

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

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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.

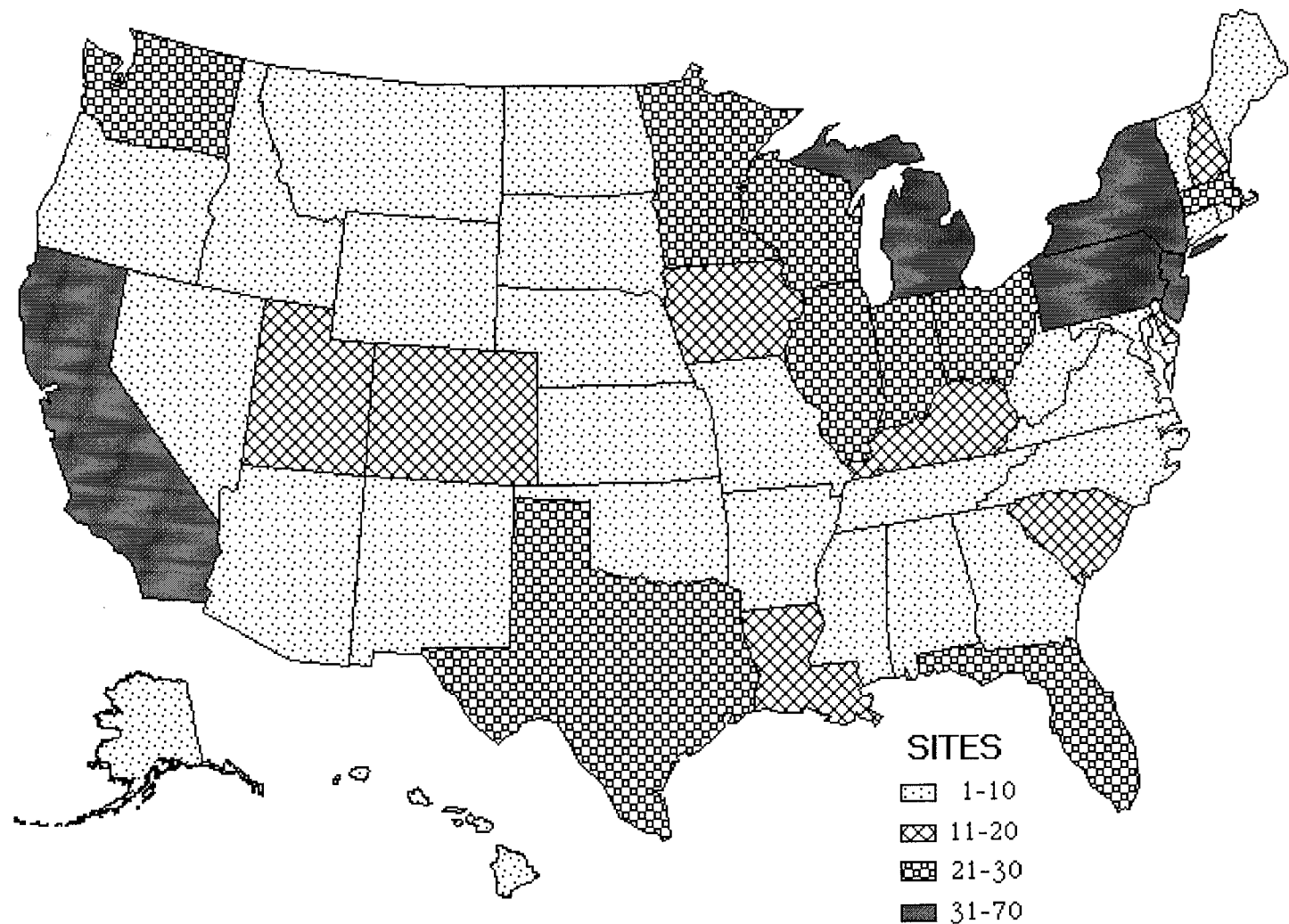
Members of the general public with potentially high exposures include individuals who live in proximity to former mercury mining or production sites, secondary production (recycling) facilities, municipal or medical incinerators, or coal-fired power plants. Other populations at risk of exposure include 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

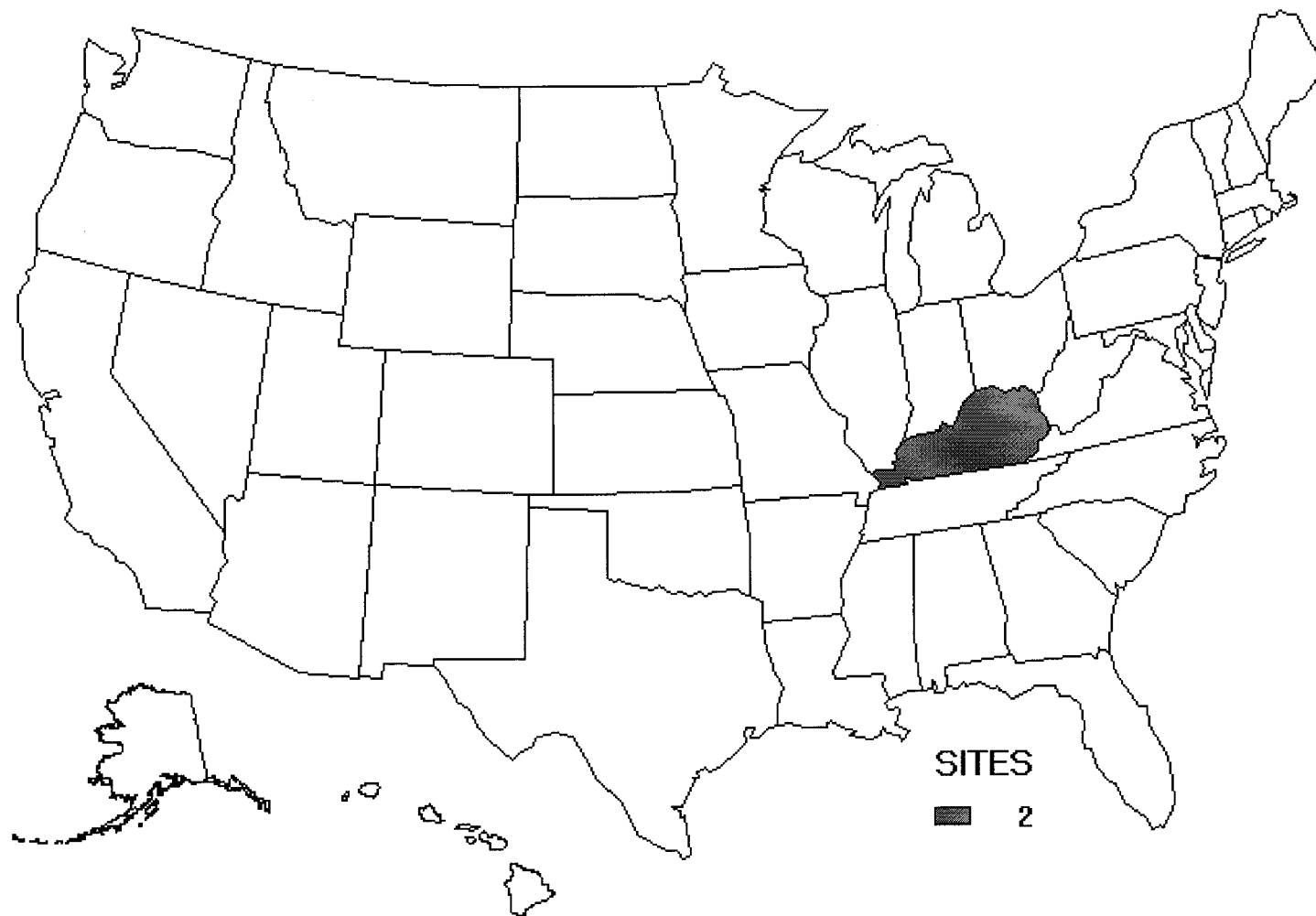
Mercury is released to the environment by both natural processes (e.g., volcanic activity and weathering of mercury-containing rocks) and anthropogenic sources. Anthropogenic releases are primarily to the

