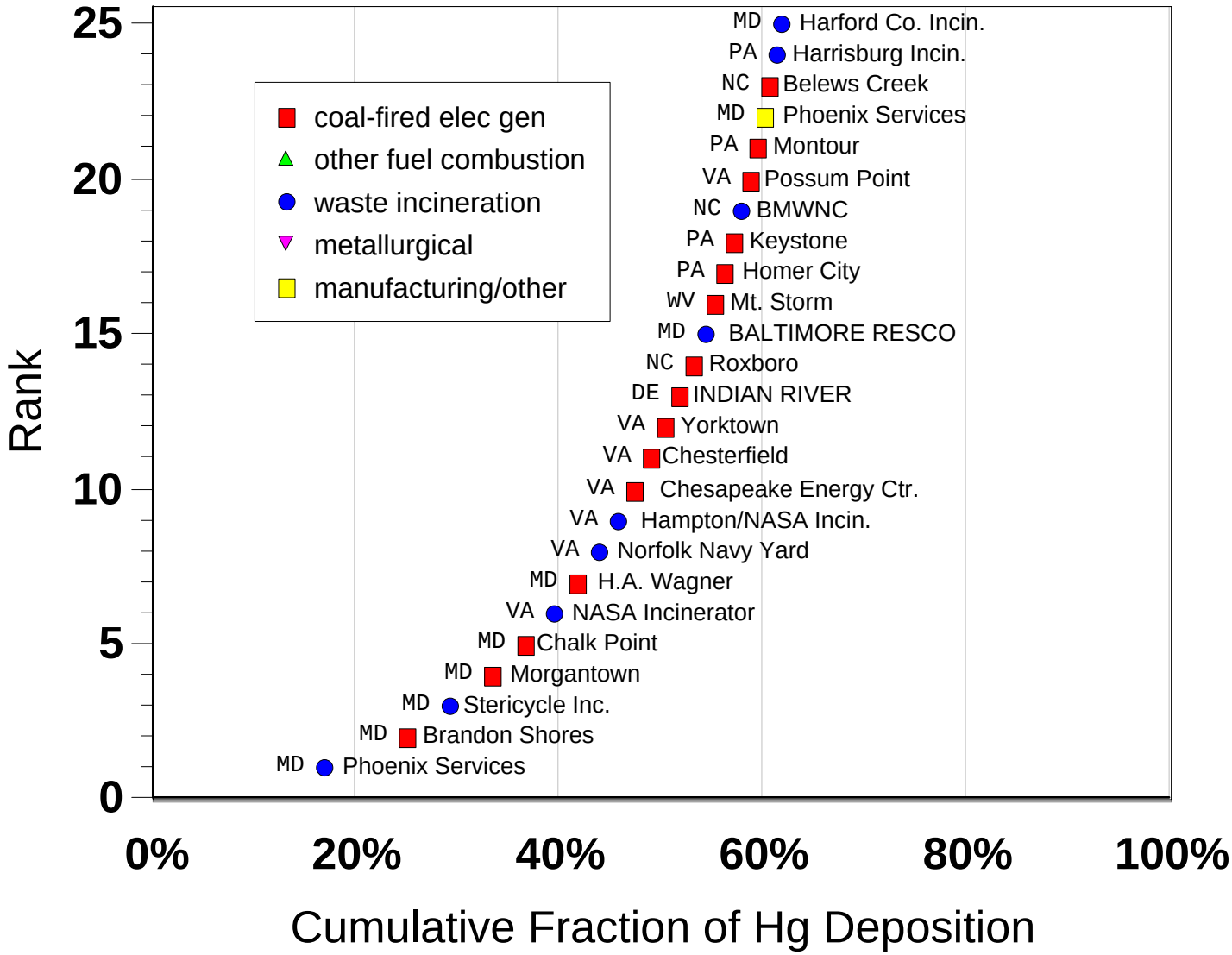
Largest Regional Individual Sources Contributing to 1999 Mercury Deposition Directly to the Chesapeake Bay



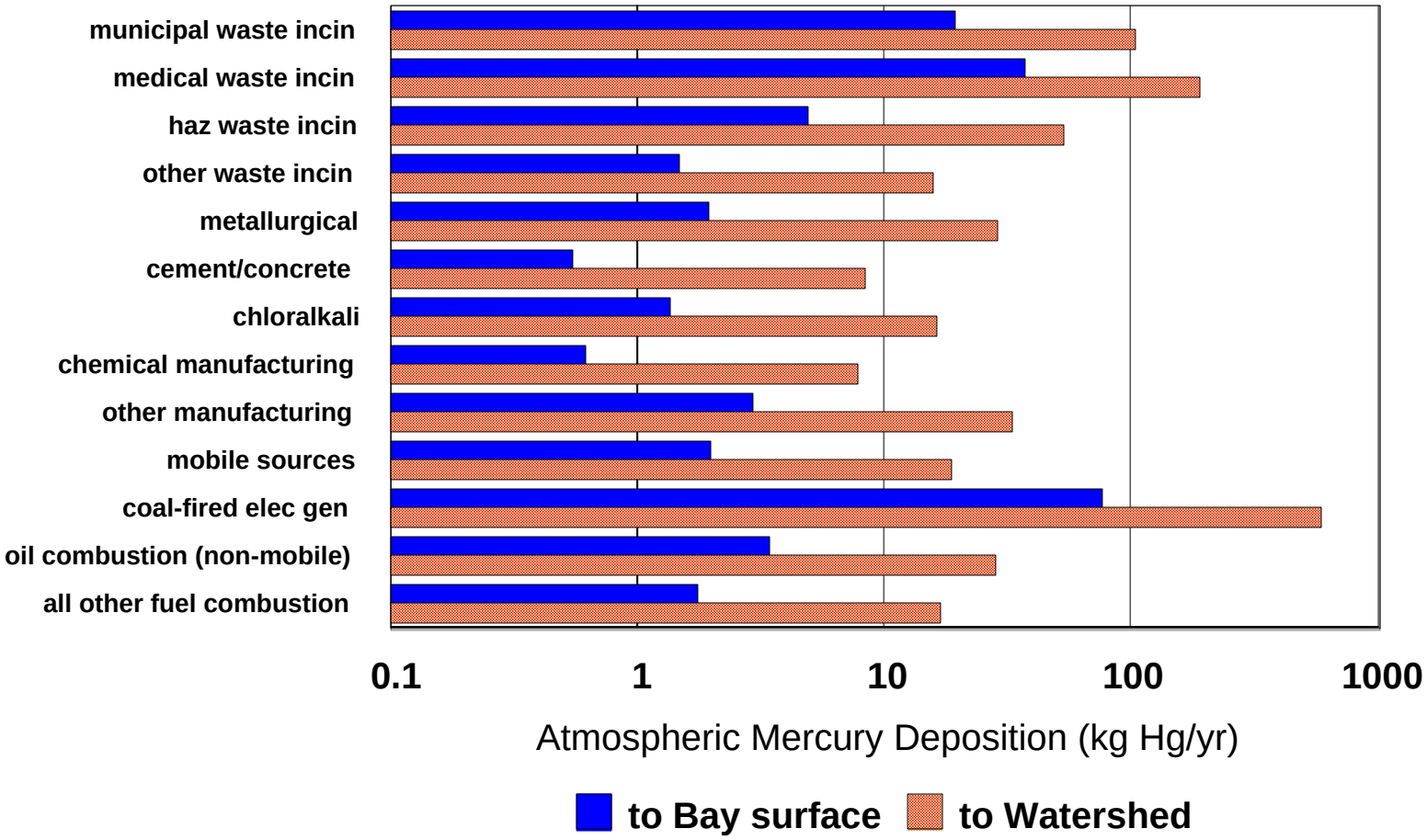
Largest Local Individual Sources Contributing to 1999 Mercury Deposition Directly to the Chesapeake Bay



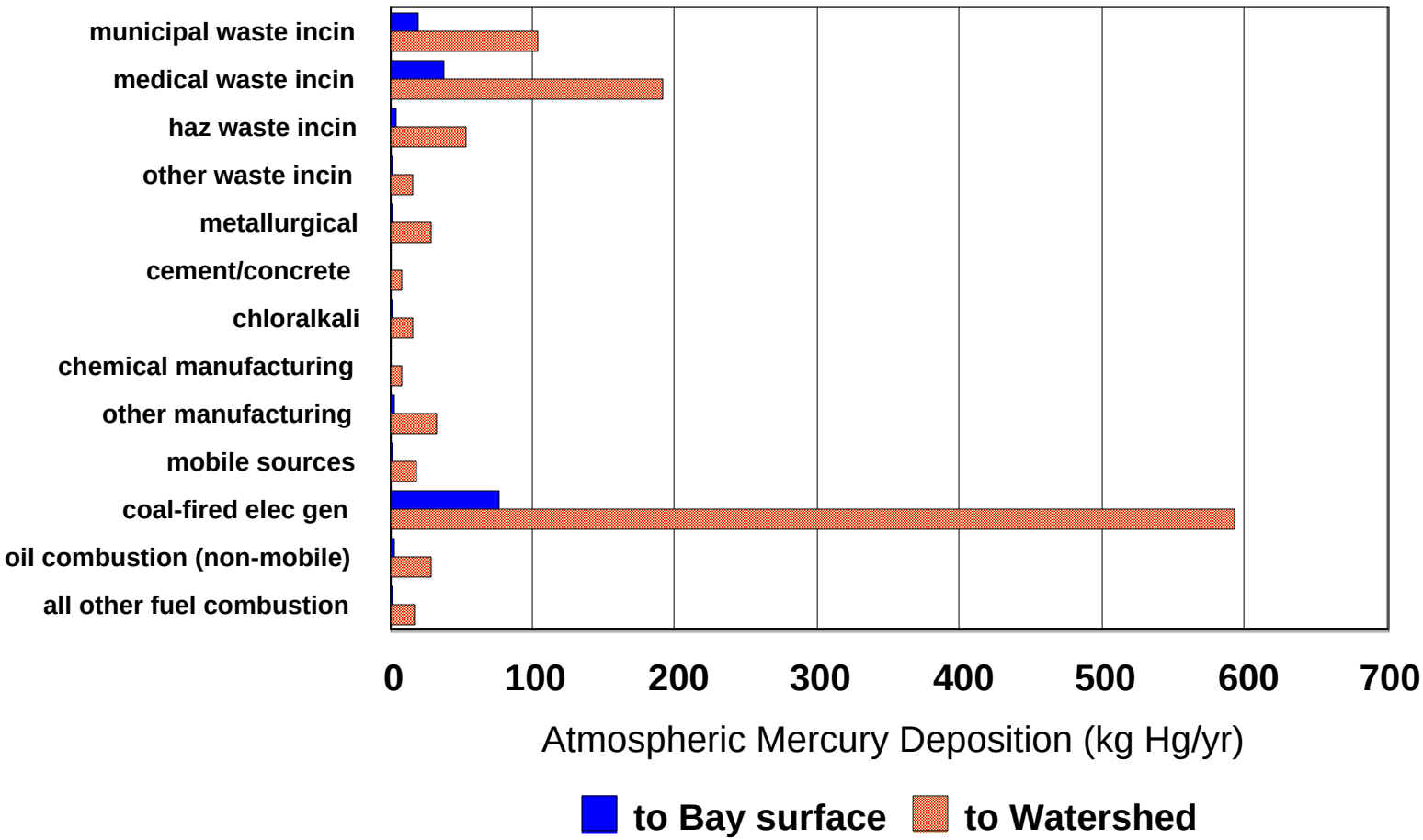
Top 25 Contributors to 1999 Hg Deposition Directly to the Chesapeake Bay



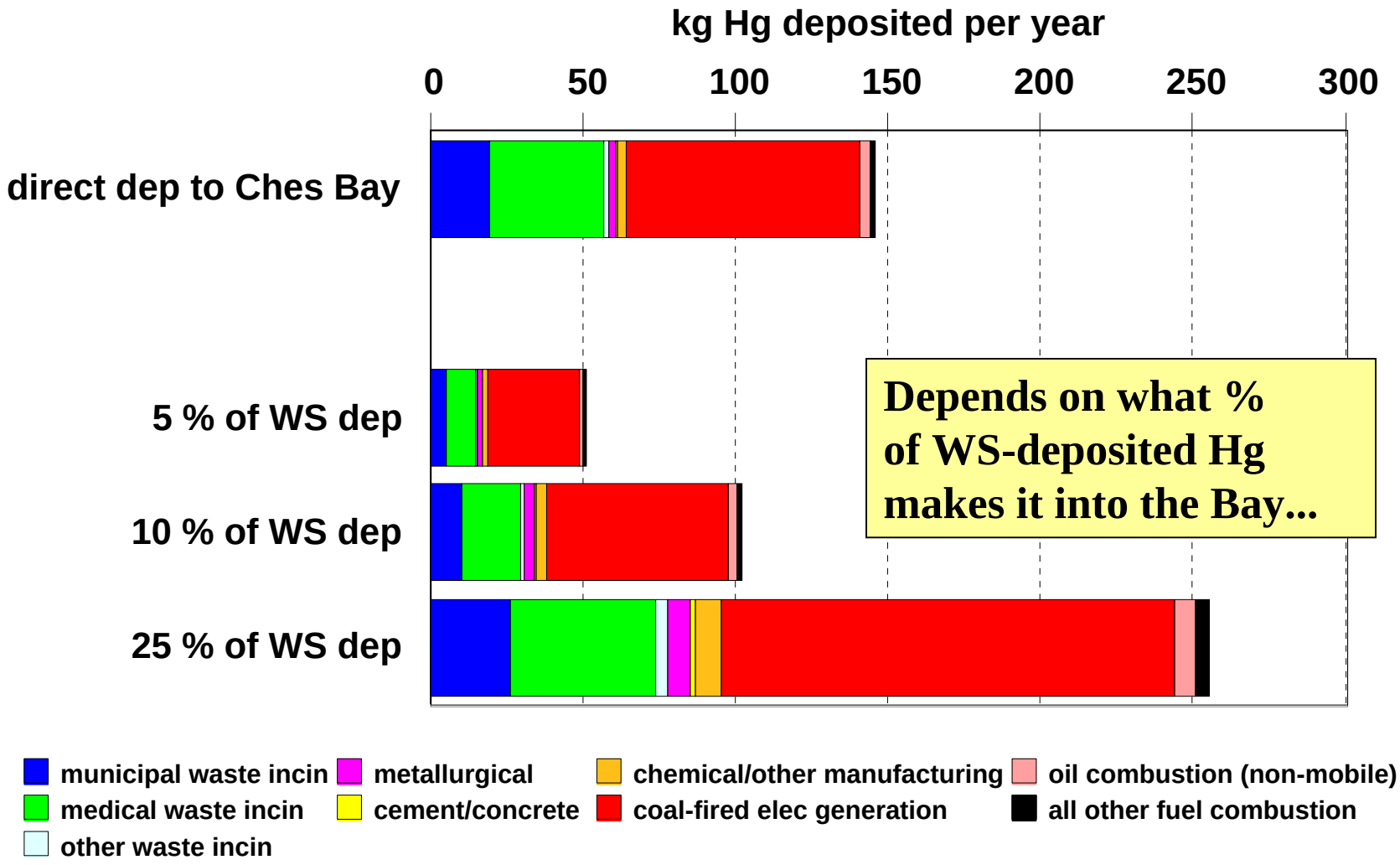
Deposition to the Chesapeake Bay and to its Watershed (~1999) (logarithmic graph)



Deposition to the Chesapeake Bay and to its Watershed (~1999) (linear graph)

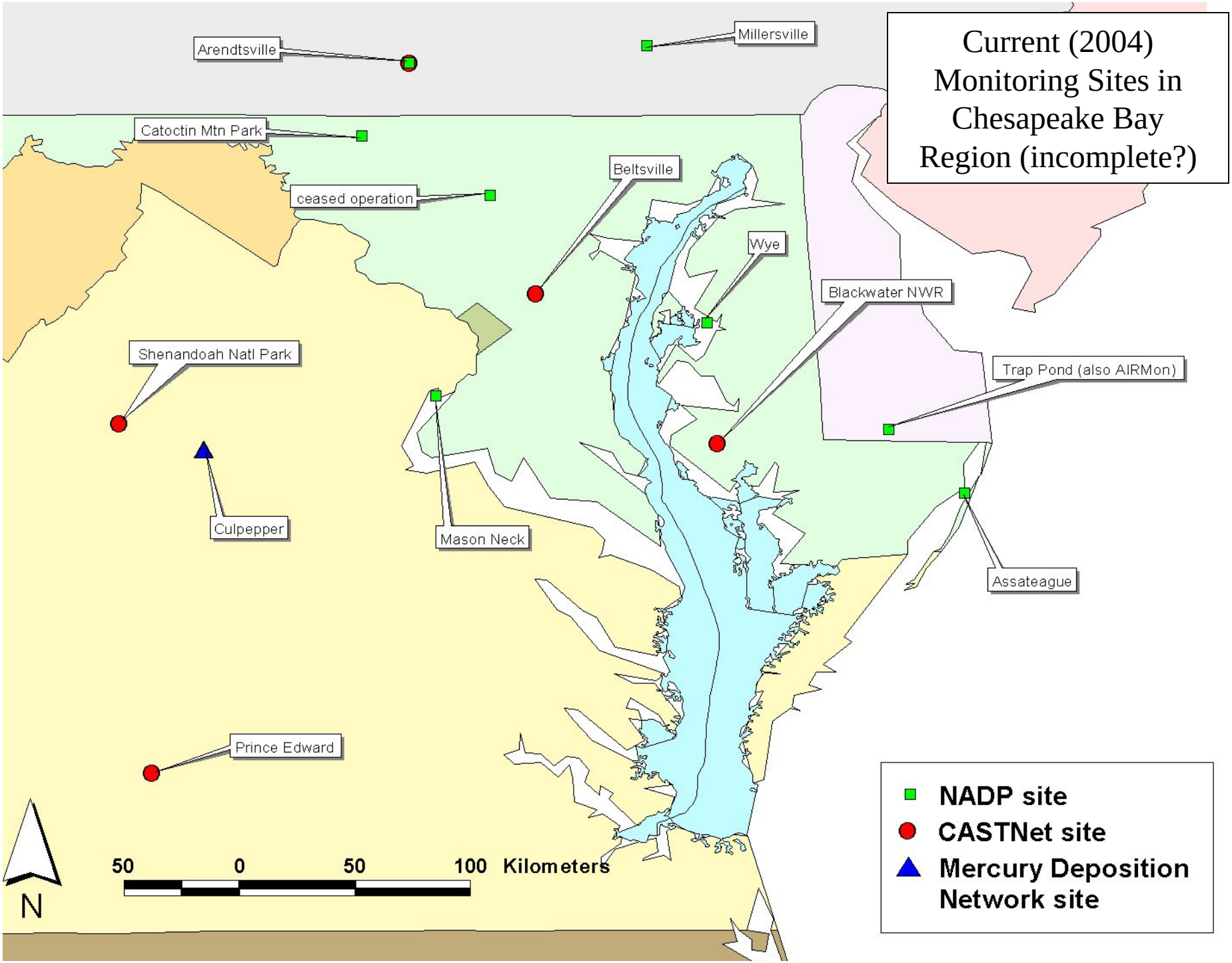


What is Relative Importance of Hg Deposited Directly to Chesapeake Bay Surface vs. Deposition to Watershed (?)



Some Next Steps

- Use more highly resolved meteorological data grid
- Expand model domain to include global sources
- Simulate natural emissions and re-emissions of previously deposited Hg
- Additional model evaluation exercises ... more sites, more time periods, more variables (e.g., not just wet deposition).
- Sensitivity analyses and examination of atmospheric Hg chemistry in the marine boundary layer and at upper elevations...


Current (2004) Monitoring Sites in Chesapeake Bay Region (incomplete?)
Arendtsville
Millersville
Catoctin Mtn Park
ceased operation
Beltsville
Wye
Blackwater NWR
Shenandoah Natl Park
Trap Pond (also AIRMon)
Culpepper
Mason Neck
Assateague
Prince Edward
50 0 50 100 Kilometers
N
NADP site
CASTNet site
Mercury Deposition Network site

INSTITUTE OF CLEAN AIR COMPANIES

1660 L Street NW
Suite 1100
Washington, DC 20036-5603
Telephone 202.457.0911
Fax 202.331.1388

David C. Foerter, *Executive Director*
Email: dfoerter@icac.com

Chad S. Whiteman, *Deputy Director*
Email: cwhiteman@icac.com

June 29, 2004

Honorable Michael Leavitt, Administrator
U.S. Environmental Protection Agency
EPA Docket Center (Air Docket)
Mail Code: 6102T, Room B-108
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Attention Docket ID No. OAR-2002-0056

Dear Administrator Leavitt:

The Institute of Clean Air Companies (ICAC) is the national trade association of companies that supply air pollution control and monitoring technology. Our members include nearly eighty leading suppliers of air pollution control and monitoring technologies for stationary sources. These companies operate and provide environmental solutions for affected industries as well as employment opportunities across the U.S.

The Institute congratulates EPA's efforts to propose a much-needed rule that provides for the reduction of mercury emissions from coal- and oil-fired electric generating facilities in order to protect public health. The Institute has a few observations concerning the proposed Utility Mercury Reduction Rule specifically concerning the performance of control technologies, technology guarantees, commercial availability, control costs, by-product disposal, and availability of construction resources. The Institute has submitted two separate sets of comments addressing mercury control and measurement technologies.

The Institute recommends that EPA pursue a regulatory framework that fully encompasses the capability and capacity of the air pollution control and measurement industry to achieve substantial reductions in mercury emissions, and then, provide regulatory flexibility to enable the most cost effective application of a range of technologies. Based on our thorough understanding of technology capabilities and the capacity of our industry to supply these technologies, we believe

that a 50 to 70 percent reduction in current mercury emissions is feasible by 2008 to 2010. As a result, emissions would be reduced to a maximum of 14 to 24 tons. We note that compliance flexibility would enable a cap based on a 70 percent reduction, with some units able to achieve reductions of 90 percent and greater, and some units as low as 50 percent. Setting an appropriately stringent cap and then providing compliance flexibility would moderate any performance differences at individual units due to differences in coal, equipment, and flue gas characteristics. However, giving priority to compliance flexibility over the adequate consideration of a feasible emission cap, such as occurred in this proposed rule, fails to address the public health issues.

The attached ICAC comments provide both general and detailed comment that support the development of a mercury control rule with greater benefits.

We look forward to working with EPA on this important issue and invite you and your staff to contact me if you have any questions.

Sincerely,

David C. Foerter
Executive Director, ICAC

Enclosure a/s

GENERAL COMMENT

The rapid development of mercury control technologies over the last several years has produced a number of technologies that are available for the implementation of a national mercury control regulation for coal- and oil-fired power plants. A large number of laboratory tests and full-scale demonstrations have been conducted that provide information on the effectiveness of controls for various coal types and control configurations. Despite the current lack of a national control requirement for mercury, a number of options are commercially available while others are still in the development and testing phases.

Past experience with technology development for other pollutants (SO_2, NO_x, and PM) as well as other source categories such as mobile sources, suggests that delaying the regulation of mercury emissions from power plants would serve to delay the development of innovative control technologies. Research and development efforts are unlikely to be sustained at a vigorous level in the absence of regulatory or other drivers capable of creating a viable market for advanced control technologies. Larger markets provide more incentives for the development of technologies as well as foster competition between vendors that produces more innovative and cost effective solutions for affected sources. Smaller markets such as those that may be developed with the implementation of State regulations (e.g. Massachusetts, Connecticut, Wisconsin, New Jersey, North Carolina) are beneficial to the air pollution control industry but will be less effective in developing healthy markets than a timely implemented national program.

With the implementation of a national program, multiple control options including precombustion, combustion and post combustion technologies will contribute to meeting the required emission reductions. Coal cleaning as well as coal switching are examples of options that have the potential to reduce mercury emissions prior to fuel combustion.

Based on the recent test results, significant amounts of mercury can be removed through the use of existing controls. Existing control installations such as fabric filters, electrostatic precipitators, SO_2 scrubbers, and selective catalytic reduction (SCR) are currently achieving an estimated 36% reduction in mercury emissions even though these processes were not originally designed nor optimized for mercury capture. This is based on EPA's information collection request findings that an estimated 75 tons of mercury was contained in coal burned by power plants while 48 tons were emitted out of the stack. The current level of co-benefit control varies significantly with some combinations of control devices and coal types achieving as much as 90% removal and others not demonstrating any co-benefit control. With the implementation of mercury regulation beyond incidental co-benefit levels of control, a number of options for optimization of existing controls will be implemented to provide cost effective reductions in a short period of time.

Mercury specific control technologies such as sorbent injection systems have been demonstrated at full-scale. Multipollutant control approaches as well as other mercury specific technologies have also demonstrated significant progress and will provide additional low cost, innovative approaches to mercury control. A number of these technologies, including sorbent injection systems as well as SCR coupled with wet FGD, have achieved removal rates greater than 90% under certain circumstances.

Under the Section 112 MACT proposal, EPA also made projections for mercury control installations. These projections were based on the assumption that the Clean Air Interstate Rule/Interstate Air Quality Rule (CAIR/IAQR) would not be implemented and the co-benefit control from SO_2 and NO_x control installations would not be realized. Based on the development and enhancement of mercury removal from existing controls, significant reductions in mercury emissions are likely to occur without touching the potential of currently available control technologies. Also under the Section 112 MACT proposal, EPA did not consider ACI technology in the development of the MACT floor as EPA stated that this technology was not demonstrated nor commercially available (U.S. EPA, 2004). Activated carbon injection is commercially available and has been demonstrated on at least four full-sized coal-fired plants to-date with additional full-sized tests scheduled later this year (see details below). Outside of the United States, the Berrenrath 275 MW and the Wachtberg 166 MW plants in Germany operate on carbon injection technology to control mercury. Based on this knowledge, EPA should consider ACI in the development of the MACT floor as it is a viable technology for the electric power sector that has also been proven in other industrial sectors to control mercury emissions. What is contradictory in EPA's analysis is that they used ACI in their cost modeling exercises with the integrated planning model (IPM) but failed to recognize this technology in setting the level of mercury reductions for the MACT requirement.

Based on the current availability of mercury specific control options and the near term development of other promising technologies, EPA's own analysis (ref: Office of Research and Development submittal to the e-docket) has indicated that a reduction of 50-70% of current emission levels in the 2008 to 2010 timeframe is justifiable. This corresponds to annual mercury emissions of between 14-24 tons for the electric power sector. This level of reduction seems even more reasonable considering that EPA estimated that the co-benefit level of mercury emissions cap under the 2003 Clear Skies proposal was initially set at a first phase level of 26 tons of mercury emissions in 2010. The first phase cap is somewhat below EPA's estimated co-benefits estimate of 30 tons, and switching by units to different coal types with lower mercury content would be likely for compliance with mercury control requirements. The current mercury control proposal made under the Clean Air Act, Section 111 provisions would not create markets for technology development nor encourage innovation as the projected mercury cap level was set at the revised co-benefit level that is much higher at 34 tons. Additionally, EPA's

modeling analysis does not consider the low cost reductions that will come from enhancing existing control technologies for greater mercury capture. These innovations will reduce the cost and overall demand for mercury specific reductions. EPA's projections for mercury specific control installations under the Section 111 proposal estimate that only 1 GW, or approximately two of the more than 1000 coal-fired boilers in the U.S., would install mercury control technologies by 2010.

Concerning the regulatory mechanism used for a mercury control program, ICAC would recommend including flexible mechanisms in the regulation that would encourage innovation while providing a clear goal with meaningful reductions. Examples of these types of mechanisms include early reduction incentives, market based approaches, capital recovery programs, plant wide averaging, safety valves or other approaches. These types of incentives combined with concrete goals would encourage technology innovation and reduce impacts on generation mix.

The air pollution control industry already has considerable experience with the implementation of mercury controls for other industrial sectors. Sorbent injection has been commercially proven to augment the removal of mercury in waste-to-energy plants. Experience controlling mercury emissions has been gained in more than 60 US and 120 international waste-to-energy plants which burn municipal or industrial waste or sewage sludge. For the past two decades, sorbent injection upstream of a baghouse has been successfully used for removing mercury from flue gases from these facilities. Other reagents used include activated carbon, lignite coke, sulfur containing chemicals, or combinations of these compounds. The mercury control experience gained from the municipal and industrial waste combustors demonstrates that the air pollution control industry has been able to control mercury in the past and is able to apply their expertise to the electric power sector.

TECHNOLOGIES AND PERFORMANCE

The list of technologies provided below is not intended to be an exhaustive list of the available mercury control technologies as there are many new and emerging technologies not listed. The technologies below are just a sample of the technologies that are currently available or under development but will be available soon.

Sorbent Injection Systems

Injecting a sorbent such as powdered activated carbon, bromine, poly sulfides, or other sorbent into the flue gas represents a relatively simple approach to controlling mercury emissions from coal-fired boilers. The gas-phase mercury in the flue gas contacts the sorbent and attaches to its surface. The sorbent with the mercury attached is then collected by the existing particle control device, either an electrostatic precipitator (ESP) or fabric filter (FF) as shown in Figure 1. This combined material, consisting of 99% fly ash and 1% sorbent, is then either disposed of or beneficially used.

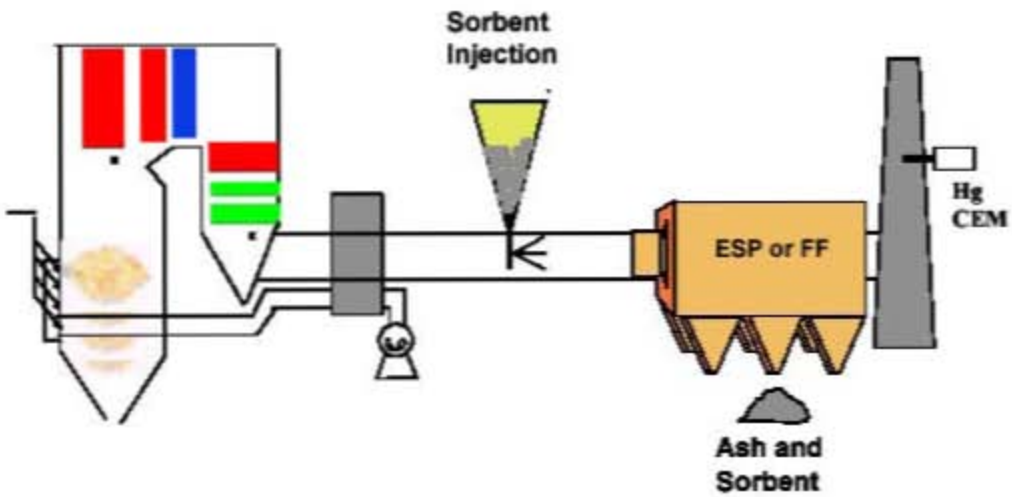


Figure 1. Schematic Diagram of Sorbent Injection Process

The type of particulate control equipment installed at the plant is a key parameter defining both the amount of sorbent that is required and the ultimate limitation of the amount of mercury that can be removed. The two primary particulate control devices are ESPs and fabric filters. When the sorbent is injected into the flue gas it mixes with the gas and flows downstream. This provides an opportunity for the mercury in the gas to contact the sorbent and be removed. This is called "in flight" capture. The sorbent is then collected in the particulate control device where there is a second opportunity for sorbent to contact the mercury in the gas. Because a fabric filter provides better contact than an ESP between the sorbent and the vapor-phase mercury, higher levels of mercury removal can be achieved at lower sorbent rates on units with a fabric filter. Currently only 10% of the power plants have fabric filters and the other 90% have ESPs.

Four full-scale demonstrations were conducted during 2001 and 2002 under a cooperative agreement from the Department of Energy National Energy Technology Laboratory (DOE/NETL), ADA-ES, PG&E National Energy Group (NEG), We

Energies, Alabama Power Company, Ontario Power, TVA, FirstEnergy, and EPRI. Follow-on funding was received for additional tests being conducted in 2003.

The first program was completed in the spring of 2001 at the Alabama Power E.C. Gaston Station (Bustard et al., 2002). This unit burns a low-sulfur bituminous coal and uses a hot-side ESP followed by a COHPAC™ fabric filter as a secondary collector for remaining fly ash. Activated carbon was injected into the fabric filter. The second program was conducted during the fall of 2001 at the WEC Pleasant Prairie Power Plant (PPPP) (Starns et al., 2002). This unit burns a subbituminous Powder River Basin (PRB) coal and uses an ESP to collect the carbon and fly ash. The third program was completed in the summer of 2002 at PG&E National Energy Group's Brayton Point Station (Durham et al., 2002). This unit burns low-sulfur bituminous coals and uses ESPs for particulate control. The fourth program was completed in the fall of 2002 at PG&E National Energy Group's Salem Harbor Station. Salem Harbor fires bituminous coals with an ESP for particulate control and an SNCR system for NO_x control.

Figure 2 presents full-scale data from two sites with ESPs; one bituminous coal and the other a Powder River Basin (PRB) coal. For both cases, mercury removal increases with increased rates of carbon injection. For the PRB coal, mercury removal was limited to 70% across the ESP. This limitation is most likely due to the trace amounts (< 1 ppm) of HCl available in the gas stream. For the bituminous coal, mercury removal exceeded 90% at the highest carbon injection rate. This coal has a high chloride content that resulted in approximately 150 ppm of HCl.

Manual mercury measurements were made at all sites following the draft Ontario Hydro method. Table 1 presents measurement results from the PRB test site with carbon injected upstream of an ESP. These tests show that the overall removal was 73% even though the majority of the mercury was in the elemental form, which is thought to be the more difficult form to capture. In fact, the collection efficiency was nearly identical for both elemental and oxidized mercury. This test is typical of all of the results that validate the capability of powdered activated carbon to capture all forms of mercury from both bituminous and subbituminous coals.

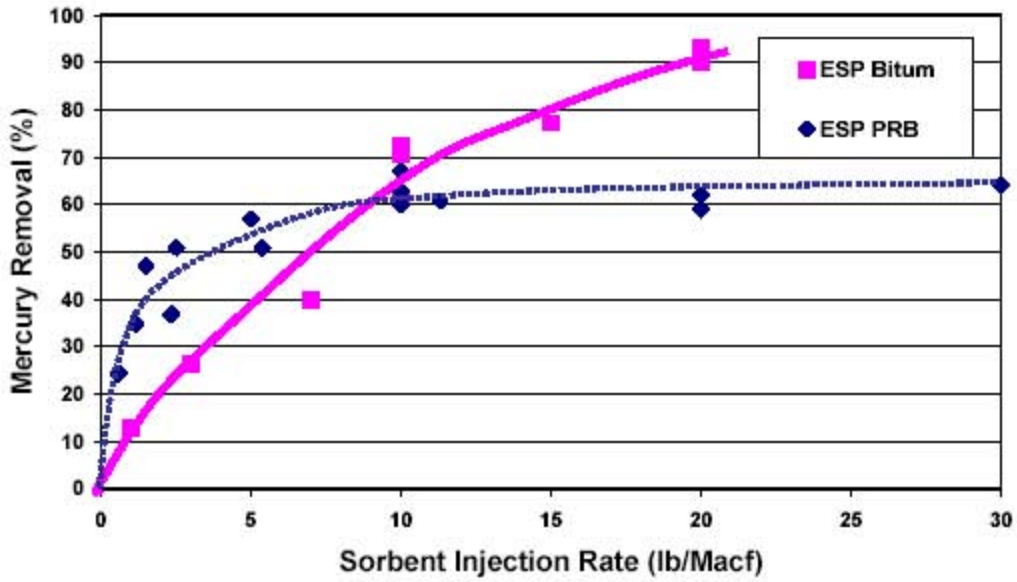


Figure 2. Mercury Removal with Activated Carbon Injection Upstream of an ESP

Table 1. Speciated Mercury Measured by the Ontario Hydro method, long-term tests with activated carbon injection concentration = 11 lbs/MMacf. Tests conducted at Pleasant Prairie Power Plant in fall 2001 (note: The configuration used at this facility made the ash unsuitable for sale).

	Particulate (µg/dncm)	Elemental (µg/dncm)	Oxidized (µg/dncm)	Total (µg/dncm)
ESP Inlet	1.0	14.7	1.7	17.4
ESP Outlet	0	4.3	0.4	4.7
Removal Efficiency (%)	100	70.7	74.5	72.9

Figure 3 shows performance of activated carbon injection (ACI) upstream of a fabric filter. This plot includes full-scale data from Plant Gaston on a bituminous, and reduced-scale tests conducted by EPRI on a PRB coal (Sjostrom, 2002a). The data from both fabric filter test programs show that ACI can produce 90% removal of mercury for both bituminous and subbituminous coals. Comparing the data from the fabric filter results in Figure 3 with the ESP results in Figure 2, it can be seen that the increased contact between the flue gas and the sorbent in the dust cake reduces the carbon feed requirements by nearly a factor of ten.

Ontario Hydro measurements of mercury removal during ACI tests with a fabric filter at Plant Gaston are presented in Table 2. As can be seen, the activated carbon is effective for both species of vapor-phase mercury.

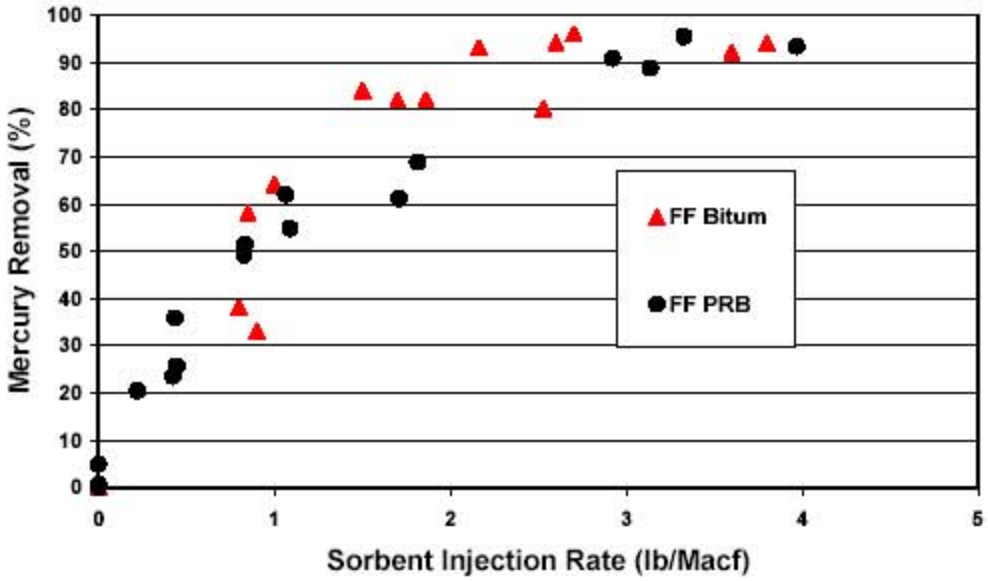


Figure 3. Mercury Removal with Activated Carbon Injection Upstream of a Fabric Filter.

Table 2. Average Mercury Removal Efficiencies Across COHPAC™ as Measured with the Ontario Hydro method.

Sampling Location	Particulate (µg/dncm1)	Oxidized (µg/dncm1)	Elemental (µg/dncm1)	Total (µg/dncm1)
COHPAC Inlet	0.2	6.4	4.6	11.2
COHPAC Outlet	0.1	0.9	0.0	1.1
Removal Efficiency (%)	50	86	99	90

Long-Term TOXECON Field Test at E. C. Gaston Station

The results of the first field test program at Gaston provided a good indication of the capabilities and limitations of the TOXECON technology for controlling mercury. However, the tests were performed for a limited amount of time, less than 200 hours of continuous operation, and did not allow for a thorough operational analysis of the use of this technology for mercury control. In the fall of 2002, ADA-ES was selected by the DOE to continue to mature the technology and conduct a long-term test program at the Gaston Station.

This program provides the first opportunity to evaluate activated carbon in the TOXECON configuration for a year of operation. Although new TOXECON units may be designed more conservatively than COHPAC units, important long-term operating data will be obtained through this test. Technical and financial support on this program will be provided by Southern Company and Alabama Power, the EPRI, Allegheny Energy, Arch Coal, Inc., FirstEnergy, Hamon Research-Cottrell, Ontario Power Generation, Duke Power and TVA (Durham, et al., 2003).

Figure 4 shows a plot of inlet and outlet mercury concentrations and overall mercury removal during four months of continuous operation. As can be seen, in spite of significant variability in the inlet mercury, the system has been able to maintain consistent levels of mercury removal with an overall average above 85%. These results further demonstrate the effectiveness of activated carbon injection for reducing mercury emissions. This technology is ideally suited for use on existing coal-fired boilers as it provides the following advantages: minimal capital cost of equipment (<$3/kW); can be retrofit with little or no downtime of the operating unit; effective for both bituminous and subbituminous coals; and can achieve 90% removal when used with a fabric filter that has been designed properly for carbon injection.

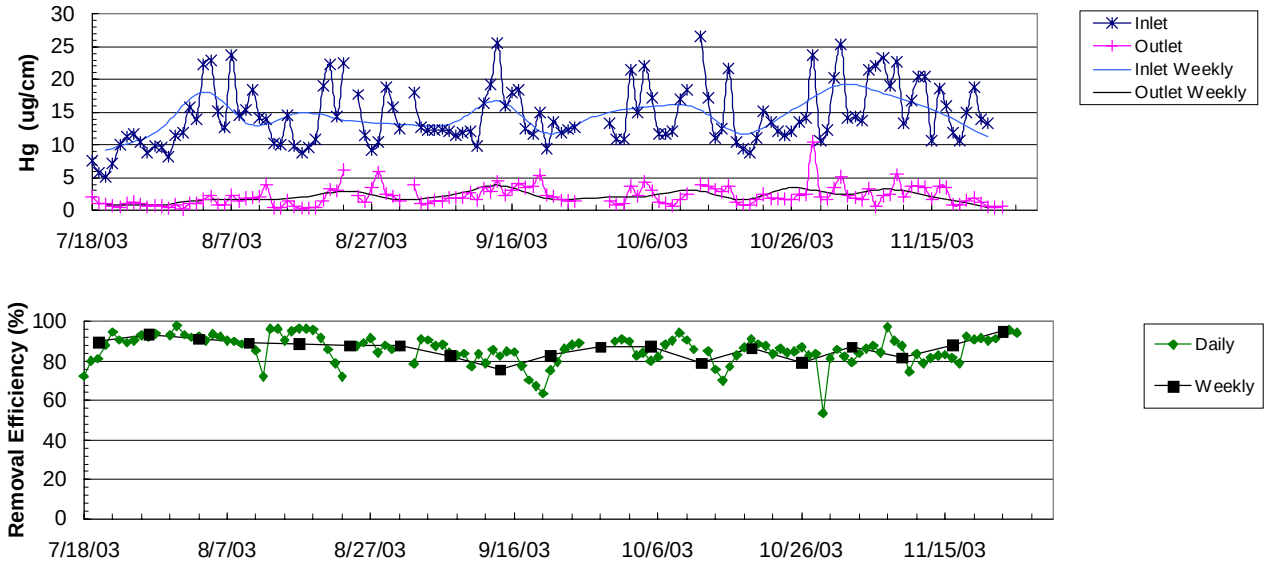


Figure 4. Inlet and Outlet Mercury Concentrations and Removal Efficiency for Toxecon Technology During Four Months of Operation at E. C. Gaston Station

Multipollutant Control Approaches

In anticipation of markets fostered by regulation or legislation, a number of multi-pollutant control technologies that also reduce mercury are currently being demonstrated. The long-term viability of these technologies for the coal-fired power

market largely depends on meaningful regulation to enable a commercial market in which these technologies would compete. That competition among a range of technologies, and enabled with flexibility within regulation, leads to the availability of increasingly cost-effective control options. Information has been included on electro-catalytic oxidation and a pre-combustion control technologies, technologies with vastly different approaches on how to address the same multi-pollutant problem on coal-fired power units.

Electro-Catalytic Oxidation (ECO) is an integrated multi-pollutant control technology that achieves major reductions in emissions of NO_x, SO_2 , fine particulate matter, and mercury from the flue gas of coal-fired power plants. The process also produces a valuable fertilizer co-product that reduces operating costs and avoids landfill disposal of waste.

ECO treats flue gas in three steps to achieve multi-pollutant removal as shown in Figure 5. In the first step of the process, a barrier discharge reactor oxidizes gaseous pollutants to higher oxides. For example, nitric oxide is oxidized to nitrogen dioxide and nitric acid, a small portion of the sulfur dioxide is converted to sulfuric acid, and mercury is oxidized to mercuric oxide. Following the barrier discharge reactor is an ammonia scrubber that removes the sulfur dioxide and the oxides of nitrogen. A wet electrostatic precipitator (WESP) follows the scrubber that in combination captures acid aerosols produced by the discharge reactor, fine particulate matter and oxidized mercury.

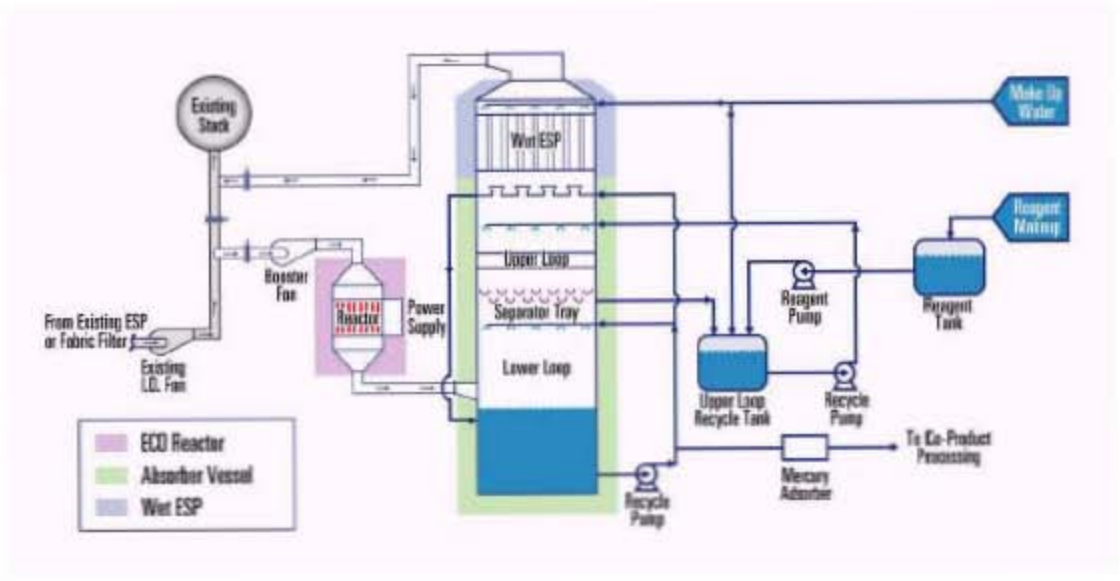


Figure 5. Schematic Diagram of Electro-Catalytic Oxidation (ECO) Process

The ECO process has undergone pilot scale testing on a 1-2 MW flue gas slipstream at FirstEnergy's R.E. Burger Plant for 2 years. In addition, a 50 MW commercial demonstration has been constructed at the same plant. The R.E.

Burger Plant burns a blend of eastern bituminous and western sub-bituminous coals with oxidized mercury being the predominant mercury species measured. The pilot testing has been successful and has shown that the ECO process consistently achieves 80 to 90% capture of the mercury contained in the plant's exhaust gas. Due to the multi-pollutant nature of this technology, the ECO pilot was also able to achieve greater than 90% NO_x removal, 98% SO_2 removal, and 96% removal of particles under 3 microns in size. A mercury semi-continuous emission monitoring system was recently installed on the 50 MW commercial demonstration unit. Although data is not yet available, total mercury removal is projected to be 80 to 90%; consistent with results obtained during pilot testing.

Pre-combustion coal scrubbing process was developed at Stanford Research Institute and currently holds the U.S. patent as *K-Fuel*. Pre-combustion cleaning of pollutants and their precursors is another mechanism to address multi-pollutant control issues on coal-fired power facilities, particularly the low rank coals such as lignite and western sub-bituminous coals. The established pre-combustion technology uses heat and pressure to physically and chemically transform low Btu, high moisture content low-rank coals, such as western sub-bituminous coal and lignite, into a high Btu, low moisture fuel. This coal processing technology increases energy efficiency of sub-bituminous coal and lignite by 30-55%. An added benefit of this technology is that 65-70% of mercury is removed, with as high as 90% reduction in some cases, and up to 30% of SO_2 and NO_x removed from the initial feedstock. During the process of removing the water, mercury is volatized and released in the gas and water off-streams. The mercury and other pollutants are captured with carbon filters and disposed of at permitted disposal sites. The result is a pollutant scrubbed high energy coal. The number and geographic scope of patents around the world that are applicable to this technology have increased dramatically in anticipation to requirements for a wide range of coal types.

As demonstrated in Table 3, the resultant coal product improves the quality of low-grade western and lignite coals, increasing efficiency of steam generating units, and offering another approach for facilities to comply with air emissions standards. Since western sub-bituminous coal is typically already low in mercury and sulfur before refining, facilities can substitute the scrubbed coal for bituminous coal feedstocks for significant emission reduction benefits.

Table 3. Product ComparisonBetween Sub-bituminous and Lignite, K-Fuel, and Eastern Coal

	Sub-bituminous and Lignite Coal[1]	K-Fuel™ Product	Eastern Compliance Bituminous Coal[2]
Heating Value (Btu/lb)	6,850-8,804	10,637-11,683	13,210
Moisture Content (%)	26.29-45	5.74-8.0	7
Mercury (ppm)	0.0289-0.342	0.008-0.163	0.15

Source: K-Fuel test data

[1] Includes four sub-bituminous and one lignite coal feedstock used in K-Fuel tests
[2] Average of Eastern Compliance Coal, USGS

The pre-combustion process employs both mechanical and thermal means to increase the quality of sub-bituminous coal and lignite by removing moisture, sulfur, nitrogen, mercury, and other heavy metals. Because these constituents are removed before burning the coal at the plant, this form of control can virtually replace the need for post-combustion controls.

The K-Fuel process diagram is given in Figure 6 below. To start the process, raw coal is delivered directly from a mine to the coal processing facility. The coal enters the first stage separator, developed using conventional coal cleaning technology, where it is crushed and screened to remove the large rock and rock material. The processed coal is then transferred to an intermediate storage facility prior to being sent via a distribution system to the specialized thermal process. This process essentially operates like a giant pressure cooker, utilizing Lurgi Mark IV vessels under high pressure and temperature to place thermal stress on the coal. The coal passes through pressure locks into the processors, and then steam is injected into the processors at 460° F and 485 psi. The coal is maintained at these conditions, and the mineral inclusions are fractured under the thermal stress, removing both the included rock (containing some mercury) and sulfur-forming pyrites. The inherent moisture of the coal also released.

After being treated for a sufficient time in the main processor, the coal is discharged into a second pressurized lock, which is sealed off from the primary reactor. After sealing, the processor pressure is vented into a water condenser to return the processor to atmospheric pressure, and to flash cool the coal to approximately 200°F. The coal is then discharged onto a belt and further cooled by convection and indirect cooling. Following cooling, the coal is sent to a second stage separator for additional screening to remove sulfur- and mercury-containing material liberated by the thermal process. Water liberated from the coal is removed at various points in the process. This water, along with some condensed process steam, is either sent directly to treatment or is reused within the process. The water treatment system removes coal fines and hydrocarbon compounds liberated from the coal in the processor, and uses carbon filtering to remove mercury and other heavy metals that were released from the coal and rock. The waste products (carbon, mercury, and heavy metals) from the filtering process are sent to a qualified, permitted disposal site for final disposition.

Third party and company tests have demonstrated the clean-burning qualities of the patented pre-combustion product. Results reported in 2002 indicated the ability to achieve 70% mercury removal when using Wyoming Wyodak sub-bituminous coal, and up to 65% mercury removal when using Louisiana Dolet Hills lignite coal.

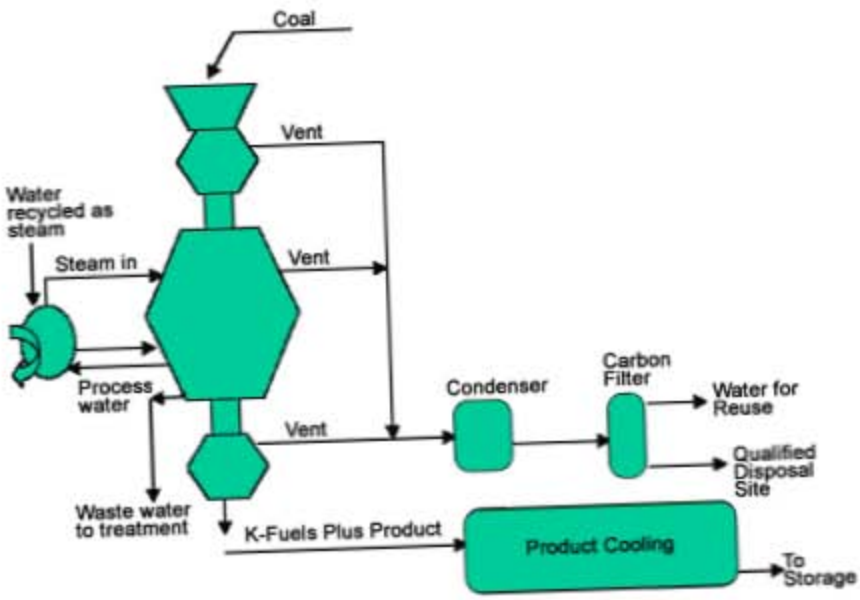


Figure 6. K-Fuel Thermal Processing Plant

The first commercial 750,000 ton per year K-Fuel plant will be in operation by the end of the first quarter of 2005 near Gillette, Wyoming. In early May 2004, the company announced an agreement-in-principle to locate the first commercial K-Fuel plant at the Buckskin Mine, north of Gillette. The Buckskin mine has the potential to be expanded to 8 million tons per year of capacity. In addition, the company announced a signed agreement to purchase the Fort Union Mine site and related facilities.

The commercial plant currently under development is fully funded and approximately two-thirds of the production from the initial 750,000 tons per year plant has been committed, with the balance to be used for test burns to facilitate future contract commitments, for plant expansions, and future coal processing facilities. There are plans to own and operate coal processing production facilities, as well as license the technology to third parties. These coal processing facilities plants are built using proven, off-the-shelf, modular equipment designed by Lurgi SA, that allows for adding capacity to each plant as demand grows.

Enhancing Control Through Existing Control Technologies

Mercury may be removed from the flue gas of coal-fired boilers to a greater or lesser extent by devices such as electrostatic precipitators, baghouses, SCRs, and SO_2 scrubbers used to control other emissions. The efficiency of such co-beneficial collection depends on the specific equipment and operating parameters, as well as

on the chemical form of mercury in the flue gas, which in turn is influenced by fuel composition and combustion parameters.

Sorbent addition in wet scrubbers has been shown to be highly effective in capturing the oxidized portion of mercury in the vapor phase in boiler flue gas. This is because vaporous mercury that is in an oxidized rather than elemental form is soluble. Scrubbers have been shown to be highly effective in capturing the oxidized mercury in the vapor phase in boiler flue gas, on the order of 60-80% depending on site specifics and fuel factors. Addition of sulfides is being used to retain the mercury in solution and prevent a small fraction of the captured mercury that can potentially be re-emitted as elemental mercury.

Wet FGD systems are currently installed on about 25 percent of the coal-fired utility generating capacity in the U.S., representing about 15 percent of the total number of coal-fired units. Depending on the effects of the operating parameters, FGD systems can provide a cost-effective, near-term mercury emissions control option with a proven history of commercial operation. For boilers already equipped with FGD systems, the incremental cost of any vapor-phase mercury removal achieved is minimal. To be widely accepted and implemented, technical approaches that improve mercury removal performance for wet FGD systems should also have low incremental costs and have little or no impact on operation and SO_2 removal performance.

Mercury Oxidation in Selective Catalytic Reduction System: The speciation of mercury is known to have a significant impact on the ability of air pollution control devices to capture mercury. The oxidized form of mercury is highly water soluble, therefore, it is easier to capture in wet FGD systems than the elemental form of mercury which is not water-soluble. The oxidized form of mercury is also more easily adsorbed than elemental mercury on unburned carbon in the flyash and on injected sorbents. SCR catalysts act to oxidize a significant portion of the elemental mercury, making it easier to remove it in downstream wet FGD systems or PM collection sevices.

SCRs are already used for reducing NO_x emissions on close to 100 GW of the approximately 310 GW of coal-fired capacity in the U.S. Additional SCR installations are projected to occur due to existing ozone-related rules including: NO_x SIP call, State Regulations and the proposed CAIR/IAQR. SCR catalyst is known to oxidize elemental mercury to oxidized mercury forms such as $HgCl_2$ that are more easily captured and removed by downstream air pollution control equipment (Lee et al., 2003). Mercury oxidation is enhanced by lower temperature, higher coal chlorine content, and increased residence time. Due to the low gas-phase chlorine in flue gases from low-rank coals (e.g. sub-bituminous coals such as PRB), the mercury oxidation level over SCR catalyst has been found not to be as high as it is for flue gases in bituminous-fired units.

A number of simple approaches can achieve more mercury emissions control in a shorter timeframe. Under the NO_x SIP call, a cap-and-trade program to reduce NO_x emissions from power plants in 19 northeastern states and the District of Columbia is being implemented starting in May 2004. The emission limits governing affected sources under the NO_x SIP call only apply during a five-month ozone season from May through September. Year-round operation of the SCRs at those units could achieve greater mercury co-benefits as soon as implemented. Additionally, if SCRs are run October to April without ammonia injection, the absence of ammonia would result in higher mercury oxidation levels and thus downstream capture for all coal types where downstream FGD or PM capture devices will be in place. It is expected that many SCR installations will go to year-round operation under the existing and proposed rules, but early implementation of this approach can provide quicker co-benefits without the addition of new equipment.

If SCRs are run October to April with ammonia injection, the co-benefit of mercury oxidation will likely not be as high as mercury oxidation is inhibited by ammonia. However, it would still contribute significant mercury emissions control co-benefits due to full-year operation versus ozone season where effective downstream capture equipment is in place such as wet FGD.

Another approach would be to add an extra layer of catalyst to the existing SCR installations. The extra layer would be effective for both NO_x and mercury oxidation. Mercury oxidation would be enhanced due to the lower ammonia concentration in the last layer. The additional benefits include both additional NO_x reduction, that would generate valuable NO_x credits to defray the cost of the catalyst or even generate a net benefit, as well as higher co-benefits for mercury emissions control. Most or maybe all SCR installations in the U.S. already have provision for one or more extra layers built into the ductwork so that no additional construction would be needed to implement this approach and would have the highest co-benefit mercury capture where wet FGD or other effective capture equipment is in place downstream of the SCR.

New technologies are being developed for mercury oxidation across an SCR that inject chloride prior to the catalyst. The enhanced mercury oxidation is due to improved thermodynamics at regular SCR operating temperatures due to higher flue gas chloride concentrations which otherwise limits the extent of Hg oxidation possible. At low chloride levels, thermodynamics limit the extent of Hg oxidation that is possible. The higher chloride concentration makes the reaction possible while the catalyst speeds it up. This new patented technology will be especially useful for low-rank coals and will be ready for implementation in a short time horizon. This technology should greatly enhance the mercury co-benefits for sub-bituminous and lignite installations that have or will have SCR installations for NO_x control and where effective downstream capture equipment is or will be in place. It is expected that chloride concentrations downstream of the SCR would be

no higher than typical chloride flue gas levels seen in average bituminous-fired units, and that chloride injection would not be necessary for flue gases already having high chloride levels.

CAPITAL AND OPERATING COSTS

Levelized capital and operating costs are generally low for mercury control approaches compared to new full-scale installation costs for control of criteria pollutants such as NO_x and SO_2. This is true for mercury control through enhancements to existing equipment such as SCR and wet FGD, as well as for installation of new equipment for mercury specific control technologies such as sorbent injection. In addition, the costs for mercury specific controls will likely decrease over time as more is learned about current approaches and new ideas are tested as has been seen in prior experience curves for pollution control equipment.

The costs for activated carbon injection technology consists of capital equipment and operating costs. The capital costs to retrofit an existing facility will depend on site-specific issues. Generally, the uninstalled cost for the sorbent injection equipment for power plants generating 100 to 500 MW is in the range of $600,000 to $1,000,000. The primary operating cost and the largest cost element for the technology is the cost of the throwaway sorbent. Figure 6 shows a plot of the sorbent costs in mils/kWh for both bituminous and subbituminous coals. For a unit with an ESP, the cost of the sorbent would be approximately 1.2 – 1.5 mils/kWh to achieve 60 to 70% mercury removal for both types of coals. If a unit has a fabric filter, it is expected that up to 90% mercury removal can be achieved at a sorbent cost of between 0.3 - 0.4 mils/kWh.

One option that a plant might consider is to trade off capital costs for operating costs by installing a fabric filter to reduce sorbent requirements. The cost of a pulse jet fabric filter designed for the collection of the injected activated carbon would be $40 - $50/kW and would result in a factor of three reduction in sorbent costs while achieving up to 90% mercury removal. This is an example of the importance of a regulation that gives the utility flexibility in how to achieve mercury reduction at each site.

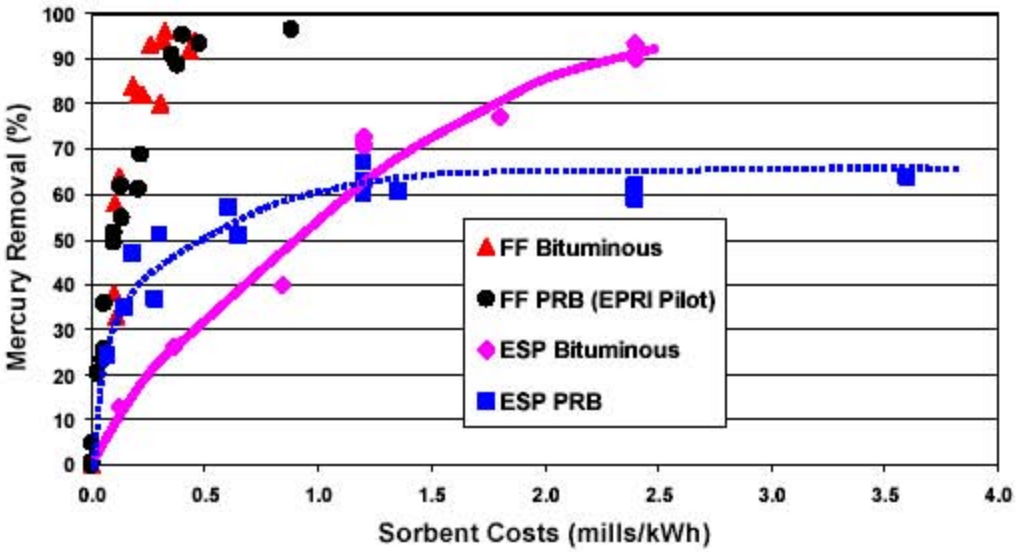


Figure 6. Costs of Sorbents for Mercury Control at Coal-Fired Power Plants.

As the ECO process is a multipollutant technology, the costs incorporate control of SO_2, NO_x, PM and mercury and are considerably higher. For the development of a compliance strategy for a particular unit, one must weigh the cost of control installations for individual pollutants such as scrubbers for SO_2, SCR or SNCR for NO_x, fabric filters for PM, etc. The capital cost associated with an ECO installation is estimated to be $200/kW, including balance of plant modifications. The operating costs are estimated to be 2.5 mils/kWh.

For the precombustion control option, the marketplace would ultimately establish the per ton price for clean, pre-combusted western sub-bituminous and lignite coal. K-Fuel would compete with eastern coal of a similar Btu heat value.