Figure 5-1. Frequency of NPL Sites with Mercury (Elemental) Contamination



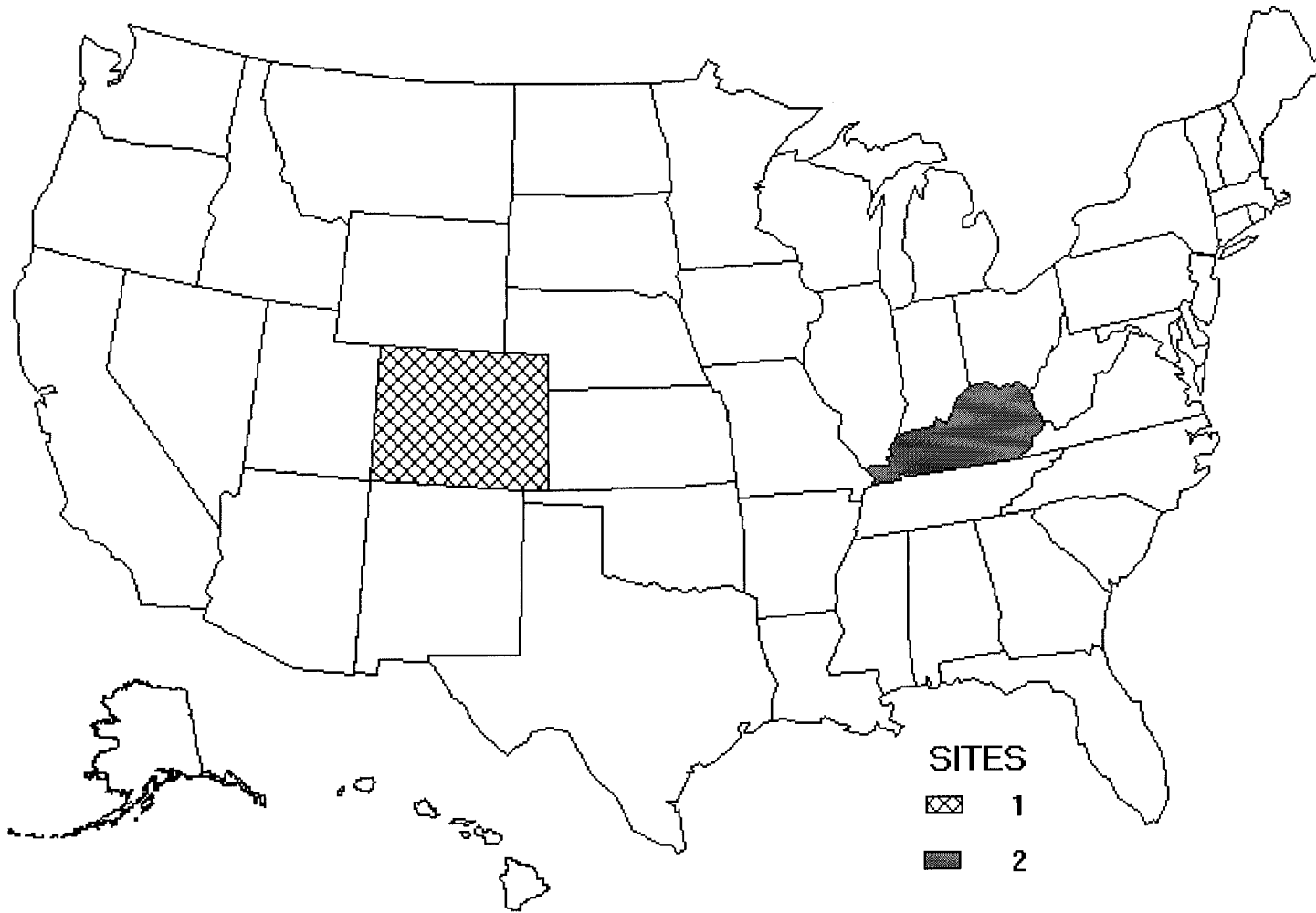
Derived from HazDat 1998

Figure 5-2. Frequency of NPL Sites with Mercuric Acetate Contamination



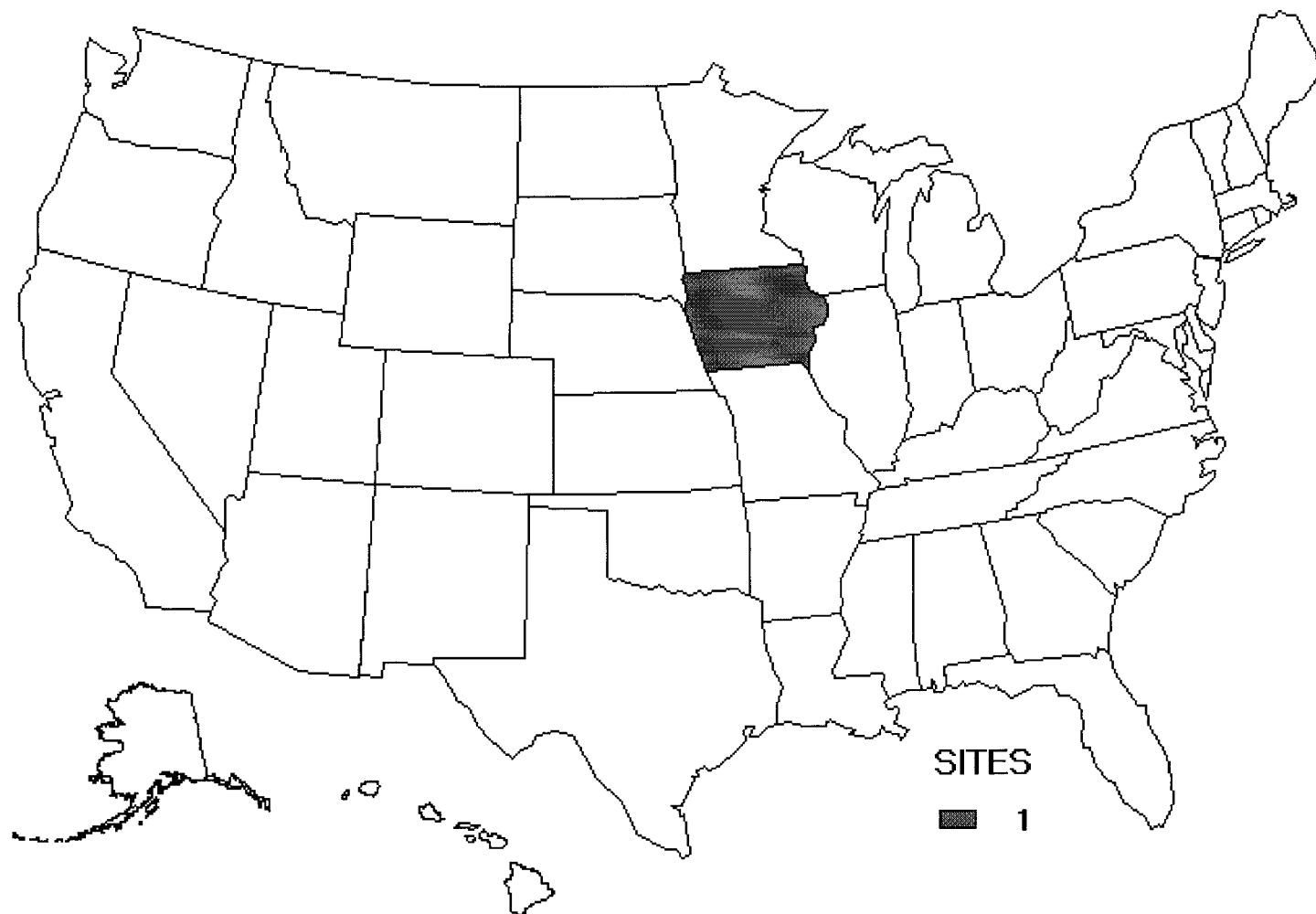
Derived from HazDat 1998

Figure 5-3. Frequency of NPL Sites with Mercuric Chloride Contamination



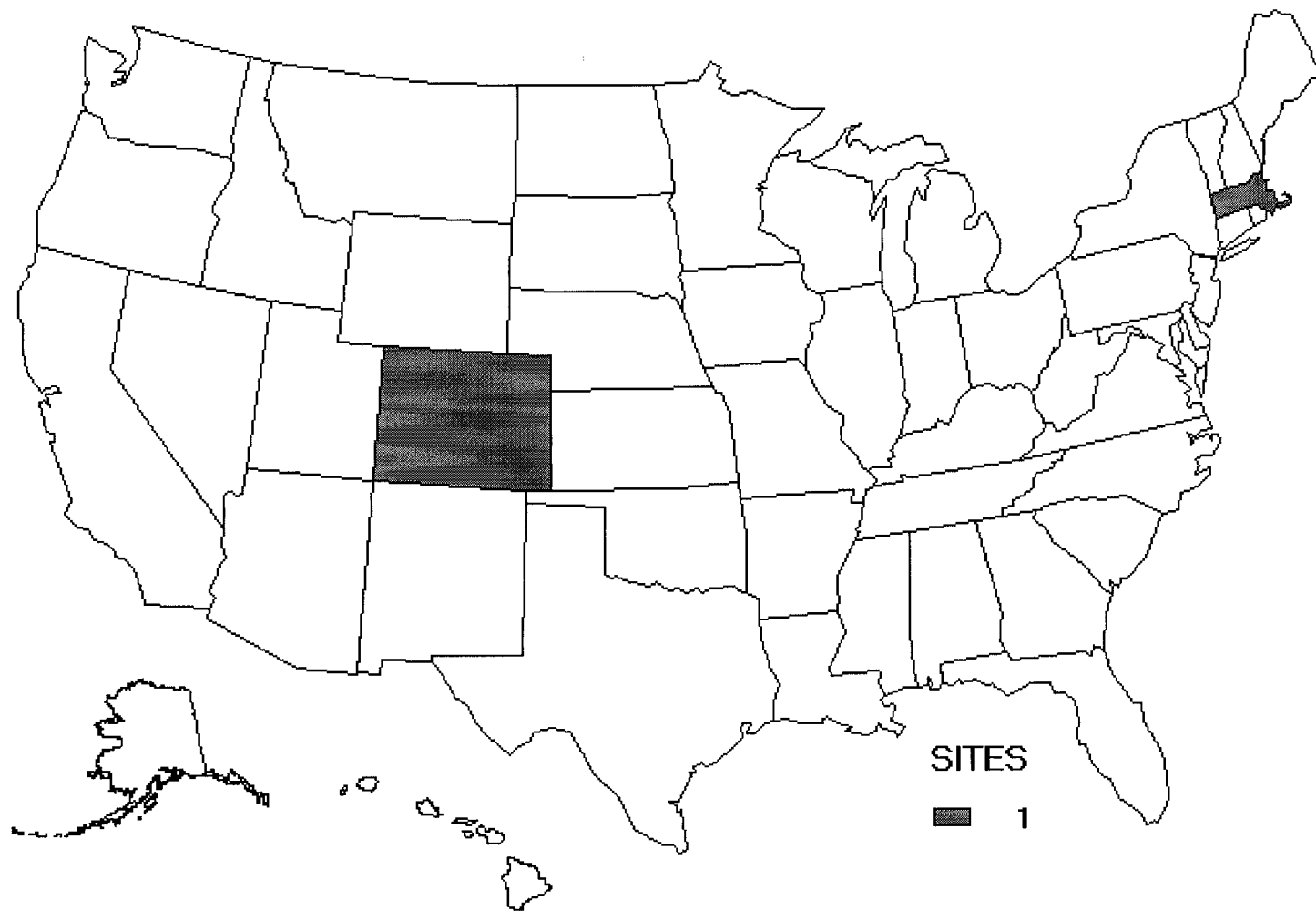
Derived from HazDat 1998

Figure 5-4. Frequency of NPL Sites with Mercurous Chloride Contamination



Derived from HazDat 1998

Figure 5-5. Frequency of NPL Sites with Dimethylmercury Contamination



Derived from HazDat 1998

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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