An analysis of the cost for the SCR mercury optimization options outlined above, as well as the new chloride injection technology is in progress and will be submitted to EPA shortly. The analysis looks only at the amount of electric generating capacity that is projected to be equipped with both SCR and wet FGD in the year 2010

SCR catalyst is already used for reducing NO_x emissions on about 100 GW of the approximately 300 GW of coal-fired capacity in the U.S. Currently, there is approximately 25 GW of coal-fired electric generating capacity in the U.S. equipped with both SCR and wet FGD. The amount of capacity equipped with both SCR and wet FGD is projected to rise to about 40 GW by 2005 and to about 93 GW by 2010 as companies install new control equipment to comply with NO_x requirements related to the NO_x SIP call and SO_2 and NO_x requirements related to the proposed Clean Air Interstate Rule. About 94 percent of the projected capacity equipped with both wet

FGD and SCR is expected to burn bituminous coal, with the remaining six percent burning sub-bituminous.

The in-progress analysis examines the mercury removal performance, associated costs of several optimization options, combinations of options for enhanced Hg oxidation, and capture for units that would be equipped with both SCR and wet FGD in the year 2010 with and without chloride injection technology.[1] *The analysis of the options for mercury control through SCR enhancements will be submitted to EPA shortly.*

Option 1—Operating SCRs Year-Round
Option 2—Installing an Additional Layer of SCR Catalyst
Option 3—Combination of Options 1 and 2, With Ammonia Injection
Option 4—Combination of Options 1 and 2, With No Ammonia Injection During the Non-Ozone Season (October 1-April 30)
Option 5--Chloride Injection

CONTROL TECHNOLOGY GUARANTEES

Mercury reduction regulations are currently being implemented in certain states such as Massachusetts, New Jersey, Wisconsin, North Carolina, and Connecticut. In addition, new power plants being planned are required to meet Maximum Achievable Control Technology (MACT) requirements for mercury. Carbon injection is being specified as the MACT technology for most of these new coal-fired power plants. Because of these state regulations and the new power projects, there is a significant amount of commercial activity for mercury control technologies. Bids are being requested from power companies for equipment to meet these emissions regulations and many require guarantees.

Guarantees of mercury control are being offered commercially in the marketplace today for many plant configurations and coal types. For mercury control technologies consisting of carbon injection systems followed by a particulate collector, such as a fabric filter or ESP, performance guarantees have been provided to firm customer specifications on commercial projects in various stages of development. These guarantees have included performance guarantees for mercury emissions and powdered activated carbon consumption that are contingent on coal type, coal mercury content, existing flue gas cleaning equipment, and plant operational data. The frequency of the compliance requirements; such as whether the guarantee would be based on hourly, daily or monthly average; would also influence the level of the guarantee. Generally, the process for developing guarantees for mercury control is the process that is common to development of guarantees for control of other air pollutants.

Vendors will guarantee a given emission rate for a specified range of coals having specified mercury content. They would generally not provide a collection efficiency guarantee since that would be progressively more difficult to meet the less mercury there is in the coal. For example, a coal with a mean mercury content of 0.15 ppm would typically be provided a guaranteed emission value between 1.5 – 7.0 micrograms Hg /Nm3 (corresponding to between 90 to 50 % removal efficiency respectively), all depending on the factors listed above. This issue could be simplified with a regulation that had a dual limit of a lower emission rate and a maximum efficiency. Given a longer compliance period, such as a yearly average, it is expected that guarantees for higher removal efficiencies would be provided for the vast majority of plants compared to shorter averaging periods.

Guarantees can be provided for the oxidation performance of mercury across an SCR. Mercury oxidation activity is adequately sustained with SCR catalyst over its lifetime for NO_x reductions. Based upon studies in the literature and data from vendor studies, the activity decline rate is similar to the De- NO_x decline rate. Coupled with predictive models for mercury oxidation that are being validated with full-scale data, performance guarantees can be provided.

At this point in the commercialization of mercury control equipment, the guarantees will likely be more conservative than in the coming few years. As with other air pollution control technologies such as wet scrubbers for SO_2 control and selective catalytic reduction for NO_x control, the more mercury control equipment that is installed and more experience gained, the tighter and more aggressive guarantees will become. In fact, some customers have not requested performance guarantees for criteria pollutant control technologies as the technologies are well established and able to easily meet the state and federal requirements.

In general, the guarantees provided for mercury controls are provided in the same manner that guarantees are provided for SO_2, NO_x and PM control technologies. The industry source will request a bid for a particular situation based on their regulatory requirements. The vendor will then submit a proposal based on the specific site characteristics defining the emission rate that is achievable and can be guaranteed for that specific application. After the control installation has been completed, testing will be performed to verify that the emissions guarantee has been met. The vendor may provide a 'make right' statement in the guarantee that the vendor will have a window of time, typically up to twelve months, to make adjustments to the technology if the guaranteed level was not initially satisfied. This is the general approach that is taken by vendors when guaranteeing a control technology installation whether it is for a SO_2, NO_x, PM or mercury control.

COMMERCIAL AVAILABILITY OF CONTROL TECHNOLOGIES

A number of air pollution control technologies for mercury control from power plants are commercially available or will be available by the end of the 2004. Mercury specific control technologies such as activated carbon injection systems are currently available and have already been used on full-scale systems for the power sector as well as for other industrial sectors. These systems can be applied to any plant configuration and coal type.

A number of mercury control approaches can be applied through the utilization of existing air pollution control equipment. These applications have the potential to provide immediate mercury control benefits and require little if any capital investment. For plants that already have an SCR installation, a promising technology that adds a reagent across an SCR catalyst is expected to be available in 2005. A U.S. patent was granted in October of 2003 that uses a chlorinating agent and ammonia to accomplish the simultaneous reduction of NO_x and oxidation of mercury over SCR catalyst. This technology has been successfully demonstrated in pilot work and will be tested on a much larger scale.

In addition, adding an additional layer of catalyst to existing SCR installations, and/or running existing SCR installations year-round, are viable options that increase the oxidized form of mercury making it easier to capture in existing downstream control equipment. This technology is immediately available to almost one-third of the coal-fired electric power sector due to the almost 100 GW of SCR installations for the NO_x SIP call, and is likely to pay for itself in the best plant configurations depending on the value of additional NO_x credited generated.

Other technologies such as the multipollutant ECO process are in the commercial demonstration stage. Pilot studies on smaller slip streams have been performed for 24 months and a 50 MW demonstration is in the beginning stages of operation. Based on successful commercial demonstration, the ECO technology is expected to be commercially available in late 2004.

WASTE STREAM DISPOSAL

Since the purpose of controlling emissions from coal-fired boilers is to reduce potential buildup of mercury compounds in lakes and streams and ultimately to protect public health, the stability of the captured mercury in the ash and other coal combustion byproducts (CCBs) is a critical component of the overall control scheme. The ICR program showed that currently approximately 30 tons per year of mercury is contained in CCBs. Pending mercury control regulations could result in an additional 20 to 40 tons per year of mercury in CCBs.

In the U.S., approximately 67% of all fly ash produced from utility coal combustion is disposed of in landfills or surface impoundments. The remaining 33% is used for a variety of commercial applications. There are approximately 600 waste disposal sites for CCBs in the U.S.; half are landfills and half are surface

impoundments. Note here that CCBs include other waste streams such as bottom ash and scrubber sludge. A 1999 EPA study estimated that about half of the CCB landfills and a little less than a third of the surface impoundments have some type of liner, the most common type being compacted clay (Senior et al., 2002).

Volatilization of mercury from landfills was estimated by EPA to be small. To date, there has been no evidence based on laboratory leaching studies for leaching of large amounts of mercury from fly ash under landfill conditions. Leaching appears to be the most likely pathway for liberation of mercury from fly ash. Volatilization may be important for certain applications of fly ash as filler in concrete applications. Volatilization is, of course, the primary pathway for mercury release to the atmosphere if fly ash is used as a raw material in cement kilns. However, volatilization will be complete in this case.

PAC-injection applied to coal-fired boilers will result in the fly ash being mixed with a certain amount of mercury-containing sorbent. This material will be sent to land disposal or used in specific applications (assuming that the presence of the sorbent is compatible with the application). Since the mercury on the spent sorbent may be present in a different form than on fly ash, it is necessary to consider what might be the most likely routes for release of mercury in sorbent-fly ash mixtures and how sorbent-containing coal utilization byproducts (CUBs) should be tested.

Senior et al., (2002) evaluated samples of ash with activated carbon from two ADA-ES field demonstration programs. The Gaston sample (the product of a bituminous coal) had a high LOI and mercury content, in spite of the low sorbent injection rate, because most of the ash was removed upstream of the COHPAC baghouse by a hot-side ESP. Thus the sample had a relatively high proportion of sorbent. The Pleasant Prairie sample (the product of a subbituminous coal) had a low LOI and mercury content. Sorbent was injected upstream of an ESP and was combined with the full ash stream. The LOI and mercury content were much lower than the Gaston sample. Little or no mercury was detected with leaching tests including TCLP, SGLP (including 30- and 60-day leaching), and sulfuric acid leach (bituminous ash). Samples were also analyzed by CONSOL as part of a DOE program. They also found no leaching of mercury from activated carbon (Withum et al., 2002).

Although the ash with activated carbon appears to be highly stable, initial testing with a PRB ash determined that the presence of even trace amounts of activated carbon in the ash rendered the material unacceptable for use in concrete. Even though the Pleasant Prairie (PRB) ash conformed to the ASTM C-618 standard for Class C fly ash, it did not pass the Foam Index test that is also required for sale of this ash for use in concrete formulation. These are field tests used to determine the amount of Air Entrainment Additives needed to meet freeze-thaw requirements. This means that with activated carbon injection, the plant

would not only lose revenues from ash sales, it would incur additional expenses to landfill the material.

For the multipollutant ECO technology, it is important to note that the mercury is removed from the co-product stream and is isolated for disposal. The stream is pumped through an activated carbon adsorption bed, which strongly adsorbs mercury compounds to the bed. The mercury is disposed of as a hazardous waste, and the spent activated carbon is replaced in the ECO process. It is estimated that the variable cost of mercury removal with activated carbon in the ECO process is $800 per pound of mercury, including the sorbent media and its disposal.

During the K-Fuel process, mercury is volatized and released from the coal in both the gaseous and liquid phases. The mercury and other heavy metals and pollutants are captured with carbon filters and disposed of at permitted disposal sites. The pre-combustion process captures these pollutants in a highly concentrated form compared to post-combustion technologies, thereby creating less waste. The process can achieve significant savings in waste disposal compared to post-combustion technology as tests have shown 5-10 times less solid waste disposal for pre-combustion technology versus post-combustion technology. The quality of the ash produced by the power plant is maintained as a usable, salable product.

RESOURCES FOR ENHANCEMENT OF EXISTING EQUIPMENT AND INSTALLATION OF NEW EQUIPMENT

SCR Catalyst: The SCR catalyst industry for coal-fired systems is a recent example of how quickly companies are able to build new production capacity in response to a significant jump in demand over a short period of time. The air pollution control industry installed close to 100 GW of SCRs over the last several years, with approximately 40 GW being installed in one year, in response to regulations requiring reductions in summer time NO_x emissions. This caused the catalyst manufacturers to more than double their SCR catalyst production to meet the market demand. SCR catalyst for enhanced SCR mercury oxidation is readily available due to overcapacity in this manufacturing sector. We estimate that extra SCR catalyst for SCR enhanced mercury oxidation approaches (extra layer and/or year-round operation) can be manufactured in one year. This would include all existing and projected SCR systems in 2010 under EPA's scenarios for existing rules plus the CAIR/IAQR. Installation manpower is not a limiting factor for catalyst addition.

Activated Carbon Injection: In general, the resources needed for the construction and operation of activated carbon injection technologies is significantly smaller than those required for the installation of SO_2 scrubbers and selective catalytic reduction units. The areas investigated for mercury control options includes the availability of sorbent (specifically activated carbon) and the

availability of skilled labor assuming fabric filter installations along with carbon injection systems.

Activated carbon injection systems consists of a bulk-storage silo and twin blower/feeder systems to convey the activated carbon from the silo to the flue gas duct. The feeder system is typically designed to deliver 750 lb/hr of activated carbon. PAC is delivered in bulk pneumatic trucks and loaded into the silo. The silo is equipped with a bin vent bag filter to prevent activated carbon from escaping during the transfer process. From the two discharge legs of the silo, the reagent is metered by variable speed screw feeders into eductors that provide the motive force to carry the reagent to the injection point. Regenerative blowers provide the conveying air to suspend the activated carbon so that it can be transferred to the flue gas duct. A programmable logic controller (PLC) system is used to control the system operation and adjust injection rates. Figure 7 is a photograph of the sorbent silo and feeder train designed and installed to treat a 150 MW boiler. Hard piping carries the reagent from the feeders to distribution manifolds located on the ESP inlet duct, feeding the injection probes.

An activated carbon injection system can be installed in less than 6 months including the design, installation, and equipment testing which is significantly less than EPA estimated in their Engineering and Economic Factors Analysis (U.S. EPA, 2002). These are the installation times that have been typical for the current set of DOE demonstration projects. The injection systems; including the silo, feeders, controllers, etc., are commonly used in numerous industries, therefore, the production capacity far exceeds the incremental demand from any mercury rule for power plants. One silo company that was surveyed by ICAC indicated that they alone could produce 1500 silos in a single year. By comparison, EPA estimates that only 2 GW of ACI systems, approximately 4 coal-fired units, would be installed by 2010 assuming the simultaneous implementation of the Clean Air Interstate Rule (CAIR). Assuming that the CAIR rule were not implemented, EPA estimated that 63 GW of ACI systems, approximately 126 coal-fired units, would need to be installed. In either case, the supply of hardware for activated carbon injection systems can easily be accommodated by existing production capacity.

The most commonly used sorbent for mercury control has been activated carbon. For the past two decades, powdered activated carbon injection upstream of a baghouse has been successfully used for removing mercury from flue gases from municipal and hazardous waste combustors. Activated carbon is carbon that has been "treated" to produce certain properties such as surface area, pore volume, and pore size. Activated carbon can be manufactured from a variety of sources (e.g., lignite, peat, coal, wood). More commonly, steam is used for activation, which requires carbonization at high temperatures in an oxygen-lean environment. As some carbon atoms are vaporized, the desired highly porous activated carbon is produced. Commercially, activated carbons are available in a range of particle sizes, as well as other characteristics that are needed for a specific application.

Figure 7. Carbon Injection Storage Silo and Feeder Trains for 150 MW.

A survey of the major suppliers of activated carbon in the U.S. and abroad demonstrates the amount of material that could be made available for this market with current production capacity (Durham, 2003). The results are presented in Table 4. The excess production capacity in the U.S. is approximately 62,000 tons per year. There is an additional 190,000 tons of AC that is available from China and Germany for this market. Chinese activated carbons are currently flooding the U.S. market and competing for the water treatment business.

The total excess capacity of activated carbon from foreign and domestic sources is approximately 250,000 tons per year. Table 5 presents an estimation of how many plants could be treated by this material in response to a mercury control regulation. The market share depends upon whether the activated carbon is used in conjunction with ESPs or fabric filters. The full-scale data indicate that mercury removal in the 50 to 70% range can be achieved with an ESP at a feed rate of 10 lb/MMacf, whereas 70 to 90% removal could be achieved with a fabric filter at one-third the feed rate. Therefore, if the 250,000 tons per year were applied to ESPs, then 120 of the 1,100 plants could be treated. However, if new fabric filters were installed on many plants, it would be possible to treat 400 units with this same amount of material without increasing the current production capacity.

Table 4. Estimates of Excess Production Capacity of Activated Carbon that could be Available for the U.S. Utility Market.

AC Source	Current excess capacity of AC production in Tons/year
NORIT Americas	22,500
Other U.S. Suppliers	40,000
Total U.S. Excess Capacity	**62,500**
Rheinbraun (Germany)	130,000
CarboChem (China)	60,000
Total Import Excess Capacity	**190,000**
Total U.S. and Import Excess Capacity	**252,500**

Table 5. Estimate of the Number of 250 MW Power Plants that could be Treated with Activated Carbon from Current Excess Capacity.

	Excess Capacity Tons/yr	ESPs (50-70%)	FFs (70-90%)
U.S. AC	62,000	30	99
Total U.S. plus Imports	252,000	120	400

A new mercury regulation would create a significant new market for activated carbon. All of the activated carbon manufacturers that were surveyed expressed a strong interest in investing significant capital in building new production facilities to provide sorbents for the utility market. In order to build new production capacity, between a two- to four-year period would be needed to expand production. However, all of the activated carbon suppliers said that they would be hesitant to invest capital resources to increase capacity based only on the promise of a new regulation. A decade or so ago, the AC industry increased capacity when EPA announced that they were going to tighten up drinking water standards. After the new capacity was added, EPA did not follow up with new regulations, which produced a glut of activated carbon. Some companies went out of business because of this, and the industry as a whole is just now recovering. As a result, it is unlikely that new AC production will move beyond the planning stages until there is the certainty of a regulation.

EPA's Economic and Energy Impact Analysis for the Proposed Utility MACT Rulemaking did not specify the number of fabric filters that would be installed along with the ACI systems to control mercury. The fabric filters, such as those used in the COHPAC and TOXECON systems would be added downstream of

the existing particulate control device and would be installed following the sorbent injection system to collect the waste sorbent. The fabric filters that would be installed for these types of control applications would typically be smaller than the fabric filter that would be built as a dedicated particulate control device. EPA estimates that only 2 GW of ACI would be installed by 2010 assuming the simultaneous implementation of the CAIR rule. This would mean that approximately four units would need a fabric filter for mercury control. If for some reason the CAIR rule were not simultaneously implemented, EPA estimates that approximately 63 GW of ACI controls would be needed. Conservatively assuming that all 63 GW would need the smaller sized fabric filter would not provide a difficult installation challenge especially under the assumption that an additional 63 GW of SO2 (49 GW of scrubbers) and NOx (24 GW of SCR) controls would not be installed if CAIR were not implemented during the same timeframe.

ECO Technology: The *ECO technology* is expected to be installed and commissioned within 24 to 30 months after order placement. The components of this system, including the WESP, are commonly used for air pollution control. A picture of the 50 MW ECO commercial demonstration is provided in Figure 8. The predominant reagent used in ECO is ammonia. The ammonia can be supplied to the system in any form—anhydrous, aqueous of any concentration, or even from systems that generate ammonia from urea. All the ammonia that goes into the ECO system becomes part of the ammonia sulfate co-product (Boyle, 2003). Ammonia sulfate is a valued fertilizer both for its sulfur content and for its nitrogen content. The largest use of ammonia in the US is as a nitrogen fertilizer. Some of the ammonia is applied to fields directly, but most of it is converted to a more convenient form of nitrogen, either a liquid such as urea ammonium nitrate or a solid, such as granulated urea. The processing of ammonia into other forms of nitrogen is becoming more common as the difficulties of handling pure ammonia in an agricultural environment increase.

Figure 8. 50-MW ECO Commercial Demonstration Unit at FirstEnergy's R.E. Burger Plant

CONCLUSIONS

ICAC would recommend that a reduction of 50-70% of current emission levels in the 2008 to 2010 timeframe be targeted and that it is achievable via enhancements in existing SCR and wet FGD pollution control technologies as well as installation of mercury specific sorbent injection systems. ICAC would also recommend including flexible mechanisms in the regulation that encourage innovation while providing clear goals and meaningful reductions. This type of approach would provide cost effective solutions and minimize impacts on generation mix.

Flexible mechanisms are important for mercury control technology for a variety of reasons. Some examples of these types of concepts include but are not limited to the following: early reduction incentives, market based approaches, capital recovery programs, plant wide averaging, safety valves or other approaches. The cost of the control technology is related to the size of the plant treated so that two plants of identical size but with a factor of ten differences in emissions would have almost the same capital and operating costs. Therefore, the cost per pound of mercury removed would be ten times higher for the low-emission plant. Flexible mechanisms would provide a means to level the playing field and actually create incentives for the power companies to treat the higher emitting plants, thus, making the largest amount of reductions in total mercury emissions while minimizing costs.

These incentives would also alleviate some of the issues related to differences and uncertainties in performances due to plant-by-plant variations in coal characteristics and equipment design. This will significantly reduce risk for both the air pollution control technology vendor and the power company purchasing mercury control technology. Certain incentives would mitigate risk and reward early compliance for plants that install equipment. This would increase the experience base which will decrease uncertainty and make it possible to provide more aggressive performance guarantees.

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INSTITUTE OF CLEAN AIR COMPANIES

1660 L Street NW
Suite 1100
Washington, DC 20036-5603
Telephone 202.457.0911
Fax 202.331.1388

David C. Foerter, *Executive Director*
Email: dfoerter@icac.com

Chad S. Whiteman, *Deputy Director*
Email: cwhiteman@icac.com

January 3, 2005

Honorable Michael Leavitt, Administrator
U.S. Environmental Protection Agency
EPA Docket Center (Air Docket)
Mail Code: 6102T, Room B-108
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Attention Docket ID No. OAR-2002-0056

Dear Administrator Leavitt:

The Institute of Clean Air Companies (ICAC) is the national trade association of companies that supply air pollution control and monitoring technology. Our members include nearly eighty leading suppliers of air pollution control and monitoring technologies for stationary sources. These companies operate and provide environmental solutions for affected industries as well as employment opportunities across the U.S.

The Institute applauds EPA's efforts to request further information on a much-needed rule that provides for the reduction of mercury emissions from coal- and oil-fired electric generating facilities in order to protect public health. The Institute has a few observations concerning the notice of data availability (NODA) for the Utility Mercury Reduction Rule specifically concerning the performance of control technologies, technology guarantees, commercial availability, control costs, by-product disposal, and availability of construction resources. The Institute continues to work with stakeholders, including the owners and operators of major generating facilities, to develop a better understanding of the control and measurement technologies that can achieve and exceed the requirements of a mercury control rule. These discussions have in part led to the strengthening of our industry's position on the need to instill flexibility in compliance requirements, to promote industrial progress without disadvantaging coal as an energy source, continuing reliable electric generation, and to encourage additional research and development of additional cost-effective control technology options.

The Institute advocates EPA's investigation and use of flexible approaches to promote innovation and early compliance with requirements. Within the MACT framework EPA has used flexible approaches that should be considered again in the utility mercury MACT program. For example, in the large municipal waste combustor MACT, EPA relied on Section 111(d) and 129 to allow State Plans backed up by Federal Plans to achieve compliance. If states did not have an approved State Plan, the Federal Plan would apply with a generic compliance schedule and "increments of progress" toward the retrofits of air pollution control by the compliance deadline. Many states have approved plans, with some inherent flexibility, and did not require the Federal backstop. There are other examples in other MACT programs where beyond the floor technologies have been used as the basis for establishing limits, but EPA has provided a backstop should the technology not perform as expected.

The rapid development of mercury control technologies over the last several years, primarily as a result in public and private investments in research and development, has produced a number of technologies that are available for the implementation of a national mercury control regulation for coal- and oil-fired power plants. A large number of laboratory tests and full-scale demonstrations have been conducted that provide information on the effectiveness of controls for various coal types and control configurations. As demonstrated in the past for a number of other pollutants, market response to a regulatory requirement will provide the single greatest push for the advancement and commercialization of control and measurement technologies. However, despite the lack of any current national control requirement for mercury, a number of options are already commercially available while others are still in the development and testing phases.

Past experience with technology development for other pollutants (SO_2, NO_x, and PM) as well as other source categories such as mobile sources, suggests that delaying the regulation of mercury emissions from power plants would serve to delay further development of innovative control technologies. Research and development efforts are unlikely to be sustained at a vigorous level in the absence of regulatory or other drivers capable of creating a viable market for advanced control technologies. Larger markets provide more incentives for the development of technologies as well as foster competition between vendors that produces more innovative and cost effective solutions for affected sources. Smaller markets such as those that may be developed with the implementation of State regulations (e.g. Massachusetts, Connecticut, Wisconsin, New Jersey, North Carolina) are beneficial to the air pollution control industry but will be less effective in developing healthy markets than a timely implemented national program.

With the implementation of a national program, multiple control options including precombustion, combustion and post combustion technologies will contribute to meeting the required emission reductions. Coal cleaning as well as coal switching are examples of options that have the potential to reduce mercury

emissions prior to fuel combustion that are not discussed further in these comments.

Based on the recent test results, significant amounts of mercury can be removed through the use of existing controls. Existing control installations such as fabric filters, electrostatic precipitators, SO_2 scrubbers, and selective catalytic reduction (SCR) are currently achieving high levels of mercury reductions even though these processes were not originally designed nor optimized for mercury capture. With the implementation of mercury regulatory requirements beyond incidental co-benefit levels of control, a number of options for optimization of existing controls will be implemented to provide cost effective reductions in a short period of time.

Mercury specific control technologies such as sorbent injection systems have been demonstrated at full-scale. Multipollutant control approaches as well as other mercury specific technologies have also demonstrated significant progress and will provide additional low cost, innovative approaches to mercury control.

It is important to note that EPA's modeling assumes no advancement in the development of mercury control technology and no reduction in the cost of mercury control technology over time. These assumptions are contradictory to both historical trends with control technology for other pollutants and the current rapid progress in mercury control technology development. As noted, the progress in advancing mercury control technologies has been rapid without increasing opportunities to lower the cost of control. Regulatory drivers are a powerful market tool that drives competition in our industry. Often lower cost solutions emerge after regulatory requirements have been established, rather than before.

There have been a number of arguments made that state that mercury control technologies are not available. Many of these perspectives invoke all too familiar arguments that have been offered in the past to dissuade EPA from promulgating an effective rule. For example, during promulgation of the NO_x Transport SIP Call in 1998 a number of commenters claimed that the prominent control technologies, SCR and SNCR, had not been fully demonstrated on large units (250 MW and larger) or domestically; was an immature technology; would not be attainable on a sustained basis; had not been adequately demonstrated on all U.S. coals; were incapable of meeting guaranteed performance; and were not able to be constructed in time for compliance due to inadequate resources to accomplish what now has already been done with considerable success. As part of these comments on NO_x control for major industrial and electric generating facilities, EPA was urged not to rely on "emerging control technologies" that provide no assurance of being able to achieve mandated emission reduction levels. However, EPA's promulgation of the NO_x SIP Call was steeped in success and advancement of these and other NO_x control technologies. Today, mercury control technology advances and commercial availability have surpassed the position we once held on NO_x control before the NO_x SIP Call. However, the addition of compliance flexibility

should reduce any perceived or real uncertainties and would provide opportunities to use additional control options.

1. POST COMBUSTION CONTROL TECHNOLOGIES

i. Sorbent Injection Technology

a. Technology Description

Injecting a sorbent such as powdered activated carbon, bromine, polysulfides, or other sorbent into the flue gas represents a relatively simple approach to controlling mercury emissions from coal-fired boilers. The gas-phase mercury in the flue gas contacts the sorbent and attaches to its surface. The sorbent with the mercury attached is then collected by the existing particle control device, either an electrostatic precipitator (ESP) or fabric filter (FF).

The air pollution control industry already has considerable experience with the implementation of mercury controls for other industrial sectors. Sorbent injection has been commercially proven to augment the removal of mercury in waste-to-energy plants. Experience controlling mercury emissions has been gained in more than 60 U.S. and 120 international waste-to-energy plants that burn municipal or industrial waste or sewage sludge. For the past two decades, sorbent injection upstream of a baghouse has been successfully used for removing mercury from flue gases from these facilities. Other reagents used include activated carbon, lignite coke, sulfur containing chemicals, or combinations of these compounds. The mercury control experience gained from the municipal and industrial waste combustors demonstrates that the air pollution control industry has been able to control mercury in the past and is able to apply their expertise to the electric power sector.

b. Performance and Applicability

EPA has requested comment concerning the availability of sorbent injection technologies to serve the electric power market. Activated carbon injection equipment is currently being sold to utilities. ACI equipment is identical for all coal types including bituminous, subbituminous, lignites and blends. Therefore, ACI equipment can be purchased for all coals and all plant configurations.

The specific sorbents may vary for different coals and operating conditions. In addition, the ability to accurately predict the levels of mercury removal that will be achieved will vary for different coals depending on the available performance data. For example, there have been a significant number of tests over the past year and a half on PRB coals and North Dakota lignites. Therefore, it is possible to estimate results for these configurations. There is less data on bituminous coals, so predictions will be less precise. Several full-scale field tests will be conducted on bituminous coals during 2005 and 2006. The first test on a Texas lignite will be

conducted in 2005. Until this occurs, it is difficult to predict performance on Texas lignite.

The performance of activated carbon injection systems for lignite, subbituminous, and bituminous coals on various coal-fired power plant configurations are given in Table 1. The mercury reduction performance for these power plant scenarios are based on results from full-scale demonstrations that have been documented in various technical papers presented at major electric power conferences.

Table 1. Activated Carbon Injection Control Technology Options

Plant Configuration	Technology	Coal Type	% Reduction				Cost		Year Commercially Available
			Min	Max	Avg. Total[a]	Avg. Incrm.[b]	Capital ($/kW)	O&M ($/kWh)	
CESP[d]	ACI[f,g]	Bit	50	90	70	70	1.5 to 3	.0012	2004
	ACI[g,h]	Sub	0	95	80	80	1.5 to 3	.0005	2004
	ACI[i]	Lig	0	80	63	63	1.5 to 3	.0005	2004
CESP/FGD	ACI[j]	Bit	50	90	70	70	1.5 to 3	.0012	2004
	ACI	Sub	0	95	80	80	1.5 to 3	.0005	2004
	ACI[k]	Lig	0	80	60	70	1.5 to 3	.0005	2004
CESP/FGD-dry	ACI	Bit	80	>90	>90	88	1.5 to 3	.00012	2004
	ACI	Sub	0	90	80	85	1.5 to 3	.00017	2004
	ACI	Lig	0	90	70	70	1.5 to 3	.00017	2004
CESP/SCR/FGD	ACI	Bit	50	90	70	70	1.5 to 3	.0012	2004
	ACI	Sub	0	95	80	80	1.5 to 3	.0005	2004
	ACI	Lig	0	80	60	60	1.5 to 3	.0005	2004
FF	ACI[l]	Bit	20	95	85	80	1.5 to 3	.00036	2004
	ACI[l,m]	Sub	20	90	90	80	1.5 to 3	.00054	2004
	ACI	Lig	20	80	80	75	1.5 to 3	.00054	2004
FF/FGD	ACI	Bit	50	95	90	70	1.5 to 3	.00012	2004
	ACI[l]	Sub	30	90	90	80	1.5 to 3	.00027	2004
	ACI	Lig	30	90	85	70	1.5 to 3	.00027	2004
FF/SCR/FGD-dry	ACI	Bit	80	>90	>90	50	1.5 to 3	.00012	2004
	ACI[n]	Sub	0	>90	>90	90	1.5 to 3	.00017	2004
	ACI[o]	Lig	0	90	75	70	1.5 to 3	.00017	2004
FF/SCR/FGD	ACI	Bit	50	95	90	70	1.5 to 3	.00012	2004
	ACI	Sub	30	90	90	80	1.5 to 3	.00027	2004
	ACI	Lig	30	80	80	70	1.5 to 3	.00027	2004
HESP[e]	TOXECON[p]	Bit	20	95	85	80	3 + 15 to 3 + 50	.00036	2004
	TOXECON	Sub	20	90	90	80	3 + 15 to 3 + 50	.00036	2004
	TOXECON	Lig	20	80	80	70	3 + 15 to 3 + 50	.00054	2004
HESP/FGD	TOXECON	Bit	50	95	90	70	3 + 15 to 3 + 50	.00012	2004
	TOXECON	Sub	30	90	90	80	3 + 15 to 3 + 50	.00036	2004
	TOXECON	Lig	30	80	80	70	3 + 15 to 3 + 50	.00027	2004

HESP/FGD-dry	TOXECON	Bit	80	>90	>90	50	3 + 15 to 3 + 50	.00012	2004
	TOXECON	Sub	0	>90	>90	90	3 + 15 to 3 + 50	.00017	2004
	TOXECON	Lig	0	90	88	70	3 + 15 to 3 + 50	.00017	2004
HESP/SCR/FGD	TOXECON	Bit	50	95	90	70	3 + 15 to 3 + 50	.00012	2004
	TOXECON	Sub	30	90	90	80	3 + 15 to 3 + 50	.00036	2004
	TOXECON	Lig	30	80	80	70	3 + 15 to 3 + 50	.00027	2004

[a] This is the percent reduction attributable to the existing pollution controls and the technology.
[b] This is the percent reduction attributable only to the technology.
[c] In EPA's modeling, is it appropriate for an economic forecast to assume an improvement in costs over time (such as through technology cost reductions or through future technology innovation).
[d] CESP – represents cold-side electrostatic precipitator
[e] HESP - represents hot-side electrostatic precipitator
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c. Availability

Companies are providing firm price proposals with performance guarantees for every coal and boiler type. Activated carbon injection equipment is currently being sold to utilities. ACI equipment is identical for all coal types including bituminous, subbituminous, lignites and blends. Therefore, ACI equipment can be purchased for all coals.

The material resources, labor and time required to install the control equipment is an additional topic to consider. With regards to the items that impact APC vendors, there are sufficient fabrication/manufacturing resources in the U.S. market to support a rapid retrofit of the industry with sorbent injection systems in addition to the systems required for the Clean Air Interstate Rule. These systems are relatively simple compared to FGD and SCR systems and the major components are commonly used in a variety of industrial processes from numerous manufacturers throughout the U.S.

As mentioned in ICAC's previous comments on EPA's proposed mercury rule, there is significant excess production capacity of powder activated carbon and a strong interest in investing significant capital in building new production facilities exists among current suppliers (both in the U.S. and in China). A new mercury regulation would create a significant new market for activated carbon. In order to build new production capacity, between a two- to four-year period would be needed to expand production. However, all of the activated carbon suppliers said that they would be hesitant to invest capital resources to increase capacity based only on the promise of a new regulation. A decade or so ago, the AC industry increased capacity when EPA announced that they were going to tighten up drinking water standards. After the new capacity was added, EPA did not follow up with new regulations, which produced a glut of activated carbon. Some companies went out of business because of this, and the industry as a whole is just now recovering. As a result, it is unlikely that new AC production will move beyond the planning stages until there is the certainty of a regulation.

Concerning resources for fabric filter systems, should the market dictate the need for secondary PM control (not all applications will require this) there will be sufficient engineering and material resources to complete the necessary projects. There are several examples where the industry has had to retrofit a significant number of boilers with APC controls to meet new environmental regulations. Examples include the retrofit of ESPs in the 1970s and the more recent retrofit of almost 100 GW of SCRs for the NO_x SIP Call. These examples support the assertion that, should the utility industry need to retrofit a large number of coal-fired boilers with mercury controls, it can be accomplished in a short period of time. The industries that support this market (APC suppliers, fabricators, construction firms, etc.) have repeatedly demonstrated their ability to meet rapidly increasing market demand. In addition, increasing demand for systems and fabrication can

also be met by foreign suppliers of silos, fabric filter systems, fabrication and supply of PAC.

If there is a bottleneck in retrofitting the U.S. fleet of coal-fired boilers, it is not likely to be in the area of the supply of capital equipment or under supply of sorbents but more likely to be impacted by issues that are within the scope of the utility or regulatory community itself. Examples include areas such as project permitting/PUC approval, availability of project financing, and unit outage scheduling. These are all items that are out of the control of APC vendors but may impact the timing for control installation.

d. Costs

EPA reported that the Edison Electric Institute (EEI) estimated that ACI would be less expensive per pound of Hg removed than EPA has estimated. Meanwhile, other power industry models assumed higher capital costs for ACI than EPA in its modeled scenarios. EPA is seeking comment on whether its assumptions for Hg control technology costs are reasonable.

EPA raised several questions in the NODA requesting information on sorbent injection technologies and how best to make modeling assumptions to reflect current and future capabilities of mercury control technologies. One of the questions raised by EPA was concerning the use of discounted variable operating costs for activated carbon injection (ACI). EPA questioned whether it would be appropriate for an economic forecast to assume an improvement in costs over time (such as through technology cost reductions or through future technology innovation), and what level of improvement in costs to assume. Specifically, EPA questioned whether a 2.5 percent annual improvement in variable operating costs for ACI should be incorporated into their modeling as has been done for similar power sector models.

In regards to decreasing costs, it is appropriate to assume that the cost of sorbent technologies will decrease with time due to equipment/technology innovation, improvements in sorbent removal efficiencies, and the reduction in sorbent production costs. The primary cost of sorbent injection technology is due to sorbent usage so the largest cost reductions are likely to be made with the sorbent costs. The capital costs for ACI are relatively low as the equipment is mechanically simple compared to FGD and SCR systems for coal-fired power plants. Activated carbon injection systems consist of a bulk-storage silo; blower/feeder system to convey the activated carbon from the silo through hard piping leading to the flue gas duct; and injection probes located in the flue gas duct. Currently, the annual operating costs for these systems will be more than the cost to construct the system.

Costs are expected to decrease as sorbents are developed specifically for the coal-fired boiler application. It is widely known that the current sorbents have much higher capacity for mercury removal than can effectively be used in a coal-fired

power plant application. This is because the injected sorbent will be rapped off the plates of the electrostatic precipitator or cleaned off the bags of the fabric filter before the absorption/adsorption capacity of the sorbent has been fully utilized. Therefore, work is being done to produce a lower capacity, lower cost sorbent that will be more appropriate for use in this industry.

It is also expected that technical innovations will lead to lower cost sorbents. For example, ADA-ES has reported improved mercury removals on full-scale tests with NORIT's new activated carbon named E-3[1,2]. These tests showed that significantly higher levels of mercury could be removed at significantly lower feed rates than earlier tests indicated. With this kind of technology improvement, the overall cost for mercury removal will decrease over this. This is especially true for Western coals (i.e. lignite and subbituminous) as sorbent injection rates are expected to be higher for those units yielding a drop in operating costs by a factor of two to four. It is expected that similar improvements in sorbents will result in similar cost reductions for bituminous coals.

As far as production costs are concerned, there will likely be a reduction in cost to produce sorbent products for the power industry due to economies of scale. Currently, activated carbon is already manufactured by numerous vendors for a wide variety of customized applications requiring inefficient and expensive materials handling to provide the different treatments and particle size requirements. In addition, the demand for activated carbon is seasonal and therefore the use of the equipment is not optimized. To meet the power industry demands, it is likely that new production facilities will be built to produce only a few products so there will be an increase in efficiency and reduction in cost. In addition, the power market will be much more consistent and predictable, which will serve to optimize the production equipment.

Once the sorbents are specifically produced for power industry applications, the pricing trend for activated carbon should act very much like other commodities. On average, pricing for most commodity items will normally stay unchanged or decrease slightly over time as market forces encourage cost reductions. Since inflation in the U.S. normally runs around 2-3 percent, any commodity that does not increase in price decreases (in real terms) by around this amount every year. It is safe to assume that activated carbon prices will decrease by at least 2-3 percent in real terms (net inflation). The most likely scenario is that prices for sorbents will initially decrease by much more than 2-3 percent as the market for this specific application grows and will reach a steady state annual reduction of 2-3 percent.

The final decrease in costs will come about through innovative equipment/technology configurations such as the EPRI TOXECON II. Currently, EPA modeling includes the cost for the loss of sale of power plant fly ash plus landfill costs to dispose of the fly ash. The EPRI TOXECON II process eliminates the cost of loss of sale of the fly ash for concrete without the need for a new fabric filter. As a result, plants will be able to avoid one of the most costly aspects of the

technology. As shown in Table 1, the capital cost of installing a COHPAC fabric filter is expected to range between $15 – 50/kW depending on the plant configuration. Also given in Table 1, the capital cost of installation ACI systems is expected to range between $1.5 – 3/kW.

When considering the combination of the decrease in cost of sorbent technologies with time due to equipment/technology innovation, improvements in sorbent removal efficiencies, and the reduction in sorbent production costs; it is safe to assume that costs for this technology will decrease over time. A more likely scenario for costs of ACI over the next three to five years would be more significant reductions in overall costs by a factor of 2 or more compared to current EPA and DOE projections of only 2.5 percent.

ii. Electro-Catalytic Oxidation (ECO)

a. Technology Description

Powerspan Corp's Electro-Catalytic Oxidation (ECO) is an integrated multi-pollutant control technology that achieves major reductions in emissions of nitrogen oxides (NO_x), sulfur dioxide (SO_2), fine particulate matter ($PM_{2.5}$), and mercury (Hg). The technology also reduces emissions of other air toxic compounds and acid gases such as arsenic, lead, and hydrochloric acid (HCl). ECO produces a commercial fertilizer co-product, reducing operating costs and avoiding landfill disposal of waste.

ECO is situated downstream of a power plant's existing electrostatic precipitator (ESP) or fabric filter. The system consists of three gas-processing steps, including a barrier discharge reactor, an ammonia-based wet scrubber, and a wet ESP. The barrier discharge reactor oxidizes SO_2, NO_x, and Hg; the ammonia scrubber removes SO_2, NO_2, and oxidized Hg creating an ammonium sulfate nitrate solution; and the wet ESP captures acid aerosols, fine particulate matter, and oxidized Hg.

Liquid effluent produced by the scrubber contains dissolved ammonium sulfate nitrate (ASN) salts, along with mercury and captured particulate matter. The ASN solution is sent to a co-product recovery system, which includes filtration to remove ash and a sulfur impregnated activated carbon adsorption bed, which removes mercury from the effluent stream. The mercury and spent activated carbon are disposed of as hazardous waste. The treated co-product stream, free of mercury and ash, can be used directly in liquid form or processed to form ammonium sulfate nitrate fertilizer in crystalline or granular form.

b. Performance and Applicability

The ECO process is currently being commercially demonstrated in a 50-MW slipstream unit at FirstEnergy Corp.'s R.E. Burger Plant in Shadyside, Ohio. The unit processes flue gas from a plant burning eastern bituminous coal. As of August 2004, ECO performance has met or exceeded most commercial objectives. Mercury

removal across the ECO system has ranged from 75 – 85 percent with total inlet mercury concentration up to 16 μg/Nm3. SO_2 removal is routinely greater than 99 percent with inlet SO_2 concentrations up to 2200 ppm and outlet concentrations below 10 ppm. NO_x removal has been as high as 82 percent with outlet levels of 0.05 lb/mmBtu.

Prior to proceeding with the 50-MW commercial demonstration unit, Powerspan conducted pilot testing in a 1-MW slipstream unit at the R.E. Burger Plant. During approximately 18 months of testing, the plant burned a blend of bituminous and subbituminous coals. Typical values for mercury concentration, chlorine, and sulfur content in the coal were 0.09 ppm mercury, 0.06 percent chlorine, and 1.9 percent sulfur. Ontario-Hydro sampling was conducted by Air Compliance Testing (Cleveland, Ohio) at the ECO pilot unit in May 2002. Ontario-Hydro testing measures gas-phase mercury (elemental and oxidized forms) and mercury bound to particulate matter in the flue gas. Air Compliance's testing consisted of three sample runs each on the inlet and outlet flue gas streams. Two of the three sets of sample runs had sample durations in each location of four hours while sampling for the remaining set of runs lasted three hours in each location. The mercury removal for particulate, oxidized, and elemental are provided in Table 2 with the overall mercury removal measured at 88 percent.

Table 2. Mercury Removal at ECO Pilot Demonstration

Hg Fraction	**ECO Inlet**	**ECO Outlet**	**% Removal**
Particle Bound Hg (μg/dscm)	0.62	0.016	97.4
Oxidized Hg (μg/dscm)	5.81	0.022	99.6
Elemental Hg (μg/dscm)	0.16	0.75	
Total Hg (μg/dscm)	6.59	0.79	88.0

Table 2 provides estimates of the ECO process performance for various plant configurations. It is expected that 80 percent mercury removal across the ECO system will be achieved with the application of the ECO process for units burning bituminous coals. The average incremental removal for fabric filter and hot-side ESP applications are expected to be similar to that demonstrated at the ECO commercial demonstration unit at the R.E. Burger Plant, which employs a cold-side ESP. The cost and performance estimates are based on results currently being commercially demonstrated in a 50-MW slipstream unit at FirstEnergy Corp.'s R.E. Burger Plant.

Table 2. Mercury Removal Capability of ECO Commercial Technology

Plant Config.	Coal Type	% Reduction				Cost			Year Commercially Available
		Min	Max	Avg. Total[a]	Avg. Increm.[b]	Capital ($/kW)	O&M ($/kWh)	% Expected Change Cost (+/-) w/ time[c]	
CESP[d]	Bit	(f)	(f)	(f)	80%	$225/kW	$0.0027	Decrease	2006
FF	Bit				80%	$225/kW	$0.0027	Decrease	2006
HESP	Bit				80%	$225/kW	$0.0027	Decrease	2006

[a] This is the percent reduction attributable to the existing pollution controls and the technology.

[b] This is the percent reduction attributable only to the technology.
[c] In EPA's modeling, is it appropriate for an economic forecast to assume an improvement in costs over time (such as through technology cost reductions or through future technology innovation).
[d] CESP – represents cold-side electrostatic precipitator
[e] HESP - represents hot-side electrostatic precipitator
[f] Measurements of the mercury content in the coal and in the flue gas upstream of the plant's ESP have not been made.

c. Availability

The ECO process is currently being commercially demonstrated in a 50-MW slipstream unit at FirstEnergy Corp.'s R.E. Burger Plant in Shadyside, Ohio. Previously, ECO was pilot tested in a 1-MW slipstream unit at the same plant. Commercial demonstration testing is planned to complete in the first quarter of 2005. Based on this project, Powerspan will offer commercial ECO systems with industry standard guarantees and warranties by the beginning of 2006.

d. Costs

It is estimated that the capital cost of the multipollutant ECO process will be $225/kW and the operation and maintenance costs will be $0.0027/kWh. These are the estimated costs for cold-side ESP application based on the experience at the Burger Plant. The cost for fabric filter and hot-side ESP applications are expected to be similar to cold-side ESP application. To estimate the cost effectiveness of the process for mercury removal, it is estimated that the variable cost of mercury removal in the ECO process is $800 per pound of mercury, including the sorbent media and its disposal. The costs are expected to decrease over time due to technology innovations; however, the level of cost reduction has not yet been estimated.

2. PRECOMBUSTION CONTROL TECHNOLOGIES

i. K-Fuel

a. Technology Description

KFx, Inc. has a patented and proven pre-combustion technology that transforms low-cost, low-grade western coal (e.g. lignite or subbituminous) into a clean, affordable, efficient energy source, called K-Fuel. K-Fuel pre-combustion technology applies heat and pressure to boost the heat value of subbituminous coal and lignite by 30-55 percent, from approximately 8,000-8,800 Btu/lb to 11,000-11,500 Btu/lb, optimizing combustion in a manner that produces more generation output per ton of coal while lowering emissions. Moisture in the coal can be reduced by as much as 80 percent from approximately 30 percent in the feedstock to seven percent in K-Fuel.

Similar to post combustion SO_2, NO_x, and PM controls, mercury emission reductions from the K-Fuel technology are a co-benefit of the pre-combustion

process. K-Fuel provides a pre-combustion mercury removal solution, reducing mercury content by up to 70 percent or more. In addition to mercury reductions, K-Fuel also reduces emissions of SO_2 and NO_x.

b. Performance and Applicability

Since the K-Fuel process reduces emissions of multiple pollutants, coal-fired facilities that will most benefit from burning K-Fuel to reduce mercury emissions include those units that will achieve the most cobenefit from SO_2 and NO_x emission reductions as well as heat rate improvements. K-Fuel will benefit units burning high sulfur bituminous coal with no SO_2 control, units burning declining supplies of Central Appalachian SO_2 compliant coal, units that have switched from bituminous to subbituminous coal to meet the Title IV Acid Rain requirements with a resulting loss in generating capacity, units with no post-combustion SO_2 or NO_x control, and small generating units that are searching for low capital cost mercury control. K-Fuel can also be burned in units currently burning subbitminous coal and lignite, the feedstocks for K-Fuel.

K-Fuel is a commercially viable pre-combustion solution and proven technology for western coal to reduce mercury emissions from coal-fired power plants. K-Fuel accomplishes mercury reduction through its coal beneficiation process. In effect, by combusting K-Fuel the utility is achieving mercury reduction for free since mercury removal has already occurred during the K-Fuel process prior to combustion by a utility.

Table 4 below provides laboratory data for various feedstocks of subbituminous coal, along with the corresponding reduction in mercury, increase in heat rate (Btu), and reduction in moisture content achieved by the K-Fuel process. The information presented demonstrates that the effectiveness of the process is dependent upon the properties of the unique coal feedstock.

To date, the K-Fuel pre-combustion process has not been optimized for mercury emission reduction but is a co-benefit of the pre-combustion process. In Table 4, the amount of mercury removal listed is the amount of mercury reduced in the coal prior to combustion and does not consider the potential additional reductions from existing control technologies (e.g. electrostatic precipitators, fabric filters, etc.). As a result, the mercury reduction numbers below are a beginning point for the ultimate mercury reduction achievable when burning K-Fuel, not accounting for plant specific characteristics.

A facility knows when it purchases K-Fuel how much mercury has already been removed and what amount of mercury is in the K-Fuel prior to combustion. Additional mercury removal above that already achieved in the K-Fuel will be dependent upon unit specific characteristics such as installed pollution control devices and boiler characteristics, as mentioned below.

Table 4. Emissions Reductions from Laboratory Tests using F-Fuel Process

Coal[a] ID	Coal As Rec. Moisture Percent	Coal As Rec. Btu/Lb	Coal As Rec. Hg Lbs/TBtu	K-Fuel As Rec. Moisture Percent	K-Fuel As Rec. Btu/Lb	K-Fuel Hg Lbs/TBtu	Moisture Removal Percent	Btu Increase Percent	Total Mercury Removal Percent
Coal 1	31.06	8520	1.98	6.06	11667	0.63	80	37	68
Coal 2	27.00	8969	24.17	5.74	11683	3.75	79	30	85
Coal 3	28.41	8536	12.58	6.46	11331	3.10	77	33	75
Coal 4	32.04	7903	7.99	7.06	11162	1.84	78	41	77
Coal 5	31.72	8126	6.30	8.00	11091	2.30	75	37	63
Coal 6	30.93	8235	3.51	6.91	11149	2.02	78	35	42
Coal 7	31.20	8032	4.05	7.09	10535	1.93	77	31	52

[a] Subbituminous coals were used for all of the laboratory tests.

c. Costs

K-Fuel does not impose any installation, capital, or operating costs in addition to the cost of K-Fuel per ton to achieve mercury reduction since mercury reduction is already achieved in K-Fuel prior to combustion in a coal-fired unit. As a solid coal fuel, K-Fuel will not negatively impact system components or by-products since there are no chemicals, additives, or other substances added to the combustion process, flue gas, or to the K-Fuel itself to enhance mercury removal. Currently, KFx conservatively estimates that K-Fuel will be sold for $33 per ton (including transportation costs), though market conditions and other factors may impact the price.

d. Availability

In June 2004, KFx announced its purchase of the Fort Union mine site near Gillette, Wyoming as the location for a commercial K-Fuel production facility. The site includes approximately 1,000 acres of land, a rail loop with load out facilities, a coal crusher, related buildings, water disposal wells and about 500,000 tons of remaining coal reserves. Private money is fully funding the project and the Wyoming Department of Environmental Quality (WYDEQ) has finalized all permits necessary for construction. The final air quality permit was granted from WYDEQ on November 8, 2004 and ground was broken on the site November 10, 2004. Concrete foundations have begun being poured as of December 2004. Fabrication of the major process components of the facility is near completion.

The feedstock coal to produce K-Fuel will be purchased from adjacent mines in the Powder River Basin. Initial output from the facility will be 750,000 tons per year and two-thirds of the output has been pre-sold with the remaining portion to be used for test burns to facilitate additional markets for K-Fuel. The K-Fuel production facility is expected to be in commercial operation in the summer of 2005. The facility can be expanded to produce up to 8 million tons per year of K-Fuel and KFx expects that with the first commercial plant in operation the development of future plants will be accelerated. KFx is examining potential commercial sites in Wyoming, Alaska, South Dakota, and other locations for additional K-Fuel

production facilities. KFx plans to own and operate the K-Fuel production facilities, as well as license K-Fuel technology to third parties in the U.S. and internationally.

# # #

The Institute looks forward to working further with you on this important issue, and invite you or your staff to contact me for further information or clarification.

Sincerely,

David C. Foerter
Executive Director, ICAC

INSTITUTE OF
CLEAN
AIR
COMPANIES

Senate Democratic Policy Committee

Hearing on EPA Proposal to Regulate Mercury Emissions from Power Plants

Testimony of the Institute of Clean Air Companies (ICAC)
Presented by David C. Foerter, Executive Director

Friday, July 9, 2004

Good morning. I'm Dave Foerter, Executive Director for the Institute of Clean Air Companies ("ICAC" or "the Institute").

The Institute is the nonprofit, national association of companies that manufacture, supply, and service air pollution control and monitoring systems for a broad range of air pollutants, including mercury from power plant and industrial sources. The Institute represents a diverse group of approximately eighty companies dedicated to air pollution control. As such, the Institute represents the full range of competing technologies, rather than any single technology. In the few minutes I have here this morning, I'll begin with the "bottom-line."

Our industry believes that a 50 to 70 percent reduction from current mercury emissions of 48 tons per year is feasible by 2008 to 2010. As a result, over the next 4 to 6 years, it is reasonable and cost-effective to achieve a utility mercury budget of 14 to 24 tons. The air pollution control industry has both the technology and the resources to exceed the magnitude of NO_x, SO_2, $PM_{2.5}$, and mercury reductions, and in a shorter time frame than proposed by the U.S. Environmental Protection Agency (EPA).

It is important to remind ourselves that air pollution control technology markets have historically worked well. Studies show that the certainty of regulatory drivers spurs technical performance and cost improvement. And total costs fall dramatically as control technology moves from R&D to full-scale commercialization. It is reasonable to assume that even with the tremendous technological achievements already made, the traditional successful operation of the air pollution control market will also apply to the development and enhancement of mercury emission controls. The key to well-functioning markets is regulatory certainty. If the goal is technological innovation, then it is important to enact a clear, certain, performance-based mandate. While the Institute advocates flexibility in meeting control requirements, that compliance flexibility should be considered only after setting emissions budgets that adequately protect public health and make use of the capabilities of control technology.

One technology in particular, activated carbon injection, has been used for at least a decade in the waste to energy industry to achieve mercury reductions of at least 80 to 90 percent. This technology has been successfully transferred to the power sector for

commercial use. Activated carbon injection provides a relatively low cost solution, with very little capital investment and relatively low operating costs. In addition, control performance can be increased and operating cost decreased, if activated carbon injection is coupled with fabric filter particulate control devices. In an intensive effort over the last five years, this technology has been rigorously demonstrated through the Department of Energy's (DOE) Clean Coal Power Initiative at full scale on electric power plants, with additional demonstrations to be completed by 2005. The demonstrations identified and addressed power sector mercury control issues, but, more importantly, dramatically removed potential barriers and enhanced the technology. R&D has already matured to full-scale demonstrations and are applicable to a wide range of coal types and existing equipment configurations. Many of these project teams include utility end-users as well as technology developers, which indicates the wide-ranging, cooperative effort underway. The success of this work and other applications, have now all but obscured the 1999 Information Collection Request (ICR) data that was used by EPA to propose the MACT floor. EPA's data shows that existing controls not intended to reduce mercury, had a side-benefit of removing other pollutants, including mercury. In fact, reliance on the 1999 ICR data promotes switching between coal types to achieve compliance, while the more current data shows economical compliance can be achieved without coal switching.

As we have informed EPA and others, a growing number of companies offer commercially available mercury control technologies for sale to the electric power sector. In fact, there are an increasing number of electric utilities actively procuring these technologies and services. Several other technologies are in various stages of development and commercial availability, ready to compete as compliance options under the Utility MACT program. We believe that Congress or EPA does not have to pick technology winners and losers; the marketplace is adept at doing so. The course of technology development is too unpredictable to say what the best approach will be and experience strongly indicates that there will not be one universal approach.

The rapid development of mercury control technologies make it feasible for the electric power sector to cost effectively reduce significantly more mercury emissions than called for under the proposed Utility MACT program. Assuming the implementation of a MACT program requiring control at each plant, it is estimated that a 50 percent reduction from the current emission level of 48 tons of mercury down to 24 tons is achievable. To achieve greater levels of control, there will be performance differences at each site due to differences in coal, equipment, and flue gas characteristics. At some power plants mercury control technology can reduce mercury emissions by 90 percent or greater. Therefore, if a mercury control program included compliance flexibility it is expected that a 70 percent reduction in emissions (down to 14 tons of emissions) is achievable.

Even within the MACT program constraints, EPA can provide compliance flexibility to achieve a high level of mercury control under the Utility MACT timeline without negatively affecting generation. Some of these mechanisms have been used in previous EPA regulations, both MACT and acid rain rules, such as: long term averaging, limits that specify a percent removal and emission rate, early reduction incentives such as those used under the Title IV NO_x provisions or Section 112 (i) (5) and (6), or a safety net approach that requires significant reduction with some flexibility for difficult applications. It is important that flexibility include the performance that is achievable by technology, rather than a prescription for a particular technology.

The air pollution control industry has already achieved commercial readiness of mercury control and measurement technologies, even without the certainty typically provided by regulatory or legislative market drivers. Mercury control technology is available today at the reasonable cost of 0.1 to 0.3 cents per kW-hr, compared to and average retail rate of 8 cents per kW-hr. Mercury control technologies are currently available for a range of coals and equipment and will be available for every utility configuration and every coal type in the near future. Mercury reductions of 50 percent (24 tons of emissions) are achievable by 2008 to 2010, and up to a 70 percent (14 tons of emissions) would be achievable by all utilities if there were some flexibility in regulation or legislation.

On behalf of the more than 130,000 men and women in our nation that work to supply air pollution control and monitoring technology for stationary sources, we congratulate efforts to develop meaningful and flexible approaches to control emissions from the electric power sector. Dollars spent on compliance are recycled in the economy, generating jobs in construction, materials fabrication, and engineering. The Institute predicted that multi-pollutant control requirements would create 300,000 new U.S. jobs.

Thank you for this opportunity to testify. I look forward to your questions.

# # #

For more information go to www.icac.com or contact ICAC at 202.457.0911

INSTITUTE OF CLEAN AIR COMPANIES

Commercial Electric Utility Mercury Control Technology Bookings

Air pollution control vendors are reporting booking new contracts for mercury control equipment for more than two dozen power plant boilers. The contracts for commercial systems are attributed to federal and state regulations, including new source permit requirements and consent decrees, which specify high levels of mercury capture. Below is a summary of the mercury control equipment that has been procured to date. Highlighted units are currently in operation. **Last Update: 09-18-2007**

	Plant Size (MW)	Location	Prime OEM Contractor	Coal	APC Configuration	Hg Control	New Plant or Retrofit	Regulatory Driver
1	90 MW ea. 270 Total	**Midwest**	Wheelabrator (Norit/ADA-ES)	PRB	TOXECON	ACI	Retrofit	Consent Decree
2	250	**East**	Wheelabrator	Bituminous	SDA/FF	ACI	Retrofit	State Regulatory
3	250	**East**	Wheelabrator	Bituminous	SDA/FF	ACI	Retrofit	State Regulatory
4	650	**East**	Wheelabrator	Bituminous	ESP	ACI	Retrofit	State Regulatory
5	740	**Midwest**	B&W (ADA-ES)	PRB	SDA/FF	ACI	New Plant	New Construction Permit
6	550	**Midwest**	B&W (ADA-ES)	PRB	SDA/FF	ACI	New Plant	New Construction Permit
7	350	**West**	B&W (ADA-ES)	PRB	SDA/FF	ACI	Retrofit	Consent Decree
8	350	**West**	B&W (ADA-ES)	PRB	SDA/FF	ACI	Retrofit	Consent Decree
9	800	**West**	B&W (ADA-ES)	PRB	SDA/FF	ACI	New Plant	New Construction Permit
10	350	**East**	ADA-ES	Bituminous	ESP	ACI	Retrofit	Consent Decree
11	350	**East**	ADA-ES	Bituminous	ESP	ACI	Retrofit	Consent Decree
12	204	**Midwest**	Dustex	PRB	TOXECON	ACI	Retrofit	Consent Decree
13	375	**East**	Wheelabrator	Bituminous		ACI	Retrofit	Consent Decree

14	650	**Midwest**	Alstom (ADA-ES)	PRB	SDA/FF	ACI	New Plant	New Construction Permit
15	156 MW ea. 315 Total	**Midwest**	Powerspan	Bituminous	Multi-pollutant	ECO	Retrofit	Construction Permit
16	750	**Midwest**	Wheelabrator	High Sul. Bit	ESP/WFGD/ WESP	ACI	New Plant	Construction Permit
17	680	**South**	Alstom (ADA-ES)	PRB	SDA/FF	ACI	New Plant	Construction Permit
18	107	**East**	BPI	Bit./Bio-Mass	FT-SNCR/CDS/F F	ACI	Retrofit	DOE Demo.
19	860	**South**	BPI	Lignite	SCR/FF/WF GD	ACI	New Plant	Construction Permit
20	860	**South**	BPI	Lignite	SCR/FF/WF GD	ACI	New Plant	Construction Permit
21	220	**West**	B&W (ADA-ES)	PRB	SDA/FF	ACI	New	Construction Permit
22	575	**Southwest**	B&W (STC)	West.Bit/S ub. Bit. Blend	HS-ESP/FF/WFG D	ACI	Retrofit	Construction Permit
23	575	**Southwest**	B&W (STC)	West.Bit/S ub. Bit. Blend	HS-ESP/FF/WFG D	ACI	Retrofit	Construction Permit
24	335	**Northeast**	ADA-ES	Bituminous	Cold-Side ESP	ACI	Retrofit	Voluntary Regional Emission Abatement Plan
25	880	**South**	Wheelabrator	PRB	ESP/FF (TOXECON)	ACI	Retrofit	Voluntary Regional Emission Abatement Plan
26	350	**Midwest**	Hamon (ADA-ES)	PRB	SCR/FF	ACI	Retrofit	State Regulatory
27	650	**Southwest**	ADA-ES	PRB	ESP/FF	ACI	Retrofit	Voluntary Regional Emission Abatement Plan
28	628	**Southwest**	ADA-ES	RPB	ESP/FF Parallel Flow	ACI	Retrofit	Voluntary Regional Emission Abatement Plan
29	855	**Southwest**	ADA-ES	Lignite/PR B	ESP/WFGD	ACI	Retrofit	Voluntary Regional Emission Abatement Plan
30	670	**Midwest**	Alstom/ADA-ES	PRB	SCR/FF/WF GD	ACI	Retrofit	Construction Permit of new unit

31	850	**Midwest**	Alstom/ADA-ES	PRB	SCR/FF/WFGD	ACI	New	Construction Permit
32	167	**East**	Sorbent Tech	E- Bitum	ESP/WFGD	ACI	Retrofit	Consent Decree
33	108	**Midwest**	Dustex	PRB	TOXECON	ACI	Retrofit	Consent Decree
34	159	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	CAMR
35	348	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
36	237	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
37	347	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
38	341	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
39	566	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
40	561	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
41	850	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
42	850	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
43	359	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
44	385	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
45	281	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
46	551	**Midwest**	NORIT	PRB	ESP	ACI	Retrofit	State Regulatory
47	400	**Southwest**	Alstom/ADA-ES	PRB	SDA/FF	ACI	New	Construction Permit
48	495	**Alberta Canada**	B&W/NORIT	Can. Sub-Bit.	SDA/FF	ACI	New	Construction Permit
49	800	**Midwest**	Wheelabrator	E. Bit	Lime Inj./ESP/WFGD/WESP	ACI	New	Construction Permit

50	800	**Midwest**	Wheelabrator	E. Bit	Lime Inj./ESP/WFGD/WESP	ACI	New	Construction Permit
51	350	**Midwest**	ADA-ES	PRB	ESP	ACI	Retrofit	Construction Permit
52	568	**Southwest**	AESI/ADA-ES	Lignite	CFB Boilers/SNCR/ACI/CDS-DFGD/FF	ACI	New	Construction Permit
53	248	**Midwest**	ADA-ES	PRB	ESP	ACI	Retrofit	State Regulatory
54	590	**Midwest**	ADA-ES	PRB	ESP	ACI	Retrofit	State Regulatory
55	608	**Midwest**	ADA-ES	PRB	ESP	ACI	Retrofit	State Regulatory
56	110	**Midwest**	ADA-ES	PRB	ESP	ACI	Retrofit	State Regulatory
57	272	**Midwest**	ADA-ES	PRB	ESP	ACI	Retrofit	State Regulatory
58	375	**Midwest**	ADA-ES	PRB	ESP	ACI	Retrofit	State Regulatory
59	100 ea. 200 Total	**Northeast**	Clyde Bergemann EEC	PRB	Dry Injection/FF	ACI	Retrofit	State Regulatory
60	200 ea. 400 Total	**Northeast**	Clyde Bergemann EEC	PRB	Dry Injection/FF	ACI	Retrofit	State Regulatory
61	200 ea. 400 Total	**Northeast**	Clyde Bergemann EEC	PRB	Dry Injection/FF	ACI	Retrofit	State Regulatory
62	300	**Midwest**	Allied/ADA-ES	PRB	CDS/FF	ACI	Retrofit	Construction Permit

December 30, 2004

William Maxwell
U.S. EPA
OAQPS, Emission Standards Division
Combustion Group (C439-01)
Research Triangle, NC 27711

Dear Mr. Maxwell:

In its Notice of Data Availability (NODA) for the Proposed National Emission Standards for Hazardous Air Pollutants, (Federal Register Vo.. 69, No. 230, pp 69864-69878, Dec. 1, 2004), the U.S. Environmental Protection Agency sought updated information on the **current state of activated carbon injection (ACI) technology** for the reduction of mercury emissions from coal-fired utility boilers:

"II.B.4. Electric Utility Modeling: Areas of ongoing EPA research
[T]he Agency is seeking updated information on issues that may be relevant to assessing the assumptions employed in our power plant modeling, (e.g.[:]

- removal efficiencies
- capital and operating & maintenance (O&M) costs
- timeline for commercialization
- balance of plant issues, etc.)

e. EEI estimated that ACI would be less expensive per pound of Hg removed than EPA has estimated. … Are EPA's Hg control technology cost assumptions reasonable? …EPA is seeking additional detailed data addressing the validity of the costs assumed for ACI.

d. …CATF assumed that ACI would be available in 2005 for all coal types, while Cinergy … assumed ACI would be available in 2005 [in most of its modeling scenarios. Yet the EPA assumed that it would not be available until after 2010.] What assumptions for ACI availability are most appropriate? Specifically, what data of availability for ACI technology is appropriate to consider in a modeling analysis, at what quantities, for what coal types, and why?"