Table 5-1. Releases to the Environment from Facilities That Manufacture or Process Mercury

STATE ^b	CITY	FACILITY	Reported amounts released in pounds per year ^a							TOTAL ENVIRONMENT ^d
			AIR ^c	WATER	LAND	UNDERGROUND INJECTION	POTW TRANSFER	OFF-SITE WASTE TRANSFER		
AL	MUSCLE SHOALS	OCCIDENTAL CHEMICAL CORP.	1,069	24	0	0	0	539	1,632	
DE	NEW CASTLE	OCCIDENTAL CHEMICAL CORP.	1,110	16	0	0	0	4,337	5,463	
GA	AUGUSTA	OLIN CHLOR-ALKALI PRODS.	1,317	7	0	0	0	7,013	8,337	
IA	MASON CITY	ALEXANDER MFG. CO.	1	0	0	0	5	0	6	
IL	FREEPORT	MICRO SWITCH	4	0	0	0	0	2,500	2,504	
IL	ROCKFORD	VALSPAR CORP.	5	0	0	0	5	760	770	
IN	EDINBURGH	UNITED TECHS. AUTOMOTIVE INC.	5	0	0	0	0	2,250	2,255	
IN	ELKHART	DURAKOOL INC.	5	0	0	0	0	0	5	
IN	ELKHART	HERMASEAL CO.	5	0	0	0	0	0	5	
KS	DE SOTO	KOCH SULFUR PRODS. CO.	0	0	0	0	0	5	5	
KY	CALVERT CITY	BF GOODRICH CO.	1,200	250	0	0	0	2,000	3,450	
KY	LOUISVILLE	DU PONT	0	0	0	0	0	1,063	1,063	
LA	GEISMAR	BORDEN CHEMICALS & PLASTICS	0	17	0	9	0	13,121	13,147	
LA	LAKE CHARLES	PPG IND. INC.	1,230	22	0	0	0	73	1,325	
LA	PLAQUEMINE	DOW CHEMICAL CO.	20	0	0	0	0	0	20	
LA	SAINT GABRIEL	PIONEER CHLOR ALKALI CO. INC.	1,204	23	0	0	0	8,752	9,979	
ME	ORRINGTON	HOLTRACHEM MFG.	351	6	1	0	0	2,453	2,811	
MI	JACKSON	ELM PLATING CO.	5	0	0	0	5	10	20	
MI	ROMULUS	KERR CORP.	10	0	0	0	0	5,599	5,609	
NC	RIEGELWOOD	HOLTRACHEM MFG. CO. L.L.C.	1,446	11	0	0	0	104	1,561	
NY	ALBANY	MERCURY REFINING CO. INC.	255	5	0	0	0	520	780	
OH	ASHTABULA	ASHTA CHEMICALS INC.	1,653	5	0	0	0	682	2,340	
OK	TULSA	SINCLAIR OIL CORP.	0	20	2	0	0	0	22	
PA	ALLENTOWN	ADVANCED ENVIRONMENTAL	0	0	0	0	0	255	255	
PA	HELLERTOWN	BETHLEHEM APPARATUS CO. INC.	5	0	0	0	0	0	5	
PA	MONACA	ZINC CORP. OF AMERICA	130	0	0	0	0	10,700	10,830	
TN	CHARLESTON	OLIN CORP.	1,294	40	534	0	0	0	1,868	
TX	DEER PARK	OCCIDENTAL CHEMICAL CORP.	1,040	6	0	0	0	3,343	4,389	
WA	BELLINGHAM	GEORGIA-PACIFIC WEST INC.	1,460	45	0	0	0	205	1,710	
WI	PORT EDWARDS	VULCAN MATERIALS CO.	1,143	4	0	0	0	98	1,245	
WV	NEW MARTINSVILLE	PPG IND. INC.	1,130	40	0	0	0	191	1,361	
TOTALS			17,097	541	537	9	15	66,573	84,772	

Source: TRI96 1998

^a Data in TRI are maximum amounts released by each facility^b Post office state abbreviations used^c The sum of fugitive and stack releases are included in releases to air by a given facility^d The sum of all releases of the chemical to air, land, and water, and underground injection wells; and transfers off-site by a given facility

POTW = publicly owned treatment works

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Table 5-2. Comparison of Environmental Releases of Mercury (pounds per year) from Facilities That Manufacture and Process Mercury Reported to the Toxics Release Inventory (TRI) in 1991, 1994, and 1996

Year	Air	Water	Land	Underground injection	POTW transfer	Off-site waste transfer	Total environmental releases
1991 ^a	21,288	681	5,294	9	314	619,310	646,896
1994 ^b	13,885	326	1,351	7	15	67,480	83,064
1996 ^c	17,097	541	537	9	15	66,573	84,772

^a Source: TRI91 1993

^b Source: TRI94 1996

^c Source: TRI96 1998

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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 Pacyna (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 methylmercury 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 μkg (ppb) and 0.3 to 8.3 μkg (ppb), respectively. Both inorganic and methylmercury were transported from the

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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/m³ 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.

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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^0), 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

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Table 5-3. Atmospheric Mercury Emission Inventory for the United States by Anthropogenic Source Type^a

Source type	Mg/yr ¹	% of total emissions	% mercury species		
			Hg ⁰	Hg ²⁺	Hg _p
Medical waste incineration	58.6	26	20	60	20
Municipal waste collection	49.8	22	20	60	2
Electric utility boilers (coal, gas, oil)	48.5	22	50	30	20
Non-utility power and heat generation	28.5	13	50	30	20
Non-ferrous metal smelting	8.7	4	85	10	5
Chloralkali factories	6.5	3	70	30	0
Other point sources	16.2	7	80	10	10
Area sources (e.g., dental amalgams, fluorescent lighting fixtures)	6.9	3	100	0	0
Total	223.7	100%	41%	41%	18%

^a Emission rates are specified in units of megagrams per year (Mg yr⁻¹)

Hg⁰ = elemental mercury vapor; Hg²⁺ = mercuric form; Hg_p = mercury associated with particulates

Source: Bullock 1997

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Table 5-4. Estimates of U.S. Mercury Emission Rates by Category

Source of mercury	1990–1993 Mg/yr ^a	1990–1993 tons/yr ^a	% of total inventory
Area sources	2.8	3.1	1.3
Flourescent lamp breakage	1.4	1.5	0.6
General laboratory use	0.7	0.8	0.3
Dental preparations and use	0.7	0.8	0.3
Mobile sources	b	b	d
Paint use	c	c	e
Agricultural burning	b	b	d
Landfills	b	b	d
Point sources	217.3	239.4	98.7
Combustion sources	186.9	205.9	84.9
Medical Waste Incinerators ^d	58.8	64.7	26.7
Municipal Waste Combustors	50	55	22.7
Utility boilers	46.5	51.3	21.2
Coal	(46.3) ^e	(51.0)	(21.0)
Oil	(0.23)	(0.25)	(0.1)
Natural gas	(0.002)	(0.002)	(0.0)
Commercial/industrial boilers	26.3	29.0	12.0
Coal	(20.7)	(22.8)	(9.4)
Oil	(5.5)	(6.0)	(2.5)
Residential boilers	3.2	3.5	1.4
Coal	(0.5)	(0.6)	0.2
Oil	(2.7)	(3.0)	(1.2)
Sewage Sludge Incinerators	1.7	1.8	0.7
Crematories	0.4	0.4	0.2
Wood-fired boilers ^h	0.3	0.3	0.1
Hazardous waste combusters	b	b	b

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Table 5-4. Estimates of U.S. Mercury Emission Rates by Category (continued)

Source of mercury	1990–1993 Mg/yr ^a	1990–1993 tons/yr ^a	% of total inventory
<i>Manufacturing sources</i>	29.1	32	13.2
Primary lead production	8.2	9.0	3.7
Secondary Hg production	6.7	7.4	3.1
Chlor-alkali production	5.9	6.5	2.7
Portland cement production	5.9	6.5	2.7
Primary copper production	0.6	0.7	0.3
Lime manufacturing	0.6	0.7	0.3
Electrical apparatus	0.42	0.46	0.2
Instruments	0.5	0.5	0.2
Carbon black production	0.23	0.25	0.1
Fluorescent lamp recycling	0.005	0.006	0.002
Batteries	0.02	0.02	0.0
Primary Hg production	b	b	b
Mercury compounds	b	b	b
Byproduct coke	b	b	b
Refineries	b	b	b
<i>Miscellaneous sources</i>	1.3	1.4	0.6
Geothermal power	1.3	1.4	0.6
Turf products	c	c	c
Pigments, oil, etc.	c	c	c
Total	220.1	242.5	100.0

^a Numbers do not add exactly because of rounding.