The following are Part I of comments by Sorbent Technologies Corporation intended to assist in the fulfillment of this request. A Part II with Balance-of-plant effects data and other pertinent information for the EPA and public will also be eDocketed. Note that a third group of comments containing Confidential Business Information (CBI) are also being submitted through the EPA's separate channels for CBI and are intended to be read in concert with these non-confidential comments.

Respectfully submitted,

Sid Nelson Jr.
President
Sorbent Technologies Corporation
1664 E. Highland Rd.
Twinsburg, OH 44087
SNelsonJr@sorbenttechnologies.com
(330) 425-2354

The Current State of Activated Carbon Injection Technology

Summary

A revolution has occurred over the last year in power-plant mercury-control technology. Coal chlorine content has now been made irrelevant. Consequently, the currently-proposed mercury MACT structures, limits, and timetables need to be radically modified.

The EPA's current MACT proposals were based on a fundamental assumption that the low chlorine content of Western subbituminous coals and lignites severely limited the performance of mercury reduction technology with these fuels.

For the traditional MACT proposal, this resulted in:

- differing mercury emission floor limits subcategorized by coal rank.

For the cap-and-trade proposals, this resulted in:

- differing emission-allowance allocation adjustment-factors by coal rank.

Treating the different coal ranks differently because of their chlorine contents resulted in highly divergent standards which would lead to significant marketplace inequities and distortions:

	Proposed Emission Standards		Allocation
	Existing Plants	New Plants	Adjustment
Coal Type	(lb Hg/TBtus)	(10^{-6}lb Hg/MWh)	Factors
Lignite	9.2	62	3
Subbituminous	5.8	20	1.25
Bituminous	2.0	6	1

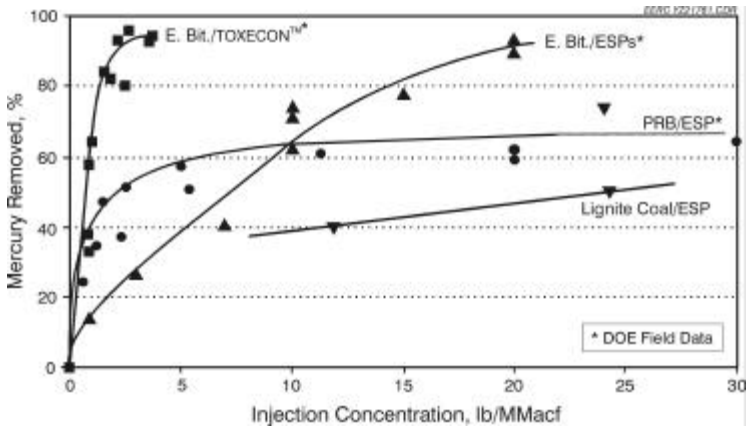
Fortunately, vast improvements in the performance and cost-effectiveness of activated carbon injection technology (ACI) for low-chlorine subbituminous coal and lignites have recently been conclusively demonstrated. Multiple DOE-co-sponsored full-scale retrofit demonstrations by different contractors of ACI with brominated carbons at plants burning low-chlorine fuels this past year consistently achieved mercury emission rates of less than 1.0 lb Hg/TBtus at very low costs. Moreover, these new brominated carbons are now commercially available for use by any plant. Consequently, the currently-proposed mercury MACT structures, limits, and timetables have to be radically modified to account for these developments.

At the very least, the MACT floor levels or adjustment factors for subbituminous coals and lignites now have to be lowered to the levels currently proposed for bituminous coals. More responsibly, subcategorized standards based in plant particulate-control equipment, rather than by coal rank, should be promulgated, with resulting MACT floor levels of 1.0 lb Hg/TBtu or below. If a cap-and-trade system is proposed instead, its 2010 cap should be in the range of 8 to 10 tons of mercury per year, not 34.

The attached comments on the results of this year's ACI demonstrations provide greater detail on these revolutionary developments.

Overall Mercury Reduction Results

Previously, at plants with just electrostatic precipitators (ESPs), the dominant industry configuration, maximum mercury reductions of only 50% to 70% were observed with lignites and subbituminous coals (e.g. PRB) at sorbent injection rates of 10 lb or more of powdered activated carbon (PAC) per million cubic feet of flue gas (lb/MMacf). See, for example, the graph below from the June 2003 Congressional testimony of Dr. Steven Benson of the University of North Dakota's Energy & Environment Research Center. High removal rates were previously observed with bituminous coal, but not with PRB and lignite.

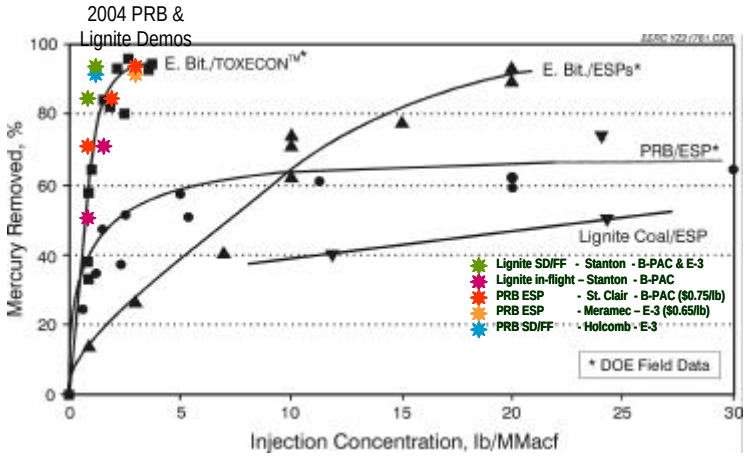


In 2004, the U.S. Department of Energy co-sponsored five full-scale ACI utility demonstrations, outlined below. The four with low-chlorine subbituminous coal and lignite used new, inexpensive, pre-brominated carbons, instead of plain carbon.

2004 Full-Scale DOE Activated Carbon Injection Trials

	Plant	Utility	Coal	Configuration	Prime Contractor
1	**St. Clair**	Detroit Edison	**Subbit. (85%)**	ESP	Sorbent Technologies
2	**Meramec**	Ameren	**Subbituminous**	ESP	ADA-ES/Alstom
3	**Holcomb**	Sunflower	**Subbituminous**	DFGD/FF	ADA-ES
4	**Stanton**	Great River Energy	**Lignite**	DFGD/FF	UND EERC
5	**Yates**	Southern Cos.	**Bituminous**	ESP	URS

The four 2004 ACI trials with the low-rank fuels, summarized in color below, demonstrated extremely high mercury reductions at very low costs with the new brominated carbon technology. In these trials, 90% to 95% mercury reductions at injection rates of 3 lb/MMacf or less were observed. The new brominated PACs that were used in 2004 are estimated to cost only a fraction more than current plain PACs.



Not only was greater than 90% mercury removal performance observed in each and every one of the low-chlorine coal trials, but net costs to achieve these levels with an ESP were estimated at **around $ 10,000 per pound of mercury removed, over 80% less** than DOE estimates of $50,000 to $70,000 /lb Hg removed only a short time ago.

Each of the trials had the same format: first with baseline testing, without ACI; then parametric testing, with short-term (ST) tests of a few hours each with different sorbents or different injection rates; and then a long-term (LT) continuous trial with one sorbent at one injection rate for a period of approximately one month.

A summary of the latest year's DOE ACI results that have been publicly released so far appears below.

Summary of 2004 Full-Scale DOE Activated Carbon Injection Trials

Plant	**Coal**	Configuration	Duration	**Avg.%Hg Removal**	Inj. Rate (lb/MMacf)	**Sorbent**	**Emissions (lb/TBtu)**	**$/lb Hg removed***
St. Clair	**Subbit. (85%)**	ESP	LT	**94%**	3	**B-PAC**	**0.3**	**$ 9,600**
Meramec	**Subbit.**	ESP	ST**	**93%**	3	**E-3**	**0.7****	**$ 8,400**
Holcomb	**Subbit.**	DFGD/FF	LT	**93%**	1.2	**E-3**	**0.8**	**$ 3,400**
Stanton	**Lignite**	DFGD/FF	ST	**95%**	1.5	**B-PAC**	**0.4**	**$ 3,500**
"	**"**	"	LT	70%***	1	**E-3**	**2.5**	**$ 2,600**
Yates	**Bituminous**	ESP	ST****	**<50%**	5.2	FGD	**1.6**	**$20,000**

* Sorbent consumption costs, with B-PAC=$0.75/lb, E-3=$0.65/lb, & FGD=$0.50/lb and a flue-gas mercury concentration at sorbent injection of 8.5, 6, and 7 μg/Nm^3 for the average lignite, subbituminous, and bituminous plant respectively. See details in further sections.

** Hg emissions data is for the final four weeks of the long-term (LT) run. Other LT run data has not yet been publicly released.

*** This relatively low removal rate was intentionally targeted by the project team in the LT run.

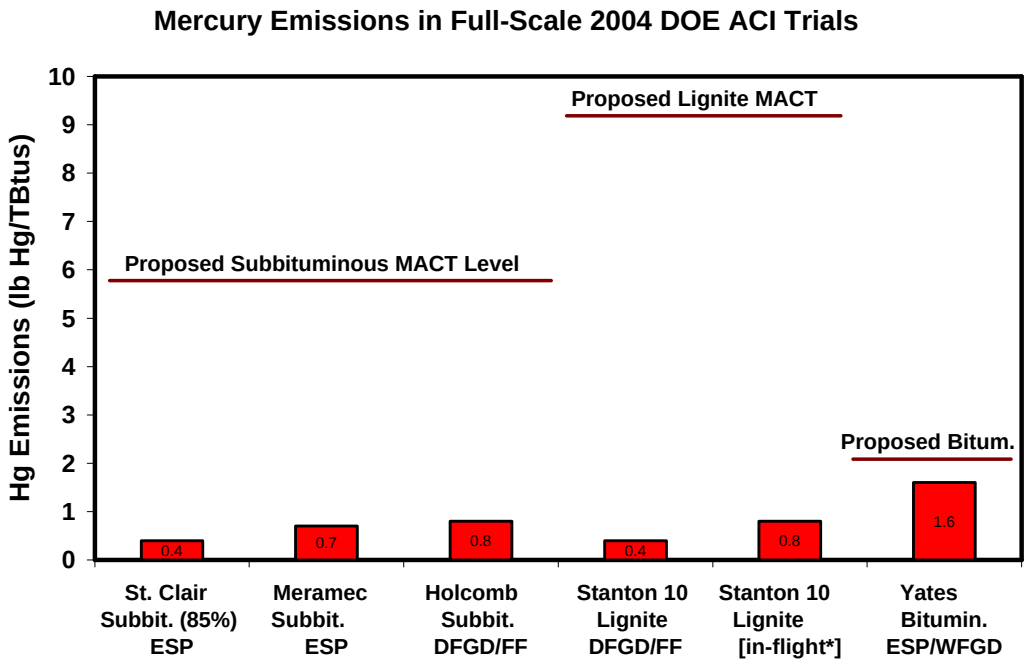
**** Long-term data is not yet available. Brominated carbon was not tested at this plant.

The two brominated PACs that were used in these trials are called "B-PAC™" and "E-3," respectively from Sorbent Technologies Corporation, which pioneered the new technology, and Norit Americas, the largest U.S. PAC manufacturer. Sorbent Technologies performed the St. Clair plant trial and will provide extensive details in these remarks.

Note that those running the one new bituminous coal ACI trial in 2004, at Southern Companies' Plant Yates, explicitly chose not to include a brominated PAC in their testing matrix (despite offers of free B-PAC), so data with the latest technology is not available for comparative purposes for this plant and coal type. It may be reasonable to assume that brominated PAC would perform similarly at this plant, with its low-chlorine and low-sulfur fuel, as well.

The results achieved in this year's ACI trials are plotted below relative to the currently-proposed "Maximum Achievable Control Technology" limits under §112 for each coal rank. As can be seen, these old MACT levels, at least for subbituminous coal and lignites, are ridiculously high relative to the low emission levels that were demonstrated. In some cases, the "Maximum Achievable" rates are more than **ten times** or 1,000% higher than those actually demonstrated last year.

The national and state caps and plant-by-plant emission-allowance allocations in the cap-and-trade proposals are similarly about **500% too high** based on this most-recent year's worth of data.



Moreover, these demonstrations, like the many that have been performed previously, reinforce how quickly ACI has become "commercially available." Each of the full-scale trials could easily have been permanent installations with little added effort or expense. At least one of the new brominated carbons, B-PAC™, from Sorbent Technologies, is now commercially available for continuous supply to power plants with removal-rate guarantees. A second company, ADA-ES, has quoted on over 50 ACI systems for new installations. Consequently, the EPA's previous assumptions regarding the timing of ACI availability also need dramatic modification.

Individual Demonstration Details

St. Clair Plant – 100% Subbituminous Coal & 85% Subbituminous Blend

Following previous short-term testing at two earlier plants, Lausche and Cliffside, and slipstream testing at three more power plants, Sorbent Technologies Corporation tested its B-PAC™ brominated PAC mercury sorbent, at DTE Energy/Detroit Edison's St. Clair power plant in the late summer of 2004. The St. Clair plant has only a cold-side ESP for air pollution control and typically burns a blend of 85% PRB subbituminous coal/15% bituminous coal, occasionally switching to 100% subbituminous during periods of low power demand. Under baseline conditions, with no ACI, native Hg removal at the plant averaged about 30% with the 85% subbituminous bend and about 15% with 100% subbituminous coal.

B-PAC was injected into the ductwork leading to one of the plant's 80MW ESPs. See below left. All that was required was blowing the B-PAC through a 2-inch-ID pipe to a 6-hose splitter (below center) and then through six open-ended lances sticking through three holes in the ductwork (below right).

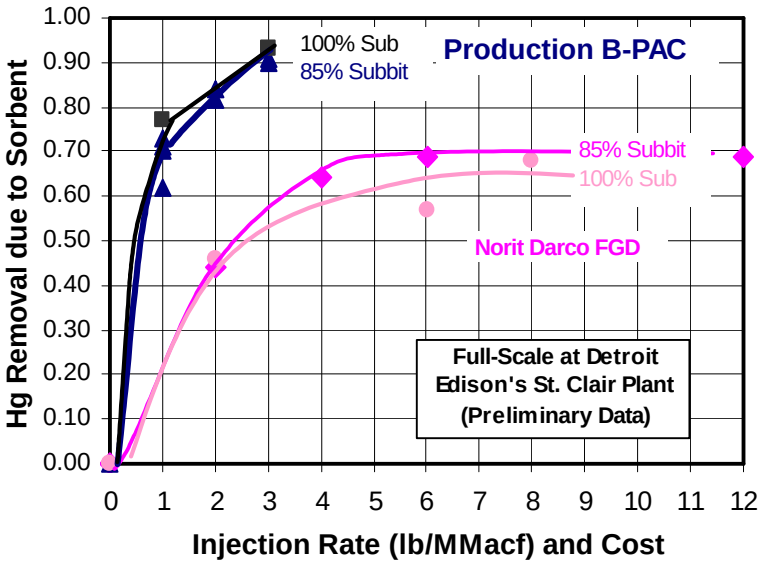
An independent, well-experienced project member, Western Kentucky University, analyzed the flue gas mercury concentrations at points before the sorbent injection and at the ESP exit using the latest generations of Baldwin inertial separators and PS Analytical Hg continuous mercury monitors (CMMs).

A summary of the project results appears below.

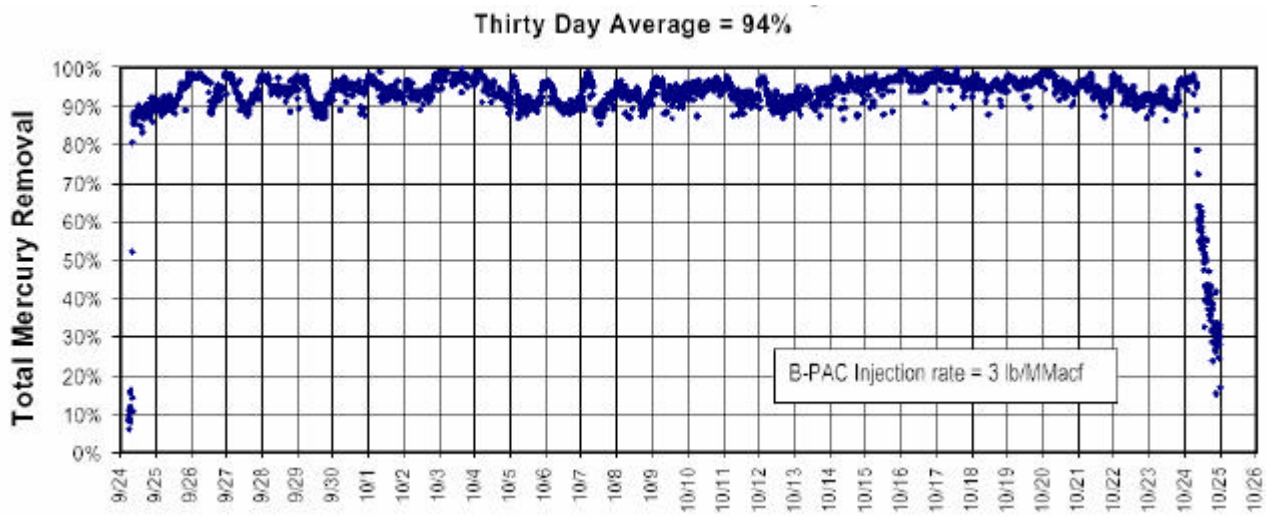
Plant	Coal	Config.	Duration	%Removal due to ACI	Inj. Rate (lb/MMacf)	Sorbent	Emissions (lb/TBtu)	$/lb Hg removed*
St. Clair	**Subbit. (85%)**	ESP	LT*	**94%**	3	**B-PAC**	**0.3**	$ 9,600
St. Clair	**Subbit.(100%)**	ESP	ST	**96%**	3	**B-PAC**	**0.3**	$ 9,400
St. Clair	**Subbit. (85%)**	ESP	ST	**70%**	1	**B-PAC**	**1.5**	$ 4,300
St. Clair	**Subbit.(100%)**	ESP	ST	**75%**	1	**B-PAC**	**1.3**	$ 4,000

* LT Hg removal rates are always total removal rates, which include native removal at the plant, because in LT runs, with changing plant operations, there is no way to gauge, and separate out, native removal.

When plain PAC (Norit Darco FGD®) was tested at St. Clair, mercury removal due to the sorbent with both 100% subbituminous coal and the 85% subbituminous/15% bituminous blend topped out at about 70% PAC consumption rates of 6 to 8 lb/MMacf. B-PAC on the other hand, achieved 75% removal with only 1 lb/MMacf on the pure subbituminous coal and 70% with the blend. Total Hg removal at 1 lb/MMacf including the native removal from unburned carbon in the fly ash, was around 80% for both fuels. Moreover, at 3lb/MMacf the B-PAC itself removed about 94% of the Hg with the pure PRB coal and about 90% with the blend. See the graphs and data below. Note that with brominated-carbon performance is actually higher with 100% subbituminous coal than it is when bituminous coal is blended in. This is due to the added sulfur and chlorine of bituminous coals, which compete for mercury-reactive sites on the sorbents.



B-PAC was then injected continuously at 3 lb/MMacf for 30 days as the plant went through its normal operations, typically ramping up to full power in the mornings and down to lower output at night, usually burning the 85/15 blend. Over the course of the month, total mercury removal averaged 94%. See below.

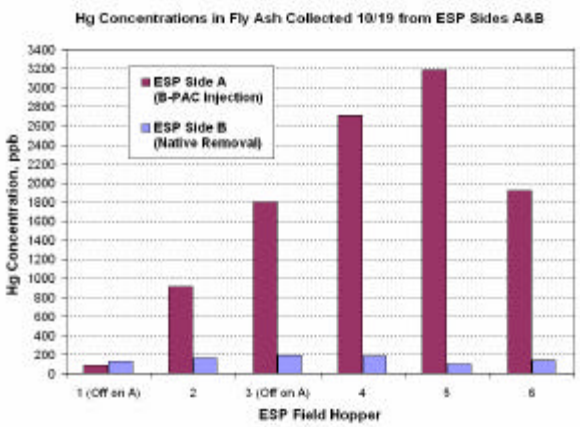


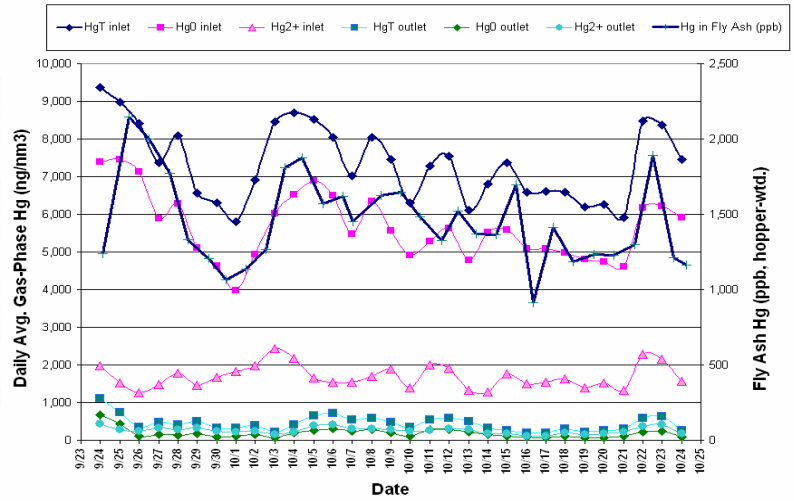
While instantaneous mercury removal rates fluctuated between about 88% and 98%, depending on the plant load and the coals burned, overall B-PAC performance was very consistent. Average weekly mercury removal rates were:

1st week: 94%; 2nd week: 94%; 3rd week: 94%; 4th week: 94%.

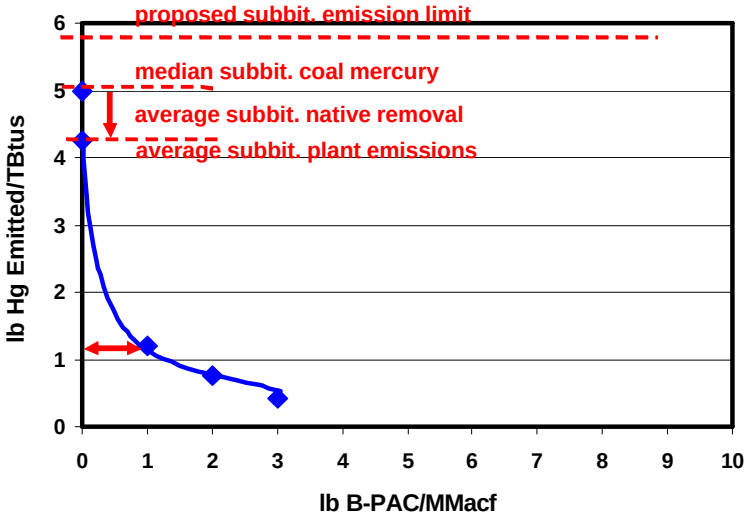
Many fly ash samples were taken from the ESP hoppers at St. Clair to confirm the mercury removal performance. The mercury captured from the flue gas ends up sequestered in the sorbents in the ash. On the left, below, are the Hg concentrations of the fly ash from the hoppers of the ESP on the A side, where B-PAC was injected, and on the B-side ESP, without sorbent.

The mercury mass balances were excellent. The darkest lines above on the right, for example, compare the average daily gas-phase Hg measured before the injection system (left axis) and the average daily hopper-weighted Hg concentrations of the resulting fly ash (right axis) for every day during the 30-day trial. Because essentially all of the mercury was captured by the B-PAC, the two curves trended tightly together for the whole month.





The results at St. Clair with subbituminous coal can be translated into MACT floor units of lb-Hg/TBtu-emitted. The median subbituminous coal being delivered to U.S. power plants, based on over 8,000 plant samples in the 1999 ICR, contains 5.0 lb Hg/TBtu. If the native Hg removal at subbituminous plants is only 15%, then the average emissions would be 4.25 lb/TBtu without any ACI. Based on the removal rates due to B-PAC observed at St. Clair, it would only take slightly over 1 lb/MMacf of B-PAC injection to bring the average subbituminous plant with just a cold-side ESP down to a 1 lb Hg/TBtu MACT floor level. B-PAC injection at 3 lb/MMacf would, on average, achieve 0.4 lb Hg/TBtu. Note that the currently-proposed limit for such plants is 5.8 lb Hg/TBtu's, more than 10 times the long-term the emissions observed at St. Clair.



How much did these levels of control at St. Clair cost?

Brominated PAC doesnot cost much more than commercial coal-derived PACs. Norit Darco FGD®, for example, a common yardstick PAC, costs about $0.50/lb, delivered, today. In large-scale production, B-PAC™ has been estimated to cost utilities approximately $0.75/lb. Norit's brominated E-3, has been quoted at $0.65/lb.

At an injection (consumption) rate of 1 lb/MMacf, the B-PAC removed 70% to 75% of the flue gas Hg, and counting the native removal from unburned fly ash, yielding 80% removal in all. There was about 6 µg/Nm^3 (at the actual O_2 level) of gas-phase Hg present at the sorbent injection point, a representative, average level for subbituminous coals. Total sorbent costs can be calculated thus:

$$\left(\frac{1\,lb\ sorbent}{1{,}000{,}000\,acf}\right)\left(\frac{Nm3}{(72\%)\,6mg\ Hg}\right)\left(\frac{\$0.75}{lb\ sorbent}\right)\left(\frac{1.5\ acf\ @\ 300F}{1\ scf}\right)\left(\frac{35.3scf}{Nm^3}\right)\left(\frac{10^9\ mgHg}{2.2\,lb\ Hg\ removed}\right)=\$4{,}000\,/\,lbHg\ removed.$$

At an injection rate of 3 lb/MMacf, the B-PAC removed 90% to 93% of the flue gas Hg, and counting the native removal from unburned fly ash, yielding 94% removal in all. For this level of mercury emissions of about one-third the previous rate, total sorbent costs were:

$$\left(\frac{3\,lb\ sorbent}{1{,}000{,}000\,acf}\right)\left(\frac{Nm3}{(91\%)\,6mg\ Hg}\right)\left(\frac{\$0.75}{lb\ sorbent}\right)\left(\frac{1.5\ acf\ @\ 300F}{1\ scf}\right)\left(\frac{35.3scf}{Nm^3}\right)\left(\frac{10^9\ mgHg}{2.2\,lb\ Hg\ removed}\right)=\$10{,}000\,/\,lbHg\ removed.$$

Other O&M and the amortization of the cost of the PAC silo and other sparse equipment needed add less than 10% to these costs, based on the EPA Cost Model methodology.

Note that these costs for about 80% and 95% mercury removal respectively with just a cold-side ESP, are **about 90% and 85% less** than recent DOE average estimates of $60,000/lb Hg removed.

Cost calculations can also be made on $/kW and $/kWh bases according to the EPA's current CUE Cost Model, as modified for mercury control, version 01/01/03 by Andover Technology Partners, originally based on EPRI's Technical Assessment Guide:

Hg Removal	PM Control	B-PAC Rate (lb/MMacf)	Total Capital (incl. CMMs)	O&M
70%	CS-ESP	1.5	$1.50/kW	$0.30/kWh
70%	FF	0.5	$0.77/kW	$0.11/kWh

Stanton Station - Lignite

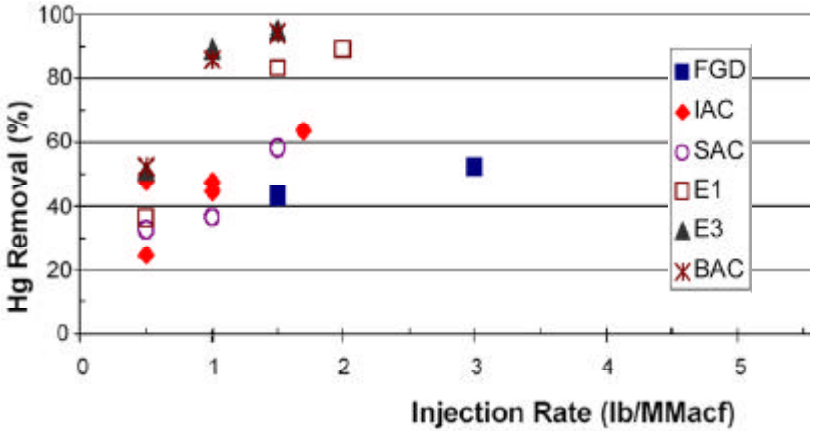
In the spring of 2004, the EERC of the University of North Dakota, along with subcontractor Apogee Scientific, ran ACI trials with both Sorbent Technologies' B-PAC sorbents and Norit's E-3 carbon, as well as other advanced carbons at Great River Energy's Stanton Station Boiler No. 10.

This unit burns lignite and has a spray dryer and fabric filter for air pollution control. This was thought to be one of the toughest configurations for mercury removal, because the spray dryer removes what little halogens there are in the flue gas to help powdered activated carbons remove gas-phase Hg, which is predominantly in the elemental form with lignites. The sorbents were injected before the spray dryer and gas-phase Hg measurements were taken both after the fabric filter and immediately before the fabric filter. The latter measurement provided a proxy for similar testing on an ESP, as all that was available was a few seconds of sorbent residence time "in-flight."

Because this plant had a fabric filter, which allows for high gas/sorbent contact time and excellent mass transfer, the injection rates need for high removal are significantly lower than for an ESP. Note that a high fraction of lignite boilers already have fabric filters for particulate control, 30%, which makes mercury control significantly easier and less expensive. Only 7% of bituminous boilers have fabric filters.

Mercury removal results with the fabric filter are presented below.

Figure 3. Results from Full-Scale Parametric Testing of Sorbents



From: Machalek, T., *et al.,* "Full-Scale Activated Carbon Injection for Mercury Control in Flue Gas Derived from North Dakota Lignite," Power Plant MegaSymposium, Wash., DC, Sept. 1, 2004.

Note that the two brominated PACs, BAC [sic] and E-3 performed extremely well, achieving 95% Hg removal at an injection rate of only 1.5 lb/MMacf on this supposedly difficult coal and plant situation. More specific parametric run information, including emissions per trillion Btus and cost-effectiveness results, follow.

Plant	Coal	Configuration	Duration	%Removal due to ACI	Inj. Rate (lb/MMacf)	Sorbent	Emissions (lb/TBtu)	$/lb Hg removed
Stanton	**Lignite**	DFGD/FF	ST	**95%**	1.5	**B-PAC**	**0.4**	**$ 3,500**
Stanton	**Lignite**	DFGD/FF	ST	**95%**	1.5	**E-3**	**0.4**	**$ 3,000**
Stanton	**Lignite**	DFGD/FF	LT	70%*	1	**E-3**	**2.5**	**$ 2,600**
Stanton	**Lignite**	in-flight [~ESP]	ST	**70%**	1.5	**B-PAC**	**2.5**	**$ 5,000**
Stanton	**Lignite**	in-flight [~ESP]	ST	**90%****	3	**B-PAC**	**0.8****	**$ 7,000****

* Because of the easy standards in the proposed regulations for lignite, this relatively low removal rate was intentionally targeted by the project team in the LT run.

** Extrapolated values.

Perhaps more importantly, the B-PAC also performed excellently in the in-flight-only mode across the spray dryer, simulating conditions with an ESP. Unfortunately, the highest B-PAC injection rate that was tested was only 1.5 lb/MMacf, where 70% Hg removal was observed with this typical lignite fuel. See the graph below on the left. If the in-flight results are plotted on a typical 1-e-x fashion, however, a good-fit extrapolation to 80% removal at 2 lb and 90% removal at 3lb/MMacf is obtained. See below on the right. The resulting Hg emissions and cost-effectiveness values for the 90% figure are calculated above.

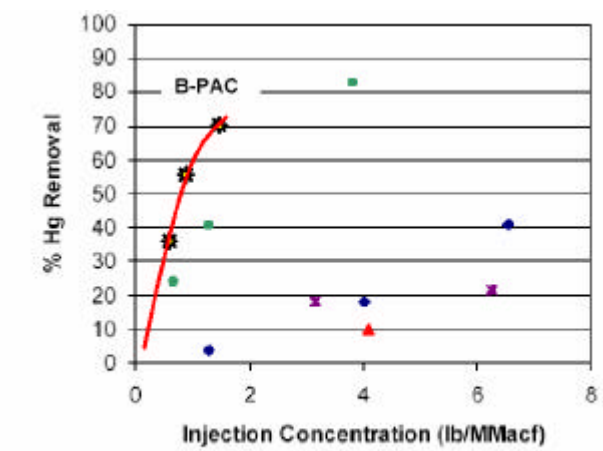
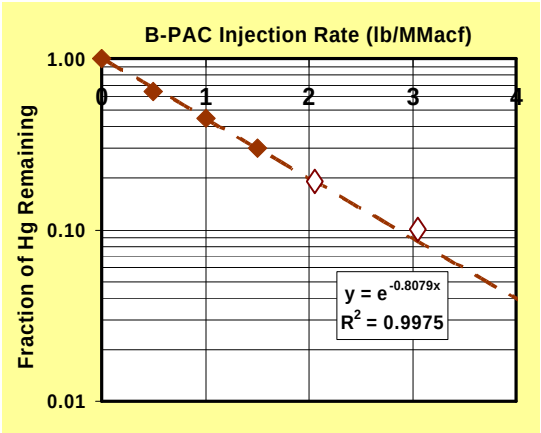


Figure 3. Mercury removal measured across the spray dryer at Star Station during parametric testing.



B-PAC in-flight data superimposed on: Sjostrom, S., et al., "Full-Scale Evaluation of Mercury Control at Great River Energy's Stanton Generating Station Using Injected Sorbents and a Spray Dryer/Baghouse," Air Quality III, Sept. 2002.

Previous B-PAC Testing at Power Plants

Cliffside and Stanton 10 are not the first full-scale tests of B-PAC. Earlier large-scale tests took place at the Lausche Plant, with high-sulfur bituminous coal and a cold-side ESP and at Duke Energy's Cliffside Plant, both in 2003 and 2004, with a low-sulfur bituminous coal and a hot-side ESP. These short-term Cliffside tests have been the only successful ACI trials anywhere with hot-side ESPs.

In all, the injection of brominated powdered activated carbon (B-PAC™) into power-plant flue gases for mercury removal has now been tested at seven different power plants, some of these were in slipstream tests. In all, these plants have burned bituminous, subbituminous, lignite coals, and blends and include testing with cold-side ESPs, hot-side ESPs, spray dryers, and fabric filters. Mercury-removal performance at these sites has varied between 70% and 98% at sorbent consumption costs of approximately $2,000 to $20,000 per-lb-of-mercury-removed. So far, B-PAC™ has consistently demonstrated high mercury removal rates at relatively low injection levels across a wide variety of coals and configurations. See the table below.

B-PAC Results to Date Indicate Applicability Across All Coals and Plant Configurations

Coal	PM Unit	Hg Removal	@lb/MMacf	@ Plant	Scale	Data
Bitum. Low-S	FF	**94%**	**0.5**	Valley	Slipstream	Apogee
Bitum. High-S	CS-ESP	**70%**	**4.0**	Lausche	Full-Scale	SorbTech
Bitum. Low-S	HS-ESP	**~70%***	**5.0**	Cliffside	Full-Scale	SorbTech
Subbitum.Blend	CS-ESP	**90%**	**3.0**	St. Clair	Full-Scale	SorbTech
Subbituminous	CS-ESP	**90+%**	**3.0**	St. Clair	Full-Scale	SorbTech
Subbituminous	CS-ESP	**89%**	**4.9**	Pleasant Prairie	Slipstream	Apogee
Subbituminous	FF	**87%**	**0.5**	Pleasant Prairie	Slipstream	Apogee
Subbituminous	SD/FF	**82%****	**<1.8**	Holcomb	Slipstream	ADA-ES
Lignite	SD/FF	**95%**	**1.5**	Stanton 10	Full-Scale	EERC
Lignite	CS-ESP+	**70%*****	**1.5**	Stanton 10	Full-Scale	EERC

* under moderate-load conditions.
** on-fabric removal only, with no in-flight opportunity and the effective "injection rate" could have been significantly lower.
*** actually the in-flight Hg removal across the spray dryer, with an injection rate of only 1.5 lb/MMacf.

More detail can be found in the following three papers:

1. S. Nelson Jr., R. Landreth, Q. Zhou, & J. Miller, "Accumulated Power-Plant Mercury-Removal Experience with Brominated PAC Injection," Combined Power Plant Air Pollutant Control Mega Symposium Washington, DC, Aug. 30, 2004.

2. M. McCoy, W. Rogers, R. Landreth, & L. Brickett, "Full-Scale Mercury Sorbent Injection Testing at DTE Energy's St. Clair Station," Combined Power Plant Air Pollutant Control Mega Symposium, Washington, DC, Aug. 30, 2004.

3. S. Nelson Jr., R. Landreth, Q. Zhou, & J. Miller, "Mercury Sorbent Injection Test Results at the Lausche Plant," Combined Power Plant Air Pollutant Control Mega Symposium Washington, DC, May, 2003.

Meramec Plant – Subbituminous Coal

In the fall of 2004, ADA-ES and Alstom similarly successfully demonstrated ACI with Norit's new brominated E-3 carbon at Ameren's Meramec Plant, which has just a cold-side ESP and burns subbituminous coal. B-PAC was not tested. The results that are publicly available so far are below.

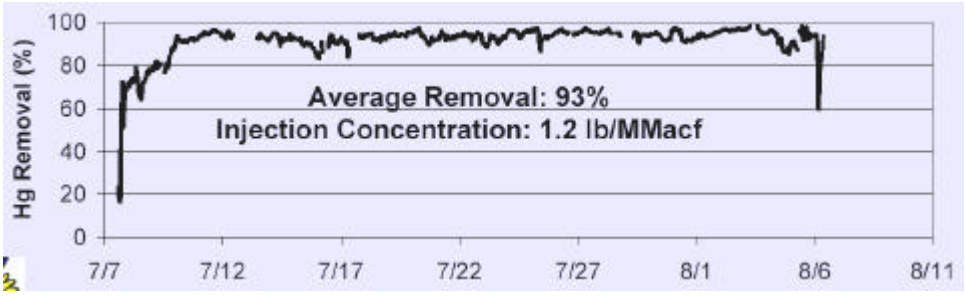
Plant	Coal	Configuration	Duration	Avg.%Hg Removal	Inj. Rate (lb/MMacf)	Sorbent	Emissions (lb/TBtu)	$/lb Hg removed
Meramec	**Subbit.**	ESP	LT**	**NA**	NA	**E-3**	**0.7****	NA
Meramec	**Subbit.**	ESP	ST**	**93%**	3	**E-3**	**NA**	**$ 8,400**

See: Starns, T., "Full-Scale Evaluation of Mercury Control by Injecting Activated Carbon Upstream of a Spray Dryer and Fabric Filter," POWER-GEN Conference, November 30, 2004, DOE DE-FC26-03NT41986. For greater detail, see also ADA-ES's upcoming January presentation at the Electrical Utility Environmental Conference and future DOE Quarterly reports.

Holcomb Plant – Subbituminous Coal

In the summer of 2004, ADA-ES similarly successfully demonstrated ACI with Norit's new brominated E-3 carbon at Sunflower Electric's Holcomb Plant in Kansas, which has a spray dryers and fabric filters and burns subbituminous coal. B-PAC was not tested. The results that are publicly available so far are below.

Plant	Coal	Configuration	Duration	Avg.%Hg Removal	Inj. Rate (lb/MMacf)	Sorbent	Emissions (lb/TBtu)	$/lb Hg removed
Holcomb	**Subbit.**	DFGD/FF	LT	**93%**	1.2	**E-3**	**0.8**	**$ 3,400**



From: Starns, T., above. See also: S. Sjostrom, "Full-Scale Evaluation of Mercury Control by Injecting Activated Carbon Upstream of a Spray Dryer and Fabric Filter," Combined Air Pollutant Control "Mega" Symposium, Washington DC, August 2004.

Plant Yates – Bituminous Coal

In the spring of 2004, URS and EPRI carried out ACI trials on Boilers ! & 2 of Southern Companies/Georgia Power's Plant Yates in Georgia, which has small cold-side ESPs and burns low-sulfur bituminous coal. No brominated PACs were not tested. A long-term run was scheduled for late 2004, but may not have run yet.

Plant	Coal	Configuration	Duration	Avg.%Hg Removal	Inj. Rate (lb/MMacf)	Sorbent	Emissions (lb/TBtu)	$/lb Hg removed*
Yates	**Bituminous**	ESP	ST	**<50%**	5.2	FGD	**1.6**	**$ 20,000**
Yates	**Bituminous**	ESP	LT	**NA**	NA	NA	**NA**	NA

* The basis for the cost-effectiveness calculation was 45% removal at 5.2 lb/MMacf with 7 μg/Nm^3 actual Hg at injection.

For more information, see: K. Dombrowski, "Sorbent Injection for Mercury Control Upstream of Small-SCA ESPs," Combined Air Pollutant Control "Mega" Symposium, Washington DC, August 2004.

Current Utility Mercury-Control Costs

Capital Costs

The capital cost of activated carbon injection (ACI) systems is very low. Unlike the gigantic, elaborate, and expensive control technologies required for SO_2 and NOx control, very little new capital equipment is required. All that is needed is a silo to store the sorbent, a weigh-feeder, a small blower to transport the sorbent, a pipes to the ductwork, and open-ended lances to distribute the sorbent into the power-plant flue gas ahead of its existing particulate collector. Such simple equipment has been around for almost one hundred years.

Coupled with a control system and continuous mercury monitors, the installed cost of such a sorbent injection systems is only about 1% that of other types of utility pollution control:

Retrofitted Control	Cost/KW
SO_2 scrubbers	$ 200
NOx SCR catalyst	$ 120
Toxecon Fabric Filter	$ 60
Hg ACI	**<$ 2**

For perspective, see the relative sizes of the equipment required for various air pollution operations below. The large spray dryer adsorbent (SDA) is a dry SO2 scrubber. The fabric filter (FF) would be required for a Toxecon I process. For ACI into existing equipment, however, all that is required is the PAC silo (Hg).

Photograph adapted from: Sjostrom, S., "Carbon Injection at Four Facilities," DOE/NETL Mercury Control Program Review, July 14, 2004.

Note that as brominated PAC decreases sorbent usage by about 80% relative to plain PAC, all of this equipment is proportionally smaller and less expensive.

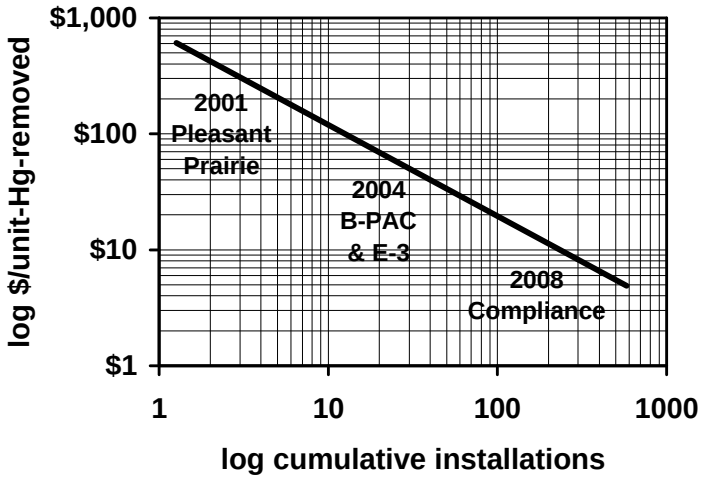
Unlike SO_2 and NOx control systems, ACI mercury-control systems require practically no trade labor for installation and are installed in a matter of days, not years. It will not take seven years, from 2007 to 2013 or 2015 as in the EPA analysis, to widely install such simple systems. With certainty of regulatory requirements, ACI silos and injection lances can easily be installed at all U.S. coal-fired power plants by 2010.

Operations & Maintenance Costs

As previously explained, the primary costs with ACI are operational costs – the cost of the sorbent, which is directly proportional to a plant's flue gas flue, or essentially, its power generation – and capital cost amortization, and maintenance costs, are effectively a rounding error. There are little advantages in economies of scale. Note that this means that emissions trading will have little beneficial effect, as there is little difference in the marginal cost of control between units. But note that this also means that smaller plants or peaking plants are not significantly disadvantaged with uniform MACT-like compliance. Trading has even less utility when required mercury removal rates are high, as they should be with mercury, because there is little opportunity for overcompliance to create allowances to trade.

The NODA asked for comment on a 2.5%-per year cost reduction assumption for ACI.

Clearly this figure is far too low. Because of the relatively nescient, unexplored nature of ACI technology, a steeply-sloped classic "experience curve" is already being observed. See the diagram below. As industry experience has moved through a factor of 10 in trials from Gaston and Pleasant Prairie, mercury control costs have similarly decreased by an order of magnitude. This will surely continue with ACI over the next few years as industry learns how to cut compliance costs further.



The EPA has used much steeper learning curves before. See, for example, the Notice of proposed rulemaking for the "Control of Emissions of Air Pollution from 2004 and Later Model Year Heavy-Duty Highway Engines and Vehicles," [Federal Register: October 29, 1999 (Volume 64, Number 209) Page 58471-58566]:

"For the long term, EPA has identified various factors that would cause cost impacts to decrease over time. First, the analysis incorporates the expectation that manufacturers will apply ongoing research to making emission controls more effective and less costly over time. … Second, research in the costs of manufacturing has consistently shown that as manufacturers gain experience in production, they are able to apply innovations [], use lower cost materials, and reduce the number or complexity of component parts. The analysis incorporates the effects of this learning curve by projecting that the variable costs of producing the low-emitting engines decreases by 20 percent starting with the third year of production [or ~6% decrease per year]… Considering this change, we believe the learning curve concept is appropriate for this rulemaking." [See also, e.g., R. Duke & D. Kammen "The Economics of Energy Market Transformation Programs," The Energy Journal, 20(4):15-64.]

For ACI, the annual cost-reduction rate should be significantly higher than the above-referenced 6% per year. If a log-log model is not used, then a 10% per year slope appears more reasonable.

Commercial Availability

ACI Hardware

ACI has been commercially used to reduce mercury emissions from municipal solid waste incinerators for over twenty years. The systems are simple and easily commercially available. Already full-scale ACI systems have been installed on at least 20 U.S. coal-fired boilers in temporary ACI trials. See the list below. About another 20 systems will be installed over the next 2 years in additional full-scale ACI demonstrations. Little distinguishes these temporary full-scale installations from permanent ACI systems except, perhaps, permanent piping rather than flexible hoses, more-permanent foundations, or larger PAC storage/silos.

Power Plant	Coal	Injected into:	Company
		Already Completed:	
Gaston (I)	Bitum.	FF	ADA-ES
Pleasant Prairie	Subbit	CS-ESP	ADA-ES
Brayton Point	Bitum	CS-ESP	ADA-ES
Abbott	Bitum	CS-ESP	Apogee
Salem Harbor	Bitum	CS-ESP	ADA-ES
Stanton 10 (I)	Lignite	SD/FF	Apogee
Laskin	Lignite	Particulate Scrubber	Apogee
Lausche	Bitum	CS-ESP	Sorbent Tech.
Stanton 1 (I)	Lignite	CS-ESP	ADA-ES?
Coal Creek	Lignite	CS-ESP (ToxII)	ADA-ES
Cliffside (I)	Bitum	HS-ESP	Sorbent Tech.
Stanton 1 (II)	Subbit.	CS-ESP	Apogee?
Stanton 10 (II)	Lignite	SD/FF	EERC
Gaston (Year-long)	Bitum.	FF	ADA-ES
Arapahoe	Subbit	FF	ADA Tech
Holcomb	Subbit	SD/FF	ADA-ES
Leland Olds (I)	Lignite	CS-ESP	EERC
St. Clair	Subbit	CS-ESP	Sorbent Tech.
Yates 1 (I)	Bitum	CS-ESP	URS
Yates 2	Bitum	CS-ESP	URS
Stanton 1	Lignite	CS-ESP	Apogee
Cliffside (II)	Bitum	HS-ESP	Sorbent Tech.
Meramec	Subbit	CS-ESP	ADA-ES/Alstom
		Early 2005 (DOE):	
Yates 1 (II)	Bitum.	CS-ESP	URS
Buck	Bitum	HS-ESP	Sorbent Tech.
Miami Fort	Bitum	CS-ESP	ADA Tech
Conesville	Bitum	CS-ESP	ADA-ES/Alstom
Antelope Valley	Lignite	SD/FF	EERC
		After Mid-2005:	
Lee 1	Bitum.	CS-ESP	Sorbent Tech.
Crawford	Subbit.	CS-ESP	Sorbent Tech.
Will County	Subbit.	HS-ESP	Sorbent Tech.
Lee 2	Bitum.	HS-ESP	Sorbent Tech.
John Sevier	Bitum.	CS-ESP	GE-EER
Dave Johnson	Subbit.	CS-ESP	Alstom
Leland Olds (II)	Lignite	CS-ESP	Alstom
Portland	Bitum.	CS-ESP	Alstom
Presque Isle	Subbit.	FF	ADA-ES
MidAmerican I	Bitum.	HS-ESP	ADA-ES
MidAmerican II	Subbit.	HS-ESP	ADA-ES
Entergy	Subbit.	CS-ESP (ToxII)	ADA-ES
AEP	Bitum.	CS-ESP (ToxII)	ADA-ES
Big Brown	Lignite	FF	EERC

Note: (I) or (II) denote separate trials with separate set-ups.
There may have been additional trials without public knowledge (e.g. EPRI).

Brominated PAC Supply

B-PAC™ brominated ACI is now commercially-available. Sorbent Technologies Corporation is both willing and able to continuously supply B-PAC ACI to any U.S. power plant that desires to reduce its mercury emissions. Once the capacity of the company's existing manufacturing plant is spoken for, additional capacity can be supplied within 6 months.

Sorbent Technologies Corporation's first B-PAC™ mercury-sorbent manufacturing plant production-line became available for continuous power plant use in September of 2004. See the photograph below. Consequentlu, all EPA utility mercury policymaking deliberations, including modeling analyses, should consider brominated ACI technology available for full-scale retrofit implementation with every coal type, on every coal-fired utility boiler, in 2005.

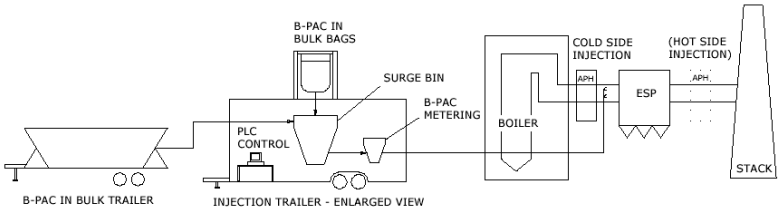
Moreover, brominated carbon supply can be easily expanded, without any necessary technological advances (although these are bound to occur), with only six-months notice. (Indeed, it is physically possible to build enough brominated-carbon production capacity in twelve to eighteen months to fully supply every unscrubbed coal-fired power plant boiler in America, although for economic efficiency reasons a timetable of three to five years would be more appropriate.)

The supply of ACI hardware and plain powdered activated carbons (PAC) have **already** been fully commercially-available from numerous vendors (e.g. ADA-ES and Norit Americas) for some time. ADA-ES has quoted systems for at least 50 individual utility boilers, mostly for new power plants, and has at least one firm order.

Consequently, the assumption employed by CATF, and mostly employed by Cinergy, of ACI supply availability by 2005 is entirely correct and appropriate and should be used by the EPA in all of its utility mercury policymaking deliberations, including modeling analyses.

Guarantees

Sorbent Technologies will offer mercury removal-rate performance guarantees on its B-PAC ACI installations and sorbent supply. These are not nearly as risky as performance guarantees on traditional air pollution equipment which takes years to build and which can be phenomenally expensive to rebuild or replace power from. This is because the performance guarantees will be based on actual full-scale trials on the actual unit with the actual coals to be used. Such trials can be inexpensively performed before long-term sorbent supply contracts are signed. See a mobile injection trailer of Sorbent Technologies that can perform trials on boiler streams to 500 MW.



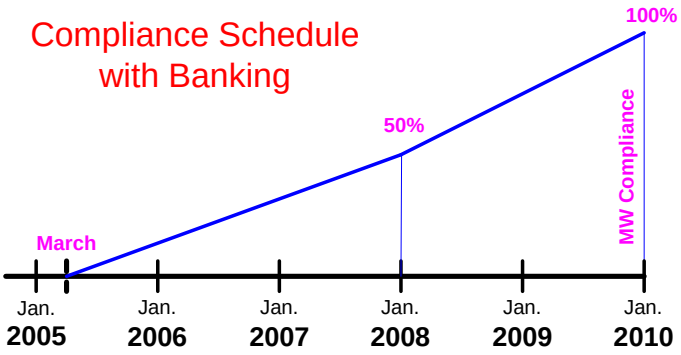
The business model is similar to that of Fuel Tech N.V. and their SNCR urea injection installations. All that is effectively being warranted is B-PAC sorbent quality. And if it takes 3.2 lb/MMacf rather than 3.0 lb/MMacf, to reach 90% Hg removal, Sorbent Technologies simply provides the extra B-PAC at no charge.

Nationwide ACI with Brominated Carbon

Sorbent Technologies is both willing, and, it believes, able, to alone supply the entire U.S. fleet by 2010 should it prove necessary. In reality, however, Norit Americas/ADA-ES and a number of other firms will likely similarly compete in supplying this marketplace.

How long would it take to outfit and supply the whole U.S.?

Using brominated PAC technology, full industry compliance with low emission-rate standards is possible by, on average, January 1, 2008. With compliance banking, where earlier compliance at some plants can be traded off for later compliance at other plants, this would extend the full industry response time out to 2010. This would allow five long years for complete compliance, leaving more time for the more-difficult plant situations.



With ACI retrofitted using only existing particulate collectors, as envisioned here, all that is required is a bin to hold the brominated PAC, a feeder, a blower, piping, and open-ended lances sticking into the ductwork. Permanent ACI systems can be installed at power plants in a couple of weeks, at most, with minimal downtime, if any, and essentially no needs for scarce boilermakers or special trade labor.

How much brominated PAC would be required by 2010 if every unscrubbed power plant in the U.S. installed ACI to comply with a strict mercury-removal standard?

To calculate this, we can make the following assumptions:

a.) 200 Gigawatts of eventual B-PAC use (i.e. assumes scrubbed boilers get enough Hg out on their own);
b.) 0.70 capacity factor (the capacity factor of unscrubbed units is lower than ~0.85 for scrubbed units);
c.) 3500 acfm/MW (a weighted-average over different coalt ypes and plant loads); and
d.) 2 lb/MMacf = avg. B-PAC injection (consumption) rate (a weighted-average for ESPs & FFs required for high mercury removal, but considering non-control reductions (NCRs) of Hg as well).

Taken together:

$$\left(\frac{200{,}000\ MW}{}\right)\left(\frac{3500\ acf}{\min ute * MW}\right)\left(\frac{2\ lb\ B-PAC}{1{,}000{,}000\ acf}\right)\left(\frac{365*24*60\min utes*0.70}{year}\right)\left(\frac{Ton}{2000\ lb}\right)$$

= ~ <u>250,000 Tons of brominated PAC/year in 2010.</u>

If B-PAC costs $0.75/lb ($1500/T) and 250,000 TPY are required, the total industry mercury sorbent costs would only be only **<u>$375 million per year</u>**. (Even if it sold for $1.00/lb, total sorbent costs would be only $500 million/yr.)

Based on the EPA's own cost models, if other ACI O&M costs and the amortization of the minor utility capital equipment that is needed are added, total control costs to achieve the resulting high level of control would only add about 10% to sorbent costs, for total control-related costs of still only around $500 million per year. Together with the costless co-benefits achieved at scrubbed plants and with essentially costless non-control reductions (NCRs) obtained from coal distribution rationalization, this would effectively be the total national cost of achieving a net 80% or 90% utility mercury reduction.

<u>Note that this total cost is only15% to 20% of year-ago EPA estimates</u> and is on the order of 1% of the estimate costs of the Clean Air Interstate Rule.

FISH AND WILDLIFE SERVICE CLASS I REFUGES

- Highest Air Pollution Threat
- Moderate Air Pollution Threat
- Lowest Air Pollution Threat
- IMPROVE Site Only
- IMPROVE and NADP Site
- IMPROVE, NADP, and MDN Sites

Map produced by the Fish and Wildlife Air Quality Branch 7/11/1997

October 27, 2005
News Release 05-20
Brad Peck, Corporate Communications Officer
(509) 377-8247 / 727-2808

Port of Kalama chosen for Pacific Mountain Energy Center

RICHLAND, Wash. – Energy Northwest, a Joint Operating Agency of 19 public power organizations in Washington, has selected the Port of Kalama for a proposed 600 megawatt electric power complex slated for initial operation in 2012. The agency's Executive Board today approved a 50-year lease for an 80-acre industrial site owned by the Port.
The Integrated Gasification Combined Cycle (IGCC) complex will produce its own "synthesis gas" to fuel two, 300-megawatt power plants; one owned by public power interests, the other under private financing and ownership.

Construction of the complex, officially named the Pacific Mountain Energy Center, is expected to draw several hundred workers and create approximately 100 permanent family-wage jobs related to operating and maintaining the facilities. The site provides adequate space for future expansion of the operation to include research and testing of new environmentally friendly energy processes, potentially creating additional jobs.

Regulated emissions from the complex are expected to rival, and potentially outperform, those of a natural gas plant. The clean burning synthesis gas will be produced by gasifying – rather than burning – coal, pet coke (a by-product of crude oil refining), and potentially other carbon-based feed stocks in a fully enclosed process.

Estimated design and procurement cost for the power complex is approximately $1 billion. The costs include $35 million to make the facility compatible with potential future technologies to remove and capture carbon dioxide from the feed stocks. Efforts to develop those technologies include research into permanently sequestering captured carbon dioxide in underground geologic formations, rather than releasing it into the atmosphere.

The Kalama site was chosen from several sites studied in Western Washington. Project Manager Tom Krueger said Kalama was one of the few sites with all the necessary characteristics, including access to the Western Washington power grid. "Kalama offers easy access to the power grid in an area where our operations are likely to help alleviate, rather than compound, transmission congestion. That will be a significant benefit to BPA and the region. The site also offers an attractive mix of adequate space, appropriate industrial zoning, grid access, utility infrastructure, diverse feedstock transportation options, and highly professional Port management," he said.

Krueger said the next major step will likely be a 20-month process to secure environmental certification of the site by the Washington Energy Facility Site Evaluation Council (EFSEC). "The operating parameters of the proposed complex are all well within industry standards. We're looking forward to working closely with EFSEC to ensure they have a comprehensive view of the entire proposal and its expected benefits to the region," he said.

Dan Porter, head of Energy Northwest's generation project development business sector emphasized Energy Northwest's ongoing commitment to conservation and renewable energy sources. He points to the agency's 64-megawatt Nine Canyon Wind Project and White Bluffs Solar Station, both in Southeastern Washington, as evidence of that commitment. "We continue to believe that conservation

and renewable energy solutions must be at the core of our collective efforts to meet future demand for electricity. However, we don't see those avenues as sufficient, by themselves, to meet any reasonable estimates of future demand. In reality, we believe even modest economic growth in the region over the next decade – and the resulting increased demand for electricity – will far exceed the capacity of conservation and full-on development of renewable energy projects. That is particularly apparent when you consider that wind power projects, the most prominent new source of renewable energy today, only produces electricity about one-third of the time," he said.

Porter noted that, "As a public power agency we are charged with providing economically and environmentally responsible options to our members. This IGCC project has the potential to let us use abundant domestic petroleum coke, coal and potentially other solid fuel feed stocks to produce large quantities of electricity in an environmentally responsible manner. We also have a strong desire to help move carbon dioxide capture and sequestration from the research world to commercial application. Our alliance with the Big Sky Carbon Sequestration Partnership (BSCSP) – which is pursuing such a program – is further evidence of our simultaneous commitment to economics and the environment."

Krueger noted that some people have tried to label IGCC as nothing more than a coal plant under a new name. "They would be mistaken. The differences are real and meaningful. IGCC has been in use in Europe for decades where higher energy prices made gasification a reality much sooner than here in the U.S. The Europeans chose IGCC not as the lowest cost option, but as the realistic, environmentally responsible option for producing large quantities of base load power at affordable rates; exactly what we believe our region will need in the coming decade.

"Furthermore, with three large corporate partnerships emerging to offer standardized IGCC plant designs with guaranteed prices and performance warranties, the cost gap between gasification and coal plants has narrowed dramatically. At the same time, questions regarding IGCC readiness for commercial deployment have been answered by the entry of those and other major players in the market," he concluded.

Energy Northwest's IGCC power complex proposal includes features to ready the plant for additional equipment to allow the capture and sequestration of carbon dioxide, if and when it becomes commercially feasible. The BSCSP includes other partners, such as Pacific Northwest National Laboratory, who are actively working on processes to safely and permanently sequester captured carbon dioxide. One such initiative is focused on storing carbon dioxide in underground geologic formations common to the Pacific Northwest.

Energy Northwest is working with the Port of Kalama to ensure community members have full access to information about the proposed power complex, including potential environmental and economic benefits to the area. A public meeting is slated for Wednesday, November 9, at the Kalama Community Building, 126 North 2nd Street in Kalama, at 7 p.m. Energy Northwest and Port representatives will be on hand to share information and answer questions.

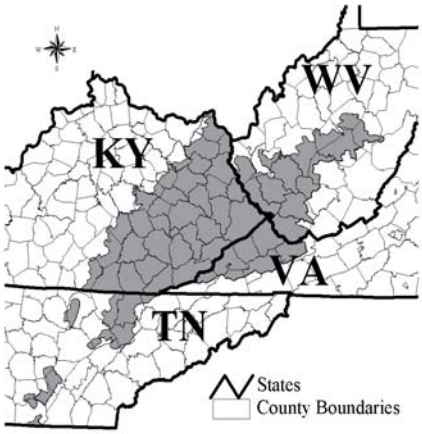
III. AFFECTED ENVIRONMENT AND CONSEQUENCES OF MTM/VF

This chapter includes a description of the physical setting, Appalachian lotic and lentic aquatic systems, relationship of surface mining and water quality, Appalachian forest communities, Appalachian microhabitats, threatened and endangered species, coal mining methods, mountaintop mine characteristics, excess spoil disposal, and economic information. Supporting information is provided in the appendices.