^b Insufficient information to estimate 1990 emissions.

^c Mercury has been phased out of use.

^d In the course of Medical Waste Incinerator rulemaking, with the receipt of new data, US EPA expects to revise the mercury emission estimate for Medical Waste incinerators downward.

^e Parentheses denote subtotal within a larger point source category.

^f Includes boilers only; does not include residential wood combustion (wood stoves).

Source: EPA 1996b

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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 LeMoyné, 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 additional 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).

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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 $\mu\text{g}/\text{kg}$ (ppb) and 0.3–8.3 $\mu\text{g}/\text{kg}$ (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

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Table 5-5. Estimated Discards of Mercury in Products in Municipal Solid Waste (in tons^a)

Products	Amount in tons ^b						
	1970	1975	1980	1985	1989	1995	2000
Batteries							
Alkaline	4.1	38.4	158.2	352.3	419.4	41.6	0.0
Mercuric oxide	301.9	287.8	266.8	235.2	196.6	131.5	98.5 ^c
Others	4.8	4.7	4.5	4.5	5.2	3.5	0.0
Subtotal batteries	310.8	330.9	429.5	592.0	621.2	176.6	98.5
Electric lighting							
Fluorescent lamps	18.9	21.5	1.1	0.7	0.8	1.0	11.6 ^d
High intensity lamps	0.2	0.3	23.2	27.9	26.0	14.7	1.2
Subtotal lighting	19.1	21.8	24.3	28.6	26.7	15.7	12.6
Paint residues	30.2	37.3	26.7	31.4	18.2	2.3	0.5
Fever thermometers	12.2	23.2	25.7	32.5	16.3	16.9	16.8
Thermostats	5.3	6.8	7.0	9.5	11.2	8.1	10.3
Pigments	32.3	27.5	23.0	25.2	10.0	3.0	1.5
Dental uses	9.3	9.7	7.1	6.2	4.0	2.9	2.3
Special paper coating	0.1	0.6	1.2	1.8	1.0	0.0	0.0
Mercury light switches	0.4	0.4	0.4	0.4	.04	1.9	1.9
Film pack batteries	2.1	2.3	2.6	2.8	0.0	0.0	0.0
Subtotal other sources	91.8	107.8	83.7	109.8	61.1	35.1	33.3
Total discards	421.7	460.5	537.5	730.4	709.0	227.4	144.4

^a EPA (1992a) (except fluorescent lamps estimates)

^b Discards before recovery, 1 ton equals 2,000 pounds

^c The estimates for the years 1995 and 2000 do not reflect recent state, Federal, or battery manufacturers' efforts to reduce the mercury content of batteries. Since 1992, several states have restricted mercury use in batteries and/or banned the sale of mercuric oxide batteries. Federal legislation to restrict mercury use in batteries is pending. The battery industry has eliminated mercury as an intentional additive in alkaline batteries, except in button cells.

^d The estimated contribution of mercury from fluorescent lamps disposal to MSW was calculated based on industry estimates of a 4% growth rate in sales in conjunction with a 53% decrease in mercury content between 1989–1995, and a further 34% decrease in mercury content by the year 2000 (to 15 mg mercury per 4-foot fluorescent lamp (National Electric Manufacturers Association (1995).

Source: EPA 1996b

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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.

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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

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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 $\mu\text{g}/\text{m}^2/\text{year}$ of mercury deposited) is absorbed to the soil and does not leach down into the groundwater (Krabbenhof 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 $\mu\text{g}/\text{L}$ (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

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0.001 mg/m³ 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 reabsorbed 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 (HgCl₂ and CH₃HgCl) results in high

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Table 5-6. Bioconcentration of Various Mercury Compounds by Freshwater and Saltwater Organisms

Species	Tissue	Chemical	Duration (days)	Bioconcentration factor ^a
Freshwater species				
<u>Mercury (II)</u>				
Rainbow trout <i>Salmo gairdneri</i>	Whole body	Mercuric chloride	60	1,800
Fathead minnow <i>Pimephales promelas</i>	Whole body	Mercuric chloride	287	4,994 ^b
<u>Organomercury compounds</u>				
Rainbow trout <i>Salmo gairdneri</i>	Whole body	Methylmercuric chloride	60	11,000
Rainbow trout <i>Salmo gairdneri</i>	Whole body	Methylmercuric chloride	75	85,700
Brook trout <i>Salvelinus fontinalis</i>	Muscle	Methylmercuric chloride	273	11,000–33,000
Brook trout <i>Salvelinus fontinalis</i>	Whole body	Methylmercuric chloride	273	10,000–23,000
Brook trout <i>Salvelinus fontinalis</i>	Muscle and whole body	Methylmercuric chloride	756	12,000
Fathead minnow <i>Pimephales promelas</i>	Whole body	Methylmercuric chloride	336	44,130–81,670
Saltwater species				
<u>Mercury (II)</u>				
Eastern oyster (adult) <i>Crassostrea virginica</i>	Soft parts	Mercuric chloride	74	10,000
American lobster (adult) <i>Homarus americanus</i>	Soft parts	Mercuric chloride	30	129
<u>Organomercury compounds</u>				
Eastern oyster (adult) <i>Crassostrea virginica</i>	Soft parts	Methylmercuric chloride	74	40,000
Eastern oyster (adult) <i>Crassostrea virginica</i>	Soft parts	Phenylmercuric chloride	74	40,000

^a Results are based on the concentration of mercury, not the concentration of the mercury compound to which the animal was exposed.

^b From concentrations that caused adverse effects in a life-cycle test

Source: ASTER 1997

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Table 5-7. Comparison of the Biomagnification of Methylmercury and Inorganic Mercury in a Freshwater Food Chain (Little Rock Lake)

Medium or trophic level	Methylmercury	Inorganic mercury	% Methylmercury
Water	1	10	10
Phytoplankton	10^5	$10^{5.7}$	15
Zooplankton	$10^{5.5}$	$10^{5.9}$	30
Fish	$10^{6.5}$	10^5	95

Source: Watras and Bloom 1992

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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 *Thalassiosira 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 $\mu\text{g g}^{-1}$ dry weight in the plant/ $\mu\text{g mL}^{-1}$ 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

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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., dimethylmercury) 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;

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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

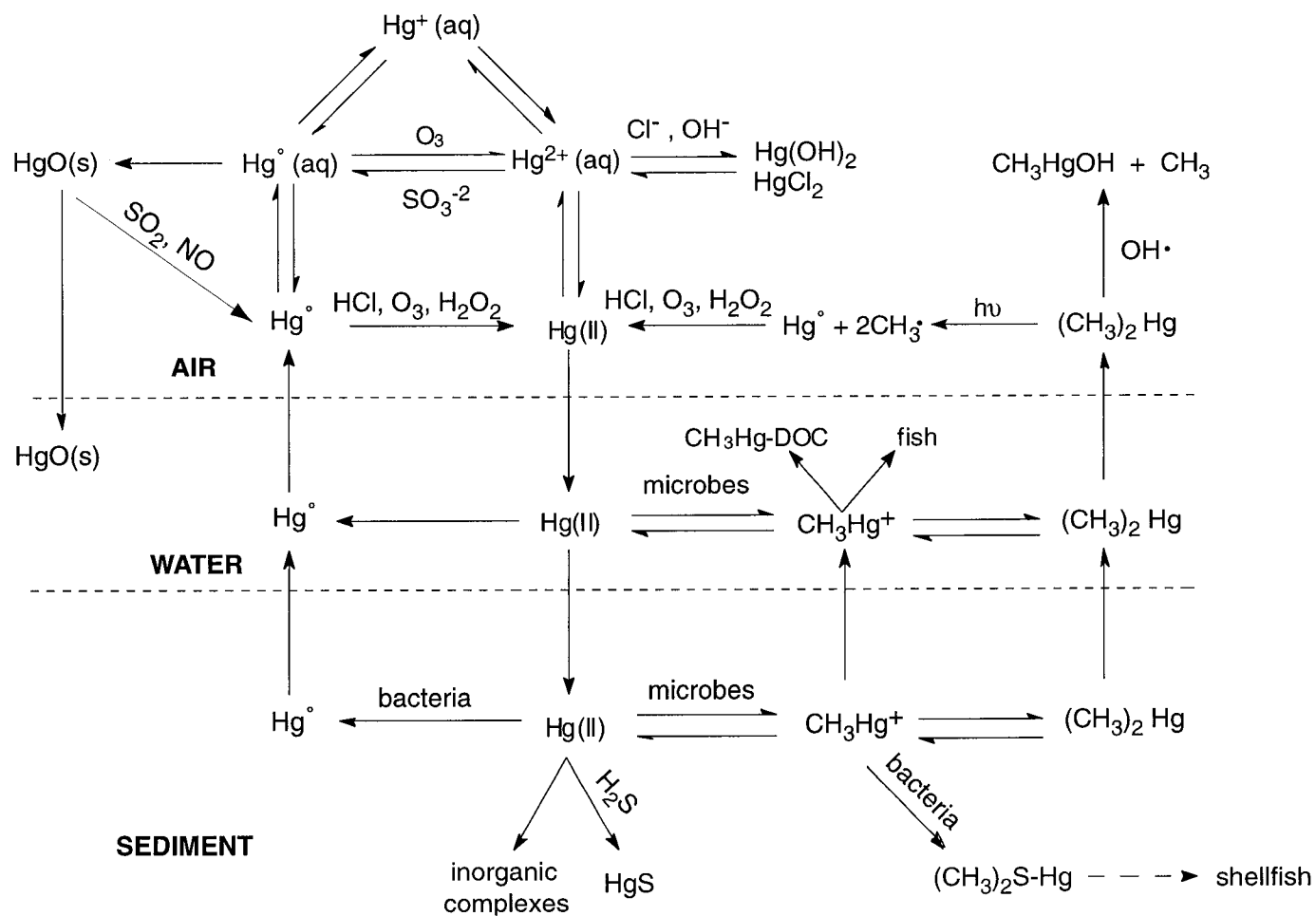
The primary form of atmospheric mercury, metallic mercury vapor (Hg^0), is oxidized by ozone to other forms (e.g., Hg^{+2}) and is removed from the atmosphere by precipitation (Brosset and Lord 1991). The oxidation/reduction of mercury with dissolved ozone, hydrogen peroxide, hypochlorite entities, or 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 ($\text{Hg}[\text{OH}]^2$), 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

Figure 5-6. Transformation of Mercury in Air, Water, and Sediment



Dashed lines represent the boundary between environmental compartments.

aq = associated with aqueous; DOC = dissolved organic carbon; s = solid

Source: Stern et al. 1996

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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).

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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).

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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 ($0.3 \mu\text{g}/\text{m}^3$) was found to be significantly higher ($p < 0.0001$) in the exposed homes (range, not detectable to $1.5 \mu\text{g}/\text{m}^3$) than in the unexposed homes (range, not detectable to $0.3 \mu\text{g}/\text{m}^3$). Among the exposed homes, there were 7 in which paint containing $< 200 \text{ mg/L}$ had been applied. In these homes, the median air mercury concentration was $0.2 \mu\text{g}/\text{m}^3$ (range, not detectable to $1 \mu\text{g}/\text{m}^3$). Six exposed homes had air mercury concentrations $> 0.5 \mu\text{g}/\text{m}^3$. 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 $< 200 \text{ mg Hg/L}$ (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 $0.92 \mu\text{g}/\text{m}^3$ (ATSDR 1990).

Ambient air concentrations of mercury have been reported to average approximately $10\text{--}20 \text{ ng}/\text{m}^3$, 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 $2\text{--}6 \text{ ng}/\text{m}^3$ and $0.01\text{--}0.1 \text{ ng}/\text{m}^3$, respectively (Brosset and Lord 1991). Higher levels ($10\text{--}15 \mu\text{g}/\text{m}^3$) have been detected near point emission sources, such as mercury mines, refineries, and agricultural fields treated with mercury fungicides. Atmospheric

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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 $2\text{--}6 \text{ ng/m}^3$ and particulate mercury at $0.002\text{--}0.06 \text{ ng/m}^3$ 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, $14\text{--}130 \text{ ng/L}$). 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 ($>2.5 \mu\text{m}$) 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 $15\text{--}1,200 \text{ pg/m}^3$. 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 ($<2.5 \mu\text{m}$ and $>2.5 \mu\text{m}$) at the rural site were 0.3 ng/m^3 (range, $<0.1\text{--}0.9 \text{ ng/m}^3$) and 0.2 ng/m^3 (range, $<0.1\text{--}0.5 \text{ ng/m}^3$), respectively, as compared to 1.0 ng/m^3 (range, $<0.1\text{--}0.7 \text{ ng/m}^3$) and

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0.5 ng/m³ (range, <0.1–1.5 ng/m³), respectively, in Chicago and 0.7 ng/m³ (range, <0.1–20 ng/m³) and 0.5 ng/m³ (range, <0.1–1.5 ng/m³), 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/m³ versus 2.0 ng/m³). Furthermore, a diurnal pattern was observed in the vapor phase mercury levels measured at the Chicago site. The average concentration (ng/m³) 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/m³). Particulate phase mercury concentrations were also higher at the Chicago site than at the South Haven site (98 pg/m³ versus 19 pg/m³). 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/m³; range, 1.2–4.2 ng/m³), and the concentrations were consistent across all seasons. Particulate phase mercury concentrations averaged 11 pg/m³, 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/m³. These values decreased to 0.006 to 0.071 µg/m³ by 1990, while background levels near the facility remained at 0.006 µg/m³. 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/m³) with the exception of one station. Mean mercury levels at the control site ranged from 5 to 6 µg/m³, while levels at the on-site stations were 6–11, 11–143, 68–174, 71–109, and 4–46 µg/m³. Mean

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particulate mercury levels were $0.00003 \mu\text{g}/\text{m}^3$ at the control site, compared with mean concentrations at the on-site stations ranging from 0.00006 to $0.00024 \mu\text{g}/\text{m}^3$ (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 $200 \text{ ng}/\text{L}$ (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 $15.2 \text{ ng}/\text{L}$ (ppt). Mercury concentrations in precipitation collected in Minnesota during 1988 and 1989 averaged $18 \text{ ng}/\text{L}$ (ppt) for an average annual mercury deposition of $15 \mu\text{g}/\text{m}^2$ (Glass et al. 1991). Antarctic surface snow contained a mean mercury concentration of less than $1 \text{ pg}/\text{g}$ (ppt) (Dick et al. 1990). In Ontario, Canada, mercury present in precipitation at an average concentration of $10 \text{ ng}/\text{L}$ (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 $7.57 \text{ ng}/\text{L}$ (ppt) in February 1992 to $17.4 \text{ ng}/\text{L}$ (ppt) in April 1992. Burke et al. (1995) reported that the average concentration of mercury in precipitation samples measured over Lake Champlain was $8.3 \text{ ng}/\text{L}$ (ppt) for the sampling year, and the average amount of mercury deposited per precipitation event was $0.069 \mu\text{g}/\text{m}^2$. 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 $0.020 \text{ ng}/\text{L}$ (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 $5 \text{ ng}/\text{L}$ (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 $500 \text{ ng}/\text{L}$ (ppt) (Glass et al. 1990). Water samples from lakes and rivers in the Ottawa,

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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 Babiarez 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).

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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 $\mu\text{g/g}$ [ppm]) than sediments at lower sediment levels (0.04–0.07 $\mu\text{g/g}$ [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 $\mu\text{g/L}$ (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 $\mu\text{g/g}$ (ppm) (mean concentrations ranged from 0.05 to 1.61 $\mu\text{g/g}$ [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

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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

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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 $\mu\text{g/L}$ [6 ppb]).

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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

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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).

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Table 5-8. Mercury Concentrations (ppm) for Largemouth Bass Collected in Various States Throughout the United States (1990–1995)

State	Number of fish	Minimum	Mean	Maximum
Alabama	914	0.100	0.393	1.630
Arizona	35	0.700	1.369	2.620
Arkansas	1190	0.030	0.675	3.170
California	537	0.020	0.305	1.800
Delaware	14	0.060	0.101	0.200
District of Columbia	11	0.037	0.153	0.458
Florida	2008	0.020	0.642	4.360
Georgia	968	0.010	0.262	1.650
Illinois	305	0.010	0.018	0.880
Iowa	38	0.080	0.189	0.480
Kentucky	120	0.124	0.581	1.460
Louisiana	452	0.001	0.391	1.883
Mississippi	606	0.090	0.647	2.630
Nebraska	182	0.080	0.343	0.920
New Hampshire	35	0.210	0.573	1.400
New York	53	0.050	0.462	0.950
North Carolina	1569	0.020	0.532	3.600
Ohio	56	0.001	0.988	1.400
Oregon	140	0.030	0.332	0.980
Pennsylvania	139	0.090	0.560	2.850
South Carolina	505	0.190	0.992	3.330
Tennessee	64	0.100	0.255	0.830
Texas	58	0.030	0.190	0.460
Washington	20	0.024	0.137	0.350
Wisconsin	346	0.050	0.369	1.500

Tissue concentrations shown in **bold type** exceed the FDA action level of 1 ppm

Source: EPA 1997b

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Table 5-9. Mercury Concentrations (ppm) for Channel Catfish Collected in Various States Throughout the United States (1990–1995)

State	Number of fish	Minimum	Mean	Maximum
Alabama	702	0.100	0.214	0.660
Arkansas	114	0.010	0.473	2.570
Delaware	19	0.020	0.050	0.133
District of Columbia	17	0.055	0.091	0.240
Georgia	658	0.010	0.081	1.110
Indiana	112	0.050	0.178	0.780
Iowa	323	0.030	0.104	0.410
Kansas	56	0.020	0.107	0.220
Louisiana	76	0.001	0.111	0.732
Maryland	157	0.006	0.029	0.179
Mississippi	157	0.040	0.272	2.100
Missouri	198	0.002	0.052	0.350
Nebraska	238	0.001	0.099	0.450
New Jersey	21	0.050	0.163	0.767
New Mexico	78	0.100	0.297	1.800
Oklahoma	171	0.100	0.186	0.540
South Carolina	42	0.250	0.345	1.610
Tennessee	138	0.100	0.173	0.650
Texas	44	0.030	0.148	0.830
West Virginia	65	0.030	0.139	1.583

Tissue concentrations shown in **bold type** exceed the FDA action level of 1 ppm

Source: EPA 1997b

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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,

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Table 5-10. Combined Data on Mercury Concentrations in Selected Fish Species Sampled in the Northeast^a

Species	Number of samples ^b	Mean Hg concentration	Minimum–maximum Hg ^c concentration range (ppm)
Largemouth bass	1,019	0.51	0– 8.94
Smallmouth bass	738	0.53	0.08– 5.0
Yellow perch	1,346	0.40	0– 3.15
Eastern chain pickerel	157	0.63	0– 2.81
Lake trout	877	0.32	0– 2.70
Walleye ^d	257	0.77	0.10– 2.04
Brown bullhead	421	0.20	0– 1.10
Brook trout	200	0.26	0–0.98

^a Northeastern states include ME, VT, NH, MA, RI, CT, NY, NJ.