THESE ECOREGIONS ARE UNIQUE IN THE WORLD BECAUSE THEY COMBINE CHARACTERISTICALLY NORTHERN SPECIES WITH THEIR SOUTHERN COUNTERPARTS, AND THUS BOAST ENORMOUS RICHNESS AND DIVERSITY

A. DESCRIPTION OF THE STUDY AREA

Figure III.A-1
Study Area



The Appalachian Coalfield Region encompasses the coal-bearing areas of Pennsylvania, Ohio, Maryland, North Carolina, Georgia, West Virginia, Virginia, eastern Kentucky, Tennessee, and Alabama. The Bituminous Coal Basin lies within the Appalachian Plateau physiographic province, extends in a northeast to southwest direction along the Appalachian Mountains, and encompasses the most historically important coal mining areas of the Appalachian Coalfield Region (USDOI OSM, 1983). The study area is located within the Appalachian Coalfield Region of the Appalachian Plateau physiographic province and Bituminous Coal Basin. As the name implies, this region is known for the substantial deposits of coal that lie beneath the surface. Consistent with the EIS purpose, the study area includes watersheds where excess spoil fills, otherwise known as valley fills, have been constructed or are likely to be constructed in the future.

Physically, two factors must be coincident in order for mountaintop mining to occur and for excess spoil to be generated: steep terrain and sufficient contiguous coal reserves located close enough to the tops of mountains and ridges to justify large scale mining. In West Virginia, this close combination exists in the southern half of the state and is most frequently aligned with the existence of the Coalburg coal seam. In Kentucky, Virginia and Tennessee, this combination of factors also exists but delineation is not quite as simple because of more complex geology. The study area is approximately 12 million acres and extends over portions of West Virginia, Kentucky, Virginia, and Tennessee [Figure III.A-1 - Study Area].

The rugged terrain of this region is generally characterized by steep mountain slopes, confined river valleys, and narrow ridge tops. The geologic processes and climatic conditions responsible for the formation of these land forms, have as a result, helped to determine the past and present land use and land cover of the region. The regional topography, and the coal it contains, have been significant driving forces behind human settlement and development patterns throughout the region. The very

history of the region has been shaped by coal, and the region itself marked by the continuous attempts to extract it. Federal law also requires the maximum utilization of the natural resource so that disturbing the land in the future will not be necessary.

THE RUGGED TERRAIN OF THIS REGION IS GENERALLY CHARACTERIZED BY STEEP MOUNTAIN SLOPES, CONFINED RIVER VALLEYS, AND NARROW RIDGE TOPS

Settlement patterns in the Appalachian Coalfield Region were constrained by the dominant topographic features of the area, such as rivers, streams, mountains, and valleys. Communities settled along rivers and within valleys primarily for transportation and agricultural purposes. The coal deposits, as well as the physical limits to other types or forms of development, have defined the locations and extent of settlement and distribution. Within the study area, there is a relative scarcity of land suitable for agriculture and conventional residential, commercial, and industrial development. As a result, the limited settlement and development of the region has occurred almost exclusively on valley floors along stream and river courses. The current road and rail transportation networks generally follow the network of streams. Although the land was largely unsettled, there was significant timber cutting, and today's forests are largely second and third growth. Private and public forests provide lumber and pulpwood, recreational opportunities, wildlife habitat, and the opportunity for harvesting non-traditional forest products.

Water is relatively abundant throughout the study area. Figure III.A-2 depicts major rivers within the study area. Most of the major rivers and tributaries in the United States east of the Mississippi originate in the mountains of the Appalachian regions (USDOI OSM, 1999a). Outside of urban areas, shallow groundwater wells provide most of the water for domestic use (Heath, 1984). Vital to the health of an aquatic ecosystem is the quality of its water.

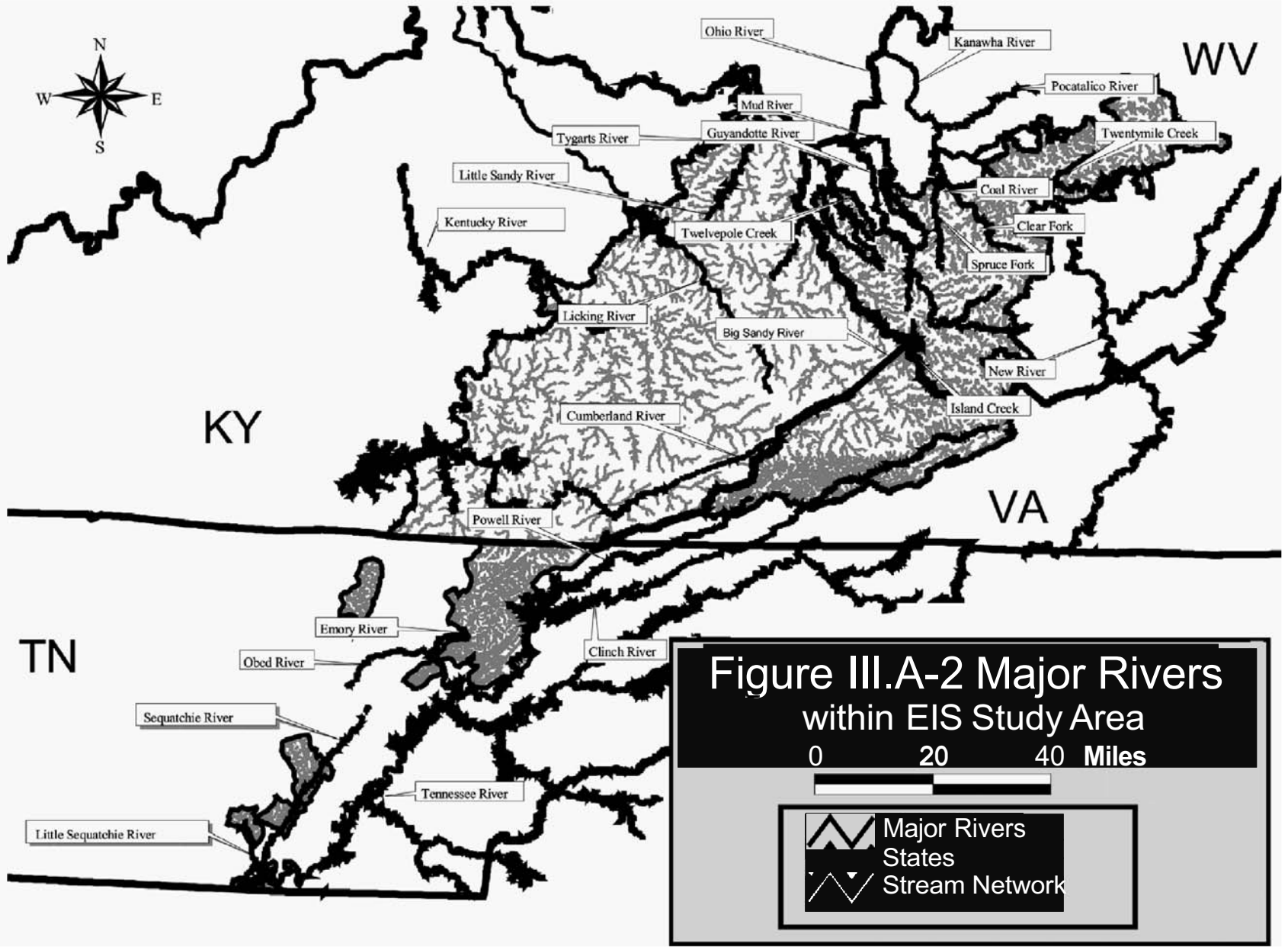


Figure III.A-2 Major Rivers within EIS Study Area

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The regional history of coal mining extends back well over a century. Remnants of earlier mining operations, as well as mines which are in operation today, have influenced the natural environment of the region. Of particular environmental concern are those resources which have the most potential for being significantly affected by the adverse impacts of coal mining. For example, the rivers and streams, and the aquatic ecosystems they maintain. An aquatic ecosystem is composed of three components: the biological, the chemical, and the physical, and any or all of them may be impacted by mining activities.

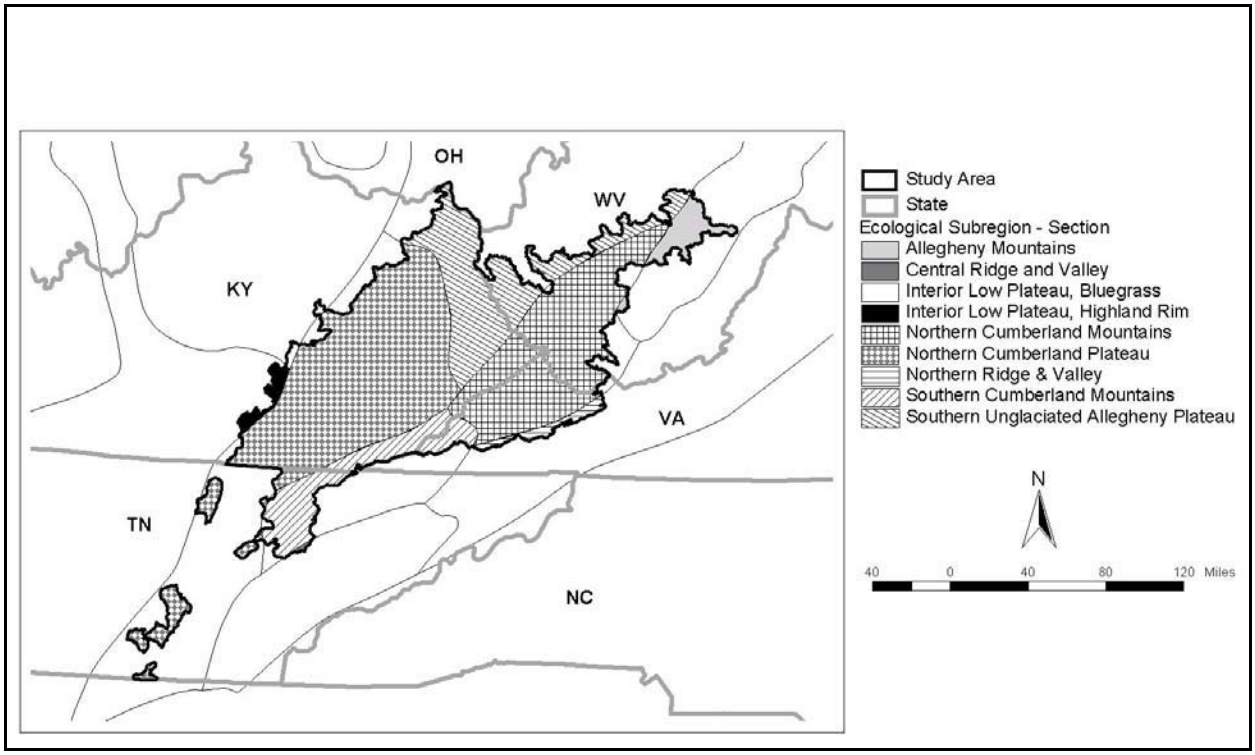
To assess the programs that monitor and govern the impacts that mining may have on aquatic and terrestrial ecosystems, it becomes necessary to consider and discuss issues in "natural" terms. By identifying and organizing environmental issues within natural boundaries, instead of partitioning areas based on arbitrary political boundaries such as state or county lines, natural resources may be considered within their own context. Two such "natural" categories of division are watersheds and ecoregions.

Watersheds are a clearly-defined unit of land that represents the area drained by a stream and all its tributaries. A watershed can include lakes, rivers, wetlands, streams, the surrounding landscape, and may also include ground water recharge areas. The watershed approach is useful because it focuses more specifically on drainage patterns, water quality, and aquatic ecosystems. The use of ecoregions and watersheds as "natural" units of area can depend highly on the scale of observation. For example, an ecoregion may contain countless small watersheds, while conversely, a large watershed (such as the Ohio River) may contain many ecoregions. For the purposes of classification, the study area watersheds are referred to individually by their 11 digit Hydrological Unit Code (HUC) assigned by the United States Geological Survey (USGS). For example, the Clear Fork watershed is located entirely within Raleigh County, West Virginia. The watershed consists of the Clear Fork itself, all the tributaries that flow into and contribute to the Clear Fork, and all the surrounding land from which the runoff and groundwater flow into the Clear Fork and its tributaries. Beginning at the highest points which surround the Clear Fork, headwater streams form which serve as the surface collection points for all surface and ground water within the watershed. As these headwater streams flow downhill, they join other headwater streams to form larger tributaries. Depending on their relative size and prominence, tributary streams may or may not be named. Further information on representative streams is provided in section III.C. of this EIS.

Ecoregions are areas of relatively similar landscapes. Across an ecoregion, one will find that climate patterns, physiography, geology, soils, and vegetation vary little. Ecoregions can be further subdivided into subregions, landscapes, and land units, each at a different planning and analysis level scale. Analysis at the ecological subregion level is of considerable value when the purpose is for strategic, multi-forest, statewide, and multi-agency assessment because several variables are considered when defining the boundaries of each ecological subregion (USDA, U.S. Forest Service 2002). The ecological units of an ecological subregion analysis are termed *sections*. Within an ecological subregion section geomorphology, lithology, soils, vegetation, fauna, climate, surface water characteristics, disturbance regimes, land use, and cultural ecology are generally similar.

The study area is located within portions of nine ecological subregion sections [See Figure III.A-3 - Ecological Subregion Sections]. Characteristics of each ecological subregion section of the study area are summarized in Table III.A-1 - Ecological Subregion Section Characteristics.

Figure III.A-3
Ecological Subregion Sections



Ecoregional analysis at a national level has highlighted the biological significance of the Appalachian ecoregions. These ecoregions are unique in the world because they combine characteristically northern species with their southern counterparts, and thus boast enormous richness and diversity. That, in combination with relatively mild environmental conditions, have provided a perfect setting for the evolution of unique species of plants, invertebrates, salamanders, crayfishes, freshwater mussels, and fishes. These species include great numbers of organisms, including terrestrial, aquatic, and plant species, which are supported by the Appalachian ecoregions (Stein et.al., 2000). The southern Appalachians have one of the richest salamander faunas in the world (Petranka 1998, Stein et.al., 2000). The Appalachian ecoregion forests represent some of the last remaining stands of a forest type that was once widespread in the northern hemisphere. These rich deciduous forests have been profoundly altered over the past few centuries and are becoming increasingly threatened.

Table III.A-1
Ecological Subregion Section Characteristics

Ecological Subregion	Geomorphology (Province)	Natural Vegetation (Forest Type)	Climate (Mean Annual)
Allegheny Mountains	Appalachian Plateaus	Northeastern Spruce-Fir Northern Hardwoods Mixed Mesophytic Oak-Hickory-Pine	Prec: 46-60" Temp: 39-54˚F
Central Ridge and Valley	Ridge and Valley	Appalachian Oak	Prec: 36-55" Temp: 55-61 ˚F
Interior Low Plateau, Bluegrass	Interior Low Plateaus	Oak-Hickory	Prec: 44" Temp: 55 ˚F
Interior Low Plateau, Highland Rim	Interior Low Plateaus	Oak-Hickory	Prec: 44-54" Temp: 55-61 ˚F
Northern Cumberland Mountains	Appalachian Plateaus	Mixed Mesophytic Appalachian Oak Northern Hardwoods	Prec: 40-47" Temp: 45-50 ˚F
Northern Cumberland Plateau	Appalachian Plateaus	Mixed Mesophytic Appalachian Oak	Prec: 46" Temp: 55 ˚F
Northern Ridge and Valley	Ridge and Valley	Appalachian Oak Oak-Hickory-Pine Northern Hardwoods	Prec: 30-45" Temp: 39-57 ˚F
Southern Cumberland Mountains	Appalachian Plateaus	Appalachian Oak Mixed Mesophytic	Prec: 46" Temp: 55 ˚F
Southern Unglaciated Allegheny Plateau	Appalachian Plateaus	Mixed Mesophytic Appalachian Oak	Prec: 35-45" Temp: 52 ˚F

Source: U.S. Forest Service, USDA, 2002

B. PHYSICAL SETTING

1. Physiographic Province

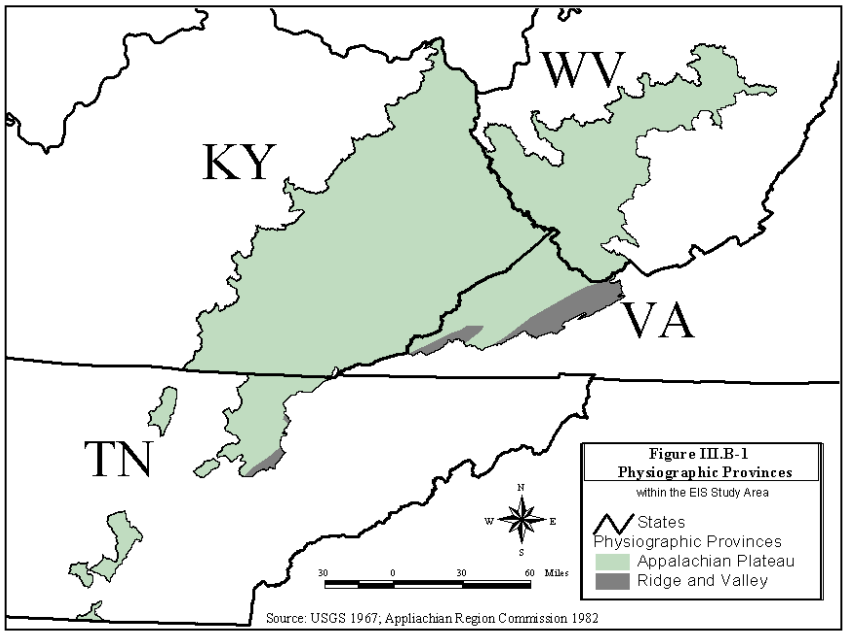
Physiographic provinces are a useful means of grouping land forms. The definition of a physiographic province is a geographic region in which climate and geology have given rise to an array of land forms that are notably different from those of surrounding regions. The feasibility and methods of coal mining in a given region are highly dependent on geologic conditions and land forms, so physiographic provinces are also useful in grouping the extent and various styles of mining within the Appalachian coalfields.

The Appalachian mountains form a wide belt (exposed width between 93 and 373 miles) that trends from Newfoundland to Alabama. The Appalachian mountains can be divided into three sections: (1) a northeastern section covering northern Maine and the maritime provinces of Canada; (2) a New England section covering portions of Vermont, New Hampshire, and New York; and (3) the Appalachian Highlands. The Appalachian Highlands section is comprised of the Ridge and Valley Physiographic Province and the Appalachian Plateau Physiographic Province. As shown on Figure III.B-1, the majority of the study area for this EIS is within the Appalachian Plateau Province.

The Allegheny Front separates the Appalachian Plateau Province from the Ridge and Valley Province. The Allegheny Front is a zone of transition between the tightly folded strata of the Ridge and Valley Province and the nearly horizontal sedimentary rocks of the Appalachian Plateau Province.

The Ridge and Valley Province is characterized by northeast-southwest trending mountains and valleys. In general, the valleys and lowlands are underlain by shales and limestones, and the ridges are composed of more resistant sandstones and conglomerates.

The Appalachian Plateau Province of the Appalachian Highlands includes the Pocatalico River, Coal River, New River, and the main stem of the Kanawha River drainage basins. It also includes small parts of the Ohio River and Bluestone River drainage basins. Differential stream erosion and repeated regional uplifts have given the plateau a rugged topography characterized by high, rounded or flat-topped ridges, rolling hills, steep valley slopes, and narrow valley floors.



2. Geology

Since coal mining involves the extraction of a geologic deposit, geologic conditions are an important factor in determining the extent and practicality of coal mining on a given site. Geologic considerations for coal mining include the depth, sequence, and thickness of coal seams, coal quality, and physical nature of the overburden (soil and rock that overlies coal) above and interburden (rock in between coal seams). The volume of excess material generated corresponds directly to the swell factors of the rock, which make up the overburden. Therefore, the potential for generating larger volumes of earth is greater with coarser-grained rocks, such as sandstone which has a higher swell factor than with finer-grained rocks, such as shale which has a lower swell factor. The chemical nature of coal and overburden, particularly with regard to pyrite content and the potential for acid mine drainage formation, is also a geologic consideration. These factors are influenced by the original conditions under which the coal deposits were formed, referred to as their environment of deposition, and subsequent deformation by tectonic processes. This section provides a brief overview of the history of formation of the Appalachian coalfields, and a summary of the general geologic conditions found in the four states of the study area. Detailed descriptions of the coalfield environment of deposition, tectonic history, chemical factors controlling acid mine drainage formation, and coal-bearing rock units are contained in Appendix C of this EIS.

a. Regional Geologic History

The Appalachian coalfields were formed during a long period of mountain building along the area of the modern east coast, with the coal beds deposited primarily from 300 to 250 million years ago. Sediments shed from these ancestral Appalachian mountains as they eroded were carried to a large inland sea occupying much of the area of the Appalachian mountain states and known as the Appalachian Basin. Large swamps formed along the margins of this sea and decayed plant matter, or peat, built up within them over time. As sea levels fluctuated, these coal swamps migrated with the shoreline and were buried by additional sediments carried from the mountains. Long-term burial pressures then converted the peat deposits into coal. This coal swamp migration and burial formed multiple layered coal seams typifying the Appalachian coalfields today.

Toward the end of the mountain building period, the collision of the North American and African continents deformed the eastern portion of the Appalachian Basin and produced the steeply folded bedrock characteristic of the Valley and Ridge Province. Further west, the Basin was only slightly deformed, producing gentle anticlines and synclines in the Appalachian Plateau Province. Over time, both the eastern mountains and the basin area were worn flat by erosion and buried by additional sediments. Regional uplift of the eastern states then re-exposed the coal-bearing bedrock to erosion, producing long valleys and ridges in the tightly folded bedrock of the Valley and Ridge Province. The erosion created deeply incised dendritic stream valleys in the relatively flat-lying bedrock of the Appalachian Plateau Province. In the Valley and Ridge Province, uplift and weathering eroded away the coal deposits from much of the area, with the remaining steeply-dipping coal seams more suited to underground mining methods. The same uplift and erosion in the Appalachian Plateau Province resulted in shallow, flat-lying exposures of coal-bearing bedrock that are amenable to surface mining within the study area.

b. State Geology Summaries

The following provides a basic description of the location, form, and structural features of the coal-bearing rocks within the study area.

b.1. Kentucky

Kentucky possesses two major coalfields at the eastern and western ends of the state, separated by a large area of older rocks exposed in a structure known as the Cincinnati Arch (USDOI OSM, 1998a). MTM/VF mining occurs in the eastern coalfield, where coal-bearing rocks underlay approximately the eastern quarter of the state and form a broad, shallow trough or synclinal basin (Kiesler, USGS 1983). Bedrock dips at 5° or less along the margins of the trough and is essentially flat-lying in the central portion of the trough (Kiesler, 1983). Upper Mississippian and Pennsylvanian coal-bearing rocks thicken towards the southeast, reaching their maximum thickness at the southeastern margins of the basin along a structure known as the Pine Mountain Thrust Fault zone. Coal units are disrupted and offset along this fault zone.

b.2. Tennessee

The Tennessee coalfields are in the east central portion of the state and trend northeast to southwest from Kentucky to the Alabama border. As with Kentucky, these coalfields form a broad, shallow trough or synclinal basin that is bounded to the west by a structure known as the Highland Rim escarpment and to the east by the Ridge and Valley Province. These coalfields are generally divided between the northern steep-slope areas of the Cumberland Mountains and the southern, flatter Cumberland Plateau, where area mining dominates (USDOI OSM, 1998b). Bedrock units primarily have a shallow southeasterly dip and thicken to the southeast near the basin's trough adjacent to the Valley and Ridge Province (Gaydos, 1982).

b.3. Virginia

With the exception of a small region in south-central Virginia which is not mined, coal-bearing rocks are present only at the westernmost end of Virginia and are contiguous with the Kentucky and West Virginia coalfields. These are relatively flat-lying rocks bounded on the northwestern and southeastern basin margins by the thrust-faulted and uplifted rock units (Rader, 1993 and Harlow, 1993). Along the northwestern coalfield margin is the Pine Mountain Thrust fault. The southeastern margin is bounded by a series of thrust faults. The Russel Fork fault divides the basin into two regions: (1) the relatively flat-lying rocks northeast of the fault and (2) the gently folded and faulted rocks located southwest of the fault that were moved as part of the Pine Mountain thrust sheet (Harlow, 1993). The rocks of both regions are nearly flat-lying and have an average northwesterly regional dip of 1.4 percent. Due to steep topography, Virginia mines are predominantly underground or contour surface operations, with a limited number of mountaintop removal and area-type operations (USDOI OSM, 1997).

b.4. West Virginia

Coal-bearing rocks underlay much of central West Virginia, extending into Ohio, Pennsylvania, and Maryland. One structural fold known as the Hinge Line separates the Dunkard and Pocahontas Geologic Basins of West Virginia. These basins are characterized by differences in the total thickness of their rocks, as well as by the orientation and distribution of their ancient swamps, lacustrine marine environments, and alluvial deposits (Arkle, 1974). The Dunkard and Pocahontas Basins approximately coincide with the northern and southern coalfields (younger and older mining districts, respectively) of West Virginia. The various formations of sedimentary rocks exhibit local differences in strata north or south of the Hinge Line in response to different depositional environments. For example, the Allegheny and Conemaugh formations in the Dunkard Basin represent a sequence of marine and coastal environments, including deltaic, offshore, and alluvial depositional conditions. In the Pocahontas Basin, these formations predominantly include the alluvial facies of non-marine sandstone, shales, and channel deposits that generally include only limited coal seams. Due to steep topographic conditions, contour, area, mountaintop-removal, and multiple-seam mining operations are the most common methods of surface mining in the state (USDOI OSM, 1998c).

3. Soils

Soils are a critical natural resource and essential for plant life in the natural environment. This resource is a particular concern for surface mining because, by definition, the practice will remove surface materials overlying the coal, including any soil present on the existing land. SMCRA requires that mine operations either preserve and replace soils on the reclaimed land surface to restore their vegetative cover or use an acceptable soil substitute. As discussed in Section III.J, surface mining operations use two methods to restore a vegetative growth substrate to reclaimed mine lands: topsoil removal and redistribution, and topsoil substitution. Both methods result in surface conditions markedly different from those present prior to mining. This section provides background on soils in general and the specific types of soils found within the study area.

a. Soil Characteristics

Because of their importance to agriculture, soils have long been studied to determine their characteristics and the factors that govern their formation and productivity. The following provides a brief overview of the soil formation process, soil profile, and the soil classification system.

a.1. Soil Formation

Soils are a fragile natural resource that require very long periods of time to form, most on the order of thousands to tens of thousands of years or longer. All soils are developed as a result of the interactions between five formation factors: (1) parent material, (2) climate, (3) living organisms, (4) time, and (5) topography. In the study area, the dominant formation factors have been topography, parent material, and time. Parent material is the bedrock, collovium (material moving down hillsides in response to gravity), or alluvium (material deposited by rivers and streams) on which a soil forms. Physical and chemical weathering and biological activity are the processes that form soils on the parent material, the rate of both being related to climate. Weathering is faster in warm, wet climates than in cold, dry climates. Soil

formation is an ongoing process, with weathering continuing to attack underlying parent material to form more soil, therefore, the longer that a parent material is exposed to the elements, the greater the weathering and the thicker the soil.

a.2. Soil Profile

Most soils show a distinct layering with individual layers referred to as horizons. There are many internal subdivisions that soil scientists use to characterize soil horizons, but the three basic groupings are called the A, B, and C Horizons. The A Horizon is the surface soil layer usually referred to as topsoil. It is the most weathered portion of the soil column and in vegetated areas will typically have a cover of decayed plant matter and high organic content known as the O Horizon. The B Horizon often referred to as the subsoil, typically contains less organic matter than the A Horizon and more clay. The C Horizon is the slightly weathered or unweathered parent material underlying the B Horizon. The individual horizons represent the downward progression of weathering in the soil formation process, and boundaries between them may be very distinct to very subtle depending on the nature of the soil. In general, older soils will have better developed horizons than younger soils.

a.3. Soil Classification

Soils are classified in the United States by a well-defined taxonomy, with a distinct hierarchy that follows from order, sub-order, great group, subgroup, family, and finally to series. There are twelve soil orders in the US, and in the study area the Inceptisol and Ultisol orders dominate. Inceptisols are immature soils that have weakly expressed horizons and retain a close resemblance to their parent material. They may form from highly resistant parent material or in alluvial floodplains, occur on extreme landscape positions, such as steep slopes and depressions, and have geomorphic surfaces so young as to limit soil development. Ultisols form in humid regions from parent material that has not been affected by glaciation, and thus develop on landscapes that are geologically old compared to glaciated areas. They are highly weathered soils that have a low nutrient content and base status.

b. Study Area Soils

To characterize the soils across a wide region such as the Appalachian coalfields, it is necessary to use soil series associations, rather than list specific soil series. For this study, two primary sources of information were used to collect pertinent data: the USDA, Geological Survey Series on the Hydrology of the Eastern Coal Province (Areas 1 to 23), and the USDA, Natural Resource Conservation Service (formerly Soil Conservation Service) County Soil Surveys.

Important points necessary to note when discussing MTM/VF region soil resources include:

- Historically, soil data have been collected and analyzed primarily for agricultural purposes, with less attention given to soils with lower attached economic value.

- Soil is an extremely heterogeneous material with high degrees of variability possible in its physical properties over a short distance. This is especially true with the non-agricultural

soil associations where many different soils with different properties are lumped together.

- The rugged topography of the MTM/VF region has made data collection difficult, and its low agricultural use has made it an area less studied than more intensively farmed regions.

b.1. Distribution

Soils typically encountered in the study area are predominantly colluvial in nature. Soil associations are shown in Table III.B-1 for the study area. These associations/complexes are typified as occurring on steep side slopes of higher mountains and formed on residuum or creep material from acidic sandstone, siltstone, and shale. These soils are very thin, with a typical topsoil layer of only 0 to 3 inches over varying amounts of colluvial material/subsoil ranging from 1.5 to 5 feet thick before reaching bedrock. These thin steep side hill colluvial soils' productivity and erodability can be increased and decreased, respectively, with proper planning. The presence of deeper colluvial and residual weathered deposits on southwest slopes that face the prevailing weather patterns make the region susceptible to land slides. A dominant land use in parts of the study area is forestry, which, depending on if and when it was last harvested, may have adversely affected the thickness of the topsoil layer.

Table III.B-1
Summary of Major Soil Associations in the Study Area

State	Hydrology Area Number	Primary Soil Associations
West Virginia	9	Clymer-Dekalb-Jefferson
West Virginia	9	Dekalb-Gilpin-Ernist
West Virginia	9	Gilpin-Ernist-Buckhanon
West Virginia	9	Clymer-Gilpin-Upshur
West Virginia	9	Gilpin-Dekalb-Buckhanon
West Virginia	12	Clymer-Dekalb-Jefferson
West Virginia	12	Clymer-Gilpin
West Virginia	12	Clymer-Gilpin-Upshur
West Virginia	13	Clymer-Dekalb-Jefferson
Kentucky	13	Jefferson-Shelocta
Kentucky	13	Dekalb-Berks-Weikert
Kentucky	14	Jefferson-Shelocta
Kentucky	14	Lathan-Shelocta
Kentucky	14	Jefferson-Dekalb
Kentucky	15	Jefferson-Shelocta
Kentucky	15	Lathan-Shelocta
Kentucky	15	Jefferson-Dekalb
Kentucky	15	Shelocta-Gilpin
Tennessee/ Kentucky	17	Muskingum-Gilpin-Jefferson/ Lathan-Shelocta
Tennessee/ Kentucky	17	Ramsey-Hartsells-Grimsley-Gilpin/ Jefferson-Shelocta
Tennessee	18/20	Ramsey-Hartsells-Grimsley-Gilpin
Tennessee	18/20	Muskingum-Gilpin-Jefferson
Virginia	13/16	Berks-Pineville-Rock Outcrop
Virginia	13/16	Kimper-Shelocta-Hazelton
Virginia	16	Berks-Weikert-Ladig
Virginia	13/16	Wallen-DeKalb-Dry Pond

State	Hydrology Area Number	Primary Soil Associations
Virginia	13	Jefferson-Wallen-Gilpin
Virginia	13/16	Fredrick-Carbo-Timberville
Virginia	16	Groseclose-Litz-Shottower
Virginia	16	Mommaw-Jefferson-Alonzville
Virginia	16	Murrill-Westmoreland-Frederick
Virginia	16	Carbo-Chilhowie-Frederick
Virginia	16	Catache-Berks-Shouns

source: http://www.va.nrcs.usda.gov/soils

Not appearing on Figure III.B.1 are the narrow bands of valley soils along the flood plains, which are both colluvial and alluvial in nature. The unconsolidated materials forming these soils can range from a depth of 5 feet along narrow streams to100 feet along large rivers. The soils in these locations often are inceptisols showing only limited horizonation. These soils are typically very productive and can qualify as prime farmland soils.

4. Soil Productivity

This portion of the environmental impact statement (EIS) addresses soil quality and forest productivity at reclaimed mountaintop mine sites and is based on a technical study performed by OSM to support the EIS. This study involved collecting available published literature, papers presented at conferences and symposiums, interviews with prominent researchers, and documenting the collective knowledge and experience of the Soil Quality and Forest Productivity team members.
Several milestones were identified in the work plan and accomplished as shown:

1) examine soil properties--evaluated on the basis of the literature and team experience
2) evaluate the effectiveness of current sampling and testing protocols--evaluated on the basis of the literature and team experience
3) establish the effectiveness of current reclamation methods--dropped from consideration as inappropriate within the study time frame
4) evaluate long-term indices for determining forest productivity on reclaimed mined lands--evaluated on the basis of the literature, team experience and interviews with researchers
5) interview prominent researchers--accomplished
6) review regulations--accomplished
7) determine which factors limit tree production on mined lands--accomplished
8) conduct field verification of site conditions if the information gathered warrant such investigations–this task was not warranted, given the experience of the team

The study report outline is based on the activities described above. Four major topic areas were identified:

1) Review and identify applicable regulations--This is not simply a restatement of the regulations, but an assessment of whether the rule has positive or negative effects on the reclamation of mined lands;
2) Mine soil forest relationships–A technical perspective on different mining techniques use to create a growth media conducive to reforestation;
3) The third topic deals with the effect of mycorrhizal relationships on planting stocks–Evidence supports inoculating tree stock with mycorrhiza in order to improve the growth and survival of planted trees. Other researchers argue that native organisms found in topsoil are important to tree growth. The study will look at this issue and report the results;
4) The fourth topic is about planting trees on mined lands. Here again, the idea is to extrapolate from existing literature a brief description of the state-of-the-art, risks, hazards, and probable replanting rates in an attempt to identify changes that could be implemented to encourage planting of more trees.

There are also other factors that influence tree planting on mined lands that will only briefly be mentioned here. The stability of growth media placed on backfill must be considered when selecting reclamation techniques. Although this factor deals with topsoil/substitute placement, it is more of an engineering issue. Cost is another consideration that will have a great influence on whether or not changes will be made that allow increased, more effective tree reclamation to occur on mine sites. The challenge is to find more cost effective ways to create new forest on mined lands.

a. Applicable Regulations and Observations on Implementation

SMCRA, OSM regulations, and state regulations (which must be as effective as OSM regulations), contain elements that may work at cross purposes. For example, when regulatory authorities strictly enforce erosion control regulations as a means of protecting water quality, there is a strong tendency for operators to use quick-germinating, vigorously-growing grasses and legumes to stabilize the soil and prevent erosion. Such vigorous herbaceous vegetation, however, has the unfortunate side effect of discouraging tree establishment. Additionally, in most of Appalachia, grass and legume stabilized areas are considered adequately vegetated to meet the requirements for the pasture land use. Thus, an operator could obtain bond release without further revegetation work. A further disincentive to reforestation is the fact that grass/legume mixtures commonly used in reclamation tolerate a wide range of soil chemical conditions, as well as the excessively compacted soils, that typify reclamation in Appalachia. Thus, the use of grasses and legumes serves as the low cost, low-risk option for bond release. Even when the reclamation plan calls for the planting of trees, excessive compaction of the rooting medium, which severely reduces tree growth, is the norm.

The following sections use West Virginia soil handling regulations to illustrate barriers to effective reforestation. The other steep slope Appalachian regulatory programs contain similar provisions but, for brevity, will not be restated here.

38CSR2.14.3 Topsoil

14.3a. Removal. Prior to disturbance of an area, topsoil shall be removed from the area to be disturbed in a separate layer and if not immediately redistributed, it shall be segregated and stockpiled in a separate stable location as specified in the preplan. Stockpiled topsoil shall remain in place until used for redistribution unless otherwise approved by the Director. Stockpiled topsoil shall be protected from excessive compaction. Where the removal of vegetative material, topsoil or other materials may result in erosion, the Director may limit the size of the area from which these materials are removed at any one time.

Historically, Post Mining Land Uses (PMLU)in the mountaintop mining area of West Virginia were predominantly grasslands, which led to the soils rarely being salvaged. The soils were generally characterized by permit applicants as too thin and/or too poor in quality to justify salvaging for the PMLU.

14.3.b. Redistribution. Prior to redistribution of topsoil, the regraded land shall be treated, if necessary, to reduce the potential for slippage of the redistributed material and/or to enhance root penetration. Topsoil and other materials shall be redistributed in a manner that prevents excess compaction and that achieves an approximate uniform, stable thickness, consistent with the approved postmining land uses, contours, soil density, and surface water drainage system. Immediately after redistribution all topsoil areas shall be protected from wind and water erosion.

Excessive compaction is a well-known impediment to revegetation in Appalachia and other coal regions of the country. As noted above in 14.3.b, the conflict between not over compacting soils and stability or soil erosion is a concern. That is, soil and soil substitutes are often compacted to the point of seriously reducing root penetration when the objective is to maximize stability or reduce erosion of those soils. The negative impact of this compaction on biomass production is greater for trees than for grasses and legumes.

14.3.c. Top Soil Substitutes. Any substitute material used for top soiling must be capable of supporting and maintaining the approved postmining land use. This determination of capability shall be based on the results of appropriate chemical and physical analysis of overburden and topsoil. These analyses shall include at a minimum depth, thickness and areal extent of the substitute structure or soil horizon, pH. Texture class, percent coarse fragments and nutrient content. A certification of analysis shall be made by a qualified laboratory stating that:

14.3.c.1 The proposed substitute material is equally suitable for sustaining vegetation as the existing topsoil;

14.3.c.2. The resulting soil medium is the best reasonably available in the permit area to support vegetation;

14.3.c.3. The analyses were conducted using standard testing procedures.

14.3.d. Soil Amendments. Nutrients and soil amendments in the amounts determined by soil tests shall be applied to the redistributed surface soil layer so that it supports the approved postmining land use and meets the revegetation requirements of Section 9 of this rule. These tests shall include nutrient analysis and lime requirement tests. Results of these tests shall be submitted to the Director with the final planting report as required by this rule.

In practice, selective overburden handling in the mountaintop mining area of West Virginia is conducted so as to prevent the deposition of acid toxic materials on the surface. The predominant PMLU has included a bias towards salvaging fine-textured, high pH soil materials that provide favorable chemical conditions for the growth of grasses and legumes, but have a negative impact on forest regeneration.

Approval for use of a topsoil substitute material requires a waiver, as described in 14.3.c above, and must support the PMLU. Most permits requesting the use of a topsoil substitute will indicate thin soils [III.B.1] as a reason for not saving topsoil. The permit will explain, using language something to the effect that "slopes are steep with only a thin layer of topsoil that would not be practical to save following clearing and grubbing." Furthermore, the permit will state that "the quality of the topsoil is poor with very little capacity for supplying plant nutrients." This may provide poor soils for grasses and legumes, but support a mixed hardwood climax forest. What is described as poor for one land use may be ideal for another land use.

Topsoil has nearly all of the living matter that makes the collection of sand, silt, and clay a living soil capable of sustaining plant life. It is not just soil pH and nutrients that makes a medium suitable for plant growth and development. This is the reason why the surface mining act and State regulations at 38CSR2.14.3.a. require the saving of topsoil. Recognizing that all topsoil is not created equally, topsoil substitutes are permissible, provided the new material can be shown to be as good as or better than the original topsoil.

The West Virginia surface mining law, at §22-3-10.(2)(B), and other steep slope state counterparts require an evaluation of the land's capability to support a variety of uses prior to any mining. §22-3-10.(3) and other states' similar provisions require that, relative to mined land use following reclamation, the permit include a discussion of the utility and capacity of the reclaimed land to support a variety of alternative uses.

b. Mine Soil/Forest Relationships

Prior to the passage of SMCRA, most surface-mined land in the east and midwest was reclaimed with trees. The quality and productivity of these lands varied, but, in general, reforestation was successful, and commercially valuable forests were created (Andrews et al., 1998). With the implementation of SMCRA-based rules and regulations, the percentage of land reclaimed to forest dropped significantly. The rules, as typically interpreted and enforced, resulted in intensely-graded landscapes with erosion control provided by herbaceous vegetation. In this post-SMCRA environment, reforestation was difficult and productivity of those lands reforested was disappointing.

The reclamation literature, extending from well before the passage of SMCRA, up to the present, presents a clear picture of the factors responsible for the success or failure of reforestation efforts. OSM has recently initiated a program to promote reforestation and eliminate regulatory barriers to establishing trees on reclaimed sites. The goal is to create a regulatory process that will result in successful reforestation; that is, result in the establishment of forests that are productive and economically viable for timber production.

Deep rocky soils with the appropriate chemical composition can be produced through mining and reclamation, and will support forests that are more productive than those supported by the thin natural soils typical of the Appalachian mountains. However, the mine soils that support good forest growth vary chemically over a more limited range than those that will support a good stand of herbaceous vegetation. Trees also are more sensitive than herbaceous vegetation to the negative impacts of excess compaction.

Ashby et al., 1984, states that "mine soils with differing contents of coarse fragments may have productivity equal to or greater than pre-mining soils." Indeed, a relatively small percentage of soil fines distributed through a matrix of rocky material that is not excessively compacted can function as an excellent substrate for tree growth. The "increased rooting depth on loose mine soils appears to compensate more than adequately for loss of soil volume due to stones." Additionally, appropriately constructed mine soils may have higher water infiltration rates and lower erosion rates than replaced soils. Ultimately, it is the water and nutrient supplying capacity of the rooting medium that translates into plant productivity.

Research in Appalachia on reforestation of mined lands in the post-SMCRA environment portrays the actual accomplishments and has assisted in refining the requirements for mine soils that will support productive forests. The productivity of mine soils produced post-SMCRA is characteristically reduced by excessive compaction. These soils may be further reduced in value for forest growth due to lack of selection of appropriate substrate materials or selection of fine textured materials with a high pH (which are more favorable for supporting herbaceous vegetation). However, the technology exists to produce high-quality forest soils. Burger, et al., 1998 describe this technology and identify policies designed to encourage its use. They state:

> Research by reclamation forestry groups throughout the Appalachian and Midwestern coalfields has shown that productive mine soils and forests can be restored by using a "forestry reclamation approach," described in Virginia Cooperative Extension (VCE)

Publications 460-123 (Burger and Torbert, 1992) and 460-136 (Torbert et al., 1994). The forestry reclamation approach basically entails:

1. Replacing 3 to 4 feet of surface soil and/or weathered, sandstone overburden (taken from the surface 10 feet) for the new reclaimed soil and sub-soil medium;

2. Loosely grading noncompacted topsoil or topsoil substitutes that include, when possible, woody debris and native seeds;

3. Using native and non-competitive domestic ground covers (tree-compatible) that quickly protect the site, encourage native forest plants and animals, and enhance forest succession; and

4. Planting nurse trees for wildlife and mine soil improvement, and planting valuable crop trees for their commercial value to the landowner and adjacent communities.

This forestry reclamation approach has been used operationally and has proven successful. In addition, the approach described above can cost the mine operator $200 to $500 less per acre than traditional reforestation practices, due to reduced grading costs and less expensive ground cover seed mixtures. This approach has been approved by the Virginia Department of Mines, Minerals, and Energy in a July 9, 1996, memo on reforestation guidelines. Approximately 80% of Virginia's operators/landowners are now opting for a post-mining land use of forestry. New reforestation reclamation guidelines have also been approved as a reforestation initiative by the Kentucky Department for Surface Mining and Reclamation and Enforcement in Reclamation Advisory Memorandum #124 (KY DSMRE, 1997). In West Virginia, this approach is consistent with regulatory agency criteria for approving reclamation plans to achieve a forestry post-mining land use.

Foresters judge soil quality based on the average height of trees at a given "index age," such as trees of age 25 or 50 years. Site index has a dramatic effect on the value of timber produced (Burger, et al., 1998). In Table III.B-1, reclamation technique is related to white pine productivity and stand value at 30 years.

c. Effect of Site Index on Timber Value: Oak

White pine was used in the analysis shown in Table III.B-1 because of its predominant use on post-law mined land. Although total wood volume would be less for hardwoods, the same general relationships between site quality and value per acre would hold true. A site with a white pine site index of 55 (age 25) has an average oak site index of 65 (age 50), which is an average value for oaks across most of the Appalachians. This species-to-species relationship shows that average post-SMCRA reclamation site quality for oaks would be about 50, and the site quality potential for oaks of properly-reclaimed mine sites would be about 85. This estimate is confirmed by Ashby, *et al*. (1984) who evaluated mine soil productivities for oak species.

Table III.B-2 shows the relative influence of soil and site properties on oak site index, wood yield, and harvest value. Average oak sawtimber value at age 60 on average quality sites (SI = 65) is about $4,250 per acre. If forest sites are degraded through typical post-law reclamation from SI 65 to 50, potential harvest value becomes one-fourth of what it was originally. If sites are upgraded through reclamation to SI 85, harvest value doubles. These estimates show the dramatic effect site quality has on forest land value and, it shows why landowners and the mining community should strive for proper reclamation of forest land.

Table III.B-2
The Effects of Reclamation Technique on White Pine Productivity and Stand Value at 30 Years

Case	White Pine Site Type	Site Index* (Base Age 25)	Bd.Ft.Vol. at Age 30 (MBF/ac)**	Harvestable Wood Products	Harvest Price ($/MBF)	Total Value ($/acre)
I	Average quality of an undisturbed Appalachian forest site (Doolittle 1958)	55	35.1	small sawtimber	50	1,755
II	Projected average quality of a post-SMCRA reclaimed mine soil (Torbert et al., 1994)	45	6.1	pulp	20	122
III	Actual quality of a white pine stand on a good minesoil in Virginia (Kelting et al., 1997)	70	46.4	large sawtimber	75	3,480

*Site Index = Expected tree height after 25 years.
** Board Foot Volume at Age 30 (MBF/acre). MBF = thousand board feet (Vimmerstedt, 1962).

Table III.B-3
The Relative Effect of Site Quality
on Appalachian Oak Harvest Volumes and Stumpage Value at Age 60

Site Index	Appalachian Oak Site Index (ft) (Base Age 50)	Bd.Ft.Vol. at Age 60 (MBF/ac)*	Harvestable Wood Products	Stumpage Price ($/MBF**)	Total Value ($/acre)
Average	65	11.8	sawtimber	360	4250
Poor	50	5.6	small sawtimber	200	1120
Good	85	16.2	large sawtimber, veneer	520	8425

*MBF = thousand board feet (Schnur, 1937)

The information in Table III.B-2 is corroborated by the experience of reclamation personnel and is reflected in West Virginia's recently proposed commercial forestry regulations. In estimating the likely quality of reclamation to be obtained under these regulations, we must recognize the fact that the current regulations (which have been in place since May 16, 1983) require that selected overburden substitutes for soil be "equal to, or more suitable for sustaining vegetation than the existing topsoil, and the resulting soil medium is the best available in the permit area to support revegetation." Also, soil materials are to be redistributed in a manner that prevents excessive compaction of the materials. Be this as it may, the reality of reclamation in Appalachia is that selective overburden handling is rarely practiced beyond that required to keep highly toxic material out of the rooting zone; excessive compaction is commonplace. Andrews, et al, 1998, point out that "Height growth was greater on steeper slopes. In naturally-forested stands the opposite is usually true, because steeper slopes have greater runoff, shallower soils, and more erosion. On reclaimed sites, slope steepness is related to depth and compaction. Level sites are often subjected to greater vehicle traffic, resulting in more compaction and poorer drainage and aeration."

Production of soils that will support commercial forestry as part of mountaintop mining requires selective overburden handling and replacement procedures on a scale that has never been carried out in Appalachia. Full-mine scale replacement of native soils without excessive compaction does occur however. Replacement of native soils without excessive compaction in area mining operations, or with reduction of excessive compaction by ripping, is standard practice where prime farmland is reclaimed.

d. Soil/Overburden Chemistry

Andrews, *et al.*, 1998, found that the most important chemical factor influencing the growth of white pine was soluble salts. The second-most important soil chemical property affecting white pine growth was extractable phosphate, and in general, height growth declined when exchangeable manganese levels exceeded 20 mg/kg. Site requirements for different species of trees vary widely, and there is ample opportunity to further refine the site requirements for different tree species used in reforestation. However, from a practical standpoint, it is probably adequate to reconstruct the soil medium by salvaging material

from the top 10 feet of overburden. On average, this will result in a soil medium with an adequate chemical composition and with adequate microbial inoculum from the native soils.

Burger et al., 1998, address the practical aspects of re-establishing productive forests in Appalachia, stating:

> "Our work shows that, in nearly all cases, any mix of the surface 10 feet of soil and rock makes an excellent growth medium for virtually all native species of pines and hardwoods. Applying 4 feet of this mix of material without compaction creates a topsoil substitute that is usually as productive or more productive than the original soil. Woody debris and some rocks mixed in or laying on the surface actually create microsites for native species. Less grading and seeding is needed for forestry land uses, making the use of this topsoil substitute cheaper for the mine operator."

e. Soil Compaction

Compaction of mine soils is identified as one of the chief factors reducing the value of reclaimed forest lands. We are not aware of any research on the effect of natural forces such as freezing and thawing and root action on improving compacted mine soils. However, with the increase in the size of agricultural equipment and the advent of "no till" agriculture, there has been increasing attention given to the effect of compaction on agricultural soils. Research on soils that are subject to freeze-thaw cycles during the winter and root action from crops or native and introduced grasses suggests that compaction below the plow layer may persist a century or more (Sharratt, *et al.*, 1997; Kay, *et al.*,1985; Voorhees,1983; Sharratt, *et al.*, 1998; and, Blake, *et al.*, 1976). In spite of the lack of systematic data addressing the impact of natural forces and tree roots on the compaction of mine soils, it is prudent to assume that compacted mine soils, with their well documented detrimental impact on tree growth, will behave similar to agricultural soils with compaction enduring over similar periods of time.

f. Mycorrhizal Relationships

Mycorrhizae have been widely reported to aid survival and growth of forest trees under many different site conditions (Ruehle and Marx, 1979) (Parkinson, 1978) (Danielson, *et al.*,1978). *Pisolithus tinctorius* (Pt.) was found to improve the survival and growth of pine seedlings on acid coal mine spoil by Marx and Artman (1979). Schoenholtz and Burger (1984) found that inoculation with this same fungi enhanced seedling growth to some extent, but high amounts of natural ectomycorrhizal colonization probably masked some of the effects of Pt. Cordell and co-workers (1999), indicated that specific mycorrhizal fungi provided significant benefits to the plant symbionts on drastically disturbed mine sites through increased water and nutrient absorption, decreased toxic materials absorption, and overall plant stress reduction. Other researchers have contributed to the body of knowledge concerning the effects of surface mining on soil microbial communities and algal colonization and succession (Visser, *et al.*, 1978) (Starks and Shubert, 1978) (Shubert and Starks, 1978).

The role of mycorrhizal fungi in sustaining productive forests on more favorable mine sites has also been well documented. When a readily available source of nutrients are present, seedlings would not be expected to benefit nutritionally from mycorrhizae. Marx (1977) determined that loblolly pines with roots

that are growing rapidly due to high soil fertility have a decreased sucrose content and are not susceptible to ectomycorrhizal infection. Also, Torbet, *et al*. (1985) found that mycorrhizae did not have an effect on seeding growth in spoil material that had a high soil fertility. This study also determined that mycorrhizal trees not planted with a fertilizer pellet had significantly greater volumes due to enhanced diameter growth; however, fertilized non-mycorrhizal seedlings in rock mix spoils had greater heights, diameters, and twice the volume of non-fertilized infected trees in spoils with surface treatments.

The influence of pH on mycorrhizal fungi has had substantial investigation. It has been demonstrated that conifers are better adapted and are more productive on somewhat acidic soils (Pritchett, 1979). Part of this adaptation has to do with their symbiotic association with mycorrhizal fungi, which play a significant role in the rhizosphere of conifers (Marx, 1977). Theodorow and Bowen (1969) reported that most ectomycorrhizae associated with conifers do not thrive when the soil pH exceeds 6.5. This was confirmed in a study on minesoils by Schoenholtz et al. (1987) when the rates of colonization of mycorrhizal was compared for three pine species growing in two different spoils with pH values of 5.4 and 6.1, respectively. Numbers of trees and numbers of short roots per tree colonized were consistently higher at the lower pH. The colonized trees survived and grew better. Torbet and co-workers (1990) also found that there was a distinct inverse relationship between pine growth and mine soil pH which they attributed in part to the symbiotic association with mycorrhizal fungi.

Although more recent studies generally acknowledge the benefits of mycorrhizal inoculation, there has been caution to portray it as a panacea for revegetation problems on surface mine spoil. Torbert and Burger (1990) advised that their studies show that when a site is properly reclaimed and revegetated, virtually any tree species suitable to the climate can be established without the need for containerized seedlings, mycorrhizal inoculation, fertilizer tablets, or chemical weed-control. And in keeping with this same theme, Burger (1999) concluded in a research summary that on one study site, "After 2 years, all seedlings were colonized by native mycorrhizae. Special mycorrhizal treatment was not necessary; there was no difference in survival and growth between treated and untreated seedlings."

g. Planting Trees on Mined Land

Establishment of trees on surface mined lands has been documented for at least 50 years. Changes in the mining industry in recent years or, more to the point, changes in the methods of reclaiming mined lands have been responsible for the poor results on areas reclaimed to forest lands. As the poor results became apparent, a number of researchers began a quest for solutions to the problem. Among the leaders in this research were Dr. Don Graves of the University of Kentucky and Dr. James Burger of Virginia Tech. Their contributions to the research of reforesting mined lands have been prolific and have followed parallel lines. Both Graves and Burger were also intimately involved in the development of a document designed for the state of Kentucky to address the problems of reforestation on mined lands. The goal of the guidance document was not simply to get trees to survive on mined land, but to provide an environment in which they could thrive. Much of what is known today regarding reforestation of mined lands has been brought together in the Kentucky guidance document.

The document, called Reclamation Advisory Memorandum (RAM) #124, was developed with the assistance of coal industry officials, educators, environmental leaders, forestry and wildlife officials, and

federal and State mine regulators from Kentucky. Most current reforestation literature was reviewed and utilized in producing this guidance document. It was published March 10, 1997. The following summation of RAM #124 is essentially a summary of the state of the art of tree planting on mined lands in the eastern portion of the United States.

Successful tree planting is not measured by numbers of living stems per acre but by the potential of those living stems to produce "crops" of recreation, wildlife, lumber and other values associated with forested lands. RAM #124 was developed to enhance the potential for mined lands to produce viable, productive stands of commercial timber. Associated values are likewise increased by proper site preparation of mined lands. The RAM identified three practices that inhibit the establishment of productive stands of timber. They are:

1) excessive compaction of the surface 4-6 feet;
2) selection of inappropriate rooting medium; and
3) excessive competition from herbaceous ground cover.

Conversely, the RAM identified three practices that, if followed, could promote tree establishment and growth. They are:

1) minimal grading of level to gently sloping areas;
2) use 4 - 6 feet of slightly acidic to near neutral rooting medium;
3) and selection of less intrusive species for erosion control.

The RAM addresses each limitation with guidance to avoid certain practices and establish productive practices in their place.

Excessive compaction constructs a limited rooting zone, resulting in poor root penetration, along with poor survival and reduced growth. To achieve minimal compaction, it is recommended that end-dump equipment be used to place the rooting medium in tightly placed piles. The surface is then graded by low ground pressure equipment to grade the tops of the piles and gently level the area in one or two passes. Areas utilizing drag lines are advised to similarly place material in order that grading can be accomplished in 1 or 2 passes with a tracked dozer. Steep slope operations (over 27 degree slopes) are advised to end dump material that has had large boulders removed on the outslope and grade in one or two passes.

Limited topsoil and the erosiveness and compacting qualities of topsoil often make it desirable to utilize an alternate material as a growing medium. Growth medium with low to moderate levels of soluble salts, low pyritic sulfur content, pH levels between 5.0 to 7.0, and texture conducive to proper internal drainage should be selected. Revegetation species should be selected that are compatible with the soil pH, with consideration for the wide range of acceptable pH and limited range of optimum pH for tree species.

Excessive competition from ground cover has had a negative impact on establishment of tree stands on mined lands due to the use of aggressive species such as fescue and excessive fertilization designed for herbaceous vegetation . Selection of ground cover should be based on soil pH and the growth habit of the

species. Slow growing ground cover species insures soil stabilization while allowing tree seedlings to emerge above the ground cover, ensuring their survival.

Attention to these factors and practices, along with controlled fertilization is recommended by this State RAM in order to achieve establishment and good growth of timber stands on mined lands. One aspect of tree establishment that literature addressed but that was not addressed in this RAM is the effect of mycorrhiza on tree establishment and growth. Dr. Burger stresses the use of the top10 feet of soil and rock as the growing medium in his studies, providing natural inoculation with mycorrhiza. The RAM however did not strictly adhere to that recommended practice. It did however recommend the inoculation of seedling stock.

Reforestation is also subject to risks caused by the vagaries of the weather, browsing damage, girdling by mice and improper handling and planting of nursery stock. These risks have tended to discourage the choice of reforestation as a land use option by coal operators. However, these risks may be more than offset by the potential for reduced costs when the reduced grading required for successful reforestation is factored in.

5. Topography and Geomorphology

Topography describes the actual shape of a land form, while geomorphology is the study of the characteristics, origin, and development of a land form. Topography is a very important factor in determining the extent and practicality of coal mining on a given site, and the nature of its excess spoil disposal requirements. Steep-sloped, deeply incised topography as found in much of Tennessee and West Virginia, exposes many coal seams to access by surface mining methods, but limits the practical return of spoil to mine benches. Shallower geomorphology and less coal seams does not expose as many coal seams to surface access. Underground mining is not as strongly influenced by topography, but it is favored by incised lands that allow ready access to the outcrops of coal seams deeper in the geologic formation. This section provides background on the topographic and geomorphic setting of the study area to aid in understanding the influences that these features have on the mining activities discussed in other portions of this EIS.

a. Topographic Characteristics

The study area is characterized by steep slopes and narrow valleys. Several areas within the study area have steep river gorges. However, there are areas within the study area that have rounded hilltops, stream terraces, and floodplains near large rivers. Level stream terraces and wide floodplains along rivers and some tributaries provide areas of nearly flat land. Gently sloping plateau areas are interrupted and dissected by numerous rivers and streams with steep valley slopes in portions of the study area. The majority of the study area can be characterized by consecutive ridges with slopes greater than 20° and only a few small, rounded hilltops. Many portions of the study area have mountain peaks greater than 1,969 feet (600 meters). Elevation is depicted in Figure III.B-2.

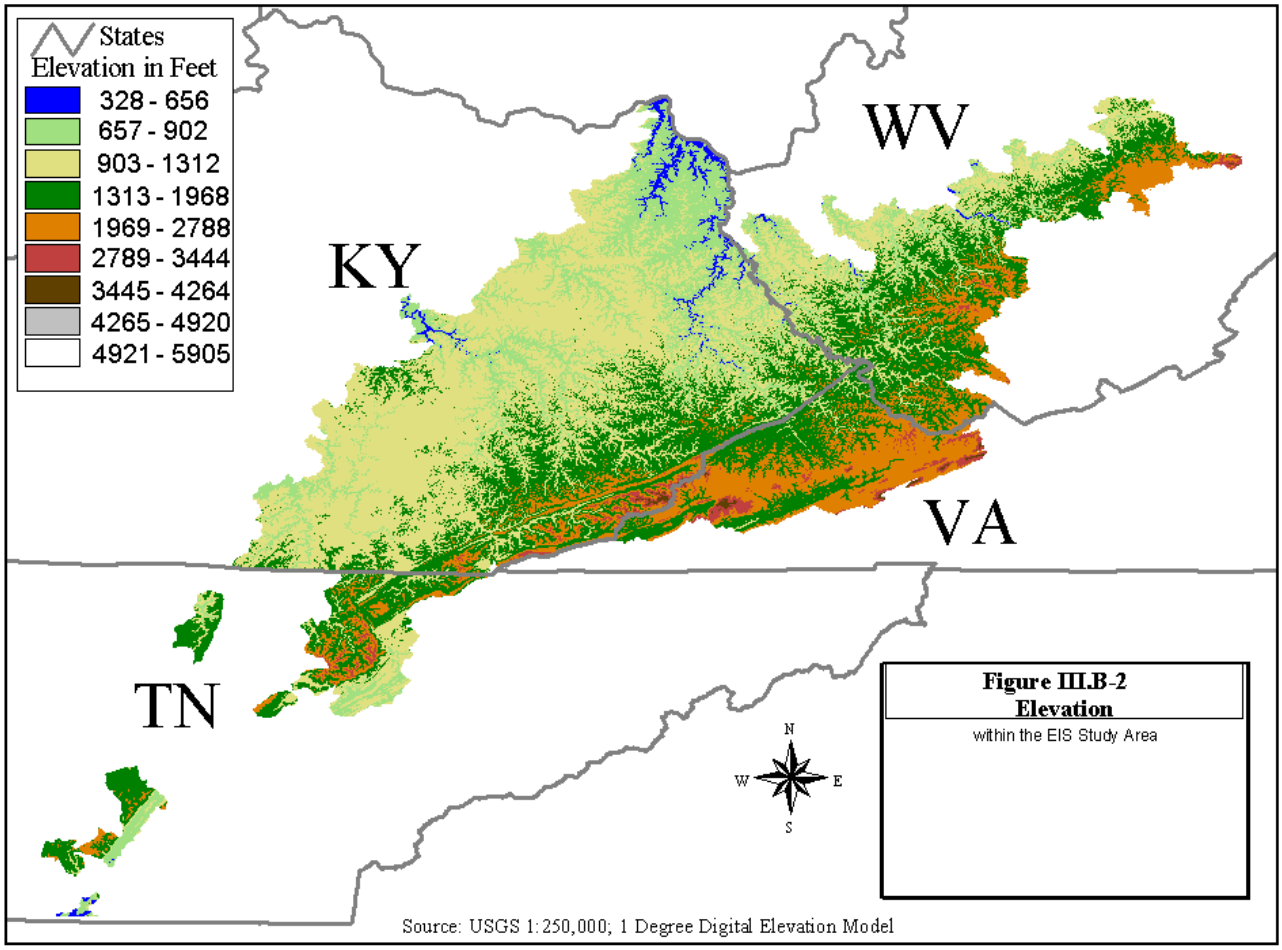


Figure III.B-2 Elevation

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b. Geomorphic Characteristics

After the mountain-building phase of the Appalachian orogeny, the study area experienced a long period of erosion stretching into the Miocene epoch. Most of the Appalachian Highland region is believed to have been beveled flat, as evidenced by the long ridge crests of equal elevation in the Valley and Ridge Province. After the Miocene, a regional uplift of undetermined origin elevated the modern Appalachian Mountain region to its approximate existing peak elevations. Rejuvenated erosion then carved into the elevated strata primarily along zones of structural or bedrock weakness. In the Ridge and Valley Province, the soluble limestone cores of breached anticlines eroded to form long, gently curving valleys, while in the Appalachian Plateau Province erosion along fracture trends was favored, forming dendritic and trellis stream patterns. The original drainage patterns of some large, pre-uplift rivers were preserved during the uplift and cross structural trends. The Susquehana River Valley is an example of such a superimposed drainage pattern. Other prehistoric drainage patterns have been abandoned over time, usually by significant drainage pattern changes occurring during glacial periods.