^b In some cases, states reported an average of values from a given location. Thus, the number of samples indicated may not represent the number of individual fish sampled.

^c Maximum tissue concentrations shown in **bold type** exceed the FDA action level of 1 ppm

^d Walleye data are from New York State only and may not be representative of walleye mercury concentrations in other parts of the Northeast.

Source: NESCAUM 1998

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possibly as a result of the increased bioavailability of organic mercury produced by aquatic microorganisms, 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 $\mu\text{g/g}$ (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 $\mu\text{g/g}$ (ppm) (wet weight) and ranged from 0.06 to 2.87 $\mu\text{g/g}$ (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 $\mu\text{g/g}$) 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 $\mu\text{g/g}$) 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 $\mu\text{g/g}$ (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 $\mu\text{g/g}$ (ppm) dry weight in oysters (*Crassostrea virginica*), 0.28 to 0.41 $\mu\text{g/g}$ (ppm) in the Hawaiian oyster (*Ostrea sandwichensis*), 0.05 to 0.47 $\mu\text{g/g}$ (ppm) in the blue mussel (*Mytilus edulis*), and 0.04 to

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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 walrus 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

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Table 5-11. Total Mercury Concentrations in Tissues of Marine Mammals in Alaska and Canada

Species	Date collected	Muscle concentration (µg/g, wet weight)	Liver concentration (µg/g, wet weight)	Number	Source
Bearded seal		0.200±0.0150		7	Galster 1971
Bearded seal			1.910±1.200	4	Galster 1971
Pacific Alaska walrus		0.020±0.005		6	Galster 1971
Pacific Alaska walrus			0.490±0.100	7	Galster 1971
Polar bear	1972	0.040±0.014		12	Lentfer 1976
Polar bear	1972	0.040±0.260		4	Lentfer 1976
Polar bear	1972		4.800±1.460	9	Lentfer 1976
Polar bear	1972		3.920±1.280	16	Lentfer 1976
Beluga whale	1977	2.120±1.150		11	Muir et al. 1992
Beluga whale	1977		30.60±20.50	8	Muir et al. 1992
Ringed seal	1972	0.230±0.110		13	Smith and Armstrong 1975
Ringed seal	1972		1.000±1.160	13	Smith and Armstrong 1975
Ringed seal	1972-73	0.720±0.330		83	Smith and Armstrong 1975
Ringed seal	1972-73		27.50±30.10	83	Smith and Armstrong 1975
Ringed seal	1976	0.910±0.380		27	Smith and Armstrong 1975
Ringed seal	1976		16.10±13.80	27	Smith and Armstrong 1975
Ringed seal	1976	0.080±0.070		37	Smith and Armstrong 1975
Ringed seal	1976		0.320±0.800	36	Smith and Armstrong 1975
Ringed seal	1976	0.310±0.170		33	Smith and Armstrong 1975
Ringed seal	1976		3.760±3.420	33	Smith and Armstrong 1975
Bearded seal	1973	0.530±0.350		3	Smith and Armstrong 1975
Bearded seal	1973		143.0±170.0	6	Smith and Armstrong 1975
Bearded seal	1974	0.090±0.040		55	Smith and Armstrong 1975
Bearded seal	1974		26.20±26.10	56	Smith and Armstrong 1975

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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 $\mu\text{g/g}$ (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,

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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 of 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.

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(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)

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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 54°C. The degreased material was then placed in an aqueous HCl solution, shaken, and centrifuged. The supernatant was then filtered through a 0.45 µm 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

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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.

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Table 5-12. Estimated Average Daily Intake and Retention of Total Mercury and Mercury Compounds in the General Population

Source of exposure	Elemental mercury vapor	Inorganic mercury compounds	Methylmercury
Air	0.030 (0.024)	0.002 (0.001)	0.008 (0.0064)
Food			
Fish	0	0.600 (0.042)	2.4 (2.3)
Non-fish	0	3.6 (0.25)	0
Drinking water	0	0.050 (0.0035)	0
Dental amalgams	3.8–21 (3–17)	0	0
Total	3.9–21 (3–17)	4.3 (0.3)	2.41 (2.31)

Note: Values given are the estimated average daily intake (in $\mu\text{g}/\text{day}$) for adults in the general population who are not occupationally exposed to mercury; the figures in parentheses represent the estimated amount retained in the body of an adult.

Source: WHO 1990, 1991

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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 $\mu\text{g}/\text{day}$ for infants ages 6–11 months, 1.3 $\mu\text{g}/\text{d}$ for 2-year-old children, 2.9 $\mu\text{g}/\text{day}$ for females ages 25–30, and 3.9 $\mu\text{g}/\text{day}$ 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 ($\mu\text{g}/\text{day}$) for 78,882 adult females and 38,075 adult males in 1990 was 8.2 $\mu\text{g}/\text{day}$ (range, 0.37–203.5 $\mu\text{g}/\text{day}$) for females and 8.6 $\mu\text{g}/\text{day}$ (range, 0.22–165.7 $\mu\text{g}/\text{day}$) 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).

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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 than 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

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Table 5-13. Mercury Concentrations in the Top 10 Types of Fish Consumed by the U.S. Population

Fish	Mercury concentration (ppm) ^a	Comments
Tuna	0.206	Mercury content is the average of the mean concentrations in 3 tuna species: Albacore tuna (0.264 ppm) Skipjack tuna (0.136 ppm) Yellowfin tuna (0.218 ppm) The FDA measured the methylmercury concentration in 220 samples of canned tuna in 1991; the average amount of methylmercury measured was 0.17 µg/g and the range was <1–0.75 µg/g) (Yess 1993).
Shrimp	0.047	Mercury content is the average of the mean concentrations in 7 shrimp species: Royal red (0.074 ppm) White (0.054 ppm) Brown (0.048 ppm) Ocean (0.053 ppm) Pink (0.031 ppm) Pink northern (0.024 ppm) Alaska (sidestripe) (0.042 ppm)
Pollack	0.150	The Pesticide and Chemical Contaminant Data Base for the FDA (1991/1992) reports the methylmercury concentration in pollack in commerce as 0.04 ppm
Salmon	0.035	Mercury content is the average of the mean concentrations in 5 salmon species: Pink (0.019 ppm) Chum (0.030 ppm) Coho (0.038 ppm) Sockeye (0.027 ppm) Chinook (0.063 ppm)
Cod	0.121	Mercury content is the average of the mean concentrations in 2 cod species: Atlantic (0.114 ppm) Pacific (0.127 ppm)
Catfish	0.088 0.160	Two data sets were collected from U.S. freshwater sources: Bahnick et al (1994): channel, largemouth, rock, striped, and white Lowe et al. (1985): channel and flathead. Neither survey included farm-raised catfish, which is the type predominantly consumed in the U.S. Mercury content of farm-raised catfish may be significantly different from feral catfish.
	0.020	The Pesticide and Chemical Contaminant Data Base for USFDA (1991/1992) reports the methylmercury concentration in catfish as 0.02 ppm.
Clam	0.023	Mercury content is the average of the mean concentrations in 4 clam species: Hard (quahog) (0.034 ppm) Pacific littleneck (0 ppm) Soft (0.027 ppm) Geoduck (0.032 ppm)

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Table 5-13. Mercury Concentrations in the Top 10 Types of Fish Consumed by the U.S. Population (continued)

Fish	Mercury concentration (ppm) ^a	Comments
Flounder (flatfish)	0.092	Mercury content is the average of the mean concentrations in 9 flounder species: Gulf (0.1487 ppm) Summer (0.127 ppm) Southern (0.078 ppm) Four-spot (0.090 ppm) Windowpane (0.151 ppm) Arrowtooth (0.020 ppm) Witch (0.083 ppm) Yellowtail (0.067 ppm) Winter (0.066 ppm)
Crab	0.117	Mercury content is the average of the mean concentrations in 5 crab species: Blue (0.140 ppm) Dungeness (0.183 ppm) King (0.070 ppm) Tanner (<i>C. opilio</i>) (0.088 ppm) Tanner (<i>C. bairdi</i>) (0.102 ppm)
Scallop	0.042	Mercury content is the average of the mean concentrations in 4 scallop species: Sea (smooth) (0.101 ppm) Atlantic bay (0.038 ppm) Calico (0.026 ppm) Pink (0.004 ppm)

^a All concentrations determined on a wet weight basis

Source: EPA 1996e

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Table 5-14. Fish Consumption Rates of Various Populations Including General Population and Recreational and Subsistence Fishers

Consumption rate (g/day)	Description	Population	Reference
170	95 %ile (adult)	Umatilla, Nez Perce, Yakima, and Warm Springs Tribes of Columbia River Basin Washington	CRITFC 1994
140	90 %ile (adult)	Subsistence fisher (default value)	EPA 1995k
109	Mean (adult)	Native Alaskans in 11 separate communities	Nobmann et al. 1992
63	95 %ile (adult)	Wisconsin anglers (10 counties) includes both recreationally and commercially caught fish	Fiore 1989
59	Mean (adult)	Umatilla, Nez Perce, Yakima, and Warm Springs Tribes of Columbia River Basin Washington	CRITFC 1994
37	95 %ile (adult)	Wisconsin anglers (10 counties) includes only recreationally caught fish	Fiore 1989
34	75 %ile (adult)	Wisconsin recreational anglers	Fiore 1989
30	Mean (adult)	Recreational fisher (default value)	EPA 1995k
28	Mean (adult)	New York anglers	Connelly 1990
26	Mean (adult)	Wisconsin anglers (10 counties) includes both recreationally and commercially caught fish	Fiore 1989
20	Mean (child 5 years and younger)	Umatilla, Nez Perce, Yakima, and Warm Springs Tribes of Columbia River Basin Washington	CRITFC 1994
12	Mean (adult)	Wisconsin anglers (10 counties) includes only recreationally caught fish	Fiore 1989
6.5	Mean (adults)	General U.S. population	EPA 1995k

Source: EPA 1996e

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Table 5-15. Estimated U.S. Population Consuming Fish^a, Excluding Alaska and Hawaii

Population group	Estimated number of persons
Total U.S. population	247,052,000
Total female population ages 15–44 years	58,222,000
Total population of children aged <15 years	53,463,000
Percentage of respective group reporting fish consumption during the CSFII 3-day dietary survey period in 1989–1991 ^b	
Total U.S. population	30.9%
Total female population ages 15–44 years	30.5%
Total population of children aged <15 years	24.9%
Number of persons predicted to consume fish based on percentage consuming fish in CSFII 3-day dietary survey period in 1989–1991	
Total U.S. population	76,273,000
Total female population ages 15–44 years	17,731,000
Total population of children aged <15 years	13,306,000
Number of persons in highest 5% of estimated population that consumes fish ^c	
Total U.S. population	3,814,000
Total female population ages 15–44 years	887,000
Total population of children aged <15 years	665,000
Estimated number of adult pregnant women in highest 5% of estimated population that consumes fish	
Number of females aged 15–44 years times percentage of women pregnant in given year	84,300

^a Estimates based on the 1990 U.S. Census and the 1989-1991 Continuing Surveys of Food Intake by Individuals

^b Numbers of persons rounded to 3 significant figures

^c Persons who consumed an average 100 g or more of fish/day

Source: EPA 1996e

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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 feel 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.

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Dental amalgams may contain 43–54% elemental mercury (DHHS 1993). A single amalgam filling with an average surface area of 0.4 cm² 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

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Table 5-16. Estimates of Mean Daily Elemental Mercury Uptake from Dental Amalgam Restorations

Number of surfaces	Mercury ($\mu\text{g}/\text{day}$)	Reference
Not reported	27.0	Patterson et al. 1985
8-54	3.0	Langworth et al. 1988
13-48	1.7	Berglund 1990
1-16	19.8	Vimy and Lorscheider 1985
7	1.3	Snapp et al. 1989
8-54	3.0	Langworth et al. 1988
0-82	12.0	Skare and Engqvist 1994
		Clarkson et al. 1988 ^b
Not reported	17.5	Svare et al. 1981
1-16	2.9	Vimy and Lorscheider 1985
0.2-4.2 ^a	8.0	Abraham et al. 1984
Not reported	2.5	Patterson et al. 1985
		Mackert 1987 ^c
1-16	1.24	Vimy and Lorscheider 1985
		Weiner and Nylander 1995 ^d
1-75	≥ 27.0	Patterson et al. 1985
1-16	20.0	Vimy and Lorscheider 1985
1-16	1.2	Mackert 1987
Not reported	3-18	Clarkson et al. 1988
24-63	7-10	Aronsson et al. 1989
13-48	1.7	Berglund 1990
Not reported	3-17	WHO 1991
14 (avg)	≥ 1.3	Snapp et al. 1989
≥ 36	10-12	Jokstad et al. 1992

^a Occlusal surface area in cm^2

^b Clarkson's estimates based on the data from studies listed

^c Mackert's estimate based on the data from the study listed

^d Weiner and Nylander's estimate based on the data from the studies shown below

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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

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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

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Table 5-17. Total Mercury Concentrations in Human Breast Milk

Population	Year	Number of samples (% positive)	Total Hg content in whole milk (ppb) ^a	Reference
Minamata, Japan (contaminated seafood)	1968	–	63	Fujita and Takabatake 1977
Iraq (contaminated grain)	1972	44	<200 ^b	Bakir et al. 1973
Tokyo, Japan (urban population)	1974	34	3.6±2.2 (0.4–9.8)	Fujita and Takabatake 1977
Iowa, USA (general population without abnormal exposure)	1975	32 (44%)	0.9±0.23	Pitkin et al. 1976
Alaska, USA (coastal population) (interior population) (urban population)	1975	1155	7.6±2.7 3.2±0.8 3.3±0.5	Galster 1976
Madrid, Spain	1981	20 (100%)	9.5±5.5 (0.9-19)	Baluja et al. 1982
Sweden (15 women fish consumers)	1980s	NA	0.2–6.3	Skervfving 1988
Sweden (fish consumers with an average of 12 amalgam fillings)	1990s	30	0.6±0.4 ^c (0.1–2.0)	Oskarsson et al. 1996
Faroe Islands (88 women who consumed pilot whale meat)	1990s	100	median 2.45 maximum 8.7	Grandjean et al. 1995

^a Results are expressed as means ±S.D unless otherwise noted. Ranges are shown in parentheses.

^b Of the total mercury, 40% was inorganic mercury; 60% was methylmercury

^c Of the total mercury 51% was inorganic; 49% was organic mercury

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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 ($2 \mu\text{g}$ 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).

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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 $\mu\text{g Hg/g hair}$ 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 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 $\mu\text{g/g}$ (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

Table 5-18. Mercury Concentrations in Hair ($\mu\text{g Hg/g hair}$) from Residents of Various U.S. Communities

Population	Mean concentration (ppm)				Maximum concentration (ppm)				Reference
	Adults	Males	Females	Children	Adults	Males	Females	Children	
NY metropolitan area adults n=203 children n=280	0.77			0.67	14.0			11.3	Creason et al. 1978a
4 NJ communities (Ridgewood, Fairlawn, Matawan, and Elizabeth) adults n=117 children n=204	0.78			0.77	5.6			4.4	Creason et al. 1978b may have to drop
Birmingham AL and Charlotte NC adults n=282 children n=322	0.47			0.46	7.5			5.4	Creason et al. 1978c may have to drop
U.S. unidentified community males n=22 females n=16 adults n=24 adults n=31 adults n=24 adults n=79		2.7	2.6			6.2	5.5		Airey 1983b
LaJolla-San Diego CA males n=13 females n=13 adults n=8 adults n=17 adults n=5 adults n=30		2.4	2.7			6.2	5.5		Airey 1983b
Maryland adults n=11 adults n=11 adults n=11 adults n=33	1.8 1.5 2.3 1.9				3.8 3.9 4.5 4.5				Airey 1983b

Table 5-18. Mercury Concentrations in Hair ($\mu\text{g Hg/g hair}$) from Residents of Various U.S. Communities (continued)

Population	Mean concentration (ppm)				Maximum concentration (ppm)				Reference
	Adults	Males	Females	Children	Adults	Males	Females	Children	
Seattle WA									Airey 1983b
males n=9		3.3				5.6			
females n=3			2.2				4.1		
adults n=5	2.6				5.6				
adults n=3	1.5				2.1				
adults n=8	3.8				7.9				
adults n=16	3.0				7.9				

Nome AK									Lasora and Citterman 1991
females of child-bearing age n=80							15.2		

Seguim WA									Lasora and Citterman 1991
females of childbearing age n=7			0.70				1.5		

Florida Everglades									Fleming et al. 1995
adults that consumed local wildlife n=330	1.3				15.6				
Range	0.47-3.8	2.4-3.3	2.2-2.7	0.46-0.77	2.1-15.6	5.6-6.2	1.5-15.2	4.4-11.3	

Source: EPA 1996e

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(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 $\mu\text{g/g}$ (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 $\mu\text{g/g}$ (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 $\mu\text{g}/\text{m}^3$ (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 $\mu\text{g}/\text{L}$ (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 $\mu\text{mol}/\text{mol}$ 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

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Table 5-19. Total Mercury Levels in Exposed Workers and Controls

Number of subjects	Exposure group	Air ($\mu\text{g}/\text{m}^3$)	Blood (nmol/L)	Plasma (nmol/L)	Urine (nmol/mmol creatinine)	Serum (nmol/L)	Reference
26	Chloralkali workers	25–50	No data	48.0	16.0	No data	Barregard et al. 1991
26	Unexposed referents	No data	No data	7.5	1.3	No data	
89	Chloralkali workers	10–106	55	No data	14.3	45	Langworth et al. 1992b
75	Controls	No data	15	No data	1.1	4	

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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 $0.92 \mu\text{g}/\text{m}^3$ (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 $815 \mu\text{g}/\text{kg}$ (ppb) wet weight (range, $135\text{--}4,040 \mu\text{g}/\text{kg}$) in pituitary tissue of dental staff (7 dentists and 1 dental assistant), as compared to a median of $23 \mu\text{g}/\text{kg}$ (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 $1.1 \mu\text{mol}/\text{kg}$ (221 ppb) wet weight (range, $0.7\text{--}28 \mu\text{mol}/\text{kg}$ [$140\text{--}5,617$ ppb]) in the pituitary. the median mercury concentration in the pituitary of the controls was $0.11 \mu\text{mol}/\text{kg}$ (22 ppb) (range, $0.03\text{--}5.83 \mu\text{mol}/\text{kg}$ [$6\text{--}1,170$ ppb]).