The ancient Teays Valley trends east-west across the lower Appalachian Basin. Prior to the Pleistocene Period (2.5 million years ago), the Teays River flowed westward across Virginia and West Virginia along a course presently occupied in part by the New River and Kanawha River systems. The Kanawha River follows the course of the pre-glacial Teays River upstream from St. Albans. The geologic history of the abandonment of the Teays River Valley west of St. Albans is poorly understood or documented. Today, the sediments of the former Teays River and its tributaries reach an elevation of nearly 800 feet (244 meters) within the former stream valley. The fine sand, silt, and clay within the sediments average 20 to 30 feet (6 to 9 meters) in thickness and may increase to a thickness of greater than 59 feet (18 meters) locally. These sediments serve as important aquifers for residential, industrial, and municipal use.

c. Steep Slopes and Slope Stability

The most significant topographic controls on surface mining activities within the study area are the steep slopes that are prevalent in the Appalachian Plateau Province. The slopes control both the volume and stability of excess material placement during filling. Steep slopes are the places where the mass movement of earth material is most likely to occur following mining or other disturbances. Landslides along highways are generally most common where slopes range between 20 percent and 35 percent (Hall 1980, Lessing et al., 1976). In many areas, more severe slopes already have been stabilized through slides and other earth movements, whereas these lesser slopes (20 percent to 35 percent) remain unstable and sensitive to mine-related disturbances. The regulations interpreting the Surface Mining and Reclamation Act (SMCRA) define steep slope as any slope of more than 20 degrees, or a lesser slope as may be designated by the regulatory authority of a state.

SMCRA regulations contain permitting, design, and construction monitoring requirements intended to implement state-of-the-art engineering standards for excess spoil disposal. The regulations and engineering standards are tailored to ensure meeting the SMCRA goals of long-term stability, public safety and environmental protection. To perform a retrospective study definitively evaluating the mass stability of large earth and rock structures requires intimate knowledge of representative shear strength parameters of the fill and foundation material, as well as definition of the phreatic surface within the fill. With reliable excess

spoil, geotechnical strength parameters and internal pore water pressure information (along with the dimensions of the fill, foundation, and bedrock) a stability analysis can provide accurate engineering estimates for the factor of safety of the fill. Various state regulatory programs routinely evaluate the company submission of this type of information in permits, evaluate the adherence to approved plans in monthly inspections, and assess the fills for signs of incipient or actual failure prior to making bond release decisions after construction. Company engineers and consultants perform extensive tests, stake their professional reputation and licenses on fill designs, document/certify critical construction phases, and attain certification quarterly. Valley Fill stability is discussed in Section III.K.

d. Unstable Slopes

Generally, most slope failures are confined to the thin layer of soil, colluvium, or weathered rock that develops on the steep valley slopes. Rockfalls are usually associated with the excavation activities of man, but they also may occur on natural cliff faces where meandering streams erode soft rocks that underlie more resistant sandstone bluffs. Any construction activity that involves: removal of vegetation, increased loading on the slope, undercutting the slope, or alteration of the hydrologic balance (surface water and groundwater), may induce slope failure. Coal mining and its related activities commonly involve all of these. Other factors that increase the potential for slope failure are as follows:

- Bedrock Factors - For example, the red shales of the Monongahela and Conemaugh Groups are naturally weak and incompetent. These red shales weather rapidly, especially when exposed, and are the rock type most commonly associated with landslides in West Virginia.

- Soil Factors - Easily erodible soils are thin, clayey soils weathered from shales. These soils are usually on steep inclines, impede groundwater infiltration, and are easily erodible.

- Slope Configuration - Naturally occurring or artificial concave slope configurations concentrate water, that lubricates joints to cause slope failure (Lessing et al., 1976).

Of the landslides studied in West Virginia, 69 percent occurred on concave slopes. In the Coal/Kanawha River Basin, the Muskingum-Upshur association presents a serious landslide hazard on slopes over 20 percent (11°). Muskingum-Upshur, Upshur, Vandalia, and Westmoreland soils also have a high landslide risk, and Brooke soils are moderately susceptible to landslides. The Meckesville, Shelocta, and Wharton series are to a lesser degree subject to slippage. These soils are the known soils to have the highest landslide risk in the Basin (Cardi et al., 1979). In the state of West Virginia the following soil series are susceptible to landslides: Brooke, Brookside, Clarksburg, Culleoka, Dormont, Ernest, Guernsey, Markland, Upshur, Vandalia, Westmoreland, Wharton, and Zoar. These soils are considered to be slide prone due to soil characteristics, percent slope and other variables.

Long, continuous precipitation events or sudden heavy rains may reduce the shear strength of soils and colluvium and load these materials sufficiently to produce landslides on steep dip and talus slopes. During coal mining on 25 percent to 36 percent slopes, spoil placed on the downslope, even temporarily, is highly susceptible to slope failure, especially during the spring rainy season (Lessing et al., 1976).

6. Climate

The climate within the study area is temperate and is favorable for many types of plants and animals. Generally, summers are warm and humid with winters moderately cold. Valleys can have lower temperatures than the surrounding hills when cooler heavier air drains to areas of lower elevations. Precipitation is fairly well distributed throughout the year. Seasonal temperatures, rainfall, snowfall, wind, and humidity differ from West Virginia, Kentucky, Tennessee and Virginia. An approximate average of 43 to 50 inches of rain falls on the Kentucky portion of the study area each year. Anywhere from 2 to 5 inches of rain can be expected in any given month. Approximately 52 to 55 inches of rain falls on the Tennessee portion of the study area in the average year. Anywhere from 3 to 6 inches of rain per month can be expected in this area with the wettest months being March and December and the driest month being October. Approximately 84 to 95 days throughout the year will experience greater than 0.10 inches of precipitation.

In the West Virginia portion of the study area, approximately 38 to 50 inches of rain occurs per year. Monthly rainfalls of 3 to 6 inches can also be expected in this area throughout the year. The wettest month tends to be July while the driest months are usually February, October, and November. In the Virginia portion of the study area, approximately 41 to 50 inches of rain occurs per year. Between 2 and 5 inches of rain can be expected in any given month of the year with the wettest months being March, May, and July and the driest month being October. Monthly temperature and precipitation data for each state within the study area are shown in tables presented in Appendix C.

C. APPALACHIAN AQUATIC SYSTEMS

1. Lotic (Flowing) Aquatic Systems

Lotic or flowing aquatic systems are important landscape features in the Mountain Top Mining/Valley Fills EIS Study area. Lotic systems may be considered to include rivers, streams, and creeks and springs. This section will discuss the types, features and functions of lotic systems in the study area.

a. Representative Streams

a.1. Physical Characteristics

Numerous physical parameters such as flow volume, substrate (i.e., the stream bottom made up of cobbles, gravel, sand, etc.), water chemistry, and bank cover influence the biota of the aquatic systems in the study area. These parameters are determined by the climate, lithology, relief and land use in the area of a particular stretch of stream. Many of these factors have been discussed in other chapters of this EIS.

EVEN WHERE INACCESSIBLE TO FISH, THESE SMALL STREAMS PROVIDE HIGH LEVELS OF WATER QUALITY AND QUANTITY, SEDIMENT CONTROL, NUTRIENTS AND WOOD DEBRIS FOR DOWNSTREAM REACHES OF THE WATERSHED. INTERMITTENT AND EPHEMERAL HEADWATER STREAMS ARE, THEREFORE, OFTEN LARGELY RESPONSIBLE FOR MAINTAINING THE QUALITY OF DOWNSTREAM RIVERINE PROCESSES AND HABITAT FOR CONSIDERABLE DISTANCES.

a.2. Stream Classification

Streams are generally classified through a system called stream ordering (Strahler, 1957). This system classifies streams based on size and position within the drainage network. A first-order stream is defined as not having tributaries. The confluence of two streams of the same order produces the next highest order. For example, the joining of two first-order streams results in a second-order stream. The joining of two second-order streams produces a third-order stream, etc. Headwaters are usually classified as first- through third-order streams, mid-sized streams as fourth- through sixth-order streams, and larger rivers as seventh- through twelfth-order streams (Ward, 1992). First order streams in the study area account for approximately 60% of total stream miles as represented by blue lines at the 1:100,000 scale USGS topographic map (EPA Region III June 2000 comments). This classification system can be misleading when just using blue lines on printed maps to indicate stream orders. It is known that there are many more miles of first order streams actually present in the field than appear on most commonly used maps . Therefore, this classification system includes some uncertainty. Stream ordering, though useful in placing a stream reach within an entire stream system, is not necessarily a meaningful description of the physical component of the stream reach itself.

In addition to first-through twelfth-order streams, ephemeral streams and intermittent streams occur in the Appalachian region. Ephemeral and intermittent streams have been defined in various ways depending on the regulatory program. Appendix B of this EIS presents the various definitions.

Generally, ephemeral streams have a discrete channel and flow only in direct response to precipitation events. In contrast, flow in intermittent streams is periodic or seasonal and based on

the presence groundwater. Perennial streams are those streams that maintain flow year round. The starting points of the intermittent and perennial streams may vary from year to year depending how wet or dry years have effected the groundwater table. Flow is permanent, but of a relatively low volume in first and second order perennial streams with flow volumes generally continuing to increase with stream order.

a.3. Habitats in Streams

Generally, headwater streams originate at high elevations in the study area. Substrate patterns in headwater streams channels are typically comprised of coarser material such as boulders, cobble rubble and bedrock. Large, woody debris often contribute to the substrate complexity in headwater streams. Small pools with finer sediments may also be found along headwater streams. Typical substrate patterns in larger rivers are comprised of finer material such as silt and sand. Mid-sized rivers typically contain a blend of cobble and gravel with some finer sediment interspersed in areas of slower flow.

Although intermittent streams tend to go dry for a portion of the year, macroinvertebrate life still exists within its channel. In a study of intermittent and perennial streams in Alabama, assemblages of normally intermittent streams did not differ greatly from those of nearby permanent or perennial streams (Feminella, 1996). Data recently collected in conjunction with this EIS (Interagency Invertebrate Study, 2000), suggests similar findings for ephemeral/intermittent streams in the study area. These data show that biological communities in the study area streams are present as soon as there is flowing water. During periods of no visible streamflow, interstitial water flows through the material below the steam. This special hydrology creates a unique habitat, called the hyporheic zone. Specially-adapted macroinvertebrates are able to continue their life cycles by burrowing into the hyporheic zone, especially in times of drought. Other macroinvertebrates live completely within the hyporheic zone (Hynes, 1970).

The combination of substrate characteristics and varying flow rates and other flow characteristics (hydrologic cycles, flow patterns, load transport and storage) produce channel features such as riffles, runs, and pools. Riffles are erosional habitats where surface water flows over coarser substrate, creating turbulence, which causes disturbances in the surface of the water. This turbulence increases levels of dissolved oxygen by encouraging the mixing of oxygen in the air with the water. Pools are depositional areas where flow is slow or stagnant, allowing finer particulate matter to settle onto the stream bottom. Runs are moderately fast sections of streams where the water surface is not as disturbed. Headwater streams, typically consist of alternating riffles and runs though small depositional pools, may be present and represent an important microhabitat. Mid-sized rivers typically contain all three features because increased width and depth allow more variation in flow.

Stream features that are important in determining habitat for aquatic organisms include, overhanging vegetation, the presence and characteristics of leaf packs, in-stream vegetation, large woody debris, undercut banks, and exposed tree roots. Overhanging vegetation consists of riparian shrub and herbaceous vegetation on banks that grows over and sometimes into the surface water. In-stream vegetation occurs where proper substrate and flow conditions allow growth. Snags are pieces of wood that have accumulated in a stream area. Undercut banks and exposed tree roots are caused by a combination of unstable banks and fast streamflow. All of these features provide unique habitat for cover, habitat, and food for macroinvertebrates and fish.

Other in-stream features that provide additional habitat include littoral areas such as shorelines, sandbars, and islands. Typically these features exist most prominently in depositional systems such as larger rivers. These littoral areas are important shallow habitats, which provide habitat for smaller fish and macroinvertebrates that are unable to live in the deeper sections of the river.

Wetlands and riparian zones may occur along streams. Wetlands and riparian zones may influence the physical characteristics of streams, thereby affecting stream habitats. In addition, wetlands and riparian zones may be used by stream biota directly during periods of elevated flow. Wetlands are crucial transition zones between terrestrial and aquatic habitats. They are defined as areas "that are inundated or saturated by surface or groundwater at a frequency and duration sufficient to support, and that under normal circumstances do support, a prevalence of vegetation typically adapted for life in saturated soil conditions" (COE, 1987). Wetlands can be found on floodplains along rivers and streams (riparian wetlands). Typical steep geomorphology of headwater streams usually prohibits the formation of a floodplain, so wetlands are usually restricted to small depressional areas. As the gradient of the land becomes more gradual, more wetlands are found on the floodplain of the stream. Wetlands associated with rivers can take the form of forested wetlands, emergent marshes, wet meadows or small ponds. The unique characteristics and vegetative composition of wetlands provide important habitat for many species of aquatic macroinvertebrates, amphibians, and reptiles.

b. Energy Sources and Plant Communities

Aquatic ecosystem energy sources consist of allochthonous (organic material produced outside the stream such as leaves, wood, etc.) and autochthonous (instream primary production by plants, algae) sources. Allochthonous materials reach the stream either through directly falling into the stream or through indirectly being transported into the stream, commonly though wind movement or runoff. Allochthonous organic material has been found to be the predominant energy source in high-gradient streams of the southern Appalachians (e.g., Hornick et al., 1981, Webster et al., 1983, Wallace et al., 1992). Headwater energy sources are important, not only to invertebrates and vertebrates in upper reaches of the watershed, but, excess organic carbon is subsequently utilized by life forms in all stream orders down gradient. Since streams have a unidirectional flow, downstream areas are also dependent on upstream areas for portions of their energy (Vannote et al. 1980).

Plant communities of high-gradient streams live in what may be considered to be a physically challenging environment. Frequently these habitats are densely shaded and subject to high current velocities. As a result, the plant communities in high-gradient streams are reduced relative to lentic habitats and low-gradient streams (Wallace et al., 1992). However, the plant communities occurring in high-gradient streams contain flora uniquely adapted to survive in this type of environment. This habitat also supports an abundance of flora considered to be endemic (i.e., not found in other locations) to the region (Patrick, 1948). Possibly, the historic lack of direct anthropogenic (human-induced) disturbance to watersheds of high-gradient streams may have contributed to the survival of the unique and endemic flora of this region (Wilcove et al., 1998).

b.1. Vascular Plants and Bryophytes

Vascular plants, such as aquatic macrophytes or ferns, found in high-gradient streams typically have adventitious roots, rhizomes, flexible stems and streamlined narrow leaves (Westlake 1975, Wallace et al. 1992). In contrast, bryophytes (mosses and liverworts,) live closely oppressed to rocks and

boulders and are characterized by a small body size. In streams with high turbulent flow, mosses and liverworts have been found to be the dominant group of macrophytes (Westlake, 1975).

b.2. Algae

As summarized in Wallace et al. (1992), the algae of high-gradient streams are limited to species capable of anchoring to stable substrates, preferably large stable objects. Algae may temporarily colonize smaller objects during periods of low flow. The major groups of algae represented in high-gradient streams include red algae (Rhodophyta), filamentous green algae (Chlorophyta), and diatoms (Bacillariophyta) (Wallace et al.1992). Endemic and unique species of algae are common to the high-gradient streams of the southern Appalachians as described in Wallace et al. (1992).

b.3. Primary Production

Primary production is the input of energy into a system by the growth of flora living in the system. In streams, primary production is generally measured as mass of carbon or ash free dry mass, which is largely carbon, per unit area, per year. Primary production rates in Appalachian streams have been shown to vary with stream order, season, degree of shading, nutrients, and water hardness (Wallace et al., 1992). Although under some circumstances, gross primary production can be high (see Hill and Webster 1982b [in Wallace et al., 1992]), typical primary production inputs appear to range from approximately 9 to 446 pounds of carbon per acre of stream per year (Keithan and Lowe 1985, Rodgers et al., 1983, Wallace et al., 1992).

b.4. Allochthonous Energy Sources and Processing

Allochthonous energy sources consist primarily of leaves and woody material. However, dissolved organic carbon (DOC) from a variety of sources is an additional allochthonous energy source. Sources of DOC external to the stream include groundwater or runoff. Sources internal to the stream relate largely to leaching of organic matter from detritus or other organic matter. Fisher and Likens, in Science Applications International Corporation (1998), explain that over 90 percent of the annual energy inputs to small forested streams can be attributed to leaf detritus and dissolved organic carbon from the terrestrial environment. Webster et al. (1995) further discusses sources for organic inputs to streams.

The estimate of almost 3600 pounds of carbon per acre of stream per year developed by Bray and Gorham (1964) as a measure of leaf and wood litterfall into a stream per year, is considered to be a good estimate for input into high-gradient Appalachian streams. The mass of material input as leaf fall is generally greater than that input as woody material. However, in some circumstances the mass of input as woody material may equal that of leaf input (Webster et al., 1990).

Woody Material

In addition to functioning as an energy source, woody material may provide other important stream functions relating to hydrology and habitat structure. These functions may include contributing to stair-step stream bed profiles that result in rapid dissipation of the stream's energy; forming micro-pools or sieve-like structures that retain other particulate organic material, which may influence

trophic and nutrient dynamics; providing fish habitat; providing a substrate for some stream invertebrates; and functioning as a food source for wood-eating organisms (Wallace et al., 1992).

Organic Matter Processing

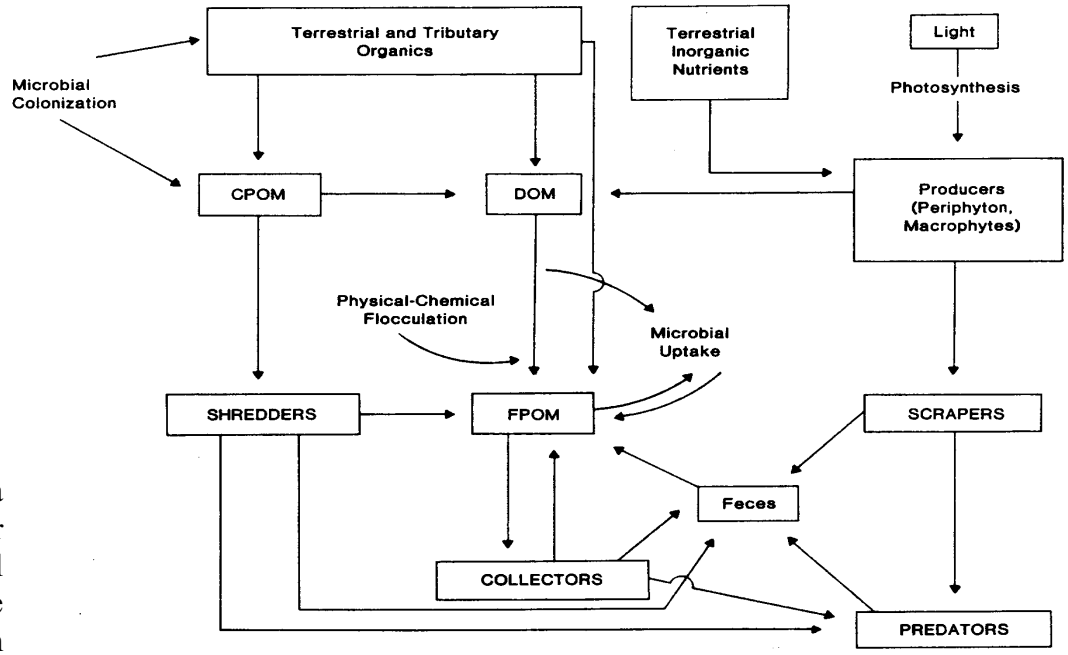
The headwater stream (first- through third-order) is the origin for energy processing within the river ecosystem. Headwater streams in the study area are located in forested areas and are characterized by a heavy leaf canopy and low photosynthetic production. Sources of energy for headwater streams are allochthonous in origin or derived from the terrestrial environment. The vast majority of this allochthonous material arrives in the streams in the form of Coarse Particulate Organic Matter or CPOM (> 1 mm or 0.039 inch in size). Smaller amounts of other allochthonous material that is transported to the stream includes Fine Particulate Organic Matter (FPOM, 50 um – 1 um in size or 0.0019 - 0.000039 inches in size) and Dissolved Organic Matter (DOM) traveling from surface and groundwater flow. Microbes and specialized macroinvertebrates living in headwater streams, called shredders, feed on the DOM and CPOM, converting it into FPOM and DOM. The FPOM and DOM are carried downstream to mid-sized streams.

Because mid-sized streams (fourth- through sixth-order) are wider than headwater streams, the canopy is usually more open and more light is able to penetrate to the stream bottom. As a result, a greater abundance of algae and aquatic plants are able to grow along the stream bottom. In general, the contribution of allochthonous material derived from terrestrial vegetation in midsized streams is less than in the headwater streams. Autochthonous material, meaning material that is derived from within the stream, becomes an important component of the energy budget in midsized streams. Autochthonous material includes both the primary productivity of the stream and the FPOM and DOM derived from upstream reaches which flow into midsized stream. Consequently, mid-sized streams may exhibit a shift from a heterotrophic to an autotrophic system, or one that generates its own energy through photosynthesis. The biological community of mid-sized streams differs somewhat from that in headwater streams in part because of the more diverse types of energy sources that are available. Specialized macroinvertebrates called collectors-filterers and collector-gatherers break down the FPOM carried from upstream reaches into Ultra-fine Particulate Organic Matter (UPOM, 0.5 – 50 mm in size or 0.019-1.97 inches in size). These macroinvertebrates, as well as microbes, also consume living plant matter (algae and aquatic plants) converting it into additional forms of energy. The UPOM derived from these energy sources is then carried downstream to larger rivers. Interestingly, collectors can actually also increase particle sizes in some cases by feeding on material in the several micron range and defecating compacted feces of a much larger particle size. These larger particles then become available to larger particle feeding detritivores (Wallace et. al., 1992).

Larger rivers (seventh- through twelfth-order) have different biological communities from lower order streams. The increased width of these rivers results in relatively insignificant allochthonous inputs. The depth, combined with suspended mineral and organic matter, prohibit much light penetration and consequent growth of algae and plants within the main channel. Collectors again become the primary macroinvertebrate community to process the particulate organic material. Larger rivers tend to be heterotrophic systems.

Figure III.C-1 illustrates the flowchart summarizing the energy processing that occurs within the river ecosystem.

Figure III.C-1
Energy Resource Categories and Invertebrate Classifications in River Ecosystems
(from Merritt and Cummins, 1996)



Several major models have been developed to describe the movement of energy and nutrients in rivers. These theories include the River Continuum Concept developed in Vannote et al. (1980) and the concept of nutrient spiraling. The development of the River Continuum Concept greatly improved the scientific communities' understanding of the ecosystem-level functions of rivers and provided direction for lotic ecosystem research over the last 20 years.

River Continuum Concept

The River Continuum Concept (Vannote et al., 1980) is a theory that details how differing energy sources are processed efficiently, progressing from headwater streams to large rivers. This theory explains that energy sources are dependent upon geomorphological, chemical, and biological factors that have evolved within the surface water ecosystem to create a balanced energy transport. The

general metabolism for the river ecosystem uses energy that is transported downstream from upstream reaches within the system.

From the headwaters to the mouth of the river, the river ecosystem is comprised of a balanced, efficient, longitudinal gradient of energy sources and processing in which the particle size of organic matter becomes more refined as the river becomes larger (Vannote et al, 1980). In each portion of a river ecosystem, some organic matter is processed, some stored, and some released (Vannote et al., 1980). Organic matter is conditioned by microbes (fungi and bacteria), and some is respired (to carbon dioxide) by microbes and animals, some converted to smaller particles and dissolved organic matter which is exported to downstream communities (Vannote et al. 1980). Macroinvertebrate communities at each section of the river ecosystem have become specifically adapted to maximize the processing of energy available in the form of organic matter. Since macroinvertebrate communities serve as a food base for higher trophic organisms (i.e., fish) in the food web, these higher trophic organisms have also evolved to fit available niches in the stream ecosystem. Figure III.C-2 summarizes the River Continuum Concept and the types of benthic macroinvertebrates mentioned that are typically distributed along the river ecosystem. General range of stream widths (in meters) are given for each order.

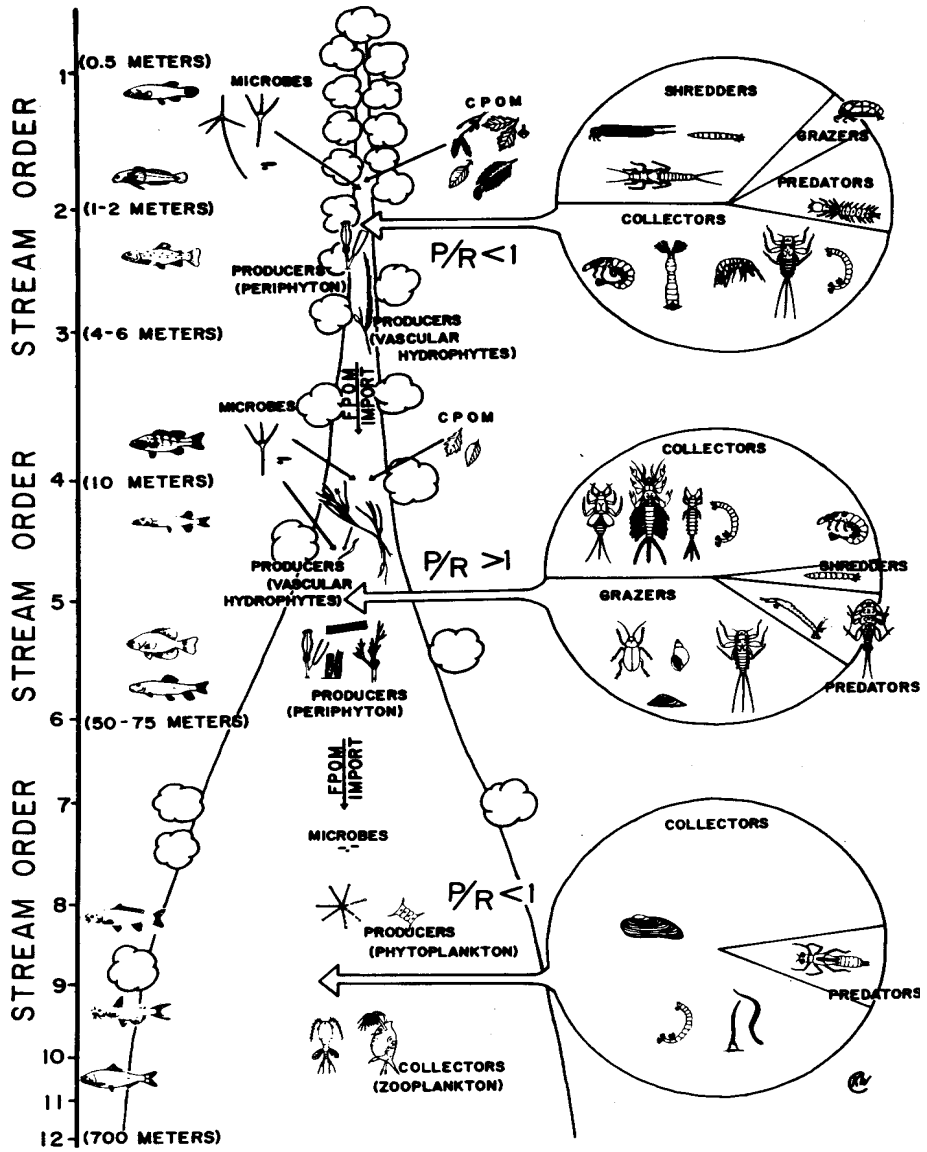
Heterotrophic systems are designated by the P/R ratio (gross photosynthesis to community respiration ratio) < 1, and autotrophic systems are designated by the P/R > 1.

c. Animal Communities

c.1. Invertebrates

Stream order typically dictates the community structure of the resident aquatic life. Headwater streams harbor primarily benthic macroinvertebrate communities who are specialized to feed on the CPOM deposited in the system. Examples of benthic macroinvertebrates include crayfish, worms, snails and flies. The majority of benthic macroinvertebrates in headwater streams are classified as shredders and collectors, who feed on the CPOM and FPOM, and predators who feed on the other macroinvertebrates. Typical benthic macroinvertebrates found in headwater streams in the study area include insects such as mayflies (Ephemeroptera), stoneflies (Plecoptera), caddisflies (Trichoptera), dragonflies and damselflies (Odonata), beetles (Coleoptera), dobsonflies and alderflies (Megaloptera), true bugs (Hemiptera), springtails (Collembola), and true flies (Diptera). Other macroinvertebrates that have been collected include crayfish (Decapoda), isopods (Isopoda), worms (Oligochaeta and Annelida) and snails (Gastropoda) (FWS, 1998; Science Applications International Corporation, 1998).

Figure III.C-2
Diagrammatic Representation of the River Continuum Shown as a Single Stream of Increasing Order
(Vannote et al., 1980)



In the southern Appalachian Mountains, macroinvertebrates of several orders including Ephemeroptera, Plecopter and Trichoptera have been found to be rich in species, including many endemic species and species considered to be rare. This diversity and unique assemblage of species has been attributed to the unique geological, climatological and hydrological features of this region (Morse et al., 1993, Morse et al., 1997). Many biologists agree that the presence of a biotic community with such unique and rare populations should be considered a critical resource.

Stream macroinvertebrates are typically classified on the basis of their functional feeding group (Cummins 1973, Cummins and Klug 1979, Merritt and Cummins 1984). Insects within a functional feeding group share similarities in their morphology, feeding behavior and feeding mechanisms (e.g., scraping, collecting, shredding, filtering, etc.). Typical functional feeding groups are described below.

Scrapers

Scrapers are adapted to scape materials such as, algae or periphyton and its associated microflora from rock or organic substrates, such as leaves (Wallace et al., 1992). Typically scrapers include certain taxa of snails, mayflies, caddisflies, beetles and fly larvae.

Shredders

Shredders chew primarily large pieces of decomposing vascular plants (≥ 1 mm or 0.039 inch diameter) along with its associated microflora and fauna. They may also feed directly on living vascular hydrophytes or gouge decomposing wood submerged in streams (Wallace et al., 1992). In addition to aquatic insects, many omnivorous crayfish in the study area are facultative shredders. Shredders are important because their mode of feeding causes the generation of large quantities of small particles. These particles are more easily transported downstream and may be acted on by microbes more easily due to the increase in the surface area to volume ratio. Common shredders in the study area are certain taxa of stoneflies, caddisflies and fly larvae.

Collector-gatherers

Collector-gatherers feed primarily on fine pieces of decomposing particulate organic matter (FPOM ≤1 mm or 0.039 inch diameter) deposited within streams (Wallace et al., 1992). Many chironomidae larvae are collector-gatherers.

Collector-filterers

Collector-filterers have specialized anatomical structures (setae, mouthbrushes, fans, etc.) or silk and silk-like secretions that act as sieves to remove particulate matter from suspension (Jorgensen 1966, Wallace and Merritt, 1980) (Wallace, 1992). Some mayflies, caddisflies and fly larvae are collector-filterers.

Predators

Predators feed on animal tissues by either engulfing their prey or by piercing prey and sucking body contents (Wallace et al., 1992). Predators include dragonflies, hellgrammites, some taxa of stoneflies, caddisflies, beetles, fly larvae and some crayfish.

c.2. Vertebrates

Two groups of vertebrates, fish and salamanders are the major stream-dwelling vertebrates in the study area. Typically, salamanders occupy small, high-gradient headwater streams while fish occur

farther downstream. Predation by fish is believed to restrict salamanders to the smaller streams or the banks of large streams (Wallace et al., 1992).

Fish species present in headwater streams tend to be representative of cold water species, and primarily sustained by a diet of invertebrates (Vannote et al, 1980). As found with invertebrates and amphibians, the fish assemblages of the Appalachians tend to contain a relatively large number of endemic and unique species. Some fish species collected in the pristine headwaters of West Virginia include blacknose dace (*Rhinichthys atratulus*), creek chub (*Semotilus atromaculatus*), and slimy sculpin (*Cottus cognatus*) (FWS, 1998).

Many different kinds of amphibians and reptiles live in or near streams and wetlands. Many types of amphibians in particular are unique to the Appalachian regions. The West Virginia Division of Natural Resources has published a pamphlet, "Amphibians and Reptiles of West Virginia: A Field Checklist." This list mentions 46 amphibious species and 41 reptilian species, the vast majority of which are most likely located throughout the study area within suitable habitat of Kentucky, Tennessee, and Virginia. These species include mole, dusky, woodland, four-toed, green, spring, red, mud, and brook salamanders as well as newts, hellbenders, and mudpuppies, which can frequently be found near aquatic habitat. Skinks, a lizard species, can also be found around aquatic habitats. Toads as well as cricket, chorus, true, leopard, pickerel, and treefrogs are associated with aquatic habitats. Snapping, spotted, map, musk, mud, and painted turtles as well as sliders, cooters, redbellies, and softshells can also be found in these areas. Water, crayfish, brown, garter, ribbon, and kingsnakes are associated with aquatic habitats. Many of these amphibious and reptilian species may be primarily terrestrial, but live in proximity to aquatic areas such as streams and wetlands. In addition, several species strictly rely on the presence of streams or wetlands for at least part of their life cycle (Conant and Collins, 1991).

The diversity and distribution of fishes in West Virginia is intimately related to drainage divides. The Potomac and James rivers drain the Atlantic Slope, while the remainder of the state drains to the Gulf of Mexico via the Ohio and Mississippi rivers. The fauna of all West Virginia systems draining into the greater Ohio River are similar in composition and have an interrelated history. The greater Ohio River drainage is chiefly comprised of the Monongehela, Little Kanawha, Kanawha, Guyandotte, and Big Sandy/Tug Fork rivers. The upper Kanawha (New) River system above the Kanawha Falls has a unique fauna with six endemic species; the bigmouth chub (*Nocomis platyrhynchus*), the New River shiner (*Notropis scabriceps*), the Kanawha minnow (*Phenacobius teretulus*), the candy darter (*Etheostoma osburni*), the Kanawha darter (*Etheostoma kanawhae*), and the Appalachia darter (*Percina gymnocephala*); all but *E. kanawhae* occur in West Virginia. For this reason, the New River is treated separately from the greater Ohio River drainage with respect to fish distribution. In the ichthyological literature, New River refers to all of the Kanawha River drainage above Kanawha Falls (Stauffer and Ferreri, 2002).

A shift in the fish community from cold-water to more warm-water fish species occurs in mid-sized streams. Generally, the fish community becomes more diverse and more piscivores (fish-eaters) coincide with the invertivores (Vannote et al, 1980). Studies have determined that approximately 277 native freshwater fish species, distributed among 22 families exist within the central Appalachian drainages (EPA, 1983). Minnows, suckers, catfishes, sunfishes, and perches are the five predominant families. (EPA, 1983). The lack of modifications, combined with numerous geological, climatic, and hydrological events in eastern Kentucky have allowed the rivers to harbor

a fairly diverse fish community (EPA, 1983). In addition, the geological events associated with the development of the river system within the MTM/VF EIS study area have resulted in a unique fishery system which has importance in the evolution and speciation of North American freshwater fishes (Stauffer and Ferreri, 2002).

d. Ecosystem Function

The value of headwater streams in the study area was the subject of a symposium held in April 1999. The proceedings of this symposium have been included in Appendix D and are summarized below.

The changes in invertebrate communities from stream headwaters to mouth have been well documented. However, local conditions may exert as great or greater an influence on the biotic communities as can be seen by examining stream order alone. In general, major shifts in the relative abundance of macroinvetebrates considered to be shredders, scrapers and collector-gatherers are seen from headwaters to mouth. Collector-filterers and predators are generally found in all stream orders. However, differing species may occur to occupy these niches in different stream reaches. Shredders are generally relatively abundant in headwater areas where allochthonous inputs are high, and present in lower abundance in mid-order streams, where less of the organic matter input is allochthonous. Shredders may be absent or occur in only localized conditions in higher order streams. Scrapers tend to be present at a relatively low abundance in headwater streams owing to the relatively low amount of periphyton (periphyton inhabiting the surfaces of underwater vegetation, rocks, and other substrates) present in these stretches. The relative abundance of scrapers increases in mid-order streams in conjunction with an increase in periphyton abundance, but decreases again in high order streams owing to decreases in suitable habitat and physical limitations. Collector-filters are present in all reaches of a stream. However, the species occupying these niches varies tremendously, from almost entirely arthropods in headwater streams to largely molluscs and arthropods, especially aquatic insects, in high-order rivers.

Small streams play a pivotal role in lotic ecosystems. Small streams:

- Have maximum interface with the terrestrial environment with large inputs of organic matter from the surrounding landscape
- Serve as storage and retention sites for nutrients, organic matter and sediments
- Are sites for transformation of nutrients and organic matter to fine particulate and dissolved organic matter
- Are the main conduit for export of water, nutrients, and organic matter to downstream areas (Wallace in Symposium on Aquatic Ecosystem Enhancement at Mountain Top Mining Sites, January 2000)

The major functions of headwater streams can be summarized into two categories, physical and biological (Wallace in Symposium on Aquatic Ecosystem Enhancement at Mountain Top Mining Sites, January 2000):

Physical

- Headwater streams tend to moderate the hydrograph, or flow rate, downstream
- They serve as a major area of nutrient transformation and retention

- They provide a moderate thermal regime compared to downstream waters- cooler in summer and warmer in winter
- They provide for physical retention of organic material as observed by the short "spiraling length"

Biological

- Biota in headwater streams influence the storage, transportation and export of organic matter
- Biota convert organic matter to fine particulate and dissolved organic matter
- They enhance downstream transport of organic matter
- They promote less accumulation of large and woody organic matter in headwater streams
- They enhance sediment transport downstream by breaking down the leaf material
- They also enhance nutrient uptake and transformation

In summary, light and the input of allochthonous material are the two limiting factors in the contribution of energy to a river ecosystem as a whole. When an energy source is altered or removed in the upstream reaches, downstream biological communities are also affected. The value of headwater streams to the river ecosystem is emphasized by Doppelt et al. (1993): "Even where inaccessible to fish, these small streams provide high levels of water quality and quantity, sediment control, nutrients and wood debris for downstream reaches of the watershed. Intermittent and ephemeral headwater streams are, therefore, often largely responsible for maintaining the quality of downstream riverine processes and habitat for considerable distances."

2. Lentic (Non-flowing) Aquatic Systems and Wetlands

a. Overview

Lentic aquatic systems are defined as non-flowing water bodies such as lakes and ponds. Strausbaugh and Core (1978) states that there are no natural lakes and ponds in West Virginia (other than beaver ponds). This statement highlights several features of the lentic systems found in the study area. Virtually all lentic systems in the study area have been formed by impounding flowing water systems. The majority of the lentic systems in the study area are small ponds. Small impoundments are constructed for agricultural use, community water supplies, recreational areas, or flood control, or may have resulted from road construction or surface mining activities (Menzel and Cooper, 1992).

There is no clear distinction between a pond and a lake. Attempts have been made to classify lentic water bodies as ponds or lakes depending on depth and on surface area. A reasonable distinction between ponds and lakes may be made on the type of lake mixing that occurs. Water bodies may be considered lakes when the wind plays the dominant role in mixing. In ponds, gentler convective mixing predominates (Goldman and Horne, 1983).

ON A REGIONAL SCALE, SMALL PONDS OR IMPOUNDMENTS IN THE APPALACHIANS PROVIDE HABITAT FOR COMMON ANIMAL AND PLANT POPULATIONS THAT REQUIRE AQUATIC CONDITIONS FOR FEEDING OR REPRODUCTION.

Wetlands are also a water-related system that occurs throughout the study area. As per section 404 of the Clean Water Act, wetlands are defined as:

> Those areas that are inundated or saturated by surface or groundwater at a frequency and duration sufficient to support, and that under normal circumstances do support, a prevalence of vegetation typically adapted for life in saturated soil conditions.

As can be seen from this definition wetlands and lentic aquatic systems may be overlapping. Note that this regulatory definition does not define shallow lakes and ponds as wetlands. For resource mapping purposes, the FWS (Cowardin et al. 1979) has also defined wetlands as follows:

> Wetlands are lands transitional between terrestrial and aquatic systems where the water table is usually at or near the surface or the land is covered by shallow water. For purposes of this classification, wetlands must have one or more of the following three attributes: 1. At least periodically, the land supports predominantly hydrophytes; 2. The substrate is predominantly undrained hydric soils; and 3. The substrate is non-soil and is saturated with water or covered by shallow water at some time during the growing season of each year.

In this definition, shallow lakes and ponds are included as wetlands. Wetlands are frequently mapped using the classification system developed by Cowardin et al. (1979). In this system, some types of lentic systems (i.e. lakes) are designated as deepwater habitats as distinct from wetlands, while ponds are typically considered to be a type of palustrine wetland.

b. Physical Environment

Four elements play a major role in defining the structure of a lake or pond. These include the physical characteristics, chemical characteristics, biological characteristics and the watershed for a particular pond or lake.

The hydrology of the lentic systems in the study area is dependent, in many cases, on both surface runoff, as most ponds are formed by damming a small stream, and by groundwater input. Springs and other gains from groundwater may provide the majority of the water to some ponds (Menzel and Cooper, 1992). Studies have found that water levels in Appalachian impoundments tend to remain fairly constant over the year. However, sediment inflows may greatly reduce the capacity of impoundments, especially in the years immediately following impoundment construction.

Watershed conditions can greatly affect conditions in Appalachian impoundments. For example, ponds located in a forested setting would tend to receive more allochthonous input than ponds located in agricultural settings. Depending on the variation in inputs to ponds, i.e., terrestrial detritus versus algae in more open reaches, the change in energy base can also influence the food base and the community structure of ponds.

Small impoundments in this region are usually classified as soft water with dissolved solids less than 120 mg/L and hardness less than 60 mg/L as Ca CO3 (Geraghty et al., 1973). Even in limestone regions dissolved solids rarely exceed 350 mg/L with a maximum hardness of 120 mg/L (Menzel and Cooper, 1992). Impoundment pH typically ranges from 4.1 to 10. Most Appalachian impoundments are found to be phosphorus limited, as is true for most freshwater bodies (Menzel and Cooper, 1992).

c. Energy Sources and Plant Communities

Plant communities in ponds and lakes consist of submerged, floating and emergent vascular plants, phytoplankton, and periphyton. Autotrophic bacteria may also occur in lentic systems and contribute to the primary production of these systems.

c.1. Phytoplankton and Benthic Dwelling Micro-organisms

Phytoplankton

All major groups of algae are found in small ponds. However, the species distribution of small ponds generally differs from that of large impoundments and lakes. In small ponds, benthic algae and periphyton may detach and become part of the planktonic community (Menzel and Cooper, 1992).

If nutrient enrichment is present, blue-green algae (i.e., cyanobacteria) in small ponds may become dominant. This results in negative impacts from several perspectives. Blue-green algae is often considered noxious to humans and are often rarely consumed by planktivores.

Bacteria and Fungi

Bacteria and fungi are the major decomposers in small ponds. Although these organisms may occur as part of the planktonic community, the vast majority of bacteria and fungi are found on or in the top several centimeters of sediments. Bacteria and fungi may also represent a food source for benthic dwelling organisms.

c.2. Vascular Plants

Vascular plants in small impoundments include species with submergent, float-leaved or emergent growth forms. Submergent macrophytes are found rooted in benthic sediments at depths from 3 to 12.5 feet depending on light penetration. Submergents may occur in patches or may cover the entire bottom of ponds.

Floating or floating-leaved vascular plants may be very abundant in small ponds if nutrients are present. Where these plants are found in abundance, they may reduce the photosynthesis in the hypolimnion, (cold lower layers of a body of water) resulting in an increase in water column respiration. This may result in anoxic (low amounts of oxygen in the water) conditions in the water column, with elimination of fish in the pond (Menzel and Cooper, 1992).

Emergent macrophytes typically occur where sedimentation or benthic morphology has resulted in sediments located at a suitably shallow depth from the surface of the water. Examples of emergent species common to ponds in the Appalachian Mountains include cattails (*Typha latifolia*) and willows (*Salix sp.*). Emergent macrophytes are an important energy source for small impoundments and provide habitat for numerous vertebrate wildlife (Menzel and Cooper, 1992).

Small ponds tend to fill with sediments as they age. This results in changes in the plant community beginning with sparse populations of non-persistent emergents and submergents in the first several years after impoundment. Pond vegetation 8 to 25 years after impoundment may be characterized as latter successional wetland plant communities consisting of woody vegetation on the pond margin, emergent persistent vegetation located inside the woody margin, and a pond surface and substrate largely covered by submergent or floating-leaved species or absent entirely (Gunn, 1974).

c.3. Primary Production

Most ponds found in the southern Appalachians tend to be highly productive, eutrophic systems, (having concentrations of nutrients optimal or nearly so for plant or animal growth), although some small impoundments in this area may be oligotrophic (low concentrations of plant nutrients and hence low productivity). Submergent or emergent vegetation is the primary source of primary production in these systems (Menzel and Cooper, 1992). The presence of nutrients, light penetration, and temperature appear to be the major factors influencing primary production in small impoundments in the study area.

d. Animal Communities

Animal communities may be arbitrarily divided into two groups: those dwelling in the benthos and those dwelling in the water column. Often organisms move between these two zones during their lifecycle (Menzel and Cooper, 1992). Invertebrate groups found in small impoundments include zooplankton and insect larvae. Major vertebrate groups include fish and reptile. Birds may heavily utilize vegetated portions of the benthos for feeding and breeding.

d.1. Invertebrates

Pond invertebrates may function as primary consumers or secondary consumers and also represent a major food source for fish.

Zooplankton

Major groups of zooplankton include the Cladocera, Copepoda and Rotifera. Zooplankton populations exhibit seasonal population cycles, which may be controlled by a variety of factors. Zooplankton may feed on phytoplankton, detritus or other zooplankton. They are considered to be important in the nutrient cycling dynamics of small ponds.

Zoobenthos

Major groups of benthic dwelling organisms in ponds include aquatic oligochaetes (worms), crustaceans and immature insects. Feeding modes for zoobenthos include herbivorous, carnivorous and detrital feeding. Organisms feeding on detritus may actually obtain a majority of their energy from the microbial fraction of the detritus (Walker, Olds and Merritt, 1988). Zoobenthos greatly increase the secondary productivity in ponds through exhibiting high growth rates (Cooper, 1987). For example, some Chironomidae (Midge flies) may experience up to 10 life cycles per year in southern Appalachian ponds (Cooper, 1987).

d.2. Vertebrates

Five major groups of vertebrates are found in small impoundments in the southern Appalachians including fish, amphibians, reptiles, birds and mammals. These animals inhabit or use freshwater ponds for feeding or breeding during at least some part of their lifecycle. Available literature indicates a limited species diversity in all groups except birds (Menzel and Cooper, 1992).

Fish are generally the dominant predators in ponds. Predominant types of fish in small impoundments include bluegill and other sunfish, brown bullhead, bass, yellow perch and golden shiner. Frogs, turtles, and water snakes are other commonly occurring vertebrate species found in small impoundments.

e. Ecosystem Function

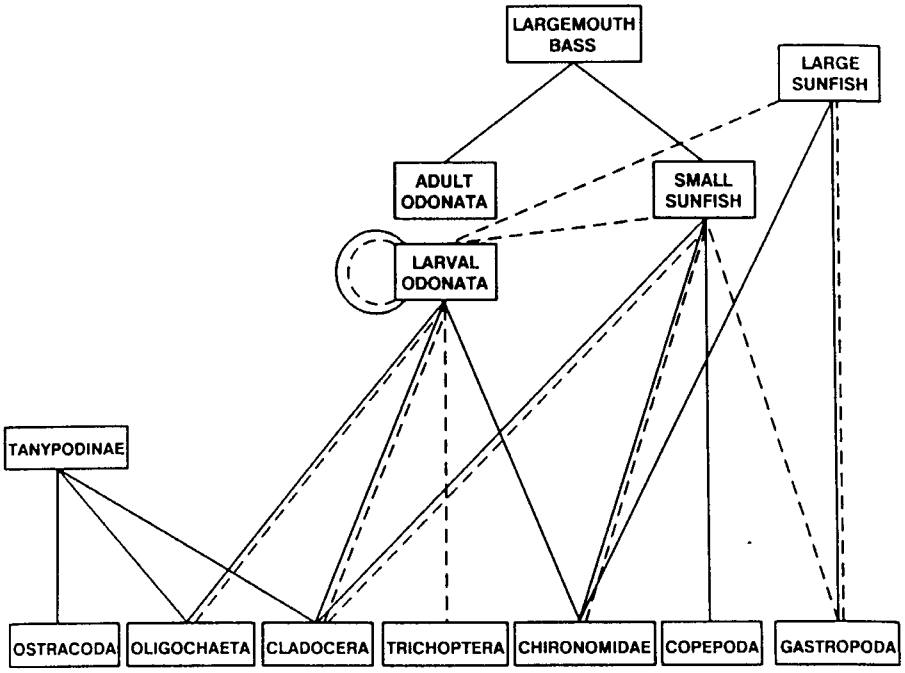
Small ponds or impoundments serve a variety of functions within the regional ecosystem, but also exhibit distinct internal ecosystem dynamics. On a regional scale, small ponds or impoundments in the Appalachians provide habitat for common animal and plant populations that require aquatic conditions for feeding or reproduction. These may include animal species such as beaver, waterfowl, fish or pond-dwelling obligate aquatic plant species. Small impoundments may contribute to flood control, and may improve the water quality of riparian systems downstream from the impoundment through the temporary removal of organic and inorganic nutrients and toxic materials from water that pass through them (Mitsch and Gosselink, 1993).

Ecosystem-level functions occurring within small ponds and impoundments include food web and the related energy flow relationships. Food webs in pond systems are well-developed and have been well studied (Johnson and Crowley, 1989). A typical food web of a small pond or impoundment system is summarized in Figure III.D-1, Major links in the food web of littoral zones. Compared to small streams, ponds are relatively self contained and have a limited ability to cycle nutrients on a watershed scale.

This figure summarizes a study of the feeding web occurring in the littoral zone of Bays Mountain Lake, which is located in Sullivan County, Tennessee. The watershed of this lake was classified as forested mountaintop (Crowley and Johnson, 1982). This lake is anticipated to be similar to natural ponds found in the study area. As shown in this figure, insect larvae, crustaceans, oligochaetes, gastropdods (snails), and ostracodas (minute fresh-water crustaceans with a bivalve, hinged shell) accounted for the majority of the secondary productivity in the shallow area of this pond. These organisms were consumed by predacious midge larvae (Tanypodinae), larval dragonflies and damselflies (Odonata), and small sunfish.

Large sunfish also consumed some benthic immature insects and gastropods, but were found to feed on larval odonates (dragonflies and damselflies) as well. The top predators within the pond were largemouth bass. These fish fed primarily on small sunfish and adult odonata. Food webs of other ponds and small impoundments in the study area have been found to exhibit similar types of food webs as illustrated in the figure. As summarized by Menzel and Cooper (1992), “Thus, while specific producers and consumers of importance may be dictated by habitat, abiotic parameters, or geographic location, the generalized pond food web is predictable.”

Figure III.C-3
Major Links in the Food Web of Littoral Zones — Prey Comprising at Least 10% of the Diet of Predators — Statistically Significant Depletion of Prey Populations in Enclosure Experiments
(from Johnson and Crowley, 1989)



f. Wetlands in Study Area

The wetlands and deepwater habitats in the MTM study area are almost entirely riverine (rivers and streams) or palustrine (e.g., marshes, swamps and small shallow ponds) (Tiner 1996). In West Virginia, palustrine wetlands, primarily ponds, have been found to be the most abundant type of wetland (Tiner 1996). Nearly all (99%) of the state's wetlands fall within the palustrine system. West Virginia's wetlands are mostly comprised of ponds, forested wetlands, and emergent wetlands (Tiner 1996). Reviewing wetland inventory summary maps available on the web (www.dep.state.wv.us/watershed), it can be seen that palustrine wetlands are common in areas of the state with extensive riverine wetlands. However, many isolated palustrine wetlands occur in areas lacking riverine systems as well.

A qualitative assessment of the occurrence of wetlands in areas subjected to surface mining compared to areas which had not experienced surface mining was performed as part of this EIS. National Wetland Inventory (NWI) maps produced by the FWS for the States of Virginia and West Virginia and the Commonwealth of Kentucky were used in this evaluation. One observation from this evaluation is that areas with surface mining frequently contain numerous, small ponds (indicated as wetlands classified as PUB or PUS, palustrine unconsolidated bottom or palustrine unconsolidated shore, respectively). Areas lacking surface mining did not appear to have as many small ponds as did mined areas. It is likely that these ponds were created as a result of surface mining activity. Additionally, in the review of the NWI maps for this area, it is clear that these

numerous, small pond-type wetlands on surface mining sites are not directly connected to the stream system in the region. Most of these wetlands appear to be formed in isolated small depressions on the formerly surface mined area. As such, these isolated pond-type wetlands would not be expected to contribute to energy flow or nutrient cycling in the stream system of the watershed. Ongoing research is being conducted on techniques for developing pond-type wetlands that would be more integrated with watershed-level aquatic functions (see Atkinson et al. 1997). However, most of this work is still in the conceptual stages. Programs such as the Powell River Project (http://als.cses.vt.edu/prp/index.html) are pursuing research to improve techniques for wetland construction/restoration on surface mining sites.

Existing information on surface mining techniques indicates that some surface mining practices do tend to result in pond formation both before and after mine restoration while other practices do not result in the formation of ponds (Atkinson and Cairns 1994, Atkinson et al. 1996). It is also important to note that the NWI maps generated for West Virginia were developed based on aerial photography from the early 1980's. In the past 15 to 20 years, it is likely that many of the wetland/ponds mapped as PUB may now contain emergent vegetation such as cattails.

Other types of palustrine wetlands such as forested swamps or shrub swamps were also observed in the study areas associated with creeks or rivers as marked in the NWI maps. It is believed that these areas are largely naturally formed wetlands and are not related to mining practices based on their position in the landscape and the maturity level of the vegetation in these wetlands (Tiner 1996).

The ecosystem functions of created lentic systems were discussed and summarized during a symposium on aquatic ecosystem enhancement held in January, 2000 by the MTM/VF EIS work group investigating this technical study area (EPA et al. March 20, 2000). Several presenters from academia, coal companies and environmental consultants discussed the values of man-made pond and wetland systems.

Characteristics and functions of man-made ponds and wetlands, as summarized by Dr. Wallace in EPA (March 20, 2000) include:

- Less of an interface with terrestrial environments than seen with headwaters streams
- Autochthonous primary productivity, primarily from algae and aquatic plants
- Energy systems tend to be closed with less linkage, if any, to other areas, or downstream ecosystems
- Disturbance in a pond will tend not to affect other ecosystems such as downstream areas
- These systems can be important sites of nutrient storage and uptake provided that a sufficiently vegetated littoral zone is present
- Under post-mining conditions, biological communities appear to resemble natural communities and are not as indicative of disturbance as is found in headwater streams

REI Consultants evaluated aquatic habitat functions provided by sediment control ponds and ditches (in EPA March 20, 2000). They found that functions present depended on the age of the structure with the number of functions increasing with structure age. The establishment of functions also

depended somewhat on water quality though older ponds tended to exhibit better water quality in most cases. Functions provided by the ponds and ditches included:

- Habitat for groups of aquatic insects typical to lentic habitats
- Water filtration/nutrient fixation
- Wildlife habitat including fish habitat for fish typical of small ponds
- Possibly, water treatment through filtration and precipitation. This function may be of increased importance for ponds developed in channels leading to headwater streams

In summary, functions of man made ponds and wetlands exist and may be considerable. While these functions differ from those of headwater streams, these functions do have their own inherent values. In fact, the establishment of ponds or wetlands on benches or at the toe of mined areas may tend to limit the effect of disturbances on the downstream watersheds (Wallace, B. in EPA et al. March 20, 2000).

3. Interrelationship Between Headwater Streams and Native Forests

Riparian (water-edge) habitats are transitions (ecotones) between terrestrial and aquatic environments and constitute a transition zone through which energy, nutrients, and species are exchanged. These areas typically are especially productive biological communities in which both species diversity and species densities are high (Warner, 1979). Characteristic woody vegetation exists in narrow bands along the streams that dissect this rugged landscape and include such species as black willow (*Salix nigra*), silver maple (*A. saccharinum*), box-elder (*A. negundo*), hackberry (*Celtis occidentalis*), sycamore (*Platanus occidentalis*), and cottonwood (*Populus deltoides*). In the rich alluvial soils along the streams, many species of shrubs and herbaceous plants can be found. Riparian habitat in the study area is limited to the narrow bands along the numerous streams because of the mountain and valley topography (WVDNR Water Resources 1976a).

THE SOUTHERN APPALACHIANS HAVE THE RICHEST SALAMANDER FAUNA IN THE WORLD

The headwater streams of the study area have a profound influence on the surrounding terrestrial habitat-- just as the terrestrial habitat influences the headwater streams. Leaves tend to blow across the forest floor and collect in the headwater streams which are wet depressions in the landscape. Very little of this coarse organic material in the form of leaves is transported downstream; most is processed by living organisms. The importance of the relationship between streams and the native forests is highlighted by the difference in coarse organic material inputs between streams flowing through forests and streams flowing through grassy areas. Streams flowing through grassy areas have much lower inputs of coarse organic material than streams flowing through forests (Sweeny, USFWS 2000). Also, different kinds of leaves from different species of trees affect the production and biomass of invertebrates. In addition, as precipitation percolates through leaves on the forest floor, it extracts organic compounds from the leaves. These dissolved organic compounds drive a major portion of the aquatic system's productivity (USFWS 2000). In aquatic ecosystems, the degree of land-water interaction between the terrestrial environment and the aquatic environment influences ecological processes and food web interactions (Adams and Hackney, 1992). The headwater streams of the study area have maximum terrestrial-aquatic interface ratios. Thus, the interconnection of the terrestrial and aquatic environments is greatest in these headwater streams. As mentioned previously, allochthonous organic matter typically dominates in headwater streams and other aquatic ecosystems with high ratios of land-water interaction. Therefore, the importance of surrounding forests to these streams can be easily understood in terms of generating energy for the aquatic ecosystem in the form of dead leaves and other organic matter. In addition to this relationship are the interrelationships between terrestrial wildlife and the aquatic environment of headwater streams in the study area.

The southern Appalachians have one of the richest salamander fauna in the world (Petranka 1998, Stein et al., 2000). Many species of salamanders are aquatic or semi-aquatic and utilize headwater streams at some point in their life histories. These aquatic and aquatic-phase (some larvae) salamanders are entirely predaceous and generally include a large proportion of aquatic insects in their diets (Wallace et al., 1992). The dusky salamander (*Desmognathus fuscus*), a semi-aquatic species, is a stream-side inhabitant of mountain brooks and seeps in the Appalachians. The dusky salamander spends the majority of its time in the terrestrial-aquatic environment interface zone,

along the margin of streams and seeps, opportunistically foraging on insects, slugs, and other invertebrates (Burton, 1976). Salamanders constitute a large portion of the animal biomass in eastern forests, in particular, in headwater streams. Biomass of the genus *Desmognathus* alone ranges from 1.673 to 2.683 g/m^2 (0.484 oz/yd^2 to 0.078 oz/yd^2) from four studies of headwater streams in the Southeastern United States (Wallace et al., 1992).

Many purely terrestrial species also depend on the headwater streams in the study area for their survival and the terrestrial-aquatic ecotone results in a diverse flora and fauna for these locations. For example, unique avifauna assemblages can be found along the riparian zone of headwater streams. The acadian flycatcher (*Empidonax virescens*) is commonly encountered throughout the study area (Buckelew and Hall, 1994), but is seldom found in upland forests, favoring the understory vegetation along small headwater streams where it feeds on emergent aquatic insects (Murray and Stauffer 1995). Neotropical migrant songbirds are also often attracted to headwater stream areas for breeding areas because of the diversity of the habitat and the availability of emergent aquatic insects. The Louisiana waterthrush (*Seirus motacilla*) neotropical migrant song bird is considered an obligate headwater riparian songbird because its diet is comprised predominantly of immature and adult aquatic macroinvertebrates found in and alongside these streams and it builds its nest in the stream banks (Mulvihill 1999). The Louisiana waterthrush is one of the earliest arriving migrants to the study area that places its nest among vegetation along flowing streams . The Louisiana waterthrush is also an area-sensitive species, requiring undisturbed forest tracts of 865 acres to sustain a population (Buckelew and Hall 1994). Therefore, preservation of large tracts of forest containing headwater streams is needed for the conservation of the Louisiana waterthrush in the central Appalachians (Murray and Stauffer 1995).

D. IMPACT PRODUCING FACTORS TO HEADWATER STREAMS FROM MOUNTAINTOP MINING

1. Studies Relating to Direct and Indirect Surface Water Impacts from Mountaintop Mining and Valley Fills

Surface mining operations in steep slope terrain generate excess spoil that is often placed in adjacent valleys. Mining operations and associated fills can directly impact headwaters by mining through or burying streams and eliminating existing terrestrial, riparian, and aquatic habitats. These operations also have the potential to indirectly impact stream conditions downstream from fills through physical or chemical changes. . In scoping discussions held to evaluate the impacts of MTM/VFs on headwater streams, eight potential impact factors were identified and are listed below.

Potential Impact Factors

1. Loss of linear stream length
2. Loss of biota under fill foot print or from mined stream areas
3. Loss of upstream energy from buried stream reaches
4. Changes in downstream thermal regime
5. Changes in downstream flow regime
6. Changes in downstream chemistry
7. Changes in downstream sedimentation (bed characteristics)
8. Effects to Downstream Biota

These factors fall into two categories: those occurring from the direct filling or mining of headwater streams (Factors 1, 2 and 3 in part), and those factors that manifest their effects through changes in characteristics of the stream located downstream from filled or mined areas (Factor 3 in part and Factors 4 through 8). These factors are related to the functions performed by headwater streams within the ecosystem. This section will focus on studies relating to each of these potential impact factors.

a. Loss of Linear Stream Length from Filling and Mining Activities Associated with Fills

Three studies examined the loss of stream length from valley filling. The findings of these studies are summarized below.

The EIS steering committee commissioned a study to determine the extent of valley fills in the EIS study area. This study, known as the fill inventory, includes a variety of information regarding valley fills constructed from 1985 to 2001, including the feet of stream under valley fill footprints. This study measured streams based on a synthetic stream network defined on a 30-acre watershed accumulation threshold over the National Elevation Dataset (NED). The NED for each state was processed to enforce hydrologic integrity. A flow accumulation grid was prepared and queried to define a drainage network over the entire region. The synthetic stream network represents all drainage for watersheds greater than 30 acres. The fill inventory study (USDOI OSM 2002) is presented in detail in Section III.K. This study estimated that between 1985 and 2001 approximately 724 miles (1.23%) of stream in the EIS study area were directly impacted by valley fills (i.e.,

covered by fill).

A study performed by the USFWS (USFWS 1998) evaluated stream miles permitted for filled with excess spoil and other coal mining wastes in Kentucky, Pennsylvania, Virginian and West Virginia between 1986 to 1998. This study found that at least 900 stream miles were permitted for filling in this time period. The study did not evaluate actual stream miles filled which are believed to be less than the number of miles permitted to be filled. The geographic area evaluated in this study was larger than that of the EIS study area. However, since 91% of the stream miles approved for fills were located in West Virginia or Kentucky, the results are applicable to this EIS. Other uncertainties relating to the accuracy of this estimate are presented in study. Only blueline streams from USGS topographic maps were included in this evaluation. This study did not evaluate miles of stream filled that were not marked as blueline streams, nor was an estimate made for the number of miles of streams mined through.

A cumulative impact study of the length of stream directly impacted within the study area was performed by the USEPA (2002). The stream lengths evaluated were based on the same synthetic stream network as the OSM fill inventory which includes streams located upslope from the USGS blueline streams. This cumulative impact study differed from the previously discussed studies in that the estimate of stream length impacted was based on length of stream filled and length of stream mined through. This study estimated 1,208 miles of direct impact to stream systems in the study area based on permits issued in the last ten years (1992-2002). This estimated of filled or mined through streams represents 2.05% of the stream miles in the study area.

It has been suggested that streams have been, or could be, created during the reclamation of mined or filled sites. It was not the intent nor design of these studies to assess any re-creation of streams. Due to the current lack of data to support creation of viable streams on mining operations, studies exploring the amount of, or possibility for, creation of streams should be considered.

b. Loss of Biota under Fill Foot Print or from Mined Stream Areas

When streams are filled or mined all biota living in the footprint of the fill or in the mined area are lost. There is little question that perennial streams support viable aquatic communities that could be lost from valley fills. However, prior to investigations performed in support of this EIS, the existence of aquatic communities in streams classified as “ephemeral” or “intermittent” was questioned. In fact, the points on the slope of a watershed at which ephemeral, intermittent and perennial streams originated were very poorly understood. Numerous studies in and around the MTM/VF study area had documented the existence of aquatic communities in “headwater stream” systems (See USFWS 1999) but not at the level of geographic detail needed to address questions on the existence of aquatic communities in the upper most stream reaches in the study area.

b.1. Primary Literature Review of Aquatic Communities in Streams with Ephemeral or Intermittent Flow Regimes

Literature results indicated that aquatic organisms could potentially exist in streams with ephemeral and intermittent flow regimes. In western Oregon taxa richness of invertebrates (>125 species) in temporary forest streams exceeded that in a permanent headwater stream (100 species) (Dietrich and Anderson 2000). Dietrich and Anderson (2000) also found that only 8% of the species in the total

collection were only found in the permanent headwater. A total of 25% were restricted to the summer-dry streams and 67% were in both permanent and summer-dry streams. In other words, most of the aquatic life found in the temporary streams was also found in permanent streams, clearly indicating that the temporary streams support aquatic life similar to that found in permanent streams. These researchers concluded that the potential of summer-dry streams with respect to habitat function is still widely underestimated.

In several northern Alabama streams of varying flow permanence, including a stream that was normally perennial, Feminella (1996) found little differences in the invertebrate assemblages. Presence-absence data revealed that 75% of the species (171 total taxa, predominantly aquatic insects), were ubiquitous across the 6 streams or displayed no pattern with respect to permanence. Only 7% of the species were found exclusively in the normally intermittent streams. Again, this study clearly indicates that intermittent streams support aquatic life.

Many researchers have found that intermittent streams, spring-brooks and seepage areas contain not only diverse invertebrate assemblages, but some unique aquatic species. Dieterich and Anderson (2000) found 202 aquatic and semi-aquatic invertebrate species, including at least 13 previously undescribed taxa. Morse et al (1997) have reported that many rare invertebrate species in the southeast are known from only one of a few locations with pea-sized gravel or in springbrooks and seepage areas. Kirchner (F. Kirchner pers. comm. 2000 and Kirchner and Kondratieff 2000) reports 60 species of stoneflies from eastern North America are found only in first and second order streams, including seeps and springs. Approximately 50% of these species have been described as new to science in the last 25-30 years.

Williams (1996) reported that virtually all of the aquatic insect orders contain at least some species capable of living in temporary waters and that a wide variety of adaptations across a broad phylogenetic background have resulted in over two-thirds of these orders being well represented in temporary waters. This researcher goes on to say that "perhaps the concept of temporary waters constraining their faunas is based more on human perception than on fact".

b.2. Studies in the MTM/VF Study Area

The USGS (2002 Draft) is completing their "E-point, P-point" study to characterize the size of watersheds located upstream from the starting point of perennial, intermittent and ephemeral headwater streams within the MTM/VF study area. The following table summarizes their results.

Boundary	Median Drainage Area Upstream of Boundary (acres)	Range of Drainage Areas Upstream of Boundary (acres)
Ephemeral-Intermittent	15.2	6.3 to 45.3
Intermittent-Perennial	40.8	18.0 to 150.1

Field work on aquatic communities was performed by OSM and USGS biologists in some of the same watersheds used for the USGS (2002-Draft) "E-point, P-point" study to assess the potential limits of viable aquatic communities in small headwater streams in southern West Virginia

(Interagency Invertebrate Study 2000). Most of the small streams sampled in the study were not indicated by a blueline on existing 1:24,000 scale USGS topographic maps. The study found that all eight of the target orders of insects selected were found within the headwater reaches evaluated. Furthermore, the study found that a number of taxa that were found in the extreme headwaters have multi-year life cycles. This would suggest that sufficient water is present for long-lived taxa to complete their juvenile development prior to reaching the aerial adult stage in these areas. Although only contiguous flow areas were considered for this study, the field work took place in the winter, and it was considered probable that these extreme headwaters were subject to annual drying.

As part of the work to describe stream conditions in southern West Virginia for this EIS, the EPA found that intermittent streams supported diverse, healthy and balanced invertebrate populations preceding and following a severe drought in the summer of 1999 (USEPA, 2000). During the summer and fall 1999 index periods, many of the reference streams in this EPA study were flow limited, with only trickles of water in their channels, and some of these streams were found to go completely dry. In the spring 1999 index period, preceding the drought, and in the winter 2000 index period, following the drought, all of the intermittent streams could be sampled, and all of the intermittent reference streams were in good or very good condition with diverse and balanced benthic invertebrate assemblages (USEPA, 2000). Clearly these streams, though intermittent for several months in some cases, supported diverse and balanced aquatic life.

b.3. Conclusions Regarding the Existence of Aquatic Communities in Streams Potentially Impacted by Direct Filling or Mining Activities

As can be concluded based on results from the primary literature and from studies performed for this EIS, filling or mining stream areas even in very small watersheds has the potential to impact aquatic communities some of which may be of high quality or potentially support unique aquatic species. It has not been determined if drainage structures associated with mining can provide some benefits (i.e.; increased flows at toe of fills, retaining drainage structures) that could offset aquatic impacts.

c. Loss of Upstream Energy from Buried Stream Reaches

Considerable information regarding the energy cycling functions of headwater streams has been presented in this EIS in Section III.C. The extent to which valley fills eliminate energy resources that may be used by downstream aquatic communities is not well documented. There is a lack of information on the degree to which length of stream directly correlates with the amount of energy in the form of fine-particle organic material or coarse-particle organic material leaving a particular reach of headwater stream. The Value of Headwater Streams: Results of a Workshop, (Appendix D) emphasizes the importance of headwater streams in energy and nutrient spriraling down through a watershed ecosystem. The following is a summary from information provided in Appendix D. Reference citations from primary literature are presented in Appendix D. Forest leaf litter is particularly important to macro invertebrates that process organic matter for downstream reaches. Experiments demonstrate the reliance of stream biological communities on energy inputs from the surrounding forests. When leaf litter was excluded from a stream, the primary consumer biomass in the stream declined, as did invertebrate predators and salamanders. Leaf litter exclusion had a profound effect on aquatic productivity, illustrating the direct importance of terrestrial-aquatic ecotones.

Other experiments illustrated that, although invertebrates and microbiota in headwater streams are only a minute fraction of living plant and animal biomass, they are critical in the export of organic matter to downstream areas by converting leaf litter to fine particulate organic matter, which is much more amenable to downstream transport than the leaves themselves. The extent to which energy loss may be offset by input from reclamation of the mine site and adjacent undisturbed areas is unknown. Impacts that this type of net energy "change" would have on the downstream aquatic environment is uncertain and requires further investigation.

d. Changes in Downstream Thermal Regime

Valley fills have the potential to impact a variety of water quality parameters. One study of thermal impacts of valley fills was performed by the USGS (USGS 2001c) on one stream below a valley fill site and one stream below an unmined site. This study recorded stream temperature at a valley fill site and at an unmined site on a daily basis. Water temperatures from the valley fill site exhibited lower daily fluctuations and less of a seasonal variation than water temperatures from an unmined site. Water temperatures were warmer in the winter and cooler in the summer than water temperatures from the unmined site. Based on the data from this study, it appeared that the maximum daily difference between the two streams was approximately 13.5 degrees Fahrenheit. This study included only two streams so it cannot be determined if the observations made would be true for a number of streams below valley fills. It is also difficult to predict the possible impacts of this moderated thermal regime on the downstream aquatic communities. This issue remains as an uncertainty that requires further investigation.

e. Changes in Downstream Flow Regime

Valley fills have the potential to alter the flow regime of streams downstream from fill areas. One study of the impact of valley fills on stream flows was performed by the USGS (USGS 2001c) on one stream below a valley fill site and one stream below an unmined site, and comparing one flow parameter at many streams with and without filling in the watershed. Low stream flows were investigated by comparing 90-percent flow durations, daily stream flow records, base-stream flows and storm flows. Generally, the 90-percent flow durations at valley fill sites were 6 to 7 times greater than the 90-percent flow durations at unmined sites. Some valley fill sites, however, exhibited 90-percent flow durations similar to unmined sites and some unmined sites exhibited 90-percent flow durations similar to valley fill sites. Daily stream flows from the one valley fill site evaluated generally were greater than daily stream flows from the one unmined site evaluated during periods of low stream flow. The valley fill site evaluated had a greater percentage of base-stream flows and lower percentage of storm flows than did the one unmined site evaluated.

This study included only two streams except for the evaluation of 90-percent flow durations, so it cannot be determined if the observations made would be true for a number of streams below valley fills. It is also difficult to predict the possible impacts of this moderated and elevated flow regime on the downstream aquatic communities. This issue remains as an uncertainty that requires further investigation.

f. Changes in Downstream Chemistry

Mining and associated valley fills have the potential to alter the water chemistry of streams

downstream from fill areas. It is possible to relate water chemistry to biological functions of streams where Federal or State Ambient Water Quality Criteria exist.

f.1. Studies Addressing this Impact Factor

The USEPA (2002) conducted a study of the stream chemistry associated with sites classified as mined, unmined, filled and filled/residence. Detailed descriptions of each of the EIS classes were presented in the report. In summary, unmined sites were not located downstream from mines or fills. Mined sites were located downstream of older mine project with no fills, filled sites were located downstream from mined sites with valley fills and filled/residence sites were located downstream from mined, filled sites with residential dwellings in the watershed. The data from this report indicate that MTM/VFs increase concentrations of several chemical parameters in streams. Sites in the Filled category had increase concentrations of sulfate, total dissolved solids, total selenium, total calcium, total magnesium, hardness, total manganese, dissolved manganese, specific conductance, alkalinity total potassium, acidity and nitrate/nitrite. There were increase concentrations of sodium at sites in the filled/residence category which may be caused by road salt and /or sodium hydroxide treatment of mine discharges. Results for all other parameters were inconclusive in comparing among EIS classes.

Comparisons to AWQC were performed with a subset of the total data set as explained in USEPA (2002a). Selenium concentrations from the Filled category sites were found to exceed AWQC for selenium at most (13 of 15) sites in this category. No other site categories had violations of the selenium limit. No other constituents exhibited violations of the AWQC for any category.

In a study conducted in 1998 as part of the National Water Quality Assessment (NAWQA) program of the U.S. Geological Survey, surface water quality was sampled in 12 study areas in the Appalachian Coal Region to measure changes in water quality from baseline conditions that had previously been monitored in 1979-81. Each sample collected during the July-September 1998 sampling period was matched to a 1979-81 sample considered to be most similar in discharge and season. About 180 sites were sampled to assess changes. Sites were selected for sampling on the basis of a three-factor categorical design of geology, mining method, and mining date within the surface drainage basin above each site. Geology was represented by the contrast between the Allegheny-Monongahela River and the Kanawha River Drainage basins. (This corresponds roughly to the northern and southern coal fields in West Virginia terminology.) The mining method was identified as underground, surface, or both. The mining date was identified as before the historical sample, after the historical sample, or both. The reference conditions in both study areas were identified as basins that had never been mined, and particular effort was spent in identifying these basins. While the study did not focus on mountaintop mining specifically, its results are considered relevant to the topic area and are therefore worth reviewing.

The study found that the median pH of summer base flow in these streams increased about 0.5 unit from 1980 to 1998 in both the northern and southern parts of the study area since pH is a logorithmic scale, a change of 0.5 pH is a big change. During the 1998 sample period, the median pH among all sample sites was 7.9 in the north and 7.4 in the south. Alkalinity of the streams also increased and was reflected in decreased concentrations of iron and manganese. These effects would be expected on a regional basis as a result of increased compliance with permit limits and with increasing efforts to control the worst cases of acid drainage from abandoned mines. While

improvements in pH, iron, and manganese were seen, median concentrations of sulfate among all sites increased from 38 mg/L to 56mg/L in the north, and from 46 mg/L to 77 mg/L in the south. Sulfate is a good indicator of the total disturbance of a basin by mining and other large scale earth moving activities because most sulfate is produced by oxidation of pyrite minerals to acidic iron sulfate, and these types of activities increase the amount of pyrite minerals that are available for oxidation. Among 52 basins where mining occurred both before and after 1980, for example, the sulfate concentration more than doubled in 13 basins, including greater than five-fold increases in 5 basins. In both northern and southern basins, sulfate concentrations of less than 20 mg/L were common in unmined areas. Acid loads from the pyrite reaction are neutralized at a regional scale by both alkaline minerals naturally present in mined areas and by engineered additions of alkalinity. Acid production will continue, however, in proportion to the amount of available pyrite, and after mining ends, acid production will gradually decrease as the amount of pyrite is consumed.

A study was also conducted by OSM on the cumulative off-site impacts from a large area mine in southeastern Ohio over a twelve year period. The location of the study was on the Central Ohio Coal Company (COCCO)property where a dragline was used. OSM used the 1980 data submitted by COCCO and data collected between 1987 and 1999 by the Ohio Environmental Protection Agency (OEPA) to evaluate the impacts. Although this study was not in this EIS study area it was included to show how mining activities without valley fills can impact water quality. The chemical analysis of the impacted streams indicated similarly elevated levels of hardness, sulfates and conductivity as did the EPA 2002 study. (USDOI OSM 2000)

f.2. Summary and Conclusions

In summary, mining and valley filling activity appear to be associated with some downstream changes in surface water chemistry. These changes include increases in a number of cations that are known to be associated with surface mining such as sulfate, total dissolved solids, total calcium, total magnesium, hardness, total manganese, dissolved manganese, specific conductance, alkalinity, and total potassium. The majority of these constituents may also increase in many other types of large scale earth moving activities.

In the USEPA (2002a) stream chemistry study, selenium was found to exceed AWQC at Filled sites only, and was found to exceed AWQC at most Filled sites included in the study. The existence of selenium at concentrations in excess of AWQC at most of the filled sites indicates a potential for impacts to the aquatic environment and possibly to higher order organisms that feed on aquatic organisms.

While changes in water chemistry downstream from mined, filled sites have been identified, it is not known if these changes are resulting in alterations to the downstream aquatic communities or whether functions performed by the areas downstream areas from mined, filled sites are being impaired. Question exist as to how the downstream chemistry is affected by factors such as time, method of mining, reclamation practices and size of operation. Further evaluation of stream chemistry and further investigation into the linkage between stream chemistry and stream biotic community structure and function are needed to address the existing data gaps.

g. Changes in Downstream Sedimentation (Bed Characteristics)

Valley fills have the potential to alter geomorphological features of streams downstream from fills such as sediment particle size. One study of the impact of valley fills on sediment particle size was performed by the USGS (USGS 2001c). Particle sizes were measured at 54 small stream sites in four watersheds. Valley fill sites had a greater number of particles less than two millimeters in size, a smaller median particle size and about the same 84th percentile particle size as compared to the mined and unmined sites. Results were based on visual comparisons of box and whisker plots developed for each data class.

Similar results on sediment particle size at stream sampling stations below fills were obtained from USEPA (2000). Valley fill sites had a greater number of particles less than two millimeters in size and a smaller mean particle size. However, the mean substrate size class was found to be very similar between unmined, filled, filled residential and mined EIS class sites. The authors stated that these data indicate that the valley fills do not seem to be causing excessive sediment deposition in the first and second order streams that were sampled but cautioned against generalizing this finding to higher order streams or to reaches downstream in these watersheds. In contrast, sampling downstream of mountaintop mining/valley fill sites in Kentucky revealed greater sediment deposition and smaller substrate particle sizes than in reference streams (EPA 2001).

In the OSM study of Central Ohio Coal Company (COCCO) property, stream habitat was evaluated in 1987 and 1999 using OEPA's Qualitative Habitat Evaluation Index (QHEI). The author stated that the QHEI may be somewhat subjective, but it is still a good indicator of habitat quality. The QHEI indicated impairments from heavy to moderate silt cover and substrate embeddedness in two streams studied in 1987. However, the 1999 sampling showed that the streams had improved sufficiently to support warm-water biota (USDOI OSM 2000).

While these studies illustrate that mining and valley fills may alter the sediment composition of streams, it is not known if this change may impact functions of streams downstream or how long these changes may persist. Assessment of stream sediment characteristics should be included in any further evaluations or monitoring program for streams downstream from mining and valley fills.

h. Effects to Downstream Biota

MTM/VFs have the potential to impact aquatic biota since mining and filling activities may occur within streams. A review of the literature available for this EIS on this topic has revealed that there are at least four types of studies which have been performed to evaluate the impact of mining in general and MTM/VF in particular on aquatic macroinvertebrate biota. These four types of studies include: A. Comparisons of results from stream sites upstream of mine input to downstream results; B. Comparisons of Pre-mining results to post-mining aquatic community results; C. A multivariate analysis study on a regional basis of potential impact producing factors to stream systems; and D. Studies of stream sites located downstream from mined or valley filled areas in comparison to reference locations.

Several studies evaluating the potential impacts of mining or mined-valley filled areas on fish communities address the issue of potential impacts of mining and associated fills to aquatic biota. These studies have been summarized below.

Most studies evaluated basic water chemistry and field water chemistry parameters, and habitat

characteristics including substrate conditions. Stream order was included as a criteria for establishing the study but was not evaluated further in most cases. Neither the size nor age of mining or associated fills were included as evaluation criteria in any study summarized here.

h1. Summary of Results from Upstream-Downstream Comparison Type Studies

Four studies of this type were made available for use in this EIS from coal companies, particularly from Pen Coal Corporation. These studies included studies evaluating macroinvertebrate communities downstream from mine influences to upstream sites for Twelvepole Creek (Pen Coal 1998; Pen Coal, 2000c), Honey Branch (Pen Coal, 1999a) and Trough Fork Creek (Pen Coal, 2000a). These studies assessment evaluation metrices relating to the abundance, number of taxa, proportion of sensitive species present, and diversity and evenness of the aquatic macroinvertebrate community at stream sampling locations above and below the influence of mining. Usually water chemistry and habitat characteristic evaluations were performed in concert with the biotic evaluation.

Overall, the abundance of macroinvertebrates was found to be similar in upstream and downstream stations or to be slightly higher in downstream stations. As discussed in these studies and other studies (see Arch Coal in prep 2002), this increase in abundance may be related to the presence of releases from sedimentation ponds or other releases of solids into the stream. The number of taxa were found to be similar in upstream or downstream stations or to decrease at downstream locations near to the influent area from the mines. The largest difference seen between upstream and downstream locations was the change in proportion of sensitive groups. All four studies reported a decrease in the proportion of sensitive organisms in the stream sampling locations downstream from the mining influent. In addition, other metrices that evaluate the diversity, evenness and degree of pollution tolerance of the aquatic community were found to become more indicative of an impacted stream condition (i.e. diversity and evenness decreased, pollution tolerance increased).

Two types of physio-chemical factors were singled out by these studies as potentially contributing to these community changes. Several studies indicated that sedimentation was greater downstream from the point of mine influent. All studies noted increases in the water chemistry parameters sulfate, conductivity and hardness. Selenium was not an analyte in any of these studies.

These studies did not specifically address the presence of or potential impacts from valley fills. Given the current status of these studies, fills were probably part of the mine complexes evaluated by these studies but it is not known whether all downstream locations in these studies were downstream from fills or just from mining areas.

h2. Results of Comparisons of Pre-mining Biotic Conditions to Post-mining Aquatic Communities

Two studies comparing pre-mining biotic conditions to post-mining aquatic communities from the same stream sampling locations were made available for use in this EIS from coal companies, particularly from Pen Coal Corporation. These studies included studies on Trough Fork (Maggard and Kirk, 1999 and Pen Coal, 2000a), and Honey Branch (Pen Coal,1999a). These studies assessed evaluation metrices relating to the abundance, number of taxa, proportion of sensitive species present, and diversity and evenness of the aquatic macroinvertebrate community at stream sampling locations before mining was initiated and after or during the development of a mine. Usually water

chemistry and habitat characteristic evaluations were performed in concert with the biotic evaluation.
The evaluation for Honey Branch was complicated by the fact that the historic data from 1987 appeared to have been derived from sampling performed using different sampling techniques than are currently employed. The authors of this report stated that a qualitative comparison of current to past results suggests that the aquatic macroinvertebrate community has undergone a shift to a more tolerant, less sensitive community.

The evaluation of Trough Fork is an ongoing project. Sampling was initiated in 1995 prior to mine initiation. This study included sampling sites upstream and downstream from the influent from the mine complex. Between 1995 and 1999 the upstream sampling locations showed increases in abundance, taxa richness, the number of EPT genera and slight decreases in the proportion of sensitive organisms and community diversity. These changes may reflect the natural variation present in aquatic communities over time since there should be no direct effects from mining input to the upstream stations. Changes in the downstream station were similar to those seen at the upstream station for abundance and taxa richness. However, the diversity and evenness of the downstream macroinvertebrate communities decreased notably and the proportion of tolerant organisms increased notably in comparison to the 1995 results and the upstream station.

Water chemistry did not change much between the 1995 and 1999 sampling periods for the upstream sampling station. However, for the downstream sampling station, increases in conductivity, TDS, TSS, hardness, alkalinity, sulfates, sodium, calcium and magnesium were found the 1999 sampling period compared to the initial 1995 results. Selenium was not included as an analyte in these samples.

Anecdotally, the investigator noted that base flow had increases at the downstream location. The report stated that this should have a positive impact on the aquatic community, but results from the 1999 sampling period do not appear to indicate that a positive change is occurring at the stations downstream from the mine (Maggard and Kirk, 1999).

These studies did not specifically address the presence of or potential impacts from valley fills. Given the current status of these studies, fills may not be complete at this point. This on-going project represents an opportunity to investigate the relationship between fill age and downstream impacts.

h3. Results of A Multivariate Analysis Study on Benthic Invertebrate Communities and Their Responses to Selected Environmental Factors

An extensive study of invertebrate communities and their responses to environmental factors in the Kanawha River basin was performed by the USGS (USGS, 2001a). This study included in entire Kanawha River basin and, on a regional basis, focused on relationships between macroinvertebrate community characteristics with land use types and other stream-related factors such as stream chemistry and habitat characteristics. A variety of multivariate statistical analyses were used to explore the potential relationships among variables.

Results from this study indicated that in the Kanawha River Basin the effects of coal mining, such as changes in stream water chemistry and benthic habitat quality, strongly shaped aquatic

macroinvertebrate communities especially in basins of less than 128 square miles. Coal mining appeared to influence invertebrate communities through two factors: 1. Increasing habitat degradation through decreasing the median particle size of streambed material, and 2. Increasing the specific conductance and sulfate concentration of surface water. On a positive note, this study found little evidence of classic acidic mine drainage in the Kanawha River Basin.

The increase in specific conductance and sulfate concentration was associated with a proportional decrease in the sensitive taxa in the stream macroinvertebrate communities. The study also indicated that the decrease in median particle size of streambed sediment was the habitat characteristic that most strongly correlated to loss of sensitive taxa groups and increases in tolerant taxa. It was noted that other landscape level alterations such as large construction projects and stream dredging also decreased median particle size.

While this report did not focus on valley fills, potential impacts from valley fills to stream chemistry and possible alterations to stream geomorphology were discussed as areas in need of further investigation.

h4. Studies of Macroinvertebrate Communities in Stream Sites Located Downstream From Mined or Mined/Valley Filled Areas in Comparison to Reference Locations

A fourth type of study is available relating to the potential impacts of mining and valley filling on downstream aquatic invertebrate communities. Typically, these studies evaluated stream communities located downstream from mining plus valley fills, or mining alone in comparison to various reference locations.

This type of study originated with the USEPA (2000) study of numerous watersheds throughout the MTM/VF study area. A followup to this study using a variety of comparative statistical approaches is being prepared by the USEPA (2002 in prep). Also in preparation is a supplemental study of the sampling stations used in USEPA (2000) relating to mining performed by Arch Coal, Inc. A draft version of this report was released in August of 2000 but Arch Coal has indicated that a revised version of this report will be released shortly (Arch Coal, conference call of May 29, 2002). A supplemental evaluation of sampling stations used in USEPA (2000) relating to valley fills in the vicinity of the Hughes Branch was developed by Cannelton Industries (Cannelton, June 2000). Finally, EPA Region 4 has completed an evaluation of the impacts of MTM/VF to streams in Kentucky (USEPA Region 4, 2001).

Summary of the USEPA Stream Survey Study

The EPA streams study (USEPA 2000) was performed as part of this EIS to more fully evaluate what changes, if any, are occurring in benthic communities, stream chemistry, and aquatic habitat downstream of mining operations. These studies were designed for the express purpose of providing a synoptic description of stream conditions in five representative watersheds across the primary mountaintop mining area within the study area. These watersheds were defined by the West Virginia Geological and Economic Survey (WVGES) and include Twentymile Creek, Clear Fork, Island Creek, Mud River, and Spruce Fork.

The selected study sites were monitored for benthic macroinvertebrate populations, water chemistry, and physical habitat when adequate flows allowed. Benthic macroinvertebrate populations were

sampled in the five study watersheds using the RBP single habitat sampling protocol (USEPA 1999). Samples were collected over a period of five seasons: spring 1999, summer 1999, fall 1999, winter 2000, and spring 2000. Most of the unmined streams could not be sampled during the summer and fall 1999 sampling seasons due to stream flows being either too low to allow benthic sampling or the streams lacked flows altogether.

Methodology and results of the invertebrate component of the stream study are reported in the draft report "*A Survey of the Condition of Streams in the Primary Region of Mountaintop Mining/Valley Fill Coal Mining*", dated November 2000.

The primary objectives of this study relating to the impact of MTM/VF on stream communities were:

1. Characterize and compare conditions in three classes of streams: 1) streams that are not mined (termed "unmined"); 2) streams in mined areas with valley fills (termed "filled"); 3)streams in mined area with valley fills and residences (termed "filled-residential") and 4) streams in mined areas without valley fills (termed "mined").

2. Characterize conditions and describe any cumulative impacts that can be detected in streams downstream of multiple fills. Owing to conditions encounter no definitive conclusions were reached regarding this second objective.

This study evaluated benthic macroinvertebrate assemblage data, physical stream habitat assessments, quantitative estimates of substrate size, and limited field chemical/physical parameters.

Biological conditions in the unmined sites generally represented a gradient of conditions from good to very good, based on the WVDEP SCI scores. These sites are primarily forested, with no residences in the watersheds. One site scored in the high-end of the fair range in the summer of 1999, one site scored in the poor range in the fall of 1999, and one site scored in the high-end of the fair range in the winter of 2000. The authors believes these sites scored lower primarily because the drought and lower flows impeded their ability to collect a representative sample. They observed no other changes at these monitoring sites that could account for the changes in the condition of the streams, other than the low flows. When these sites were sampled in later index periods, they scored in the good or very good range.

Biological conditions in the mined sites generally represented very good conditions, although a few sites did score in the good and poor range. One site that scored in the poor range was believed to be naturally flow-limited even during periods of normal flow. The authors believed this site was ephemeral and only flowed in response to precipitation events and snow melt. The other mined sites generally had only a small amount of mining activity in their watersheds.

Biological conditions in the filled sites generally represented a gradient of conditions from poor to very good. One site scored in the very poor range in the spring of 2000. Over the five seasons, filled sites scored in the fair range more than half of the time. However, over a third of the time, filled sites scored in the good or very good range over the five seasons. The authors believe water quality explains the wide gradient in biological condition at the filled sites. The filled sites that scored in the good and very good range were found to have better water quality, as indicated by lower median

conductivity at these sites. The filled sites that scored in the fair, poor and very poor ranges had degraded water quality, as indicated by elevated median conductivity at these sites.

Biological conditions in the filled/residential sites (filled sites that also have residences in their watersheds) represented a gradient of conditions from poor to fair. Over the five seasons, filled/residential sites scored in the poor range more than half of the time. The remainder of the filled/residential sites scored in the fair range. No sites in the filled/residential class scored in the good or very good range. All sites in the filled/residential class had elevated median conductivities.

In general, the filled and filled/residential classes had substantially higher median conductivity than the unmined and mined classes. It is important to note that the filled sites generally had comparable or higher conductivity than the filled/residential sites within a watershed, indicating that the probable cause of the increase in the total dissolved solids at the filled/residential sites was the mining activity upstream rather than the residences. Presently, there are no aquatic life criteria for conductivity or total dissolved solids.

Biological conditions in the filled and filled/residential classes were substantially different from conditions in the unmined class and were impaired relative to conditions in the unmined class, based on the WV SCI scores.

The filled/residential class was the most impaired class. The causes of impairment in this class could include several stressors (e.g. the valley fills, the residences, roads). It is impossible to apportion the impairment in this class to specific causes with the available data.

The general patterns of stream biological condition presented in the previous paragraphs were clear in all three seasons that have complete data sets (spring 1999, winter 2000 and spring 2000)including sampling results from unmined sites.

The Rapid Bioassessment Protocols habitat assessment data did not indicate substantial differences between the stream classes. The habitat in the filled class and the filled/residential class was slightlydegraded relative to the unmined class. Individual sites in the filled and filled/residential classes had degraded habitat and excessive sediment deposition.

In general, the substrate characteristics of the filled, filled/residential, and mined classes were not substantially different from the unmined class. The data from this study did not indicate excessive fines in the filled or the filled/residential classes as a whole, however, there were specific sites within these classes with substantially higher percentages of sand and fines compared to the unmined class. It should be noted that many of the filled sites were established in first and second order watersheds in order to limit the potential stressors in the watershed to the valley fills. These data indicate that the valley fills and associated mining activity did not cause excessive sediment deposition in the upper reaches of these watersheds. The authors noted that it would not be appropriate to extrapolate this conclusion to reaches farther downstream in these watersheds or to larger order streams.

Correlations in this study between the benthic metrics and selected physical and chemical variables indicated that the strongest and most significant associations were between biological condition and conductivity. Physical habitat variables were more weakly correlated with biological condition and

some of these associations were not significant. Water quality appeared to be the major factor limiting the benthos in the impaired streams. This study also discussed findings related to flow and noted that perennial flow conditions were not needed to support high quality aquatic communities.

Summary of the Other Studies Relating to Impacts of MTM/VF on Stream Biota

A followup to the USEPA (2000) study using a variety of comparative statistical approaches is being prepared by the USEPA (2002 in prep). This study is analyzing data from the USEPA report along with data provided by various coal companies. Thus far, preliminary results, using only those sample periods from all sites where flow was sufficient to allow sampling, support the findings of the USEPA (2000) study. The Filled and Filled-Residential sites have been found to differ significantly from the unmined and mined sites in six to nine of the nine evaluation metrices. All differences observed are in the direction of impairment (e.g., decreased diversity, increase proportion of tolerant organisms in the community etc).

In preparation is a supplemental study of the sampling stations used in USEPA (2000) relating to mining performed by Arch Coal, Inc. A draft version of this report was released in August of 2000. Arch Coal presented some preliminary results from the revised version of this report in a teleconference (Arch Coal, conference call of May 29, 2002 and 2000). Based on this presentation, results appear to be similar to those in USEPA (2000). Arch Coal found filled and Filled-Residential sites showed decreases in EPT taxa and increases in the proportion of tolerant organisms in the community compared to reference sites. This study also measures abundance but results on this evaluation metric are not yet available for inclusion in the EIS. In their evaluation of physio-chemical parameters that might explain community changes observed, Arch Coal noted that the moderated thermal regime may have increase the degree-date accumulation of the stonefly populations resulting in emergence earlier in the season than had previously been observed. Although it is not known if such a change would result in changes to the community, it is interesting to note that changes to the thermal regime downstream from valley fills may be exhibiting a population level impact.

A supplemental evaluation of sampling stations used in USEPA (2000) relating to valley fills in the vicinity of the Hughes Branch was developed by Cannelton Industries (Cannelton, June 2000). This study looked at three stations below valley fills and other mining influences. These stations were evaluated using the WV SCI. SCI results ranged from good to very good. This study also found very low percentages of mayflies (ephemeroptera) at this sites and elevated surface water conductivity, hardness and sulfates. All findings presented were similar to the findings of the USEPA (2000) study.

EPA Region 4 conducted a one time sampling of streams in Kentucky and evaluated those samples for impacts from MTM/VF. This study compared sampling stations located downstream from mined-filled areas to reference streams. Severe impacts to the mayfly fauna was exhibited at all mined-filled sites. Decreases in pollution-sensitive macroinvertebrates were also observed at mined-filled sites. Also, decreases in taxa diversity were observed at mined-filled sites. Mined-Filled sites generally had higher conductivity, greater sediment deposition, and smaller substrate particle sizes. Strong negative correlations were observed between conductivity and indications of macroinvertebrate community health. (USEPA 2001)

OSM Report on the Cumulatative Off-Site Impacts from a Large area Mine in Southeastern Ohio.

OSM conducted a study of a Central Ohio Coal Company (COCCO) mine in Southeastern Ohio to determine the off-site impacts from a large area mined. This study, although not in this EIS study area, provides information to consider if the cause of the impacts being seen below MTM/VF studies were due to the SMCRA defined "valley fills" or could be expected from area mining "backfill". The study used both COCCO's samples from 1980 and Ohio Environmental Protection Agency (OEPA) samples from 1987 and 1999. The preponderance of the mining was done post-1972 (SMCRA) and completed in 1987 (Rannells Creek) and 1992 (Collins Fork).

OSM obtained fish study results, macroinvertebrate study results, water quality analysis, and Quality Habitat Evaluation Indicators (QHEI) from the OEPA samplings. Comparative surveys of macroinvertebrates on Collins Fork and Rannells Creek indicate similar results to those in the filled and filled/residence class sites of MTM/VF studies (i.e.; elevated conductivity, sulfates, hardness and a decline in pollution sensitive species). Evaluations of the invertebrate community quality appeared unchanged between the two OEPA sampling periods. It is particularly noteworthy that none of the macroinvertebrate samples in 1987 or 1999 showed any significant numbers or kinds of mayflies. This absence of mayflies has also been observed in recent surveys by the USEPA 2002 study in West Virginia in mining areas with acceptable pH's, but with high conductivities. (USDOI 2000)

i. Impacts of MTM/VF on Fish Assemblages

Two studies relating fish communities to potential impacts from mining and or mining and valley filling are available for use in this EIS. The USFWS MTM Fish Assemblage Characterization Report (Stauffer and Ferreri, 2002) directly addressed this issue.

An extensive study of fish communities and their responses to environmental factors in the Kanawha River basin was performed by the USGS (USGS 2001b). This study included in entire Kanawha River basin and, on a regional basis, focused on relationships between fish community characteristics with land use types and other stream-related factors such as stream chemistry and habitat characteristics. A variety of multivariate statistical analyses were used to explore the potential relationships among variables.

The USGS (2001b) found that stream size and zoogeography masked any potential water quality effects of land use on species composition and relative abundance of fish communities in the study. This and other factors relating to natural characteristics of fish communities in this region limit the usefulness of this study to evaluate mining impacts on fish communities.

Stream Fish Assemblage Characterization

There is little historical information regarding stream fish populations in the primary region of mountain top removal/valley fill coal mining. To address this data gap, fish communities at several pre-selected sites in the MTM/VF study area were sampled (Stauffer and Ferreri, 2002). The objectives of this study were to 1) characterize the fish communities that exist in the primary region of mountain top removal/valley fill coal mining in West Virginia and Kentucky, 2) determine if any

unique fish populations exist in this area, and 3) evaluate the effects of these mining operations on fish populations residing in downstream areas.

During 1999-2000, fish assemblages were sampled in 58 sites in West Virginia located on 1st through 5th order streams, and in 15 sites in Kentucky located on 2nd, 3rd, and 4th order streams. The majority of the sample sites were selected in consultation with personnel from U.S. Environmental Protection Agency (USEPA) Region III and Region IV. A few sites were added in the field to enhance the characterization of the fish communities in the primary region of mountain top removal/valley fill coal mining. Sites in West Virginia were assigned an EIS Classification based on U.S. EPA Region III classification. Sites in Kentucky were assigned an EIS Classification based on Region IV classifications. Two sites, Stations 6 and 22 (a 2nd order and a 4th order stream) in the Mud River watershed, were sampled during each year, and it was determined that collections at these sites were comparable between seasons. However, results from the 1999-2000 sampling effort indicated that there were not enough reference sites to adequately assess the potential effects of mountain top mining/valley fill operations on fish communities in the area. A strong relationship was found between stream size (as described by stream order) and the total number of fish species present. All of the unmined sites that were to serve as reference sites were located on 1st and 2nd order streams, while sites classified as mined, filled, filled/residential, and mined/residential occurred primarily on 3rd and 4th order streams making direct comparisons between mined and filled sites inappropriate. As a result, in Fall 2001, eight sites in the Mud River that were classified as filled or filled/residential were re-sampled along with five sites in the Big Ugly and three sites in the Buffalo Creek drainages that were chosen to serve as reference (of the unmined condition) sites in the Guyandotte River system.

Due to the confounding effects of drought, small stream size (low stream order), and human impact on reference sites in West Virginia, reference (unmined) sites could not be directly compared to filled sites directly during the 1999/2000 sampling season. Thus, results were developed based on Kentucky sites and 2nd order streams in the New River Drainage where comparable reference (unmined) and filled sites were available. Comparison of unmined sites and filled sites in Kentucky and in 2nd order streams in the New River Drainage indicate that mountain top removal/valley fill coal mining has had an impact on the condition of streams. In general, the number of total species and number of benthic fish species were substantially lower in filled sites than in mined sites in both Kentucky and 2nd order streams in the New River Drainage.

In 2001, the fish samples taken in the mined sites in the Mud River were compared with reference sites sampled in the Big Ugly drainage. Both the Mud River and Big Ugly rivers are part of the Guyandotte River system. Both the total number of species and the total number of benthic fish species were greater in the reference sites (median 17 and 6 respectively) than in the filled sites collected in 2001 (median=8 and 1.5). The total number of species collected during 1999/2000 was considerable higher (median = 12.5) than the total number of species collected at the same sites in 2001 (median 8). Water chemistry analysis revealed that five of the Mud River sites sampled in 2001 had detectable levels of selenium (9.5 - 31.5 μg/l). Filled sites that were associated with detectable levels of selenium seemed to be more impaired than filled sites that had no detectable levels of selenium. Total number of benthic fish species in reference sites (median=6) was higher than those recorded in filled sites with selenium (median = 0) and without selenium (median = 3). The fisheries study noted that a multiple year collecting regimen would be needed to see if there continues to be a decrease in the number of species over time in the filled sites.

This study did not address whether there are environmental benefits of sustained flows from filled watersheds when compared to no-flow conditions in some unmined reference streams. It is possible that the altered flow regimes found downstream from valley fills (USGS 2001) may affect fish habitat for parts of the year in those cases where fish habitat had been previously limited due to seasonally dry conditions. It is also possible that potential benefits from increased flows downstream of mountaintop mining/valley fill operations are offset by changes in water quality. For example, fish collected from one lake downstream of an extensive mining complex in West Virginia were found to contain selenium concentrations much higher than would be expected to occur naturally, indicating that the selenium associated with mining operations occurs in a form that is biologically available for uptake into the food chain (U.S. FWS, unpublished data).

2. Studies Relating to Mitigation Efforts for MTM/VF Impacts to Aquatic Systems

Surface mining operations in steep slope terrain generate excess spoil that is often placed in adjacent valleys. These valley fills encroach and bury headwater stream habitats, and potentially impact stream conditions downstream from fills. Past efforts at compensatory mitigation have not achieved a condition of no-net loss of stream area or functions.

a. Definition of Mitigation

Stream habitat and functions lost through mining and filling are subject to amelioration through mitigation. The Council on Environmental Quality (CEQ) has defined mitigation in its regulations at 40 CFR 1508.20 to include: avoiding impacts, minimizing impacts, rectifying impacts, reducing impacts over time and compensating for impacts. These can be summarized into three general types: avoidance, minimization and compensatory mitigation [MOA between US Army Corps of Engineers and EPA (EPA 1990)]. The objective of compensatory mitigation for unavoidable impacts is to offset environmental losses.

Where mining and filling activities have impacted streams compensatory mitigation may be used to replace lost habitat and functions. Compensatory actions (e.g., restoration of existing degraded wetlands or creation of man-made wetlands) should be undertaken when practicable, in areas adjacent or contiguous to the discharge site (on-site compensatory mitigation). If on-site compensatory mitigation is not practicable, off-site compensatory mitigation should be undertaken in the same geographic area is practicable (i.e., in close physical proximity and to the extent possible the same watershed).

b. Mitigation Goals

In determining compensatory mitigation, the functional values lost by the resource to be impacted must be considered. Functional values should be assessed by applying aquatic site assessment techniques generally recognized by experts in the field and/or the best professional judgment of federal and state agency representatives, provided such assessments fully consider ecological functions in the Guidelines. The ecological functions of Appalachian streams are described in Chapter III.C. Headwater streams receive, process and transport a major portion of the downstream biological energy budget from leaf litter and other terrestrial sources of carbon. Downstream

biological communities are adapted to existing physical, chemical, and biological conditions within these stream arrays

Headwater streams provide habitat for lotic aquatic communities. Generally, in-kind compensatory mitigation is preferable to out-of-kind. Replacement of a mined for filled stream by restoration or creation of a similar type of stream would be more in keeping with this policy than would replacing stream systems with palustrine wetland systems.

In addition, the areal extent of impacts must be considered in the development of successful mitigation efforts.

c. Requirements for Development of a Successful In-kind Replacement Mitigation Project

Stream re-creation is a young but advancing science. In order for streams to be successfully re-created or restored, a range of natural variables must be integrated into the design including: hydrology, hydraulics, water quality, fluvial geomorphology, sediment transport mechanics, plant ecology, macroinvertebrate and fisheries biology and land use (Inter-Fluv 1998). In addition, to mitigate for values lost, size of the mitigation project must be considered.

d. Limiting Factors for In-kind Mitigation Projects

Past efforts at stream construction in association with mine restoration have found limitations in each of the parameters needed for a successful in-kind mitigation effort for headwater streams.

Stream creation on filled areas is very difficult in general due to the inability to capture sufficient groundwater flows necessary to provide a source. There is some suggestion that perennial flow could be established on a contour between the fill and the native rock by the use of some type of impermeable liner. However, no demonstration projects have yet been performed to validate this hypothetical design. Speakers at the Aquatic Ecosystem Enhancement at Mountaintop Mining Sites Symposium (Appendix D) concluded that, at best, streams recreated on mined lands would be expected to have only intermittent flow. As discussed in the USEPA Stream Chemistry Report, several chemical parameters have been found to be elevated in stream surface water downstream from filled/mined areas (USEPA 2002a). Chemical parameters elevated in excess of ambient water quality criteria may impair the aquatic productive of constructed streams.

Post-mining land use surrounding any restored stream would influence the potential functions of that stream. The cumulative impact study (USEPA 2002) found that over 80% of first to third order streams in the EIS study area are surrounded by forest. The cumulative impact study also found that land use for post-mining areas was primarily grasslands. Restored mined areas do not rapidly develop forest cover. This change in surrounding land use represents a factor that may impact the successful restoration of stream functions from a constructed stream.

Establishing aquatic communities of stream-dwelling organisms in restored or created streams depend on the extent to which the physical and chemical environment needed by these organisms has been re-created. It is possible that the elevated flow regimes found downstream from valley fills (USGS 2001) may have created additional fish habitat for parts of the year where previously fish habitat had been limited owing to seasonally dry conditions. It is not known if this increase in

stream length used by fish would be equated to greater fish product or simply represents an increase in area where fish are found.

During the development of this EIS, technical representatives from OSM and from West Virginia have suggested that groin ditches constructed along the edges of fills may represent an opportunity for in-kind replacement of streams with an intermittent or ephemeral flow regime. To date, no drainage structures observed appear to have successfully developed into a functional headwater stream (Appendix D). As discussed in the Aquatic Ecosystem Enhancement at Mountaintop Mining Sites Symposium (Appendix D), creating a more natural channel, increase the structural complexity in the mitigation design by adding boulders, logs and snags and encouraging the restoration of native plant species along a created channel such as a groin ditch would increase the potential for a successful stream creation project. However, the overall limitation to the re-construction of streams in mined and filled areas appears to be the associated with establishing suitable hydrology.

Creation of other ponds and wetland resources on mined land has shown more promise. Wallace (EPA 2000) suggested that these types of systems can be important sites of nutrient storage and uptake provided that a sufficiently vegetated littoral zone is present.

e. Types of Out-of-kind Mitigation

e.1. Onsite

The majority of past efforts at on-site mitigation have been aimed toward the development of palustrine wetland systems to replace streams destroyed through mining and valley filling activities. A review of National Wetland Inventory mapping in conjunction with status and trends information for the study area indicates that natural wetland areas typically found in the steep slope region are generally narrow linear vegetated wetlands along the stream valleys. Wetland areas are being created on reclaimed mine sites. Because steep slope areas are being flattened, it is anticipated that wetland acreage has actually increased as a result of these mining activities.

A number of studies have been performed to evaluate the functions provided by wetlands that have developed on, or been constructed on, mined and filled sites (Pen Coal, 1999 and USEPA, 2000). The results of two of these studies are summarized below.

While wetland areas may be forming on mined sites, the functions being provided by these areas are largely unknown. A technical study was performed by the USEPA to address this issue (USEPA, 2000). Field surveys were performed in November 1999 on ten wetland sites (mainly linear drainage structures and basin depressions) to assess the water quality, wildlife, and sediment trapping functions being provided by wetland areas typically being created on mined lands. The Evaluation for Planned Wetlands technique developed by Environmental Concern, Inc. (USEPA, 2000) was utilized by the field teams to perform these field assessments. The results for three habitat quality descriptors were based upon a score of 0 to 1 (lowest to highest).

Three parameters were evaluated in this study including sediment stabilization, water quality and wildlife. Sediment stabilization is the capacity to stabilize and retain previously deposited sediments. The water quality function is the capacity to retain and process dissolved or particulate materials to the benefit of downstream surface water quality. The wildlife parameter is the degree to which a

wetland functions as habitat for wildlife as described by habitat complexity. Many of the wetland systems were providing excellent sediment stabilization functions, and a few were providing good water quality and wildlife functions. These findings are expected. Generally speaking, sediment stabilization is not a difficult function to establish in a wetland system. Water quality functions are also possible to establish with modest planning. In many of these cases, we suspect that the wetland systems were largely unplanned, and that the low percent vegetative cover was a significant influence on the low degree of water quality function being provided. Finally, wildlife functions are highly dependent on the vegetative communities present, the degree of interspersion, and other physical and biological features of the system. It is not surprising, therefore, to see that this function did not score highly in many of the systems studied. Those areas that scored highly for wildlife function tended to be older systems with more complex structures. It should be noted that the wetlands studied represented wetlands with surface water connects to stream systems as well as isolated wetlands which lacked connectivity to stream systems.

A study conducted by Pen Coal, entitled *An Evaluation of the Aquatic Habitats Provided By Sediment Drainage Ditches and Sediment Control Ponds Located on Mine Permitted Areas in Southern West Virginia* (Pen Coal, 1999), examined the water chemistry and biological communities located in sediment control structures. Three sediment ponds and three sediment ditches were studied. When comparing total abundances and taxa between the ponds, the study found that two of the ponds contained large total abundances of aquatic insects and a desirable number of taxa. One pond contained relatively low abundances and low taxa diversity compared to the other ponds sampled, but this pond had only recently been constructed and may have not yet established an aquatic community. Similar results were found in the sediment ditches. One recently constructed ditch contained a low abundance but moderate taxa diversity. The other ditches contained moderate and high abundances and varied taxa diversity (one was high and the other low). In general, most of the ponds and ditches sampled were well represented by the groups of aquatic insects which are normally present in these lentic habitats. The functional feeding groups scrapers and collecters/filterers were never present, but this was not surprising since these groups need silt free environments and faster moving water. The shredder functional feeding group (those that consume leaves and other detrital material) was also not well represented, but this group is sensitive to disturbances and pollution. Alternatively, the ditches may have lacked an adequate food supply for shredders. Generally, the sites contained mostly tolerant organisms such as midges, dragonflies, and aquatic worms which can tolerate pond habitats.

While the results of this study indicate that the sediment control structures are not functioning as healthy headwater streams based upon metrics commonly used to make such an assessment, it should not be automatically assumed that these systems are of little value to downstream resources. Some nutrient cycling functions may occur in these wetlands. Merritt et al. (1984) summarized the nutrient resource utilization in a variety of aquatic habitats including headwater streams, eutrophic lakes and temporary ponds and discussed that aquatic insects in freshwater ecosystems played a role in the processing, turnover, storage and cycling of nutrients in all systems. However, published studies demonstrating the occurrence of this function in wetlands established on mining sites are lacking.

In summary, to date functioning headwater streams have not been re-created on mined or filled areas as part of mine restoration or planned stream mitigation efforts. Most on-site mitigation construction projects have resulted in the creation of palustrine wetlands that resembled ponds. Some of these

created wetlands are isolated from other surface water systems while others occur in drainage channels which connect to the original stream system at some point. On some fills, linear-shaped wetlands may develop in groin ditches. Functions potentially restored or replaced by these wetlands include sediment stabilization, wildlife support and water quality maintenance. Functions not restored include habitat for aquatic organisms that require lotic or flowing-water conditions. Palustrine wetlands are known to process organic material which may be transported to downstream if the wetland connects to the original stream. However, it is not known whether the organic matter processing that occurs in created wetlands would mimic the processing found in a natural stream system. Functions of man made ponds and wetlands exist and may be considerable. While these functions differ from those of headwater streams, these functions do have their own inherent values. In fact, the establishment of ponds or wetlands on benches or at the toe of mined areas may tend to limit the effect of disturbances on the downstream watersheds (Appendix D: Wallace).

e.2. Offsite

Past efforts by the states in the MTM/VF Study area to initiate offsite compensatory mitigation practices are discussed below. However, past efforts at off-site compensatory mitigation have not achieved a condition of no-net loss of stream area or functions.

West Virginia Mitigation Prior to 1998

The WVDEP indicates that on-site mitigation of stream impacts was not the norm for pre-settlement MTM/VF mining operations in West Virginia. The threshold for wetland mitigation was 1/3 acre of impacts. This threshold was seldom met because wetlands are typically of limited extent within the narrow hollows and valleys of most valley fill sites, and also uncommon on steep slopes or ridge crests. On-site mitigation of stream impacts was also not usually practical due to the configuration of valley fills. A stream mitigation threshold was established where the watershed, when measured from the toe of the fill, was greater than or equal to 250 acres and/or when the fill exceeds ½ acre of stream. In West Virginia, most coal companies opted to pay into a stream impact mitigation fund. Impacts were assessed at a rate of $200,000 per acre for permanent stream impacts from the toe of a fill, measured as length times width at the high water mark of Waters of the State. Temporary sedimentation ponds and culverts in stream channels were assessed at a rate of $20,000 per acre for each five-year period of channel occupancy. Coal companies could also perform other local mitigation or improvement projects in lieu of direct cash payment. Mitigation projects were usually developed in coordination with WV Division of Natural Resources.

Virginia Mitigation Prior to 1998

Prior to 1998 Virginia coal mining permits required limited terrestrial and aquatic mitigation for impacts to intermittent and perennial streams as a result of aquatic disturbances such as in-stream ponds or stream diversions/relocations. Much of this mitigation was driven by the In-stream Treatment Agreement between Virginia DMLR and the Environmental Protection Agency. This agreement states that in-stream structures with drainage areas greater than 200 acres will be mitigated. In many cases the operator would opt to leave sediment structures as wetlands to mitigate for stream disturbances. Prior to 1998 the Division of Mined Land Reclamation had no size requirements regarding fills in-stream or fill minimization procedures, however the Division did obtain terrestrial mitigation on the face of many small head of hollow fills.

Virginia did not have a system for payment into a fund in lieu of on the ground work for mitigation in the coal mining region of the state.

Tennessee Mitigation Prior to 1998

The Tennessee Water Quality Control Act including the 1994 amendments required a permit for activities resulting in alterations to the physical, chemical, radiological, biological, or bacteriological properties of any waters of the state.

Prior to 1998 an Aquatic Resource Alteration Permit (ARAP) or 401 certification was required for alterations resulting in alterations to the physical properties of waters of the state. The compensatory mitigation ratio for alterations to wetlands including fill activities was at least 3:1. Fill in waters deemed to be perennial streams was prohibited. Mitigation requirements for ephemeral and intermittent streams were established in the permit conditions of an Individual Aquatic Resource Alteration Permit for activities such as stream relocation. Isolated wetlands equal in size to 0.25 acres, not connected to other waters of the state and deemed non-jurisdictional by the USCOE, and wet weather conveyances were covered under a General ARAP without any compensatory mitigation.

The State of Tennessee has never established any system for which payments to a fund could be made in lieu of groundwork for mitigation. However, the state is currently developing guidelines for establishment of such a fund provided the proposed activity meets certain criteria.

E. COAL MINE DRAINAGE FROM SURFACE MINING

1. Study Area Water Quality Summary

The United States Geological Survey (USGS) has published a series of Open-File reports investigating the hydrology of designated watershed areas (classified by number, called Hydrologic Unit Codes, or HUCs) within the Eastern Coal Province. Many of these watershed areas fall partially or wholly within the study area, but are generally larger watersheds, e.g., 2-10 square miles, and thus may not necessarily represent typical headwater stream water quality. Generally, headwater streams have good water quality (USFWS 2000). The majority of these USGS watershed reports, date from 1981 to 1987 and are currently being updated. Recent reports are available for the Kanawha-New River Basin. These USGS reports characterize the surface water quality and quantity of mined and unmined regions in watershed areas. Watersheds that were assessed include the Little Sandy River and Tygarts Creek in Kentucky; the Clinch, Emory, Obed, Sequatchie, and Tennessee Rivers in Tennessee; the Powell and Clinch Rivers in Virginia; and the Gauley, Elk, Coal, New, Pocatalico, Guyandotte, and Kanawha Rivers as well as Twelvepole Creek in West Virginia.

THE PREDOMINANT SOURCE OF ACID MINE DRAINAGE IS PRE-SMCRA MINING.

The reports indicate that many of the watersheds were affected by coal mining activity, including surface and underground mining, construction and use of ancillary facilities such as roads, coal processing and coal transport. Many mines are located adjacent to or near streams and rivers to permit transport of coal by river barge and railroad. Most coal moves from the mines by rail or truck to a terminal near the larger rivers, and by barge or rail to the final destination. Mines, waste piles, and coal preparation plants, which are located close to streams and rivers, increase the potential for serious water-quality impairment–if improperly treated wastes are discharged. All watersheds appeared to have localized intensified areas of mining that result in moderate to severe degradation of surface water quality. Typically, there were substantial differences in measured values between mined and unmined areas. In areas of mining, decreased pH values and increased values of specific conductance, metals, acidity, sulfate, and dissolved and suspended solids were seen. These USGS reports indicate that localized surface water quality is also compromised by municipal and industrial wastewater discharges and land use changes and development (USGS, OFR 81-803). The 1980 vintage USGS studies may not represent post-SMCRA water quality. The predominant source of acid mine drainage is pre-SMCRA mining. The recent Kanawha-New River Basin studies indicate good surface water quality (Eychaner 1994 and 1998).

Streamflow in unregulated streams typically varies greatly during the year, following the precipitation and evapotranspiration regime. The greatest mean monthly flows usually occur during March, as a result of snowmelt runoff, increased precipitation, and relatively low evapotranspiration. Streamflow during spring and early summer is usually high as a result of increased thunderstorm activity. Streamflow recession during late summer and early fall results from evapotranspiration losses and decline of precipitation activity. During November and December, streamflow usually increases as evapotranspiration decreases and the winter rains begin (USGS, OFR 81-803). Flow duration is affected by many natural basin characteristics such as topography, geology, size of drainage area, climate, and by activities of man, including streamflow regulation and mining.

Surface and underground mines can affect streamflow duration when streamflow is augmented by mine drainage or pumpage (USGS, OFR 81-902).

2. Coal Mine Drainage

Coal mine drainage (CMD) is drainage from surface mining that causes water quality problems. CMD is the characteristic water that is produced from the increased weathering of minerals associated with backfilled material. In undisturbed geologic areas, groundwater flow in rock is typically along zones of secondary permeability, that is, faults, fractures and bedding planes. Minerals associated with the bedrock in the groundwater flow areas have been extensively weathered over millennia. However, during surface mining, the overlying contiguous bedrock that exists over the coal seams, also known as overburden, is broken up into smaller more homogenous rock particles. This break up increases contact of minerals by exposing new minerals to air and water. The exposure results in additional and increased weathering of minerals in the backfilled material.

Sulfide minerals, such as pyrite (FeS_2), are often associated with coal and overburden and are the primary minerals involved in the development of CMD. The oxidation of pyrite leads to the production of acidity and release of sulfate (SO_4^{2-}) and ferrous iron (Fe^{2+}) as indicated in the following reaction (Stumm and Morgan 1996):

$$FeS_{2(s)} + 3.5O_2 + H_2O \Rightarrow Fe^{2+} + 2SO_4^{2-} + 2H^+$$

Additional acidity is released from ferrous iron, as indicated in the following oxidation, hydrolysis (the splitting of a compound into fragments by the addition of water, the hydroxyl group being incorporated in one fragment, and the hydrogen atom in the other) and precipitation (the flocculation and settling of materials, in this case, such as iron hydroxides, following their chemical reaction in mine drainage) reaction (Stumm and Morgan 1996):

$$Fe^{2+} + 0.25O_2 + 2.5H_2O \Rightarrow Fe(OH)_3 + 2H^+$$

The above reaction is a simplification, in that the pH, cations (e.g., positive ions such as sodium and potassium) and anions (e.g., sulfate and chloride) affect the precipitation of ferric iron (Fe^{3+}) and precipitate formed, such as, goethite, lepidocrocite and jarosite (Nordstrom 1982). Acidity, soluble ferrous and ferric iron released from pyrite oxidation are capable of reacting with a variety of carbonate minerals (e.g., limestone, dolomite and siderite) and silicate minerals (e.g., clays, mica and feldspar) during neutralization and cation exchange processes (Rose and Crovotta*,* 1998*).* It is these reactions that can increase concentrations of a variety of common metals (e.g., calcium, magnesium, manganese and aluminum) and trace metals (e.g., copper, cadmium, nickel and zinc). The resulting CMD will vary widely in composition, depending on the characteristics of the backfilled material and reclamation practices. There are generally two categories of CMD: acidic mine drainage (AMD) and neutral/alkaline mine drainage (NAMD) (Rose and Crovotta*,* 1998). Both types reflect, to some degree, oxidation of sulfide minerals and the release of acidity, iron and sulfate.

AMD is the category of mine drainage in which mineral acidity exceeds alkalinity. In many cases there is <u>no</u> alkalinity present. The pH of AMD varies widely from 2 to 6, and acidity ranges from

0 to 1000s of mg/L (measured by standard practice in terms of $CaCO_3$ equivalency, or the amount of calcium carbonate per unit volume that it would take to neutralize the acid sample). The high acidity is a result of elevated concentrations of dissolved metals--primarily iron, aluminum and manganese. These metals can be hydrolyzed and release additional acidity. In addition to hydrolyzable metals, AMD contains a variety of anions (negative ions, in this case primarily sulfate) and cations (such as, calcium and magnesium) that are the result of the pyrite oxidation, neutralization reactions and cation exchange in the overburden. Sulfate levels will range from 50 to 1000s mg/L depending on the amount of sulfide minerals and oxidation rates in the backfilled material.

NAMD is the category containing alkalinity equal-to-or-greater than mineral acidity. Since pH is circumneutral, that is approximately 7, mineral acidity is associated with dissolved ferrous iron and manganese only. Aluminum solubility is very low, less than 0.5 mg/L, at circumneutral pH. Dissolved metals and sulfate vary considerably in NAMD, depending on whether sulfide mineral oxidation occurs prior to or after groundwater has contact with an alkaline material, such as limestone. Sulfate and dissolved metals are typically lower in mine drainage where alkalinity is present before contacting sulfide minerals, due to lower oxidation rates that occur at an elevated pH. Greater dissolved metals and sulfate result in NAMD where neutralization and alkalinity is added after sulfide minerals are oxidized, a result of accentuated mineral sulfide rates at lower pH (Moses 1987). This difference is important. There is a greater potential for trace metals and metalloids to be contained in the NAMD, formed as a result of the later process, due to increased weathering and greater solubility.

a. Indicator Parameters

As previously discussed, mining activities tend to increase weathering of rocks and, as a result, increase the amount of dissolved minerals in the contact water and in watersheds containing mining activity. A number of other anthropogenic land uses, such as, agriculture, silviculture and urbanization, are also known to increase dissolved minerals in surface waters. Two parameters, specific conductance and total dissolved solids (TDS), are used to estimate the amount of dissolved minerals in mine drainage, other contaminated and natural waters. Specific conductance is a measure of the ability of water to carry an electrical current (as measured using a current cell and meter detecting the current returned) and is proportional with the quantity of ionized minerals in solution. Specific conductance rises with increasing dissolved minerals. TDS is measured by drying the matter (suspended solids) remaining after water is passed through a filter (APHA 1989). The two parameters can generally be correlated with specific conductance typically representing about 1.1 and 1.9 times TDS in most waters. Unfortunately, sample handling and methodology can often alter TDS and specific conductivity results, which may affect direct comparison of the two parameters. There is no accepted natural range for either parameter in "uncontaminated" water, due to their dependence on surrounding geology and land use. However, natural or unpolluted freshwater generally have specific conductance between 20 and 500 micromhos (µmhos) and TDS between 10 and 250 milligrams per liter (mg/L). As reported in Rose and Crovotta (1998), CMD has been reported to have specific conductance in excess of 5,000 µmhos (TDS of 3,000 mg/L).

A common parameter used to assess water quality and evaluate impacts of mine drainage is pH (the measure of the hydrogen ion activity $\{H^+\}$ in water) and is typically estimated using an electrode and meter calibrated with known pH buffer solutions (APHA 1989). The pH scale is 0 to 14, but

pH of natural or unpolluted waters generally fall between 5 and 10. Typical convention is to consider a water with a pH of 7 as neutral, values less than 7 as acidic, and greater than 7 as alkaline. This convention may lead to confusion in evaluating impacts of mining, since waters with pH in the 5 to 7 range may occur naturally and have alkalinity present in excess of mineral acidity (parameters discussed below). It may be more appropriate to consider pH as an indicator of aquatic health-- with optimal pH for aquatic life falling between 6 and 9. This pH range is also the range of minimal solubility for most toxic metals (e.g., aluminum, copper and zinc) that may be present in water (Stumm and Morgan 1996); the exception would be reduced metal species such as ferrous iron, which are unstable under oxidizing conditions. Values of pH outside this range (less than this range in the case of mining), would be suggestive of coal mine discharge-related impacts if and only if, other indicator parameters are also present. For example, acid rain-impacted surface waters may also have lowered pH (Herlihy *et al.* 1991).

Alkalinity, usually reported as milligrams per liter (mg/l) of $CaCO_3$, is an aggregate property of water that reflects its ability to neutralize acid inputs and in natural waters is typically a measure of the bicarbonate ion (HCO_3^-). In combination with acidity (carbonate system only), the two parameters assess the ability of a water to resist pH change, which is commonly referred to as "buffering capacity." Alkalinity is measured by titrating a sample (adding a solution, with an eye dropper-like device called a pipette, drop-by-drop) with a known acid concentration to a pH endpoint between 4 and 4.5. This endpoint is known by a color change of the titrated water with a pH sensitive dye called bromocresol green, or is measured with a pH meter (APHA 1989). Natural or unpolluted waters will range from near zero buffering capacity, for smaller headwater streams and poorly buffered waters, to more than several hundred milligrams per liter buffering capacity, for larger waters and waters in predominately limestone regions. Coal mining can cause alkalinity to increase or decrease, in the receiving stream, depending on overburden characteristics and mining and reclamation practices.

Acidity in natural or unpolluted waters (usually reported as milligrams per liter (mg/l) of $CaCO_3$), is another aggregate property of water that reflects its ability to neutralize base inputs and in natural waters, is typically a measure of the presence of carbonic acid (H_2CO_3) and bicarbonate ion (HCO_3^-). In conjunction with alkalinity, these two parameters represent the buffering capacity. Carbonate acidity is titrated with a known concentration of base solution to a pH endpoint of 8.3, as determined colorimetrically with metacresol purple or with a pH electrode and meter (APHA 1989). Acidity in CMD can be difficult to evaluate, because of the potential presence of reduced forms of primarily two metals, iron and manganese, which may or may not be included in the standard acidity titration method. In evaluating mine related acidity, it is frequently necessary to measure a different type of acidity, known as hot mineral acidity, which will include the contribution of reduced forms of metals on acidity. This method uses acid to lower the pH. and remove carbonate-related acidity, hydrogen peroxide as an oxidant, and heating to increase the rate of oxidation prior to titrating to the pH 8.3 endpoint (APHA 1989). This hot mineral acidity is also an aggregate parameter of the potential of a water to depress pH from the release of hydrogen ions during the hydrolysis and precipitation of soluble metals. Difficulty arises in evaluating hot mineral acidity results due to reporting differences in coal mining-related studies (frequently reported as acidity, total acidity, mineral acidity and total mineral acidity) and as negative or zero values where alkalinity exceeds hot mineral acidity. Hot mineral acidity reported from a number of coal mined sites (abandoned and permitted) ranged from zero to several thousand milligrams per liter (Rose and Crovotta, 1998).

Sulfate is a good indicator of influence by CMD, because its presence in coal mined areas is generally indicative of sulfide mineral oxidation. Natural or unpolluted freshwater can have elevated sulfate levels in the five to twenty milligram per liter range, depending on the influence of acidic deposition and connate water. In addition, sulfate can be increased from a variety of anthropogenic sources, including treated and untreated wastewater, urban and residential runoff, and agricultural practices. Common methods for analytical sulfate concentration determination include ion chromatography and turbidimetric methods (APHA 1989). Elevated levels of sulfate in CMD can exceed several thousand milligrams per liter and, as a result, can be increased in receiving surface waters to appreciable levels, depending on the CMD source and extent of mining throughout the watershed.

The most common metal used to evaluate impacts of coal mining on surface waters is soluble or total iron, which is contained in CMD, as a result of, sulfide mineral oxidation; iron may also be present from the solubilization of siderite. In most natural or unpolluted surface waters, soluble iron is either near or less than quantifiable concentrations due to its relative insoluble properties in oxidizing and circumneutral water environments. Soluble iron can be found in unpolluted surface waters, such as lake hypolimnion (bottom waters) and groundwater where low dissolved oxygen levels persist. The impact of soluble iron on water quality is generally related to drinking water aesthetics, taste and odor. However, at high concentrations, exceeding 1 mg/L, iron oxidization and precipitation in surface waters can impact stream and lake bottoms due to the formation of "yellow boy" precipitates or staining, named for its yellowish-red appearance, which destroys habitat for aquatic insects and spawning fishes (Hoehn and Sizemore 1977). Iron concentrations are determined colorimetrically or by atomic absorption spectrometry (APHA 1989) and for CMD can range from less than one to values greater than several hundred milligrams per liter.

In addition to iron, manganese is frequently evaluated as an indicator parameter of CMD impacts on surface and groundwater. Its presence is usually considered a result of secondary weathering of carbonate minerals (Crovotta *et al.* 1994). In most natural or unpolluted surface waters, soluble manganese is absent due to its limited solubility in oxidizing and circumneutral water environments similar to iron. If present, manganese may persist in surface waters longer than iron, due to much slower oxidation rates. The effects of manganese are generally related to drinking water aesthetics, taste and odor. EPA established CMD discharge limits for manganese based on links of its presence to toxic metals (e.g., copper, cadmium and nickel) in AMD. Recent studies indicate that other parameters, such as zinc or hot mineral acidity, may be better indicators of the presence of trace metals (Unz and Royer 1997). Manganese precipitation in surface waters may cause similar impacts as "yellow boy" and higher concentrations of manganese (concentrations in excess of 20 mg/l) may be toxic to early life stages of fishes (Lewis 1976, England 1977, Lewis 1978).

Aluminum is another metal frequently found in AMD, but is typically not found in NAMD. Its present is a direct result of secondary weathering of silicate minerals (e.g., clays). The presence or absence of aluminum is a direct result of pH-dependent solubility, with aluminum solubility increasing from, much less than 1 mg/L at circumneutral pH, to greater than 100 mg/l at pH less than 3 (Stumm and Morgan 1996). In soluble form, aluminum is hydrolyzable. In this form, it can be one of the major total "hot" acidity components in AMD, but is of little importance in NAMD or AMD, where the pH is greater than 5. Aluminum, when present in soluble form, is toxic to aquatic life at concentrations as low as 0.1 mg/l (Gagen *et al* 1994), but its pH-dependent solubility limits the toxic conditions to water of pH typically less than 5.5; water of pH greater than 9 may also

contain appreciable soluble aluminum. As an indicator, aluminum would only be of value in low pH waters where other parameters would be present at levels to provide sufficient evidence of mine drainage influence. In addition, since aluminum is one of the most common elements in the earth's crust, its presence, when measured as total aluminum, may be related to suspended solids contained in the sample from various sources, including eroded sediment carried in high flow runoff events and sediments entrained during sample collection.

Total suspended solids (TSS), the measure of particulate material suspended in a sample, is frequently included in parameters used to assess CMD-related impacts. TSS is measured gravimetrically, by weighing the amount of solids captured on a filter (APHA 1989). TSS can be a useful parameter to evaluate entrainment of sediment into a sample and erroneous iron and manganese measurements, which are frequently measured as total (requiring acid digestion) concentrations. In addition, TSS in waters is an indicator of upstream erosion, which may be the result of earth disturbances such as surface mining. TSS may also be increased in surface waters from other anthropogenic activities related to agriculture, silviculture and urbanization. Changes and differences in TSS concentrations associated with surface mining are also difficult to identify and assess, because TSS typically only occurs during storm-related runoff events, and is dependent on rainfall intensity, duration and antecedent conditions.

b. Effects of Coal Mine Drainage

Coal mine drainage can have a significant environmental impact, particularly on pre-SMCRA mine sites where prevention controls were not required. Once AMD occurs, it is a long-term problem. This section provides a summary of environmental impacts of CMD in surface coal mining operations.

CMD can cause chemical toxicity to aquatic life. Most aquatic organisms have specific pH tolerance ranges within which they can survive, and changes in pH resulting from CMD may result in poor health or mortality. An example would be fish kills that occur when large precipitation events flush acidic water from abandoned deep mines into streams. Fish usually cannot survive in streams with a pH of 4.5 or less (Doyle, 1976). Similarly, at reduced pH, aluminum and manganese can reach lethal levels, as well as combinations of mineral acids and iron and sulfur ions (Gore 1985). In severely impacted streams, CMD chemical toxicity may eliminate all aquatic life.

CMD may produce physical and chemical impacts to streams as a result of chemical precipitation. As CMD discharges co-mingle with cleaner surface waters, acidity is reduced, and entrained metals and sulfate become increasingly unstable in solution. Iron and aluminum will tend to precipitate as hydroxides forming orange and white (yellow boy) sludge that coats stream bottoms. If calcium is present in solution and the pH is sufficiently elevated, gypsum ($CaSO_4$) will also precipitate. These sludge materials have the effect of smothering the stream bottom, inhibiting the feeding and reproduction of benthic macroinvertebrates (worms, nymphs, crustaceans, etc.) and destroying fish spawing habitat.

CMD can adversely affect human populations by impuring surface and ground water used for drinking water and recreational purposes. Public and private water supplies drawing from CMD-affected sources may require additional treatment processes to produce potable water, which can add significantly to the cost of the water supply. Loss of aquatic life in a water body reduces the

recreation values, with attendant economic losses to the surrounding community. In terms of aesthetics, CMD can have a significant visual impact in affected streams resulting from the unnatural appearance of the iron sludge coating. Acidic waters can also affect physical structures, increasing corrosion of steel and concrete bridges, culverts, and other in-stream structures, reducing their functional lives. OSM compiled preliminary records on post-SMCRA mine sites that have CMD problems (OSM, 2000). These include sites still active and sites where bond forfeiture occurred, as shown in Table III.E-1. Several of these sites may be in Western Kentucky and Northern West Virginia outside of the EIS study area, and as many as a third of the sites may be underground mine sites. However the information does provide a general indication of the scope and significance of CMD.

Table III.E-1 Estimates of Post-SMCRA CMD Sites for States in the Study Area

State	Active Mine Sites		Bond Forfeiture Sites	
	# Permits	# Discharges	# Permits	#Discharges
Kentucky	10	10	27	30
Tennessee	13	34	2	3
Virginia	24	26	6	6
West Virginia	363	635	119	286
Total	410	705	154	325

Table III.E-2 shows the estimated amount of CMD from the four states in the EIS study area, as well as the types of estimated "loadings" (e.g., chemical constituents per unit volume of flow) for several indicator parameters. The data presented under the average column headings are averages per site. The data presented under the total column headings are totals per state.

Table III. E-2 CMD Flow and Loading Estimates for Post-SMCRA Mine Sites in Study Area States

State	Total Flow (gpm)	Average Acidity (mg/L)	Total Acid Load (lbs./day)	Average Alkalinity (mg/L)	Average Fe (mg/L)	Average Mn (mg/L)	Average Al (mg/L)
Kentucky	1,094	341	5,534	45	78	6	16
Tennessee	1,908	110	1,892	76	51	16	1
Virginia	3,508	81	232	81	6	4	10
West Virginia	59,993	299	111,158	43	41	16	20
Total	66,503	208	118,816	61	44	11	12

3. Methods of Controlling CMD

Once established, CMD is typically a long-term problem that is technologically or economically difficult to correct. Avoidance of CMD is a provision of the hydrologic balance protection standard in SMCRA, and regulatory agencies are authorized to restrict mining if there is a risk of CMD formation. Mining in potentially toxic areas is not precluded, but the mining applicant must demonstrate that CMD formation can be avoided by mining and reclamation practices (OSM 1994). In the event of CMD formation, the permit holder becomes liable for treatment of the CMD discharge to meet CWA receiving stream standards until such time as the situation is corrected, which can represent a considerable expense for long-term treatment obligations.

The simplest form of CMD prevention is avoidance of coal or overburden containing excessive amounts of pyritic material. The permitting process for a mine site normally requires collection of overburden and coal samples to be analyzed for pyritic content (usually expressed as total sulfur content) and neutralizing potential or alkalinity (usually expressed as tons of calcium carbonate equivalent per thousand tons of material). If the acidity generation potential of the pyritic material exceeds the neutralization potential of the overburden and coal, the area represented by the samples is considered to have potential to cause CMD if mined. If the acidity generation potential greatly exceeds the neutralization potential, the site may be considered of too great a risk to mine by either the coal operator or the reviewing regulatory agency. For low- to moderately-acidic sites, various practices may be employed to reduce the risk of CMD generation, as discussed in the following section.

The annual costs for Kentucky, Tennessee, West Virginia, and Virginia sites treating CMD are estimated to exceed $37,000,000 for active mine sites and $5,600,000 for forfeiture sites. The cost to construct treatment systems at sites where discharges are untreated in the four states is estimated at $3.8 million for active sites and $3.1 million at forfeiture sites. Thus, the impact is not only environmental, but economic as well. The high, long-term cost of CMD treatment serves as a strong incentive for mining companies to avoid coal seams and overburden in known CMD-producing areas. Where avoidance does not occur, companies take special care in development of mining plans with special handling controls to prevent or minimize CMD development (see next sections). Both

SMCRA and CWA are designed to minimize CMD problems through proper planning and handling, but if all else fails, long-term treatment will be required to assure minimal impacts to the hydrologic resources.

a. Overburden Blending

If potentially toxic pyritic materials are scattered or contained in stratigraphic units that cannot be readily identified and segregated during mining, and the overburden is found to be net alkaline on a whole for a given mining area, most mining operations will use overburden blending to avoid CMD formation. This concept assumes that excavation and backfilling processes will sufficiently mix the toxic materials with other non-toxic or alkaline overburden materials to form a relatively homogeneous, "net alkaline" spoil. Alkaline materials may also be redistributed within a mine site from areas of excess alkalinity to areas of alkaline deficiency. This method is generally the most common in use for MTM/VF mining sites.

b. Isolation Methods

The concept behind isolation of potentially toxic overburden is to prevent contact between pyritic material and oxygen and water, thereby excluding both reactants necessary to form CMD. Isolation requires selective collection and placement of toxic materials during mining (a process known as *special handling)*. Toxic materials are typically segregated during mining and placed in backfill "pods," which are elevated above the anticipated postmining groundwater level and may be encapsulated by non-toxic materials to further inhibit contact with oxygen (Perry et. al. 1998). This adds to the cost of the mining process, because of the additional material handling steps and the necessity, in some cases, to create additional mine benches on toxic overburden horizons that would not be needed to recover coal seams alone. Isolation is another commonly-proposed method of CMD avoidance in MTM/VF mining.

c. Submergence Methods

Submergence of toxic overburden materials is a form of isolation, in that oxygen is expected to be excluded from contact with pyritic materials by permanent submergence under water. This requires a relatively flat isolation area with a deep, permanent, and essentially stagnant postmining water table to prevent migration of oxygen into the containment area. This method is not widely used in the Appalachian mining region (Perry et. al. 1998).

d. Alkaline Addition

A direct approach to correcting a net deficiency in overburden alkalinity is to add alkaline material during the backfilling process to serve as a neutralizing agent. This method has been applied on a number of mine sites with varying degrees of success. Crushed limestone, kiln dust, or alkaline fly ash materials are typically used as neutralizing agents, and may be placed on the pit floor prior to backfilling, mixed with spoil during backfilling, or applied to the reclamation surface during regrading. A combination of pit floor spreading and backfill blending appears to be the most effective. The most successful alkaline addition sites are those that have used substantial addition rates (500 tons per acre or more) or those with low cover overburden and very low concentrations of pyritic materials (Smith & Brady 1998). This practice requires a ready source of alkaline addition

materials--either on the mine site, or within an economical haulage distance. Because of the scale of MTM/VF mining operations, alkaline addition could represent a considerable expense for mining; and blending or isolation methods are preferred.

4. Abandoned Mine Lands

Abandoned mine lands (AMLs) are pre-SMCRA (i.e., mining occurred before 8/3/77) sites where mine operators were not necessarily required to conduct various backfilling, regrading, or revegetation techniques and where SMCRA reclamation bonds were not applied. These lands are widespread within the study area coalfields, visible as unreclaimed spoil piles, open highwalls, coal refuse piles, and abandoned mine facilities. AMLs can represent a considerable reclamation liability to the public, as pre-SMCRA mine operators are not normally under a legal obligation to reclaim them. AMLs are a primary source of AMD discharges, often representing physical hazards due to unreclaimed highwalls and unstable slopes, and are visually unattractive and are generally low productivity lands.

Under SMCRA, the OSM was authorized to oversee the implementation of and provide funding for state AML reclamation programs. Funding was established by a tax on mined coal, and AML funds are redistributed to the states based on their primacy status and the priority listing of their abandoned sites for reclamation. Kentucky, Virginia, and West Virginia administer their own AML programs, while Tennessee's AML Program is administered directly by OSM, although in cooperation with a State agency. Although these AML programs have been successful to date in remedying most "high" priority AMLs, many "low" priority sites still await funding for reclamation. The "high" priority sites are to correct safety hazards. Environmental remediation is not in the highest priority category; therefore, the ability for AML funds to correct environmental problems is extremely limited. Recent collaboration led by OSM with EPA, COE, and other agencies created the Appalachian Clean Streams Initiative (ACSI) that is addressing pre-SMCRA CMD problems through construction of passive treatment and other remedial approaches. While AML funding through ACSI has steadily increased, many mining program experts believe it will not be possible to effectively remediate aquatic resources damaged by past mining through the AML program.

5. Remining

A coal remining operation is defined by CWA Section 301(p) as "...a coal mining operation which begins after February 4, 1987 at a site on which coal mining was conducted before August 3, 1977," and a remined area is "...only that area of any coal remining operation on which coal mining was conducted before August 3, 1977." In essence, remining is new coal mining undertaken in areas of pre-SMCRA mining activities, including AMLs. The term is considered separate from new mining conducted on sites mined after August 3, 1977 because of certain water quality liability relief measures afforded by CWA Section 301(p) for potentially beneficial reclamation activities on pre-SMCRA sites.

Remining represents an avenue for achieving low- or no-cost reclamation of AMLs, with private mine operators affecting the reclamation as part of their normal mining operations on a site. Remining normally occurs where unmined coal reserves on pre-SMCRA sites have become economical to mine because of advances in equipment capabilities and mining methods. MTM/VF operations, for example, can completely recover high-cover (a large ratio of overburden to coal

volume) coal seams that could have only been economically and partially-mined from the outcrop by operations occurring decades ago. Remining operations generally must meet the same reclamation standards as other surface coal operations, and leave remined areas reclaimed to modern standards on completion. Exceptions from meeting the standards for water quality are relaxed where remining occurs in areas with pre-existing CMDs. Highwall elimination, topsoil salvage, and revegetative success standards also are adapted to remining situations.

a. Water Quality Benefits of Remining

Adverse water quality conditions on AMLs are often related to the mining technique that was employed on the sites. Pre-SMCRA surface mines often did not employ backfill regrading or special handling techniques for acid- and toxic-forming overburden, leaving spoil cast in loose, irregular piles and open mine pits where water could pool in contact with acidic pit floor materials. Pre-SMCRA underground mines also were not designed for AMD prevention and underground mine pools formed in flooding mines after abandonment, exposing to acidic materials in remaining coal to water and oxygen from open mine entries.

On surface mine sites, remining may ameliorate existing AMD conditions by regrading and revegetating the unreclaimed spoil surfaces to restore a more natural surface runoff pattern and limit the infiltration of atmospheric oxygen into the spoil. Backfilling and regrading of highwalls eliminates open pit floor pools and reduces the exposure of groundwater on the pit floor to oxygen following backfilling. Remining may extend to greater cover depths than historic operations and potentially liberate greater amounts of alkaline material, which tends to naturally weather out under low cover. Revegetation also reduces sediment runoff from sparsely vegetated or unvegetated spoil piles and pit floors. Hydrologic routing during mining and reclamation also controls the points at which infiltration and contact with CMD-forming spoil can occur.

On some sites, it is economical to mine former underground mine workings, depending on the depth of the seam and the quantity of the coal remaining. This process is known as *daylighting*, whereby some or all of an underground mine is excavated, and the void is backfilled with spoil once the coal has been recovered. This can be very beneficial to water quality, since groundwater pooling in mine voids is in contact with potentially acidic material remaining in the coal, roof, and floor materials. Ongoing collapse of mine voids tends to rejuvenate the exposure of pyritic materials over time, continuing the process of AMD formation for long periods. After surface mining and reclamation, no voids remain in the remined areas, and the pyritic material in the coal and mine roof is replaced with more homogeneous spoil, potentially with neutralizing alkaline material (if present in the overburden). Remining can also redirect groundwater movement patterns by eliminating preferential drainage paths along structural gradients in mine voids, potentially reducing the quantity of water draining to any remaining underground mine workings.

A study conducted in the Pennsylvania bituminous coalfields indicates that the majority of remining operations resulted in either no change or an improvement in water quality in terms of contaminant loading (Hawkins 1994). Loading is the mass of a contaminant carried by water, as opposed to its concentration, and is a better measure of the potential impact of a discharge on downstream water quality. Of 24 sites studied, 8 showed significant reductions in AMD contaminant loadings, as opposed to 4 that showed significant increases, with the remaining 12 sites showing no significant change in water quality. The study notes that significant increases in water quality were usually

associated with operations that daylighted substantial areas of abandoned underground mines. It is also suggested that water quality improvements may not be immediately apparent following remining due to the time necessary to equilibrate the site, and that several years of observation may be necessary to assess any long-term benefits.

b. Regulatory Aspects of Remining

One deterrent to remining by private mine operators is the presence of pre-existing AMD on previously mined sites. Under normal circumstances, the mine operator would assume responsibility for non-compliant discharges emanating from a permitted mine site. To promote remining reclamation, CWA Section 301(p) provides for federal or state mine permitting programs to allow special provisions concerning pre-existing discharges on remining sites. Briefly, applicable permitting programs may modify effluent limitations on a case-by-case basis for pH, iron, and manganese where pre-existing discharges will be affected by remining operations. Adjustments to effluent standards are made using best available technology and best professional judgement to set site-specific numeric effluent limitations. The permit applicant must demonstrate that the remining operation will result in potential water quality improvements. The applicant must also not allow pH levels to drop or iron and manganese levels to rise (above levels before remining) or exceed state water quality standards. The Interstate Mining Compact Commission (IMCC), a network of coal mining regulatory programs formed by the governors of numerous coal mining states) has a remining task force. Kentucky, Virginia, Tennessee, and West Virginia have active remining programs to promote remining. The Energy Policy Act of 1992 (modifying SMCRA) has remining provisions.

Kentucky

The Kentucky Department for Surface Mining Reclamation and Enforcement (DSMRE) strongly supports and has been encouraging remining activities for several years. Remining benefits both the people and the environment of Kentucky through reclamation of abandoned mine lands at little or no cost to the government. Kentucky Reclamation Advisory Memorandum 129 (RAM #129) discusses certain issues and procedural matters related to remining operations and implementation of the incentives. RAM #129 includes relevant definitions and explains eligibility, permitting, and bonding related to remining.

Permittees may enter into an agreement with DSMRE for reclamation of AML-eligible sites adjacent to coal mining permit areas. However, DSMRE is not obligated to enter into any Reclamation Agreement. Criteria, as listed in RAM #129, must be demonstrated for DSMRE to consider an AML Reclamation Agreement with a permittee.

RAM #129 criteria include:

- The proposed reclamation area must have been determined to be AML-eligible by the DSMRE's Division of Abandoned Mine Lands (DAML). The eligible reclamation site will be inventoried by the DAML and registered on the national Abandoned Mine Land Inventory System (AMLIS).
- The proposed area must be identified by the DAML as priority III, or greater, in accordance with KRS 350.555 and Section 403(a) of the Federal Surface Mining

Control and Reclamation Act of 1977.

- The proposed area must be causing off-site environmental impacts, but with little likelihood that the site could be addressed under the AML program in the foreseeable future.

Virginia

Virginia has an active Clean Water Act section 301 program. Virginia is actively working with the EPA in pursuing a regulation change to the Clean Water Act for the coal remining category for discharges from remining sites. Seventy percent to eighty percent of Virginia surface mining permits include at least some AML areas, according to a ongoing Virginia DMME study. Anecdotal information indicates that this percentage is on the increase, as very few first-cut surface mining operations are currently active in Virginia.

Virginia's experience documents the significant environmental benefits of remining on AML properties. Individual operations have eliminated eroding outslope areas, daylighted acid-producing AML deep mines to produce improvements in water quality, and backfilled dangerous highwall areas with excess spoil from active mines. One severe AMD/AML site, eligible for ACSI funds, has been substantially reclaimed via a remining operation and water quality has been improved dramatically–without expenditure of AML Trust Fund dollars.

Coal mine operators will not seek to permit most AML areas for fear of incurring liabilities that they will not be released from. These areas include barren and eroding outslopes, unstable highwalls, AMD seeps, open pits, and underground mine portal openings. To encourage remining of the AML areas, Virginia DMME has been providing incentives for AML reclamation by active mining operations. Several of the incentives are being formalized through rule changes proposals filed with OSM for program amendment approval. These program changes would result in increased AML reclamation via remining. For example, the “no-cost AML contract” allows active operators to backfill AML highwalls, cover acid-forming material, and to stabilize outslopes; improving environmental conditions and reducing or eliminating spoil placement in hollow fills.

The Virginia coal industry is on the decline. Coal production has fallen from 38 million tons in 1997 to 32 million tons in 1999. Without these mining operations, the opportunity to reclaim these AML sites would be lost. Once an operator mines through an area, the remaining coal reserves are depleted.

West Virginia

Remining operations are addressed by Module 13 of the WVDEP surface mine permit application. Applicants must provide an abatement plan discussing the alternatives considered, and detailing why the selected remining plan will result in water quality improvements. Applicants are required to collect consecutive bi-monthly samples for one year (24 samples minimum) from pre-existing discharges in areas to be affected by remining, and from upstream and downstream sample points on receiving streams for these discharges. Acidity, iron, and manganese effluent limitations are based on loading rather than concentration.

Daily maximum effluent limits are established for each discharge by using the third greatest concentration observed in the entire baseline monitoring data set for each parameter. Monthly average limitations are established separately for the summer/fall (May to October) and winter/spring (November to April) seasons. These are set as the average of the two median values from the data sets for each season. A trend line monitoring limit is established, setting a threshold limit, beyond which, revisions to the abatement plan may be necessary. This applies only to acidity. The trend line limit is set as the average daily loading of all baseline data for the pre-existing discharges contributing to a given watershed outlet.

A bond release limit is also established to determine the maximum annual loading that the remining area can contribute to receiving streams and still retain bond release. This is calculated as the cumulative annual average loadings for acidity, iron, and manganese. The averages are based upon baseline monitoring data on pre-existing discharges affected by the proposed remining operation.

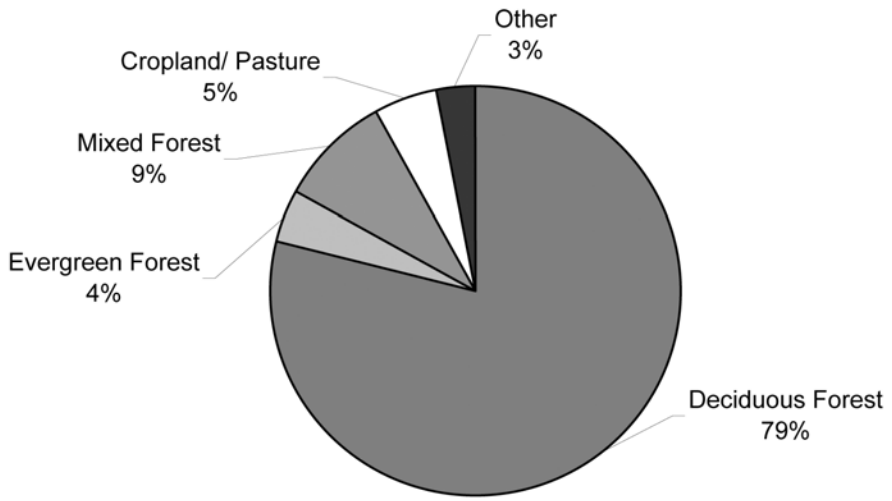
The final component of the West Virginia remining permitting process is establishment of in-stream water quality permitting conditions and in-stream water quality standards. The applicant provides minimum, average, and maximum values for pH, iron, and manganese for downstream baseline data on receiving streams. The applicant then provides in-stream water quality standards felt to be necessary to achieve bond release for the remining area, along with explanation of the methodology used to arrive at these standards. The desired standards are then used to apply for a water quality variance from the Environmental Quality Board.

Anecdotal information from the WVDEP indicates that few mine operators have opted for the remining designation to date. This is due to an earlier program that offered remining protection, but was later revoked, leaving some operators with unexpected liabilities.

F. APPALACHIAN FOREST COMMUNITIES

The study area contains many different terrestrial habitats resulting in a wide diversity of wildlife species including both game and nongame species. This diversity is due, in part, to the fact that the study area is geographically positioned between northern and southern vegetative communities, and that it has a complex and variable topography. The majority (92%) of the study area is forest land [Figure III.F-1 - Anderson Level Land Use/Cover in the Project Study Area].

Figure III.F-1 - Anderson Level Land Use/Cover in the Project Study Area



Characteristic vegetation types are found within each previously described ecological subregion section [Refer to Table III.A-1 - Ecological Subregion Section Characteristics]. The mixed mesophytic forest type is common throughout the project area. Mixed mesophytic forests are those found in habitats of intermediate moisture regime (between wet and dry). Likewise, oak dominated forest types are characteristic of each ecoregion and often co-occur with various pines. Pine dominated forest types are less common and are virtually absent from the study area. Other forest types common to these ecoregions, but not necessarily associated with the project study area, include the spruce-fir, northern evergreen, and floodplain communities (Straughsbaugh and Core, 1997; Martin et al., 1993).

Slope and aspect describe the angle and facing direction, respectively, of a mountainside. Slope and aspect have strong influences on soil moisture and thus, strong effects on vegetative communities. In the Appalachians, forest communities are distributed along both elevation and moisture gradients (Whittaker, 1956). Cove forests tend to dominate the steep-sided, mesic, (relatively moist) canyons while pine-heath communities dominate the more xeric (dry) ridges and peaks. Various oak forests dominate the flats and more open slopes that are intermediate between mesic and xeric conditions.

General forest types can be subdivided into more specific types. Ten different forest cover types are depicted in the West Virginia Gap Landcover for the West Virginia portion of the study area. Both the National Landcover forest cover types and the West Virginia Gap Landcover equivalents are presented in Table III.F.-1.

Table III.F-1.
Areas of Different Forest Cover Types
in the West Virginia Portion of the Study Area

National Landcover Dataset Forest Cover Type	Area (acres)	WV GAP Dataset Forest Cover Type	Area (acres)
Deciduous and Woody Wetlands	2,398,222	Diverse Mesophytic Hardwoods	1,852,790
		Cove Hardwoods	350,862
		Mountain Hardwoods	258,679
		Oak Dominant	193,833
		Floodplain	17,383
		Woodlands	5,716
		Total	**2,673,547**
Evergreen	52,910	Mountain Conifer	865
		Conifer Plantations	168
		Total	**1,033**
Mixed	252,520	Hardwood/Conifer	31,634
		Mountain Hardwood/Conifer	793
		Total	**32,427**
All Forest Cover Types	**2,703,652**	**All Forest Cover Types**	**2,712,723**
Total WV Study Area	**2,896,833**	**Total WV Study Area**	**3,007,623**

Note: The difference in total forest cover acres between the two data sets are a matter of scale.

The following text describes the forested communities of the study area. To avoid confusion with nomenclature related to author preference, we have listed the community types as presented by Martin et al. (1993) and placed in parentheses the forest community name used by the National Landcover Dataset and the West Virginia Gap Dataset.

1. Broadleaf Deciduous Forest Communities

a. Mixed Mesophytic Forests (Diverse Mesophytic Hardwood Forests)

Mixed mesophytic forests are found in moister habitats of north-facing slopes and in coves. The mixed mesophytic forest of the Appalachian coal fields supports one of the richest floral, breeding bird, mammal, and amphibian communities of any upland eastern U.S. forest type (Hinkle et al., 1989; cited in McComb et al., 1991); it has also been described as "the most biologically diverse ecosystem in the southeastern United States" (Hinkle et al., 1993). The diverse mesophytic forest is the dominant forest type in the study area, comprising slightly more than 68% of the forested portion of the study area in West Virginia.

Canopy species common to the mixed mesophytic forest type include American beech (*Fagus grandifolia*), yellow poplar (*Liriodendron tulipifera*), white basswood (*Tilia heterophylla*), various maples (*Acer* spp.), various oaks (*Quercus* spp.), as well as other species. The understory is usually diverse with more than 25 understory species known throughout the study area. Ferns and spring herbs are also abundant in the mixed mesophytic forest type. Among these are fragile fern (*Cystopteris fragilis*), jack-in-the-pulpit (*Arisaema triphyllum*), wild ginger (*Asarum canadens*e), and many others (Strausbaugh and Core, 1997).

Due to the abundance and variety of fruits, seeds, and nuts, mixed mesophytic forests provide excellent habitat for wildlife and game species alike. Also, an important forage source for migrant birds, especially in the spring, are invertebrates of the mesophytic forest (e.g., caterpillars, spiders, soil invertebrates). Species of birds typically present include the wood thrush (*Hylocichla mustelina*) and acadian flycatcher (*Empidonax virescens*). Additionally, many invertebrates are unique to the cove hardwoods habitat. For instance, the Diana fritillary butterfly, (*Speyeria diana*) is a denizen of the mixed mesophytic forests of southern West Virginia south to northern Georgia (Allen, 1997).

Under certain climatic and soil conditions, such as those found at the middle of north-facing slopes, eastern hemlock (*Tsuga canadensis*) or white pine (*Pinus strobus*) can become very prominent within the mixed mesophytic forest type. Although these trees provide cover for wildlife, their shade prevents the development of the understory vegetation that serves as food for game species. However, these tress do provide important habitat for various birds and small mammals. Blackburnian warblers (*Dendroica fusca*) and black-throated green warblers (*D. virens*) may inhabit areas that contain eastern hemlock and white pine.

Cove hardwoods, a subset of the mixed mesophytic forest type, are found in cool, moist valley bottoms and on lower slopes (Wilson et al., 1951, Hinkle et al., 1993). Because of their position on lower slopes, cove hardwoods form the upland forest border of the agricultural bottomlands that are scattered throughout the central section of the Appalachian Basin. The many layers of vegetation and the lush ground cover make the cove hardwoods an important habitat type for wildlife (USFWS, 1978). Cove hardwoods comprise approximately 13% of the forested lands in the West Virginia portion of the study area.

The dominant species in the cove hardwoods forest type are various maples, yellow poplar, and American beech; however, dominance is shared by a large number of species including, various oaks, hickories (*Carya* spp.), cherry (*Prunus* spp.), and black walnut (*Juglans nigra*), to name a few. This forest type is characterized by a diverse understory of trees that never attain canopy position such as, dogwoods (*Cornus* spp.), magnolias (*Magnolia* spp.), sourwood (*Oxydendrum arboreum*), striped maple (*Acer pennsylvanicus*), Paw-Paw (*Asimina triloba*) and redbud (*Cercis canadensis)*. Wildflowers are commonly found in this forest type because of the open canopy in the spring.

b. Appalachian Oak Woods (Oak Dominant Forests)

As the name implies, Appalachian oak woods are dominated by various species of oaks. The American chestnut (*Castanea dentata*) was co-dominant in this region until the chestnut blight of the early 1900's nearly extirpated this species. Most often Appalachian oak forests exist as mixed stands of assorted oaks and other species. Rarely is one species found in a pure stand; however, dominance of one species is often observed. For example, north-facing slopes at higher elevations are often dominated by red oaks (*Q. rubra*), likewise chestnut oak (*Q. prinus*) typically dominates at moderate elevations on dry slopes and ridgetops (Stephenson et al.,1993). Oak forests account for about 7% of the forested lands in the West Virginia portion of the study area.

At least 96 species of North American wildlife include the acorn in their diet (Stiling, 1996). Oak forests can support large populations of gray squirrels (*Sciurus carolinensis*) because of the availability of den trees and mast (nuts and fruits; Gill et al., 1975; WVDNR-Wildlife Resources, 1977). As many as 45 to 50 species of songbirds may breed in these forests because of the structural diversity of the vegetation (Samuel and Whitmore, 1979). Songbirds commonly present in this habitat include the red-eyed vireo (*Vireo olivaceus*), scarlet tanager (*Piranga olivacea*), red-bellied woodpecker (*Melanerpes carolinus*), downy woodpecker (*Picoides pubescens*), Carolina chickadee (*Parus carolinensis*), and many species of warblers (Allaire 1978; USFWS, 1978), all of whom forage on the invertebrates associated with this forest complex. Oak forests are considered to be prime habitat for wild turkey (*Meleagris gallopavo*) (WVDNR-Wildlife Resources, 1980a).

c. Northern Hardwoods (Mountain Hardwood Forests)

Northern hardwoods are restricted in the study area to cool, moist, north-facing upper slopes or ravines where cold air collects. They often intergrade with cove hardwoods on midslopes. The dominant species are American beech, sugar maple (*A. saccharum*), red maple (*A. rubrum*), and yellow birch (*Betula lutea*), with occasional stands of eastern hemlock or white pine (White et al., 1993). The canopy of this forest type is less open than that of the cove hardwoods type, and the lower layers are less developed. Witch hazel (*Hamamelis virginiana*), mountain laurel (*Kalmia latifolia*), rhododendron (*Rhododendron maximum*), spicebush (*Lindera benzoin*), hobblebush (*Vibernum alnifolium*), maple-leaf viburnum (*V. acerifolium*), deciduous holly (*Ilex* spp.), and elder (*Sambucus* spp.) are the typical shrubs in this community (Wilson et al., 1951). Approximately 9.5% of the forests in the West Virginia portion of the study area are the northern hardwood type.

Northern hardwoods are an important factor in the diversity of the fauna in the study area, because they support populations of plants and animals that are typical of the more northern forests. These include the golden-crowned kinglet (*Regulus setrapa*), Canada warbler (*Wilsonia canadensis*), red-breasted nuthatch (*Sitta canadensis*), northern water thrush [*Seiurus noveboracensis* (along shaded streams)], rock vole (*Microtus chrotorrhinus*), and long-tailed shrew (*Sorex dispar*) (Smith, 1974). American beech, sugar maple, and red maple may be used by wildlife as den trees (Wilson et al., 1951). Northern hardwood forests also contain the typical forest fauna assemblages of the region.

d. Floodplain Forests

Floodplain forests in the mountainous study area are generally restricted to narrow bands of vegetation along streams that have distinct woody and herbaceous components (Strausbaugh and Core, 1997). Characteristic woody species include, but are not limited to, black willow (*Salix nigra*), sycamore (*Platanus occidentalis*), ninebark (*Physocarpus opulifolius*), and box-elder (*A. negundo*). The herbaceous community often contains a mixture of climbing plants and erect herbs like greenbrier (*Smilax* spp.) and peppermint (*Mentha* spp.) to name a few. In the valleys, floodplains may be much broader but often times these areas have previously been converted to agricultural land use because of the fertility of the soils.

Floodplain forests have a great diversity of plant and animal species because of their association with water and because they serve as migration corridors. Some of the many species of wildlife that inhabit floodplain forests include waterfowl, songbirds, and a variety of reptiles and mammals. The moist soils associated with floodplain forests provide habitat for amphibians, particularly salamanders. Pools within the forest may provide habitat for amphibians, reptiles and invertebrates.

2. Other Forest Communities

a. Oak-Pine Forests (Hardwood/Conifer Forests and Mountain Hardwood/Conifer Forests)

Oak-Pine forests are located on south-facing slopes where the moisture level is between that of dry white oak woods and very dry pine woods. Characteristic of this forest type is a mix of oaks, pine, and often reduced numbers of hickories (Monk et al., 1990). Virginia pine (*P. virginiana*) and assorted oaks are the dominant canopy species (Bones, 1978); however, short-leaf pine (*P. echinata*) and loblolly pine (*P. taeda*) can reach abundant proportions in some areas (Skeen et al., 1993). Blueberry (*Vaccinium* spp.), huckleberry (*Galussacia* spp.), wild rose (*Rosa* spp.), hawthorn (*Crataegus* spp.), wild grape (*Vitis* spp.), and greenbrier (*Smilax* spp.) are the common woody shrubs. The herbaceous ground cover is sparse. The mixture of evergreen and deciduous trees makes this forest type particularly suitable for white-tailed deer (Gill et al., 1975), especially when this type of habitat is interspersed with pasture or silvicultural clear-cuts (Wilson et al., 1951, Skeen et al., 1993). This forest type is rare in the study area accounting for slightly more than 1% of the forested land in the West Virginia portion of the study area.

Because of their dependence on conifers for food and cover, the long-eared owl (*Asio otus*), pine warbler (*D. pinus*), black-burnian warbler, and red squirrel (*Tamiascurius hudsonicus*) inhabit the mixed oak-pine woods. Other birds commonly present include the great-crested flycatcher (*Myiarchus crinitus*) and the black-throated green warbler. Wild boar (*Sus scrofa*) forage for mast in this habitat during autumn and winter (WVDNR-Wildlife Resources 1977). Furthermore, this forest type harbors a diverse fauna of small mammals due to the abundance and variety of seeds, fruits, and nuts.

b. Pine Forests (Mountain Conifer Forests)

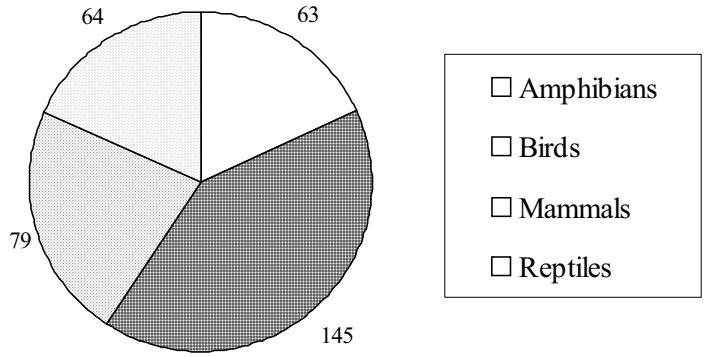
Virginia pine is the dominant species in old field habitats. Pitch pine (*P. rigida*) can become locally abundant on upper slopes with poor soils and is most often found mixed with hardwoods. Several other species of yellow pine, the short-leaf pine and loblolly pine, are of secondary importance, are essentially non-existent in the coalfields, and seldom reach dominance status outside of the Southwestern Appalachians Ecoregion. The pine forest type is often interspersed in the same part of the study area as the oak-pine forest type. The undergrowth vegetation is relatively sparse. Blueberry, mountain laurel, and dewberry (*Rubus permixtus*) are the most common species of shrubs (Wilson et al., 1951). Less than 1% of the West Virginia portion of the study area forest land is of pine forest.

The value of this habitat to most wildlife is low because of the limited availability and variety of food plants. Unless the dry pine community is interspersed with other types of habitat, it provides little more than cover (USFWS, 1978). These dry conifer stands essentially are inhabited sparsely by the same species of wildlife as those mentioned previously for the oak-pine forest type.

3. Animal Communities

Echternacht and Harris (1993) have compiled a detailed treatise on the fauna and wildlife of the southeastern United States, including the region of interest of this EIS [Figure III.F-2 - Number of Species of Terrestrial Vertebrates from the Appalachian Plateau Province]. Endemism, the localized geographic distribution of a species, is high in the region. Fourteen of the 351 vertebrate species, nine of which are amphibians, are endemic to the Appalachian Plateau Province. That is, as many as 14 vertebrate species may be found in the study area that are not found anywhere else in the world.

Figure III.F-2.
Number of Species of Terrestrial Vertebrates from the Appalachian Plateau Province



Adapted from Echternacht and Harris (1993)

The physiography of the study area allows for both northern and southern faunal components and the complex variation in local environment allows for habitat specialists. Mammal species representative of boreal (northern), temperate (warm in summer, cold in winter, moderate in spring or fall), and tropical climates are found in the Appalachian Plateau Province (Barbour and Davis, 1974).

a. Birds

Birds are amazingly diverse in the study area, due largely to the mosaic of microenvironments associated with the Appalachian Plateau Province. At least 38 families of birds can be found throughout the region. Species of birds with the greatest breeding distribution across the study area are those of forest or edge habitats and many are year round residents. Portions of the study area contain critical breeding habitat for some species of Neotropical migratory birds (Buckelew and Hall, 1994). Some of the highest concentrations of Neotropical migrant bird species like the scarlet tanager (*Piranga olivacea*), worm-eating warbler (*Helmitheros vermivorus*), Louisiana waterthrush (*Seiurus motacilla*), and wood thrush (*Hylocichla mustelina*) occur in West Virginia (Rosenberg and Wells, 2002). The mixed mesophytic forests are reported to support the richest avifauna in Kentucky (Mengel, 1965, cited in Hinkle et al., 1993) and one of the richest avifauna's in the eastern United States (Hinkle et al., 1993).

Mountaintop mining in the past has converted forest land to grasslands and in some instances shrub habitats in southern West Virginia. This change in available habitat has resulted in a shift in the distribution of birds throughout southern West Virginia with an increase in the abundance of edge and grassland bird species at reclaimed mountaintop mining sites (Wood and Edwards, 2001; Canterbury, 2001). This shift is likely apparent at mountaintop mining sites throughout the study area of this project but data supporting this claim are lacking. Many of the grassland and edge bird species now utilizing reclaimed mountaintop mining sites were once absent or rare in southern West Virginia because historically this habitat type did not occur in southern West Virginia (DeSalm and Murdock, 1993).

Eighty-four of 92 "probable" or "confirmed" breeding birds, based on data presented by Buckelew and Hall (1994) in the *West Virginia Breeding Bird Atlas*, were confirmed at mountaintop mining sites in southern West Virginia in 1999 and 2000 (Wood and Edwards, 2001)[see Appendix E for details]. The eight species not identified by Wood and Edwards (2001) are not associated with habitats associated with mountaintop mining sites (residential, urban habitats).

Species richness and abundance of songbirds is higher in shrub/pole habitats of mountaintop mining sites than in grassland, fragmented forest, and intact forest habitats (Wood and Edwards, 2001; Canterbury, 2001). The abundance of forest interior birds is significantly lower in fragmented forests near mountaintop mining sites than from intact forests near mountaintop mining sites suggesting that this bird guild is negatively influenced by mountaintop mining (Wood and Edwards, 2001). Species richness and abundance is lower on reclaimed grasslands than shrub/pole, fragmented forest, and intact forest habitats (Wood and Edwards, 2001). In general, species richness and abundance are expected to be greatest from diverse habitats, like the shrub/pole communities and lowest in the least diverse habitats, like grasslands. Studies conducted on reclaimed mountaintop mining sites in southern West Virginia support this assumption (Wood and Edwards, 2001).

Mountaintop mining sites are known to support at least ten grassland and shrub bird species not previously listed in the WV BBA (Wood and Edwards, 2001). Grassland birds are declining throughout much of the United States (Knopf, 1994). Three grassland bird species listed as "rare" in West Virginia (West Virginia Wildlife and Natural Heritage Program, 2000) are known to occupy mountaintop mining sites in southern West Virginia (Wood and Edwards, 2001). It is possible that some of the grassland bird populations on mountaintop mining sites reclaimed with herbaceous cover are existing as "sinks". "Sink" populations are maintained by immigration because death rates exceed birth rates (Pulliam, 1988). The core breeding ranges of the ten grassland birds identified on reclaimed mountaintop mining sites are in the grasslands of the Midwest. However, data suggest that the large reclaimed grassland habitats available on the mountaintop removal/valley fill mine complexes surveyed in southern West Virginia are sufficient to support breeding populations of grasshopper sparrows with nest success rates similar to populations found in other grassland habitats. Important nesting habitat characteristics included patches of dense grassland vegetation interspersed with patches of bare ground. These habitat conditions support high densities of breeding grasshopper sparrows, even on newly reclaimed sites. As ground cover develops, however, sites will become unsuitable for grasshopper sparrows unless habitats are managed to maintain the required conditions.

Some argue that mountaintop mining has the potential to negatively impact many forest songbirds, in particular neotropical migrants, through direct loss and fragmentation of mature forest habitats. Forest-interior species like the Acadian flycatcher, American redstart, hooded warbler, ovenbird, and scarlet tanager have significantly higher populations (at least one year of the two-year study) in intact forests than fragmented forests (Wood and Edwards, 2001). Furthermore, cerulean warblers, Acadian flycatchers, and wood thrush are more likely to be found in a forested area as distance from the mine increases (Wood and Edwards, 2001). These data suggest that forest-interior bird species are negatively impacted by mountaintop mining through direct loss of forest habitat and fragmentation of the terrestrial environment.

Of the 84 bird species identified on reclaimed mountaintop mining sites in southern West Virginia in 1999 and 2000, 13 species were raptors (Wood and Edwards, 2001). Of the six species typically associated with forested habitats, the red-shouldered hawk was the most common. Red-shouldered hawks were more abundant in intact forest than in fragmented forests. Of the seven species typically associated with more open habitats, the American kestrel, northern harrier, red-tailed hawk, and turkey vulture were commonly observed as expected. Rough-legged hawks and short-eared owls were observed in low numbers in the grassland habitats. They are more northern species that use large areas of open habitat and are rarely seen in West Virginia. A pair of adult peregrine falcons was observed throughout the summer on one mine in grasslands surrounding a highwall. The falcons often used the highwall for perching, but there was no evidence of breeding.

b. Mammals

There are 18 families of mammals in the project study area and mammalian diversity is greatly influenced by the presence of species from both northern and southern forest components. The variable landscape of the study area and drastic changes in elevation allow for a complex variation in the local environment over short distances. Many mammals take advantage of this complex environment and are found specializing within the project area (Wilson and Ruff, 1999). For example, the masked shrew (*Sorex cinereus*) is a common inhabitant of the coniferous and northern

deciduous forest biome, but the peak of its southern range extends into the project area where it thrives in moist, cool forests (Merritt, 1987) like the cove hardwoods.

Small mammal species richness does not differ between grassland, shrub/pole, fragmented forest, and intact forest habitats from mountaintop mine sites in southern West Virginia (Wood and Edwards, 2001) [see Appendix E for details]. Small mammal species abundance tends to be greater in grassland and shrub/pole habitats than in fragmented and intact forest habitats (Wood and Edwards, 2001). Rip-rap filled drainage ditches on reclaimed mine sites provide habitat for the Allegheny woodrat (*Neotoma floridana*) (Wood and Edwards, 2001), which is listed as threatened, endangered, or a species of special concern by the states of Indiana, Maryland, New Jersey, New York, North Carolina, Ohio, Pennsylvania, Virginia, and West Virginia. No studies are available that address the possible impact that mountaintop mining has on bats and larger mammals. There is, however, anecdotal evidence that mining has had a positive impact on white-tailed deer (*Odocoileus virginianus*) populations in the study area.

c. Herpetofauna

Five families of lizards and skinks, four families of turtles, and two families of snakes make-up the reptile assemblage of the study area. Four species of reptiles are endemic to the Appalachian Plateau Province of the study area (Echternacht and Harris, 1993). Endemism may be greater along the plateau because climatic conditions are more stable than the other ecoregions of the study area (Green and Pauley, 1987). Among the amphibians of the study area are five families of frogs and toads, and five families of salamanders. The southern Appalachians have one of the richest salamander faunas in the world (Petranka 1998, Stein et al 2000). Petranka (1993) presented a conservative estimate that there are about 10,000 salamanders per hectare of mature forest floor in Eastern forests.

Over a two-year study (2000 and 2001) of mountaintop mining sites in southern West Virginia, 1750 individuals were captured or observed using drift fence arrays, stream searches, and incidental sightings (Wood and Edwards, 2002). Of a possible 58 species expected to occur in the study area, 41 were encountered. The 41 species included 12 salamander species, 10 toad and frog species, 3 lizard species, 13 snake species, and 3 turtle species.

Amphibian and reptile species richness and abundance does not differ between grassland, shrub/pole, fragmented forest, and intact forest habitats from mountaintop mine sites in southern West Virginia (Wood and Edwards, 2001)[see Appendix E for details]. Salamanders appear to be less common in the grasslands of reclaimed mountaintop mining sites than in the nearby forests (Wood and Edwards, 2001). Herpetofaunal species, like salamanders, that require loose soil with ample ground cover, are generally absent from reclaimed mountaintop mining sites (Wood and Edwards, 2001). Salamanders are an important ecological component in Eastern forests (Burton and Lykens, 1975; Hairston, 1987) and salamander populations appear to recover slowly following forest clearing and disturbance (Bennett et al., 1980; Pough et al, 1987; Ash, 1988; Petranka et al., 1999). Mountaintop mining results in greater soil disturbance than forest clearing so a longer time may be required for recovery of salamander populations from mountaintop mined sites.

4. Interior Forest Habitat and Area Sensitive Species

A variety of wildlife species require large tracts (hundreds to thousands of acres) of continuous forest cover. Interior forest habitats are relatively rare and easily lost. Disturbance regimes, like agriculture, mining, and suburban sprawl, decrease interior forest habitat while increasing forest edge habitat, thus affecting the composition and distribution of wildlife within the region. For example, much of the avifauna (birds) of the study area depends on large areas of interior forest habitat for their survival (Robbins, 1980; Askins, 1993; Buckelew and Hall, 1994; Patton, 1994; Robbins et al., 1989). For example, the black-and-white warbler (*Mniotilta varia*) is an area-sensitive species usually not found in forest tracts less than 200 hectares (about 500 acres). Similarly, the worm-eating warbler (*Helmitheros vermivorus*) seems to require forest tracts of at least 150 hectares (370 acres). While other bird species, like the ovenbird (*Seirus aurocapillus*) and the Kentucky warbler (*Oporornis formosus*), are indirectly dependent on large tracts of interior forest because of their extreme susceptibility to brown-headed cowbird (*Molothrus ater*) parasitism in forest edge habitats.

Brown-headed cowbirds are found in very low abundance at reclaimed mountaintop mining sites in southern West Virginia; subsequently, nest parasitism is not likely a significant cause of nest loss in the study area (Wood and Edwards, 2001). Whether or not mountaintop mining has a negative effect on the breeding success of forest interior bird species through direct loss of interior forest habitat remains in question. Studies conducted at reclaimed mountaintop mining sites in southern West Virginia have yielded forest interior bird species in shrub/pole and fragmented forest habitats as well as intact forest habitats (Wood and Edwards, 2001; Canterbury, 2001). However, the abundance of forest interior bird species was significantly lower in fragmented forests than intact forest, suggesting a detrimental impact (Wood and Edwards, 2001). Canterbury (2001) suggests that studies of nesting success are needed to determine if mountaintop mining is having a negative impact on forest interior bird populations. Intuitively, it makes sense that the loss of interior forest habitat would be detrimental to wildlife populations dependent upon such habitat.

Not all large forest tracts contain interior forest habitats [Figure III.F-3 - Relationship Between Patch Shape/Size and Interior Forest Habitat]. A long, narrow forest patch may be comprised entirely of edge species. Thus, when considering the needs of area sensitive species the shape of the forest tract must be considered.

Figure III.F-3.
Relationship Between Patch Shape/Size and Interior Forest Habitat

Assume both patches are of equal area. The patch on the left contains interior forest habitat (solid shade) while the patch on the right is entirely edge habitat (dot shade).

5. Deforestation

DEFORESTATION AFFECTS WILDLIFE BY DIRECTLY REMOVING AVAILABLE HABITAT FOR SOME SPECIES WHILE OPENING THE FOREST AND PRODUCING HABITAT FOR OTHER SPECIES. FURTHERMORE, INDIRECT AFFECTS OF DEFORESTATION MAY INCLUDE INCREASED SOIL EROSION, LEADING TO SILTATION OF AQUATIC HABITATS, EUTROPHICATION OF AQUATIC HABITATS BY ACCELERATED NUTRIENT RELEASE, THE CONVERSION OF FOREST HABITATS TO RANGELANDS OR SUCCESSIONAL FIELDS, AND A CHANGE IN THE REGION'S CONTRIBUTION TO GLOBAL PRIMARY PRODUCTION (STILING, 1996).

Energy accumulated by plants is referred to as primary production. The energy remaining after plant respiration and stored as organic matter is net primary production. Globally, temperate forests produce approximately 13% of the worlds net primary production per year (Whittaker, 1975). Temperate forests also provide habitat for a large proportion of the study area's wildlife. The Land Use Assessment study concludes that approximately 5% of the West Virginia mountaintop mining study area contained evidence of having been disturbed by past or current mining [Appendix G]. Deforestation results in both habitat loss and fragmentation of the terrestrial environment.

Habitat loss is generally understood to be the single most important cause of wildlife population declines and a threat to present-day wildlife populations (Illinois Wildlife Habitat Commission, 1985). It follows that the deforestation of large portions of the Appalachians through mountaintop mining is a significant concern from the standpoint of forest-dwelling wildlife, in particular, forest interior species. On the other hand, the loss of forested habitats is equaled by a gain in other habitat types, like grasslands.

There is disagreement about what these changes in the terrestrial environment mean. Many point out that reclamation efforts have created habitat, like grasslands, edge habitat, and scattered ponds, that are important for game species such as wild turkey, bobwhite quail (*Colinus virginianus*), ruffed grouse (*Bonasa umbellus*), and white-tailed deer. Many grassland and shrub bird species, previously unrecorded as having breeding populations in southern West Virginia, are known to breed on reclaimed MTM/VF sites (Wood and Edwards, 2001). Among these grassland songbirds is the grasshopper sparrow (*Ammodramus savannarum*), which is listed as "rare" by the West Virginia Wildlife and Natural Heritage Program (2000) but is found to be abundant and breeding successfully on Mountaintop mining sites (Wood and Edwards, 2001). Two other "rare" species in West Virginia (West Virginia Wildlife and Natural Heritage Program 2000), the bobolink (*Dolichonyx oryzivorus*) and Henslow's sparrow (*A. henslowii*), were present at some mountaintop mining sites but not confirmed as breeding (Wood and Edwards, 2001). Furthermore, with the exception of a few rare species, the densities of songbirds on grassland and shrub/pole mountaintop mining sites was similar to that reported in other studies indicating that the quality of habitat and availability of resources is similar to other sites (Wood and Edwards, 2001). It should be noted that the presence of rare, threatened, and endangered species in these reclaimed habitats is more likely a result of the habitat being rare in the study region than the species being rare. That is, many of the rare species encountered at mountaintop mining sites are common or abundant in other parts of the United States where their required habitat is more abundant.

The above findings provide evidence that mountaintop mining practices provide favorable conditions for some species. However, these advantages may not surpass the disadvantages these practices have on the sustainability of plants and wildlife in the region.

Historically, vegetative communities of the Appalachians have undergone much change beginning with the replacement of pine and spruce forests by oaks, due to climatic warming about 10,000 years ago (Abrams, 1992). Humans began to alter the Appalachian vegetative communities about 1,000 to 3,000 years ago, increasing the extent of oak-chestnut forests, due to use of fire (Delcourt and Delcourt, 1998). More recently in the 1800's, logging, increased fire, clearing of forests for settlement, and the loss of the American chestnut (*Castanea dentata*) to chestnut blight fungus have led to massive changes in the vegetative communities of the Appalachians (Nowacki and Abrams, 1991). Possibly, the greatest impact to Appalachian vegetative communities was exerted by the logging industry. Clearing of forests leads to soil erosion, drying of understory, increased fires, and the depletion of soil nutrients. Logging has decreased dramatically in the study region since the 1940's, and coupling this with the abandonment of old farms has led to an increase in forest area for the region over the past 50 years (Barrett, 1995). Approximately 244,000 acres in the West Virginia portion of the study area have been disturbed by past or current mining (Yuill, 2001).

Mountaintop mining operations in the Appalachian coal fields involve fundamental changes to the region's landscape and terrestrial wildlife habitats. Prior to 1998 (the start of this EIS) with the increasing size of these operations, a single permit involved changing thousands of acres of hardwood forests into herbaceous cover. This is true even for the short-term when forest is post-mining land use. While the original forested habitat was crossed by flowing streams and was comprised of steep slopes with microhabitats determined by slope, aspect, and moisture regimes, the reclaimed mines are often limited in topographic relief, devoid of flowing water, and most commonly dominated by erosion-controlling, herbaceous communities. Islands of remnant hardwood vegetation may be present on some of the reclaimed mines, and some planting of trees and shrubs may have been undertaken.

Handel (2001) studied 55 mountaintop mining sites in southern West Virginia that were reclaimed with herbaceous vegetation and ranged in age from 6 to 24 years. Handel (2001) determined that trees and shrubs are extremely low in abundance and number on mine sites compared to surrounding forests. Reclaimed sites where trees and shrubs were invading tended to be dominated by two or three species whereas the surrounding forests were very species rich. The invasion rate of native trees and shrubs onto mined sites is most likely restricted by excessive soil compaction, large mining area, poor soil quality, and the application of grassy mixes for erosion control. Furthermore, Handel (2001) found that there were 17 fewer forest herb species on plots adjacent to mountaintop mining sites than in interior forests. This effect extended from the edge of the reclamation area 50 m into the forest.

a. Forest Fragmentation

The phrase “forest fragmentation” describes a formerly continuous forest that has been broken into smaller pieces (Jones, 1997). The disruption of continuous forest habitats into isolated and small patches may have two negative affects on biota dependent upon forest habitat: decreased area and increased isolation of the remaining patches (Meffe and Carroll, 1994). However, disruption also provides habitat for those species that thrive within the ecotone of forest and open habitat.

Fragmentation leads to a decrease in the abundance of many species of songbirds in the study area (Wood and Edwards, 2001). Wood and Edwards (2001) list ten species of forest-dwelling songbirds that are negatively impacted by forest fragmentation. Since native trees and shrubs have a slow invasion rate on mined sites (Handel, 2001) we can assume that the invasion rate of area sensitive forest-dwelling songbirds will be even slower. Similarly, we can assume that the invasion of rate of forest-floor dwelling salamanders will be slow on post-mined sites. Wood and Edwards (2001) found that taxa dominance shifted from salamanders to snakes when intact forests were converted to grasslands through reclamation of mountaintop mining sites. Populations of many eastern forest amphibian species are largely dependent upon coarse woody debris, litter moisture and depth, density of understory stems, and canopy cover (deMaynadier and Hunter, 1995). These are traits absent on most post-mining sites and traits that appear to be slow to return to reclaimed mountaintop mining sites.

b. Forest Edge Habitat, Edge Effect

Edge habitat occurs at boundaries between different types of land cover. Certain species require resources in two or more vegetation types and thus require edge habitat. The outer boundary of a habitat patch is a zone that varies in width depending on the variable being measured. For example, edge zones are usually drier and receive more sunlight than interior forests, and thus have a different floral composition, which favors shade-intolerant species. Climatic edge effects, such as this, may have a negative effect on interior species of the patch through altering of the physical environment and increasing competition for resources. On the other hand, due to the different microclimate associated with the edge ecotone; these habitats are often more diverse than the interior habitat and contain unique wildlife assemblages (Yahner, 1988).

Edge effect is used to describe the negative influence, like the microclimatic differences described above, that edges have on the interior of a habitat and on the species that use the interior habitat. However, edge effect can be used to describe the increase in edge species richness often observed at the ecotone of forest edges.

Many species of wildlife are attracted to "edges," or areas where two or more different habitat types come together. This fact has been the basis for traditional wildlife management schemes (including those recommended by State resource agencies for mine reclamation), which seek to promote edges to maximize "biodiversity." However, as explained by Heckert et al. (1993), promoting edges at the expense of large habitat blocks can lead to *lower* wildlife diversity:

> Wildlife diversity can be viewed on two different levels. On one level, diversity can be viewed as the number of species that occur on a single tract of land, such as private landholdings, single fields, or woodlots. On the other level, diversity can be viewed as the number of species that occur within a larger geographic area such as large conservation areas, counties, and watersheds.
>
> Land management focused entirely on providing abundant edge has come under recent criticism because it can exclude species that require large uniform habitat blocks or do not survive near edges. If most parcels are managed to increase edge, only those species tolerant of edges will prosper. Species needing uniform habitat blocks away from edges can be eliminated. The result of such management will be

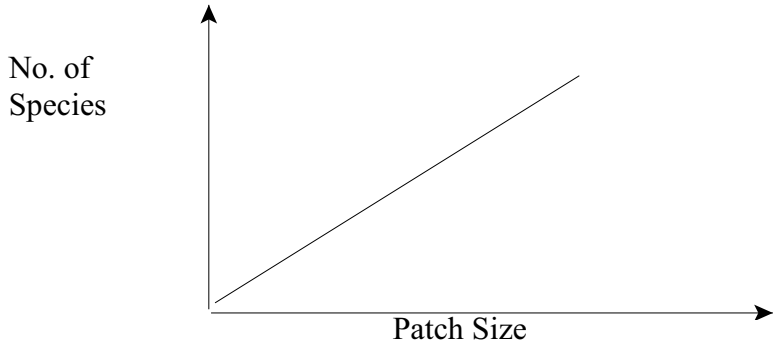
> lower wildlife diversity within large geographic regions because area-sensitive species will be lost. Conversely, the maintenance of large habitat blocks for area-sensitive species will not result in the loss of edge species as some edges will always be present in the landscape... If land use patterns and management continue to favor edge species, continued population declines and possibly local or regional extinctions of area-sensitive species are likely to occur.

Edge habitat on reclaimed mountaintop mining sites in southern West Virginia is utilized by bird species of different guilds depending upon the habitats creating the edge. For example, edge composed of grassland and fragmented forest tend to be dominated by birds of the grasslands bird guild while edge composed of forest and shrublands tend to be dominated by birds of the forest-interior and edge guilds (Canterbury, 2001).

c. Patch Size

Patch size refers to the area of a particular habitat or reserve within a landscape. The basic species-area relationship implies that larger patches capture a greater number of species of a region than do smaller patches [Figure III.F-4 - Relationship Between Species Richness and Patch or Island Area]. This is due, in part, to an increase in habitat heterogeneity as the patch size gets larger. Larger patches are also more likely to be able to accommodate disturbances than smaller patches. As patch size decreases, forest edge-to-volume ratios increase, thereby increasing edge effects and reducing the amount of true interior habitat.

Figure III.F-4.
Relationship Between Species Richness and Patch or Island Area



Adapted from MacArthur and Wilson (1963)
Adapted from MacArthur and Wilson (1967)

Another aspect of patch size is isolation. Small, isolated patches are more prone to local species extinctions than large patches and small groups of closely spaced patches, because they are less likely to be colonized. Therefore, when circumstances require or result in the creation of small patches, it is important to space them close together or to provide some form of connectivity between the patches.

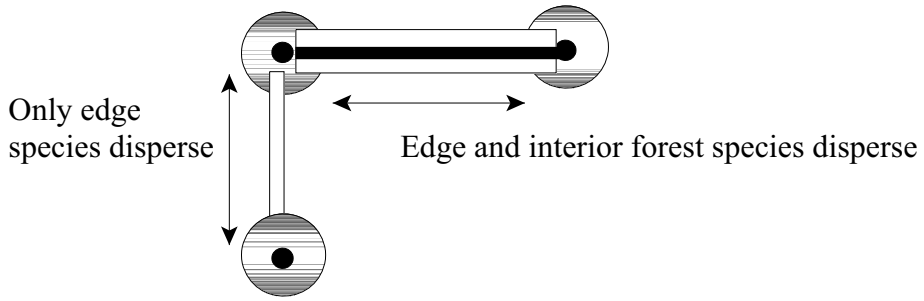
Many species require large patch sizes for their survival. For example, the cerulean warbler is a common bird of mixed mesophytic and Appalachian oak forests in West Virginia. This migratory species commonly occupies the heavily-leafed canopy of mature forests during summer months and is rarely seen. Studies suggest that a minimum area of 700 hectares (1,730 acres) is required for sustaining a viable population of this species (Buckelew and Hall, 1994). Reduction of forest patch size and the fragmation of habitats may greatly affect the distribution and abundance of the cerulean warbler. Conversely, smaller patch sizes are favored by many species, including many game species that depend upon food sources, nesting sites, and ground cover associated with small forest patches.

d. Corridors

As habitats become fragmented into small patches, there is a change in distribution and abundance of species, due to such factors discussed above as isolation and decreased interior habitat. An intuitive solution to this problem is the reconnection of these fragmented habitats through habitat corridors. Corridors allow for species movements, and thus, recolonization among isolated habitats.

Simple corridors, called line corridors, consist entirely of edge habitat and allow for the movements of edge species [Figure III.F-5 - Corridors Connect Patches in a Fragmented Environment]. In contrast, strip corridors contain some interior habitat that is required for the movements of many large animals, in particular, predatory mammals and forest interior species. Patches that are farther apart may require broader corridors in order to be effective (Harrison, 1992). Whereas, line corridors may suffice for closely spaced patches.

Figure III.F-5.
Corridors Connect Patches in a Fragmented Environment



Corridor width can act as a filter by selecting for the dispersal of some species while limiting the dispersal of other species.

Despite the obvious advantages of corridors, disadvantages do exist. For some species, like small mammals, predation may increase in corridors because of the reduction in interior habitat and cover. Furthermore, species may be pulled into a sink corridor where rates of survival and extinction differ from their source habitat (Soule, 1991). Another disadvantage of corridors is that they may provide access for unwanted species, such as, invasive exotics to invade once unoccupied areas.

Public concerns voiced before the conception of this EIS included fears that mountaintop mining may contribute to the spread of exotic and invasive species. One concern was that roads and fragmentation of the environment associated with mountaintop mining may act as line corridors aiding in the spread of exotic and invasive species. There is no evidence that mountaintop mining has contributed to the spread of invasive and exotic species in southern West Virginia (Handel, 2001).

6. Carbon Sequestration

The energy flow in terrestrial ecosystems depends on interactions between a number of biogeochemical cycles such as the carbon cycle and hydrological cycles. Terrestrial ecosystems play a role in the global carbon cycle. Carbon is exchanged between trees and the atmosphere through photosynthesis and respiration. The cycling of carbon as carbon dioxide involves assimilation and respiration by plants.

According to the World Resource Institute (1997), drawing carbon dioxide out of the atmosphere (sequestration) and into biomass is the only known practical way to remove large volumes of this greenhouse gas from the atmosphere. Reforestation could potentially achieve significant carbon sequestration. It has been estimated that temperate forests sequester 0.6 to 1.8 tons of carbon per acre per year as reported by the Intergovernmental Panel on Climate Change (2001).

G. RELATIONSHIPS OF MOUNTAINTOP MINING TO SURFACE RUNOFF QUANTITY AND FLOODING

The central Appalachian physiographic region is a highly dissected plateau characterized by high, tree-covered hills and deep, narrow valleys. Large watersheds often feed streams with narrow valleys and small flood plains. In such rugged terrain, people live near or adjacent to the streams and rivers, and they may consequently be flooded during large rainfall events.

MTM/VF mining causes alterations in the topography and drainage patterns in the mined areas. There are also changes in vegetation and ground cover that are associated with this type of mining. The combination of these alterations can impact the amount of runoff from the mined area for a given storm event. That impact and possible cumulative effects from similar or multiple projects has been raised as a concern for analysis in these watersheds.

As part of the background assessment of the effects of mountaintop mining on the environment, a number of studies were undertaken to evaluate whether MTM/VF operations resulted in an increased risk of flooding to downstream communities. The following summarizes the findings of these studies, along with an introductory background on the existing regulatory framework with respect to control of surface mine runoff and flooding risks.

1. Regulatory Background

Surface water impacts from surface mining were recognized during the development and implementation of SMCRA. These potential impacts were discussed in the Final Environmental Statement OSM-EIS-1 for SMCRA. The discussion noted that surface mining can have significant effects on surface hydrology. Removal of vegetation, new drainage patterns, storage of water on benches or in ponds, drainage of surface water into underground mines and alternate ground cover change the runoff characteristics. These changes in runoff may cause scouring and erosion of unprotected stream channels and can contribute to downstream flooding. Small tributaries with a high percentage of recently disturbed land may have somewhat higher flood levels as a result of the surface mining. Increased flooding might be attributed to inadequate reclamation or inadequate drainage control structures. However, there are also reports that document surface mining effects with a lower flood rate than a similar unmined watershed (Davis, 1967; Collier and others, 1970; Curtis, 1972, Curtis, 1977). Open pits at mines sites can provide significant runoff retention. Drainage control structures can also provide retention, plus longer travel times for overland flow. The increased infiltration provided by backfills can also retard or lessen peak flows.

Surface mining may cause isolated flooding events related to failure of erosion and sedimentation control structures. In a recent incident, a mine sediment ditch in Mingo County, West Virginia, ruptured during a rainfall event and damaged downhill properties, including fences, a bridge, and a vehicle (Associated Press, 2000). Storm water control structures on surface mine sites are designed to accommodate a given storm frequency event, a statistical abstraction of the largest storm event that can be expected to occur within a given time period. In reality, there is no reason that a larger storm could not occur within that time period, only that it is less likely, so a probability always exists that storm water control facilities on mining sites or in any other application can be

overwhelmed and fail. Mechanical failure due to improper construction is also a possible source of isolated flooding incidents.

SMCRA and USOSM regulations require that flooding potential be addressed in the design requirements of coal mine permits and the consideration of offsite impacts to the hydrologic balance. Water diversions are required to be designed and constructed to provide protection against flooding and resultant damage to life and property (30 CFR 816.43(a)(2(ii)). USOSM regulations also require the operator to make a "Probable Hydrologic Consequences Determination"(PHC) as part of the permit application (30 CFR 780.21(f)). The PHC is required to specifically address flooding and stream flow alterations as part of this determination. USOSM regulations further require the regulatory authority to provide a "Cumulative Hydrologic Impact Assessment"(CHIA) as part of the permit approval process (30 CFR 780.21(g)). This hydrologic assessment must include the impacts of the proposed operation and all anticipated mining on surface and ground water systems in the cumulative impact area. Currently, not all of the state regulatory agencies require a quantitative analysis of flooding impacts for proposed mine operations in either the PHC or CHIA assessments.

The USCOE routinely relies on state or SMCRA regulations to address flooding. The USCOE may evaluate flooding impacts from an individual mine. The USCOE districts routinely consider flooding impacts when they evaluate mining activities under the Individual Permit process. The need to do a separate flood impact analysis is determined on a case by case basis by the USCOE. Most districts will not conduct a separate flood analysis if such an analysis is required by state or SMCRA regulations.

2. EIS Peak Flow Studies

Previous studies of peak flow evaluated sites that were not specifically impacted by MTM/VF mining and were done prior to the implementation of SMCRA. To fill in this information and analysis gap several studies were done in preparation of this EIS. The EIS studies evaluate the impacts of MTM/VF mining on peak flow using computer modeling, continuous data collection using stream gages, post-flood highwater marks, on-site drainage control structure evaluation, and citizen complaint investigations. Each study analyzes discrete circumstances that help to create a more complete evaluation when coupled with the other EIS studies. The output from these efforts is summarized and discussed below. The complete studies are presented in Appendix H.

USOSM and the Army COE (Pittsburgh District) performed computer model analysis of peak flows at locations immediately downstream of several drainages where valley fills were planned in West Virginia. Specific design precipitation events were modeled for these drainages using a variety of scenarios. This study provided the predicted peak flows for several mining and reclamation plans. This is referred to as the "Peak Flow" Study.

The USGS - Water Resources Division (Charleston, West Virginia) installed and maintained three continuous recording stream gages and four rain gages in a small watershed in West Virginia. The stream gages were located to documented the stream-flows for a mined area with a valley fill, an adjacent unmined area, and the cumulative discharge downstream of these areas. This study provided the actual peak flows for the various rainfall events that occurred during the period of data collection. This is referred to as the "Fill Hydrology" study.

The USGS - Water Resources Division (Charleston, West Virginia) evaluated the peak flows for the July 8-9, 2001 flooding in southern West Virginia. Six small drainage basins were selected and the highwater marks were documented immediately after the floods. The highwater marks and stream channels were surveyed and peak flows were calculated from this data using "indirect discharge measurement techniques." This study provided the calculated peak flows for an individual extreme event that caused flooding and damage in and around the study area. This is referred to as the "July 2001 Floods" study.

USOSM and the KYDSMRE did a special study on drainage control at mine sites in Kentucky. Site selection was based on citizen complaints alleging that life-threatening "washouts" were caused by mining or otherwise significantly contributing to downstream flooding and/or flood-related adverse impacts to citizens, property or the environment. This is referred to as the "OSM/Kentucky Oversight" study.

USOSM did an evaluation of citizen complaint records for West Virginia, Kentucky, and Virginia where there were allegations of flooding from coal mine operations. Thousands of citizen complaints received and investigated by these states and those related to flooding were reviewed. This is referred to as the "Citizen Complaint" study.

a. Peak Flow Study

In November 1997, an interagency coordinating meeting of the Federal Regulatory Organization Group (FROG) was held in Berkeley Springs, WV. One of the topics for discussion was a more "pro-active" approach in response to valley fill permit applications with respect to Section 402 and 404 (CWA) permit applications, as well as related USOSM and state permitting and administrative procedures. The EPA, OSM, COE, and FWS formed a four-agency task force to evaluate valley fill issues. Flooding was one of the issues chosen for technical investigation by the four agency group.

OSM and the COE performed a model analysis of potential downstream flooding as a result of valley fills and large scale surface coal mining operations in Appalachia. The purpose of the Peak Flow Study was to evaluate the potential for flooding as a result of the construction of valley fills and the related hydrologic modifications to terrain associated with MTM/VF mining. The following summarizes the computer modeling studies that have been undertaken as part of the Peak Flow Study and the conclusions that have been reached.

Computer modeling simulations were performed to evaluate the impacts of rainfall events on three individual valley fills, as well as the cumulative impacts of two of these fills on a downstream area. The study used computer models to predict storm hydrograph peak discharges for two precipitation events (10-year and 100-year) during various scenarios of pre-mining conditions, conditions during mining, initial post-mining conditions with no change to the permitted regrading plan, future post-mining conditions with forest cover assumed for the permitted regrading plan instead, and initial post-mining conditions for a conceptual Approximate Original Contour Plus fill optimization process (AOC+ - also referred to as the WVDEP AOC Process) regrading plan. The USCOE-developed Hydrologic Engineering Center (HEC) computer model was used by the USCOE (Pittsburgh District), and the proprietary SEDCAD 4 model was used by USOSM, to evaluate three valley fill watersheds in southern West Virginia. Both models used the identical topographic and

land use conditions, which provided a useful comparison of the surface water modeling software. Both software models are readily available to private consultants. SEDCAD 4 is frequently used by the coal industry to design diversions and sediment control structures, while the HEC model is used for a wide variety of watershed hydrology studies.

The point of evaluation of the peak flows for the HEC-HMS and SEDCAD 4 modeling was the permit boundary downstream of each valley fill. The sites selected were all Arch Coal Company sites: the Samples Mine Valley Fill #1, Samples Mine Valley Fill #2, and Hobet Mine Westridge Valley Fill. The Samples Valley Fill #1 drainage area was 440 acres, with 72 percent of the area disturbed by mining operations or valley fill. The Samples Valley Fill #2 drainage area was 351 acres, with 56 percent of the area disturbed. The Hobet Westridge Valley Fill drainage area was 1600 acres, with 74 percent of the area disturbed.

As summarized by Table III.G-1, the storm runoff modeling using HEC-HMS and SEDCAD 4 both calculated that the post-mining peak flows would be higher than the pre-mining peak flows for the same design storms. However, the predicted increases in peak flow would not have caused flooding on the banks outside the receiving stream channel.

The USCOE (HEC-HMS) analysis predicted peak flow increases of about 3 percent for Samples Valley Fill #2, 13 percent for Samples Valley Fill #1, and 42 percent for Hobet's Westridge Valley Fill between pre-mining and permitted post-mining conditions. These results indicate the largest drainage area (Hobet Westridge Valley Fill) with the highest percentage area disturbed had the greatest increase in peak flow from pre-mining conditions. The results also indicate that the smallest drainage area (Samples Valley Fill #2) with the smallest percentage area disturbed had the lowest increase in peak flow.

The USCOE study also completed a cumulative analysis of the Samples Valley Fills #1 and #2. The fill drainage areas are adjacent to each other and form the headwaters of the same stream. The cumulative analysis indicates an increase in the peak flow downstream of the valley fills at a point below where the two drainages converge. However, the peak flow increase (8 percent) between pre-mining and permitted post-mining conditions represents influences of the individual valley fill drainage areas and any additional drainage area that flows to the cumulative analysis point. The influence of changes in the headwater areas will decrease as the point of analysis is moved further downstream. That is, the peak flow alteration would attenuate downstream from the mine site.

Table III.G-1
Comparison of HEC-HMS and SEDCAD 4 Models Peak Flow Results

Peak Flow Predictions in Cubic Feet Per Second (CFS)										
Storm Event	**Pre-Mining**		**During Mining**		**Permitted Post-Mining**		**AOC+ Post-Mining**		**Permitted Post-Mining Forested**	
Samples Mine Valley Fill #1										
	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4
10 Year	330	352	525	498	376	447	432	416	246	269
100 Year	742	739	931	844	832	864	932	790	580	583
Samples Mine Valley Fill #2										
	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4
10 Year	293	326	---	---	302	354	295	391	209	259
100 Year	664	768	---	---	671	688	658	744	502	563
Samples Mine Valley Fills #1 and #2 Combined										
	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4
10 Year	765	803	---	---	826	880	833	---	605	---
100 Year	1711	1693	---	---	1793	1722	1874	---	1331	---
Hobet Mine Westridge Valley Fill										
	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4	HEC-HMS	SEDCAD 4
10 Year	838	782	---	---	1193	855	---	---	---	---
100 Year	1736	1447	---	---	2459	1525	---	---	---	—

The USCOE documented channel capacity with measured survey sections for streams below each site. These measurements allow an evaluation of the effect of modeled peak flows on water levels of the receiving stream downstream from each fill. Changes in water level

are related to the flow volume and the cross-sectional area of the stream channel. The water level increases in the receiving stream were negligible for the Samples Valley Fill #2; 0.3 feet for the Samples Valley Fill #1; and 2.1 feet for the Hobet Westridge Valley Fill between pre-mining and permitted post-mining conditions. Routing design storm peak flows through these measured channel sections did not cause flooding because resultant water levels were below bank-full conditions within the receiving stream.

The same topographic and hydrologic conditions were used by USOSM to predict peak flows using the SEDCAD 4 hydrology model. Similar to HEC-HMS, the SEDCAD 4 model predicts the post-mining peak flows to be higher than the pre-mining peak flows. While the SEDCAD 4 percentage increases would not be expected to be identical to those predicted by the HEC-HMS model, the general finding that permitted post-mining peak flows will be higher was confirmed by SEDCAD 4 as well.

The one analysis of peak flows during mining for the Samples Mine Valley Fill #1 showed a 59 percent and 25 percent increase over pre-mining conditions for the 10-year and 100-year storm events, respectively. Water level increases were 1.7 feet and 1.3 feet, respectively, compared to pre-mining conditions. Again, this did not result in any predicted overbank flooding.

Predicted runoff for conceptual AOC+ conditions was 12 percent higher for the Samples Mine Valley Fill #1 than permitted post-mining configuration, whereas the peak flow was 2 percent lower for Valley Fill #2. For the combined valley fills, peak flows were 1 percent and 5 percent higher for the 10-year and 100-year storm events, respectively, for AOC+ conditions versus permitted post-mining conditions. In comparison, peak flow increases for AOC+ ranged from 1 percent less than pre-mining conditions to 31 percent more, whereas the permitted post-mining peak flows ranged from 1 percent to 13 percent more than pre-mining conditions. Water level increases ranged from negligible on the Samples Mine Valley Fill #2 to 1 foot on the Samples Mine Valley Fill #1, with no overbank flooding predicted.

The final analysis was made of future conditions if the Samples Mine sites were forested with the permitted post-mining configuration. This showed substantially lower peak flows than either the initial post-mining conditions or the pre-mining conditions. Predicted forested peak flows ranged from 22 percent to 29 percent lower than pre-mining conditions, and 25 percent to 35 percent lower than initial permitted post-mining conditions. Water levels at the receiving stream analysis points decreased from 0.4 feet to 1 foot compared to pre-mining conditions among the sites evaluated.

The storm runoff modeling using HEC-HMS and SEDCAD both calculated that the permitted post-mining and AOC+ post-mining peak flows would be higher than the pre-mining peak flows for the same design storms. However, increases in peak flow did not cause a rise in water level overtopping the receiving stream channels. Flooding typically occurs only when water levels exceed channel capacities and spread across the flood plain where residential settlements may occur. The cumulative analysis of two fills indicated an increase in the peak flow post-mining beyond the downstream confluence of the valley fill watersheds. Again, bank full capacity of the stream channel did not result. The influence of changes in the headwater areas will decrease as the point of analysis is moved further downstream.

b. Fill Hydrology Study

The USGS collected data in close proximity to several mountaintop mines to document the changes in flood peaks associated with these sites. Rainfall and runoff are being measured at four rain gages and three stream gages. The stations are in the Ballard Fork watershed near Mud, West Virginia. Data collection began in November 1999 and is continuing. The stream gages were located to document the stream flows for a mined area with a valley fill (0.19 sq. mi.), an adjacent unmined area (0.53 sq. mi.), and the cumulative discharge downstream of these areas (2.12 sq. mi.). The stream gages provide continuous records of water surface elevations for each station. These water surface elevations are converted to stream flow based on actual flow measurements taken at various water surface elevations. Peak flows and the hydrographs for each precipitation event can then be evaluated.

The precipitation gages provide a continuous record of rainfall that can be evaluated for total amount of rainfall and the rainfall intensity. These records also document and allow for the evaluation of time since the previous rainfalls to estimate the soil moisture conditions. Most of the intense rainfall in the study area occurred during summer thunderstorms.

The storm hydrographs for the mined watershed were distinctly different from the hydrographs for the unmined watershed and the cumulative watershed. The unmined and cumulative watersheds generally rose in response the rainfall events and was independent of rainfall intensity. In contrast, the storm hydrograph for the mined watershed had a double peak flow when rainfall intensity exceeded about 0.25 in/hour. The hydrograph would rise quickly to the first peak flow and recede quickly after the heavy rainfall stopped. There would then be a second peak flow that was not as high as the first but would occur hours after the first peak.

During most of the recorded storms (low intensity) the peak flows (per unit area) for the unmined watershed and the cumulative watershed were less than the mined watershed. However, during intense rainfall events the peak flows (per unit area) for the mined watershed were greater than those for the unmined and cumulative watersheds.

c. July 2001 Floods Study

The USGS investigated the effects of valley fills on the peak flows for the flood of July 8-9, 2001 in West Virginia. Six small basins (drainage areas ranging from 0.189 to 1.17 sq. mi.) within an area of about 7 sq. mi. in the headwaters of Clear Fork of the Coal River in southern West Virginia were investigated following the July floods. Three of the basins were downstream from the ponds at the toe of valley fills and three basins were not below valley fills.

The thunderstorm that produced the July 8-9, 2001 floods produced rainfall amounts between 3 and 6 inches in a 5 to 6 hour period. These rainfall amounts for this storm alone were approximately equal to the average monthly rainfall.

Within the six small drainage basins the highwater marks were documented immediately after the floods. The highwater marks and stream channels were surveyed and peak flows were calculated from this data using “indirect discharge measurement techniques.” From this information and the

roughness coefficients (ground-surface conditions) the peak flow can be calculated. These flows were divided by the drainage area for the basin to produce a unit peak flow.

The six basins were separated into a northern group and a southern group. They were grouped by geographic location and the relative difference in the unit peak flows for the unmined watersheds. There are four basins in the southern group where two had valley fills and two did not. The remaining two basins were in the northern group with one valley fill basin and one without.

The calculated unit-peak flows for the unreclaimed valley fill in the southern group was twice as high as the remaining sites. The remaining basins in the southern group had similar unit peak flows for the unmined watersheds and the reclaimed valley fill.

The calculated unit-peak flows for in the northern group showed a different relationship. The watershed without the valley fill had a unit-peak flow that was twice as high as the watershed with a valley fill.

d. Citizen Complaints Study

The citizen complaint records for West Virginia, Kentucky, and Virginia were reviewed for allegation of flooding from coal mine operations. Of the thousands of citizen complaints received and investigated by these states, a very small percentage were related to flooding. Of those flooding-related complaints found to be mining-related, the problems were caused by improper maintenance of the approved drainage control facilities or by not following approved drainage control plans. The WVDEP records for 1995-99 were assembled and reviewed where citizens alleged flooding was caused by mining. A total of 126 complaints were investigated. Sixty-two (62) complaints were associated with surface coal mine sites. Eight (8) of these investigations resulted in enforcement actions being taken to require corrections to drainage control structures. The KYDSMRE flooding complaint records for 1996-99 were also reviewed. Thirty-five (35) investigations resulted in 5 enforcement actions to require corrections to drainage control structures. The VADMLR flooding complaint records for 1995-99 showed 3 complaints investigated for surface coal mining sites. None of the investigations resulted in enforcement actions.

e. Other Studies

Two other flooding-related studies were completed in the EIS study area. The areas evaluated in these studies were in Kentucky and West Virginia. The Kentucky study, "Joint OSM-DSMRE Special Study Report On Drainage Control" was completed in December, 1999. The West Virginia study, "Runoff Analysis of Seng, Scrabble, and Sycamore Creeks" was completed in June, 2002. The studies were designed to determine whether mining caused increases in "peak flow" downstream from the mine sites and if so, the extent to which peak flows were increased. It should be noted that the West Virginia study also evaluated the impacts of logging on peak flows. In general, these two studies concluded that mining does influence the degree of runoff, but that the extent to which a change in runoff may have actually caused or contributed to flooding were site-specific. Site-specific factors may include topographic influences, stream channel conditions, distance downstream from the mine site, man-made channel restrictions, etc. The complete state studies, including conclusions and recommendations, are found in Appendix H.

Both states' studies recognized the need for the proper, thorough analysis of peak flow and flooding potential. Kentucky's mine regulatory agency has implemented a policy requiring that certain specific engineering considerations be evaluated when conducting a review of a proposed mine application. The policy has been included in Appendix K. West Virginia is evaluating their study conclusions and recommendations and considering regulations that would require peak flow analysis and other measures to minimize flooding potential downstream of mine sites and logging operations.

H. RELATIONSHIP OF MOUNTAINTOP MINING TO GROUNDWATER QUALITY AND QUANTITY

1. EIS Workshop Findings

Some public comments received during the EIS Scoping Process centered on the impacts from Mountaintop Mining/Valley Fill (MTM/VF) to the groundwater system. Principal among these were immediate and long-term changes to groundwater quality and quantity due to MTM/VF mining practices. Blasting effects to private water supplies and groundwater quality in general were concerns, as was migration of other contaminants from mine sites. In contrast, one comment expressed a belief that valley fills maintained baseflow during low flow periods by providing a more reliable groundwater reservoir.

In support of this EIS, the Workshop on Mountaintop Mining Effects on Groundwater was held in Charleston, West Virginia on May 9, 2000. The purposes of this workshop were as follows:

1. Identify potential impacts from mountaintop mining with valley fills on groundwater quality and quantity,
2. Review existing literature and current research studies focused on the effects from mountaintop mining on groundwater systems.
3. Review and assess public comments concerning mountaintop mining impacts on groundwater received during the EIS Scoping Process and,
4. In light of the recent workshop, identify potential technical and policy actions to be considered during the EIS process.

This section summarizes the results of this workshop and other available studies on the effects of MTM/VF mining on groundwater in relation to public concerns. A conceptual model of groundwater flow is examined and potential impacts from MTM/VF are explained. Note that blasting effects are discussed separately in Section III.

2. Pre-mining Appalachian Groundwater Flow System

The surficial geology of the Appalachian coal basin is dominated by layered sedimentary sequences of Mississippian and Pennsylvanian ages. These rocks encompass cyclic sequences of lithology that document the rise and fall of sea level and basin subsidence due to compaction and plate tectonics. These sequences are called cyclothem sequences and typically repeat themselves in 15 to 50 meter intervals. They emanate from changing energy conditions in the depositional environment resulting in stratigraphic facies changes (Brady et al, 1998).

Facies/lithological changes produce the layered rock sequence seen in Appalachian drill holes and road cuts. Cyclothem sequences show repeated sandstone, shale, limestone and coal lithology that vary laterally and vertically. The impacts of cyclothem sequences on the groundwater flow system are evident in the heterogeneous nature of the hydraulic properties found throughout this region.

Cyclothem sequences affect the permeability of the aquifer matrix by influencing the hydraulic conductivity and transmissivity properties of the aquifer matrix. Permeability refers to the water

transmitting properties of an aquifer unit and has two components: primary and secondary. Primary rock permeability refers to the interstitial openings between rock grains and is controlled by rock porosity. Secondary permeability refers to any form of fracture, bedding plane separation, or solution channel that occurs after sediment consolidation. Hydraulic conductivity refers to the ability of geologic strata to transmit water. Transmissivity is a related term and is calculated by multiplying the hydraulic conductivity by the saturated thickness to arrive at the total water transmitting capacity of an aquifer unit. Transmissivity embodies the ability of the unit to transmit water and the area through which it flows. As a result of cyclothem sequences, permeability varies in three dimensions, producing very heterogeneous flow systems. Aquifer testing in this region indicates a wide range of spatial attributes in hydraulic properties, often times within the same stratigraphic interval (Bruhn, 1986, USGS, 1991, Minns, 1993, Minns et al, 1995). These same studies indicate hydraulic conductivity declines with increasing depth due to changes in consolidation of the overburden. In the Appalachian basin, secondary permeability is the dominant pathway for fluid movement (USGS, 1981, USGS, 1991). The combined affect of stratigraphic changes and differing fracture density has been shown to produce lateral changes in the hydraulic properties of aquifer materials (Stoner, 1987, Minns, 1993).

An interconnected stress relief fracture network of varying density underlies the Appalachian basin. Ferguson (1967) was the first to propose a model of stress relief fracture systems in the Appalachian basin. His model indicated arching of the strata underlying valleys due to overburden unloading associated with major stream valley development. Ferguson's model shows horizontal fractures underlying stream valleys with vertical fractures along the valley walls and ridge tops. Hill (1988) proposed a distinction between wide stream valleys (> 500 ft) and narrow, V-shaped stream valleys whereby the valley floor experienced compressive stress instead of tensile stress found in broader valleys. This phenomena results in a decrease in fracture density under V-shaped valleys. Since the work of Ferguson, several researchers have proposed general models of groundwater movement for this region that incorporate the valley stress relief concept (Hobba, 1981, USGS, 1981, Kip et al, 1983, USGS, 1985). Several studies also indicate that the majority of groundwater flow occurs in the top 250 to 300 feet of strata (Stoner, 1987, USGS, 1991, USGS, 2001). Researchers have characterized Appalachian basin aquifer systems as fracture flow systems with numerous perched aquifers in the upper topographic intervals (Hobba, 1981, USGS, 1991, USGS, 1991a, Kipp and Dinger, 1991, Minns, 1993). Groundwater availability is limited on hilltops due to reduced areal recharge potential, depth to water and reduced transmissivity values (Stoner, 1987, Kipp and Dinger, 1991, Minns, 1993).

3. Impacts to Groundwater Quantity from MTM/VF

Mountaintop removal is a surface mining technique that removes a series of coal seams by removing all overlying strata down to an economical limit governed by the overburden to coal ratio. Contour and area mining of mountaintops removes <u>part</u> of the coal seams in the mountain or all of the coal seam in portions (e.g., in a narrow ridge) of the mountain—also to the economic limits of extraction. Auger mining conducted from the contour or area mining bench may remove additional coal within the mountain. As these types of mountaintop mining operations progress, overburden in excess of that required to reclaim the mine site is placed in an adjoining valley(s). The SMCRA regulations stipulate that overburden placed in valley fills must meet certain engineering criteria to ensure stability, drainage control and reclamation/re-vegetation of the valley fill. In addition, each respective state permitting program ensures any discharge from the individual mining permits adhere

to water quality standards as set forth under the various state and federal programs. The current EIS is an evaluation of the practices currently employed for MTM/VF techniques.

a. Conceptual Model of MTM / VF

Conceptually, MTM/VF mining is the complete or partial removal of mountaintops by breaking the strata into small blocks and placing the excess spoil in an adjoining valley. The physical effects of MTM/VF are clear; mountain slopes are radically decreased, both by removal of material and by filling adjoining valleys. The affects to the physical groundwater system are the elimination of the perched aquifer system in the mountaintops, and formation of an aquifer system in the valley fill.

The shallow, pre-mining perched flow system proposed by several researchers is located within the overburden strata associated with mountaintop topography (Hobba, 1981, USGS, 1991, Minns, 1993). This flow system forms the headwater areas of the region's streams and is a minor source of residential water throughout the Appalachian region due to the concentration of the majority of the population in stream valley settings. Removal of mountaintop strata removes the perched aquifer system and places the excess overburden in adjoining valleys, thus eliminating the perched system.

The placing of overburden in adjacent valleys of the MTM/VF regions of the Appalachian basin join two aquifer systems: the premining fracture flow system that underlies and adjoins the valley fill; and a postmining man made aquifer consisting of excess overburden removed during mining. Wunsch et al (1996) proposed a model of groundwater flow through a valley fill in eastern Kentucky. They determined water moved through the Star Fire mine site at differing velocities depending on the nature of spoil, preferential sorting of the spoil upon placement and degree of compaction during placement. This work corroborates work done by Carruccio et al (1984), Aljoe and Hawkins (1992), and Aljoe (1994) using pump tests and dye tracing in reclaimed surface mines. The change in spoil porosity affects the hydraulic conductivity distribution in the fill and ultimately dictates the groundwater flow regime that establishes within the fill. Groundwater gradients within the fill roughly follow the undisturbed topographic elevations; flowing along the pre-fill valleys. The type of fill material placed in these locations enhances this flow mechanism (Aljoe, 1994). Wunsch, et al (1996) noted at the Star Fire site that water recharges the site by way of surface water infiltration along the highwalls, groundwater infiltration through the highwalls, chimney drains placed in the fill, and along the headwater areas of stream courses covered during the operation. At the Star Fire site, groundwater discharges as spring flow at the toe of fill, into an adjacent active dragline pit, and into sediment ponds located on lower portions of the fill. The sediment ponds are used for dust suppression and are pumped on a continuous basis. The Star Fire site is a typical valley fill scenario.

b. MTM/VF impacts to the physical Ground Water system

Valley fills create aquifer systems that perform two functions: 1) store a larger percentage of water that would normally run off the landscape; 2) serve as separate aquifer systems. Overburden placed in valley fills consists of broken strata that are disposed of in an adjacent valley. These fills are large-scale, generally primary porosity-driven flow systems although some studies have indicated a dual porosity flow system (Caruccio, 1984, Aljoe, 1994). Water moves through them under hydraulic gradients (i) derived from the hydraulic conductivity (K) and storage (S) properties of the rock fragments. The storage (storativity) properties of the man-made aquifer are significantly greater than the original rock mass due to the increase in pore space. Total porosity may be similar

between pre and post-mining scenarios but increases in pore size and connectedness create greater *effective* porosities allowing more water to freely move through the aquifer unit. Effective porosity values for undisturbed Appalachian fractured rock aquifers range from 0.001% to 0.1% (MacKay and Cherry, 1989). Brown and Parizek (1971) found laboratory-measured porosities of coal bearing strata to range from 0.8% to 9.4% with a mean of 3.9%. Several authors have found insitu effective porosities in surface mine spoil ranging from 14% to 36% spoil aquifers (Cederstrom, 1971, Wells, et al, 1982, Hawkins, 1995). Using the laboratory derived effective porosity for insitu strata of 3.9% and an average effective porosity of spoil of 25% equates to an approximate 21% gain in porosity over premining values. A 1000-acre unmined site with a 30 foot saturated thickness stores approximately 12 million gallons of water at 3.9 % porosity, while the same size valley fill stores approximately 81 million gallons of water at 25% porosity. The valley fill site holds approximately 7 times more water than its premining counterpart.

The increase in storage of valley fill aquifers is also enhanced by a decrease in runoff volumes associated with slope reduction. Simple runoff calculations using Natural Resource Conservation Service techniques indicate runoff volumes theoretically decrease by approximately 50% for a reduction in slope from steep ($i > 8\%$) to flat ($i = 0$ to 3%) classifications and allowing the CN value (CN 70 to CN 75) to increase to account for decreased vegetation (Maidment, 1993). This decrease in runoff theoretically allows more water to infiltrate and/or re-saturate the surface of the valley fill. By diverting the runoff into the valley fill, water is effectively stored in the fill material and is released in a more subdued manner, thus affecting the peak flow volumes in adjacent streams. Wunsch et al (1996) and Wiley et al (2001) noted this phenomenon in their Appalachian basin fieldwork. Research by the USGS on stream flow characteristics in the Appalachian basin indicates similar trends (Paybins et al, 2002, Messinger, 2002).

Data from the Star Fire site indicate a greater percentage of precipitation is captured by the valley fill aquifer system compared to unmined settings. A flume located immediately downstream of the valley fill captures all the water leaving the site as discharge from the various groundwater discharge points. Measurements taken during normal baseflow conditions, that eliminate the influence of surface water, indicate 1000 gallons of water per minute (2.23 cfs) is discharging from the Star Fire site. The site has an approximate area of 1000 acres resulting in an effective infiltration rate through the valley fill of approximately 1.0 gallon per minute per acre (gpm/acre). Assuming 49.7 inches of rainfall per year, 1.35×10^6 gal/year of precipitation falls on this part of Kentucky. This total equates to 2.57 gal/min of rainfall per acre of land surface. The Star Fire site discharges approximately 1.0 gal/min/acre of valley fill, equating to 39% or 19.3 inches of the yearly precipitation falling on the land surface. Typical unmined Appalachian basin mean groundwater discharge rates range from 6.7 to 31.6 inches per year (18.8% to 50.9%) measured as the groundwater discharge component of stream baseflow in West Virginia, Virginia, and North Carolina (USGS, 1996, USGS, 2001, USGS, 2001). USGS (2001) report a band of the high mean infiltration rates (41.1% and 50.9% of total precipitation) located in a narrow band encompassing the eastern portions of West Virginia. The majority of infiltration rates cited by USGS (2001) range between 18.8% and 27.1% for the remainder of West Virginia based on 27 different stream stations. At a 39% infiltration rate, the Star Fire site directs a larger proportion of precipitation into the valley fill than is implied in recent research in unmined scenarios.

Insitu infiltration rates determined by infiltrometer studies performed on contour surface mines also indicate spoil infiltration rates increase through time; ameliorating the affects of compaction on the

surface (Jorgenson and Gardner, 1987, Ritter and Gardner, 1993). Ritter and Gardner (1993) showed through modeling, that hydrograph curves evolved over time to closely mimic runoff curves associated with saturation overland flow processes. They also concluded that runoff processes at surface mined sites are dominated by saturation overland flow which cause decreased peak runoff and increased time to peak runoff that result from the lagged response of return flow to the surface water network. Recent field studies on the effects of valley fills on peak stream discharge indicate similar trends and responses to their modeling research (Messinger, 2002).

Increases in effective porosities of spoil also lead to increases in hydraulic conductivity. Hawkins (1995) found spoil conductivities were 1 to 2.5 orders of magnitude greater than the adjacent rock mass. Herring (1977) and Weiss and Razem (1984) also noted similar findings in spoil related research. Aljoe (1994) noted that increases in the percentage of sandstone overburden in a fill also increase porosity and hydraulic conductivity. The increase in hydraulic conductivity and storativity leads to increased water velocity and reduced hydraulic head in the postmining spoil aquifer (Hawkins, 1995). The reduction in hydraulic head is related to the decrease in hydraulic energy required to drive water through spoil aquifers compared to undisturbed strata. Booth and Spande (1992) and Kendorski (1994) noted similar overburden aquifer response in longwall mining areas due to a similar increase in hydraulic conductivity and storativity.

Interaction between spoil aquifer systems and the underlying aquifer system is likely limited in areas compacted by mining equipment during active mining phases. In these areas, compaction has reduced infiltration capacity by providing an effective low permeability confining layer separating the underlying flow system from the valley fill. Wunsch et al (1996) found similar responses to rainfall runoff in areas of compacted cover material for a valley fill area in eastern Kentucky. Hawkins (Brady et al, eds., 1999) also points out similar phenomena in his chapter on hydrogeologic characteristics of surface mine spoil.

c. Impacts to Valley-bottom Groundwater Recharge From MTM/VF

Groundwater recharge to lower elevations may be impacted by mountaintop removal by reducing the amount of recharge available and/or diverting groundwater to the valley fill flow system. However, conceptual models of premining groundwater flow indicate the amount of water actually recharging valley aquifers may be limited and as such MTM/VF impacts on these aquifers would likely be similarly limited. A large percentage of precipitation falling on upland areas runs off, becoming surface flow in streams. Water that does infiltrate may or may not become part of the deeper groundwater system dependent upon existence and/or interception by valley sidewall fractures. Water that is not diverted vertically will flow horizontally on top of low permeability strata and emanate as spring flow on the valley sidewalls. Water that does get diverted into the valley sidewall fracture system infiltrates and becomes part of deeper flow systems. This water may be capable of providing a component of recharge to valley bottom aquifers. Further research needs conducted to determine the impacts from diversion / elimination of these perched systems to lower elevation alluvial aquifer systems.

4. Impacts to Groundwater Chemistry From MTM/VF

SMCRA mandates that all coal mining operations collect quarterly sampling for total iron, total manganese, total suspended solids and pH. These minimum parameters are collected at all approved mining related discharge sites and monitor the most significant components of typical coalmine drainage. The minimum list does not capture the entire expected range of chemical species emanating from coal mine drainage.

In its most basic form, overburden containing silicate and carbonate minerals is broken up, deposited into an adjacent valley, and water is allowed to flow through the fill material. The exposure of fresh mineral surfaces to a geochemically reactive material (water) produces the water chemistry produced at coal mine sites.

a. Geochemical Reactions

Coal mine drainage is produced by the oxidation of pyrite in an aqueous environment that dissociates the iron and sulfur found in the pyrite (FeS_2). Pyrite is a sulfide mineral commonly formed in the reducing environments associated with Bituminous coal fields. Coal mining and subsequent overburden removal exposes the pyrite to oxygen, which is summarized by the following reaction (1) (Brady et al, 1999):

$$FeS_2\,(s) + 3.75\,O_2 + 3.5\,H_2O = Fe(OH)_3(s) + 2\,SO_4^{2-} + 4\,H^+ + heat \quad (1)$$

Alkaline mine drainage can be produced when acidic mine water comes in contact with alkaline overburden and/or alkaline recharge migrates into the valley fill. The reaction (2) between pyrite, calcite, in limestone, and water is:

$$FeS_2 + 4\,CaCO_3 + 3.75\,O_2 + 3.5\,H_2O = Fe(OH)_3 + 2\,SO_4^{2-} + 4\,Ca^{2+} + 4\,HCO_3^- \quad (2)$$

This reaction will produce alkaline mine drainage with circumneutral pH, alkalinity greater than acidity, high sulfate and calcium concentrations and iron hydroxide as a precipitate.

Researchers have also noted high levels of sodium, magnesium, and calcium in coal mine drainage that were attributed to cation exchange (Winters et al, 2000, Perry, 2001). Divalent calcium and magnesium ions are exchanged at surface sites of clay minerals for monovalent sodium ions and can be summarized by the following reaction:

$$2\,Na^+ _\ 1\,Ca^{2+}\,(Mg^{2+})$$

Preliminary research by the EPA for the EIS document also indicates increased levels of selenium in bituminous basin discharge water (USEPA, 2002). Aluminum has also been documented in coal mine drainage at elevated levels (Brady et al, 1999).

No correlation was possible in an EPA statistical evaluation ("Ecological Assessment of Streams in the Coal Mining Region of West Virginia Using Data Collected by the U.S. EPA and Environmental Consulting Firms") of the amount and age of upstream disturbance on the character of water quality impacts; or the distance downstream that the mineralization persisted (USEPA,

2002). Further study is needed to determine the duration of the mineralization, which may be expected to decrease with time as backfill and valley fills are "flushed" of soluble materials.

b. Conceptual Geochemical Model

Overburden mineralogy determines the final geochemical signature of post mining water quality. Mining exposes fresh rock surfaces to water and oxygen allowing several reactions to occur, most notably pyrite oxidation, calcite dissolution and cation exchange. Silicate weathering may also provide chemical constituents to the final mine water chemistry, especially in acidic discharges.

Relationships between overburden mineralogy and groundwater composition lead to ionic dominance of various chemical constituents found in a water sample. Piper tri-linear diagrams provide a visual representation of the composition of the major constituents found in a water sample. Relative compositions of calcium, magnesium, sulfate, bicarbonate and chloride ions are plotted on triangular axes from which mineral provenance is estimated based on a comparison between discharge chemistry and the mineralogical composition of the aquifer matrix.

Geochemical modeling of Appalachian basin groundwater indicates several different geochemical facies are present in pre-mining aquifers. Geochemical sampling of pre-mine groundwater indicates three distinct geochemical zones within the aquifer system of the Appalachian basin. The deepest zone is characterized by sodium and chloride ions associated with brine water at depth (Rose and Dresel, 1990). Numerous studies indicate a brine – fresh water interface at depths of 1000 feet below surface with upconing under major stream valleys to depths of 100 feet (Stoner et al, 1987, Minns, 1995). The upconing area is a mixing zone but contains considerable quantities of sodium and chloride ions diluted by mixing with shallower water types. Intermediate geochemical zones are characterized by removal of the chloride ion by flushing, resulting in a sodium–bicarbonate ion dominated water chemistry. Wunsch (1993) and Minns (1995) geochemical models show this water signature was found at depths ranging from 50 to 150 feet below local base level. Shallow flow systems are dominated by calcium–bicarbonate ions due to flushing of the sodium ions from the system. Brady et al (1996) further subdivided this shallow zone into a low total dissolved solids (TDS) zone associated with stress relief/weathered regolith and a higher TDS zone associated with ridge cores. The difference between the two sub-systems is derived from water residence time and degree of weathering between the two sub-systems. Longer residence times in contact with unweathered material produces more ions in the water leading to higher TDS values whereas shorter residence time with weathered material leads to lower TDS values. Wunsch (1993) and Minns (1995) found similar geochemical zones but also found sulfate and magnesium were present in significant quantities in these shallow geochemical zones.

In Kentucky valley fills, Wunsch et al (1996) found that water emanating from the fills was a calcium–magnesium–sulfate type water resulting from pyrite oxidation and calcite dissolution along the groundwater flow path. Discharge data from Wunsch et al (1996) supports neutralization of pyrite oxidation products within the valley fill interior. Pyrite oxidation is likely occurring within the unsaturated portion of the fill as evidenced by the elevated sulfate (range: 300 to 2000 mg/l) concentrations in the discharge water quality. These oxidation products (Fe, SO^4) are then carried with infiltration and/or groundwater to the main flow paths through the fill. Alkalinity generating processes are also at work buffering the pH to approximately 6.2 (except well 14). The discharge chemistry contains significant concentrations of neutralization products (Ca, Mg, HCO^3) leading to

the calcium–magnesium–sulfate type water emanating from the Star Fire site. This conceptual flow model has been observed at surface mines throughout the Appalachian basin (USGS, 1990).

The Star Fire site also indicated increased concentrations of total dissolved solids in the discharge chemistry. This phenomena results from the release of ions due to exposure of unweathered minerals placed in the fill as spoil. Elevated TDS concentrations have been documented in surface mining discharge chemistry for more than 25 years (USGS, 1983, Quinones et al, 1981).

In the absence of neutralization materials, acidic discharges can develop whereby the main ionic constituents are iron and sulfate with lesser amounts of aluminum and manganese resulting in a sulfate–iron dominated type water. This water will have low pH (< 5.0) and very high TDS concentrations (> 2000 mg/l). This water can also be very reactive with overburden mineralogy: dissolving silicate minerals producing significant concentrations of dissolved silica, aluminum, magnesium and trace metals.

5. Summary of Groundwater Impacts

Mountaintop mining removes the perched aquifer system from the base of the target coal seam upwards. By placing this material into the adjacent valley, a new aquifer is formed. The valley fill aquifer system develops according to the physical properties of the spoil matrix and corresponding flow mechanisms that develop. Overburden placement techniques, material sorting and post-deposition compaction control the hydraulic conductivity and corresponding hydraulic gradient distribution within the valley fill. The valley fill is also capable of storing larger volumes of water compared to the original rock mass. These storage components affect stream hydrology by creating lag times in storm-induced runoff hydrographs. Sedimentary rock overburden mineralogy controls the discharge chemistry in the Appalachian basin. Exposure of fresh mineral surfaces to oxygen and water provide the geochemical mechanism for chemical evolution within the fill. The ultimate expression of the discharge is controlled by the amount and residence time of the water within the fill, which are governed by the physical properties of the spoil matrix. The Star Fire site in eastern Kentucky is a good conceptual model of an average valley fill aquifer system found in the Appalachian basin. It represents typical overburden mineralogy, mining technique and discharge chemistry of a typical Appalachian coal basin mountaintop mine.

EPA, in a 2002 statistical study of stream quality and macroinvertebrates mountaintop mine sites found correlations of stream impairment with mining disturbances upstream (USEPA, 2002). However, their report found certain data gaps for which no correlations could be evaluated. The study recommended additional evaluation to determine:

- The duration of mineralization of groundwater discharges from mountaintop mining sites. Improvements in water chemistry may be expected, with time, as the backfills and valley fills are flushed of soluble minerals on the fresh rock surfaces.
- The correlation of the size of mining disturbance and associated “mining aquifers” in a watershed with the amount of mineralization. That is, do larger backfill and valley fills increase mineralization beyond that occurring for smaller fills?

6. Groundwater Quantity and Quality Conclusions

Removal of the pre-mining perched aquifer system and associated valley fill will impact the headwater reaches of first order streams in the region by eliminating streams. Impacts to valley bottom aquifer may or may not occur depending on density of valley sidewall fractures.

Creation of valley fill aquifers change the hydrology of streams receiving baseflow from valley fill aquifers by diverting a greater percentage of precipitation into the fill, allowing water to be released at a much slower and less intense rate compared to normal storm-induced stream hydrographs (Ritter and Gardner, 1993, Wiley, 2001, Messinger, 2002).

Groundwater chemistry within valley fills changes from $Ca–HCO_3$ dominated water to a $Ca–Mg–SO_4$ dominated water reflecting pyrite oxidation and neutralization of oxidation products in the fill interior (USGS, 1990, Wunsch, et al, 1996).

MTM/VF water chemistry indicates increases in TDS resulting from groundwater contact with unweathered overburden fill material.

Further Study: Impact of MTM/VF on alluvial aquifer systems; interaction between valley fill and adjacent aquifer systems; sources of selenium in MTM/VF regions; geochemical effects from weakly buffered overburden in valley fills; correlation of mineralization characteristics with specific stratigraphic horizons, size and age of disturbance; and the duration of mineralization and distance of effects downstream.

I. OVERVIEW OF APPALACHIAN REGION COAL MINING METHODS

Mining has been conducted in the Appalachian coalfields since European settlers arrived in the region in the 1700s. Uses of coal have progressed from simple home heating and cooking, to fuel for railroads and steamships and industrial processes, and now to a predominant share of the electric power generation market. To keep pace with increasing demand, methods of mining coal have advanced from pick-and-shovel works to steam-powered equipment and now to mechanized deep mines and large-scale surface operations. National industry trends have favored surface operations over underground mining in recent decades, driven by the advent of very large earthmoving equipment, and surface methods now account for the majority of nationwide production. This trend is expected to continue, as surface mines generally provide better coal recovery than underground mines and have lower overall production costs per ton of coal.

UNDERGROUND MINING DOMINATES COAL PRODUCTION IN THE STUDY AREA

In Kentucky, Tennessee, Virginia, and West Virginia, underground mining still dominates coal production, comprising 61 percent of the combined production for the study area in 1998, while surface mining methods account for the rest (EIA, 2000). A significant percentage of these surface mines can be categorized as Mountaintop Mining/Valley Fill (MTM/VF) operations, and use of this mining method has become widespread in recent decades in response to increasing competition from western coal producers. MTM/VF operations are generally the most economical and efficient forms of surface mining in steep-slope Appalachia and provide for the highest possible recovery of multiple coal seams.

The term "mountaintop mining" used in the EIS encompasses three different kinds of surface mining operations (contour mining, area mining, and mountaintop removal mining) that create valley fills. This is a broader definition than the legal definition used in SMCRA "mountaintop removal mining." Mountaintop removal mining totally extracts underlying coal seams, and the reclaimed land is left in a flat or gently rolling configuration capable of supporting certain post-mining land uses, such as industrial, commercial, residential, agricultural, or public facilities (including recreational facilities). Since the reclamation of a mountaintop removal mine will leave flat or gently rolling land, the "approximate original contour" (AOC) standard of SMCRA does not apply. This is also true of steep slope AOC variances allowed under SMCRA-which may occur at area or contour mines. Thus, the reclamation required of a mountaintop removal or AOC variance mine is markedly different from that of an AOC steep slope area or AOC contour surface coal mine. Steep slope AOC variances and mountaintop removal operations, by their very nature, result in greater excess spoil disposal. This EIS will use the broader terms "mountaintop mining" or "mountaintop operations" to refer to all of these types of surface coal mining in the steep slope areas of the central Appalachian mountains.

Because of significant differences and much variability in geology, topography, and property ownership patterns, surface mining practices can vary from state to state within the Appalachian coal fields. For example, significant "overburden to coal" ratios often restrict the Kentucky mining industry to two or three coal seams that can be economically extracted by mountaintop mining methods. As a result, the typical surface coal mine in Appalachian coal fields of Kentucky is

approximately 350 - 400 acres in size. In West Virginia, as many as 18 seams might be mined in some permits of more than 1000 acres. In Virginia, the permit sizes are typically smaller than West Virginia and Kentucky mines, and coal removal may be limited to 3-5 seams.

Because there are a very large number of small surface owners in the eastern Kentucky coal fields, acquisition of consent to entry for the purpose of mining is often a very expensive, difficult, and very time-consuming process. This land ownership pattern is quite different than that found in adjacent coal producing states, and also serves to greatly limit both the permit size and the scale of mining conducted by the Kentucky coal mining industry.

Surface coal mining operations in Virginia differ significantly from surface coal mining operations in West Virginia and differ somewhat from those operations in Kentucky. Surface coal mining in Virginia has a long history, with most of the actively-producing coal region affected by pre-SMCRA strip mining activities. Almost all of the permit applications received by VADMLR contain AML areas that total between 50 % and 80 % of the area. Most of the streams on these proposed mine sites have been impacted by pre-SMCRA mining, and may be impacted by old spoil and/or dislocated by the prior mining. Often streams shown on the USGS topographic maps no longer exist or may have been moved by placement of spoil into the stream. Often there are long segments of stream that have no defined stream channel: the stream may spread into a wetland, it may disappear under spoil, or it may have been affected in other ways by the pre-SMCRA mining activities that occurred in the vicinity.

The size of mining operations in Virginia is limited by several factors. These include factors such as geologic conditions, steep slopes, and fragmented mineral and surface property ownership. The remaining reserves are also fragmented by prior AML and underground mining operations creating relatively small non-contiguous areas of coal available to be mined. Proposed permit areas usually consist of second cut areas that are separated by AML highwalls that cannot be mined due to prior augering, the proximity of underground mining, or excessive ratios of overburden to coal. Companies in Virginia often mine ratios exceeding 20:1 in order to recover what coal is available. These AML benches and highwalls that are not mined are used to dispose of excess spoil generated by the adjacent remining operations. There are a few permits that have first cut areas proposed, but these are usually limited in extent and are adjacent to second cut areas. VADMLR requires companies to minimize valley fills by using the excess spoil to reclaim adjacent AML highwalls and benches. Virginia mining operations reclaim nearly all areas to AOC. There are no drag lines operating in Virginia.

Current technology achieves nearly the highest possible recovery of the coal reserves beneath a typical tract of Appalachian land; however, this is neither always economically feasible nor acceptable from an environmental standpoint. Modern coal mining combines a variety of approaches to coal extraction that reflect the maximum amount of coal that can be recovered from a given land parcel within current market conditions and the regulations that govern coal mining. The two basic approaches are underground mining, where the coal is extracted without removing the overlying soils and rock, and surface mining, where this material, known as *overburden*, is removed to expose the coal for extraction.

In this section, Appalachian coal mining methods are first reviewed to provide background for further discussion. Typical mountaintop mine complexes are then described. The typical

characteristics of MTM/VF operations are presented to summarize this composite mining practice in section III.J. Section III.L presents a review of the factors influencing the feasibility of coal mining on a given site and the typical approach to developing a mine plan.

1. Underground Mining Methods

A description of underground mining methods is provided in the EIS as background to facilitate the discussion of whether underground mining methods would be able to take the place of surface mining methods. This section also provides background to the description of the synergism between underground and surface mining methods for purposes of blending coal. In underground mining, also known as *deep mining*, coal is extracted by excavating within the horizon of a coal seam and without removing the overlying overburden for reasons other than primary seam access. This approach is practical for seams of greater than 100 feet in depth, as underground mining of shallower seams can encounter difficulties with roof integrity and surface cracking (Suboleski, 1999a). Underground mines can be categorized by the manner in which access to a coal seam is made, and by the manner in which a coal seam is extracted. Access methods can include drift, slope, and shaft mines, and extraction methods can include room and pillar (conventional and continuous) and longwall mining. The method of coal extraction is not dependent on the method of access, and multiple methods of access and extraction may be present in an individual mine. Although not directly related to the focus of this EIS on surface mining valley fill impacts, underground mines are part of the overall coal industry within the study area, representing at times a constraint on the extent of surface mining or an alternative to surface mining.

a. Underground Mine Access

The method of accessing a coal seam for underground mining depends largely on its vertical position relative to the ground surface. The three basic options are summarized by Figure III.I-1. A *drift* mine enters a coal seam horizontally, requiring that the access be where the coal outcrops on the side of a slope or mountain. This is generally the simplest and most economical mine access method due to the fact that there is no significant excavation into the overburden. A slope mine utilizes an inclined entry to access the coal seam and is employed where the coal outcrop cannot be directly accessed, but is still within a reasonable vertical distance from the ground surface. Slope entries are usually driven at angles of less than 16° from the horizontal, in order to facilitate conveyor haulage, and must tunnel through the rock above the coal, or overburden, to achieve this access (Suboleski, 1999b). A shaft mine consists of a vertical opening driven from the ground surface to the coal seam and is employed where the coal seam is relatively deep or cannot be otherwise accessed due to topography or property limitations. This elevator arrangement, known as a *hoist,* is used to transport coal and miners to and from the surface through the shaft, with coal carried in hoist cars known as *skips*, and miners riding in hoist cars known as *cages*. An individual mine may have more than one of these access types, depending on safety, coal haulage, ventilation, and supply requirements.

b. Room and Pillar Mining

The defining principle of a room and pillar mine is that portions of the coal seam remain in place to support the mine roof while coal is extracted. Room and pillar mines are developed by driving parallel series of *entries*, usually four to eight in a series, with perpendicular *crosscuts* that connect the entries to form a grid-like pattern in a panel of coal, which can be more than 400 feet wide and

half a mile long. Figure III.I-2 shows an example of a typical room and pillar mining plan. The coal blocks that remain within this pattern after primary coal extraction are referred to as *pillars* or *stumps* and serve to support the roof of the mine. The coal pillars are generally 20 to 90 feet wide, and the entries average 20 to 30 feet wide. Room and pillar mines are best suited to relatively small reserves, or reserves where variable coal quality requires selective extraction within the seam, and can be applied to seams from 28 inches to 13 feet in thickness. The equipment required for room and pillar mining has a smaller capital investment requirement than that for a longwall mine and can be more easily moved to other mine sites (Suboleski, 1999a).

After a panel has been fully developed, the mining direction is usually reversed for retreat or secondary extraction. During secondary extraction, some of the remaining coal pillars are removed in a systematic manner in order to maximize the amount of the coal seam that is recovered from the panel. Secondary extraction can result in roof collapse and subsidence as the roof support of the pillars is removed. The amount of secondary mining performed at a mine depends on safety, subsidence, geology, and coal market considerations. Room and pillar mines with both primary and secondary extraction can achieve approximately 70 to 80 percent recovery of a coal seam, while primary extraction alone can achieve only about 40 to 60 percent (McDaniel & Kitts, 1999). Within this general mining type, the two basic extraction methods employed in room and pillar operations are conventional and continuous mining.

b.1. Conventional Room and Pillar Mining

Conventional room and pillar mining employs a combination of mechanical cutting machines and blasting to extract coal from coal faces exposed within an advancing panel. Once the predominant mining method in the Appalachian coal fields, it now accounts for only about 10 percent of total production (Suboleski, 1999b). The conventional process is conducted in five distinct steps:

1) Cutting – the coal face is undercut, side, center, or top cut by a mobile machine that resembles a large chain saw. Cutting of the coal allows another open face into which the rock can be blasted.
2) Drilling – the coal face is drilled in a pre-determined pattern to insert a blasting agent or compressed air.

3) Blasting – the cut coal face is blasted to free the coal for loading and hauling.

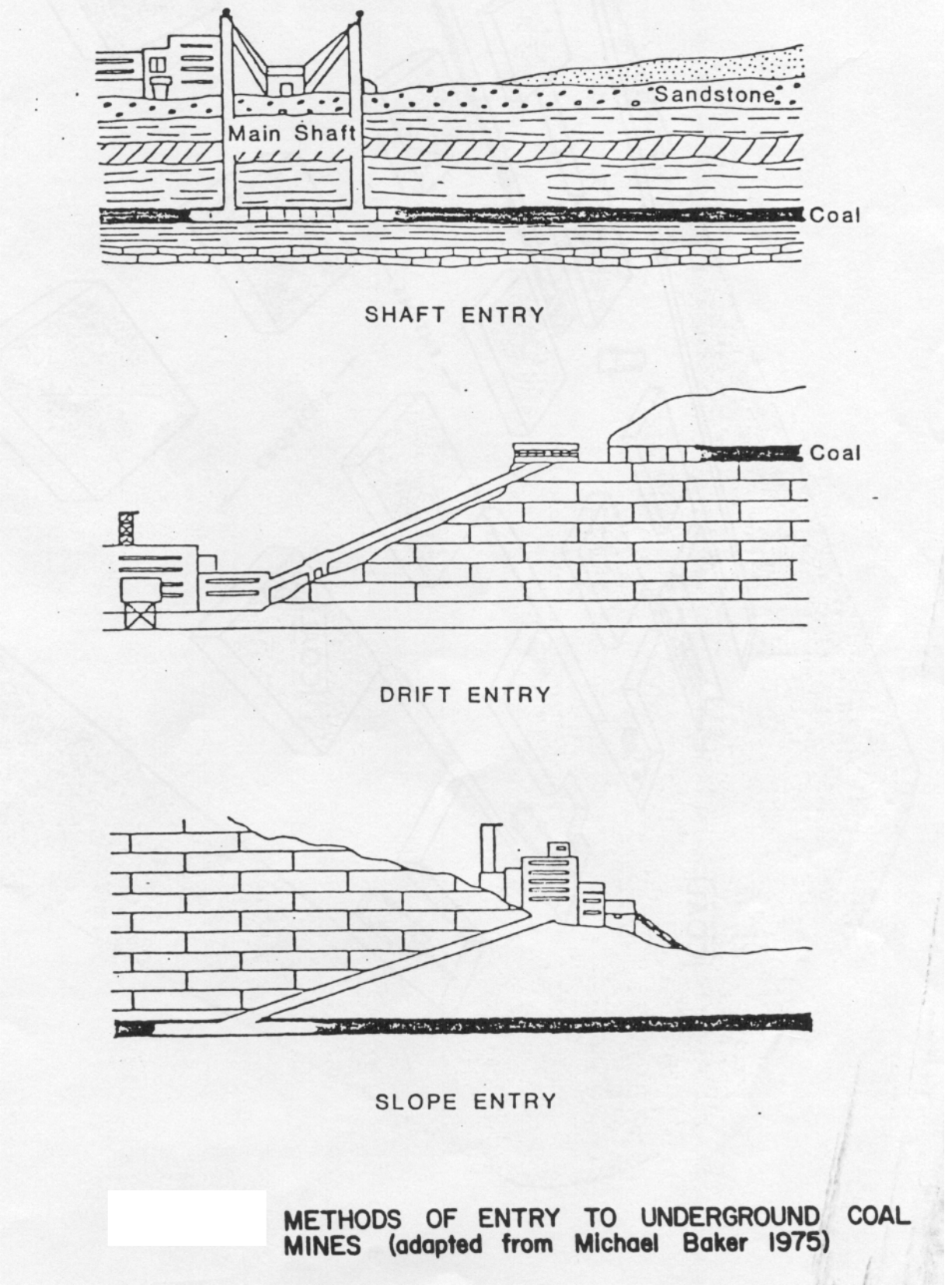



Figure III.I-1
Basic Options for Underground Mine Access