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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 µg/m³; range, non-detectable to 4 µg/m³) than for nonpainters (non-detected; range, not detected to 3 µg/m³). 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.

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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 latex 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,

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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

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Table 5-20. Estimated Number of Workers Potentially Exposed to Mercury and Various Mercury Compounds in the Workplace

Mercury compounds	Number of workers	Number of female workers
Mercury (metallic)	71,933	23,826
Mercury chloride	45,492	18,717
Mercury acetate	6,063	2,770
Mercuric sulfide	98	—
Phenylmercuric acetate	28,347	5,150
Methylmercuric chloride	14	5

Source: RTECS 1998

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Table 5-21. Calculated Mercury Absorption from Air

Route (source of exposure)	Guideline	Air concentration ($\mu\text{g}/\text{m}^3$)	Exposure ($\mu\text{g}/\text{week}$)	Exposure ($\mu\text{g}/\text{day}$)
Lung (work) ^a	OSHA PEL; NIOSH REL	50	3000	429
Lung (work)	WHO	25	1500	214
Lung (home) ^b	EPA RfC	0.03	33.6	4.8
Skin (work) ^c	OSHA PEL; NIOSH REL	50	52	7.4

^a Work exposure assumes 8 hours per day, 5 days per week, ventilation rate of 15 m³ and no other mercury exposure

^b Home exposure to ambient air assumes 24 hours per day, 7 days per week, and ventilation rate 20 m³/day

^c Skin exposure excludes respiration exposure (Hursh et al. 1989)

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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

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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

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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];

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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 $8.5 \mu\text{m}^3$ when a vacuum cleaner was not in use and concentrations rose to $69 \mu\text{m}^3$ 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

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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 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.

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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 $\mu\text{g}/\text{kg}/\text{day}$, 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 $\text{ng}/\text{kg}/\text{day}$ (Clarkson 1990; Gunderson 1988). For 2-year-old children, the intake was estimated to be approximately 100 $\text{ng}/\text{kg}/\text{day}$ (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.

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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 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 $0.25 \mu\text{g}/\text{m}^3$ (range, $0.02\text{--}10 \mu\text{g}/\text{m}^3$), and the levels of mercury in the urine of the children averaged $25 \mu\text{g}/\text{L}$ (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 $0.92 \mu\text{g}/\text{m}^3$ (ATSDR 1990). Although protective clothing was used, work gloves, clothes, and

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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

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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 that 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 $\mu\text{g/L}$ (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).

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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 $\mu\text{g/L}$ (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,

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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

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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

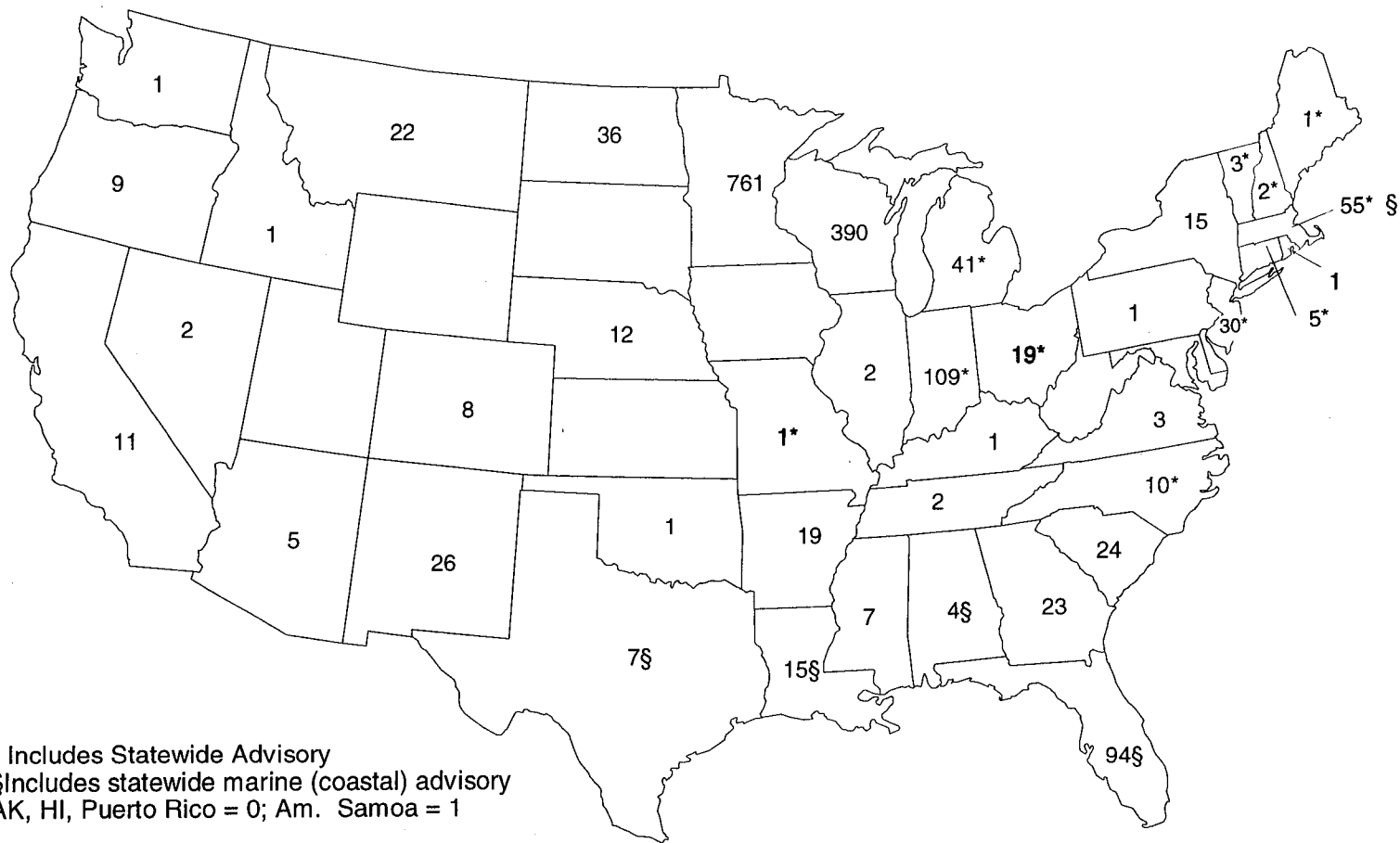
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protection of high-end fish consumers who routinely and repeatedly consume large quantities of fish from local bodies of water.

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 ($\mu\text{g/g}$ wet weight), but mean total mercury levels were

Figure 5-7. Listing of Fish and Wildlife Consumption Advisories Issued for Mercury



* Includes Statewide Advisory
 §Includes statewide marine (coastal) advisory
 AK, HI, Puerto Rico = 0; Am. Samoa = 1

Source: EPA 1998

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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

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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 $0.73 \mu\text{g}/\text{day-surface}$, with a standard deviation of $0.3 \mu\text{g}/\text{day-surface}$ and a “stimulation magnification factor” of 5.3, based on a weighed average enhancement of mercury vapor concentration following chewing, eating, or tooth brushing

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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 $\mu\text{g/L}$ [ppb]) were detected in 167 persons with dental restorations, compared to 0.000008–0.0001 $\mu\text{g/L}$ (ppb) (mean, 0.00006 $\mu\text{g/L}$ [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 $\mu\text{g/L}$ [ppb]) as compared to persons who did not eat fish and had no dental amalgams (0.2 ± 0.4 $\mu\text{g/L}$ [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

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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

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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 $\mu\text{g}/\text{m}^3$ less than 6 hours after painting, 10 $\mu\text{g}/\text{m}^3$ at 24 hours, and 6 $\mu\text{g}/\text{m}^3$ 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).

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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

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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

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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 from 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

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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

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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.

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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.

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Table 5-22. Ongoing Research Relevant to Human Exposure to Mercury

Investigator	Affiliation	Research description	Sponsor
Ford, TE	Harvard School of Public Health Boston, MA	Assessment of metal contamination and ecological implications	National Institute of Environmental Health Sciences
Myers, GJ	University of Rochester School of Medicine Rochester, NY	Child development following prenatal methylmercury exposure via fish diet	National Institute of Environmental Health Sciences
Clarkson, T	University of Rochester School of Medicine Rochester, NY	Health hazards of methylmercury	National Institute of Environmental Health Sciences
Pepper, IL	University of Arizona Tucson, AZ	Biodegradation within metal/organic contaminated soils	National Institute of Environmental Health Sciences
Fernando, Q	University of Arizona Tucson, AZ	Determination of toxic metal species with high energy ion beams	National Institute of Environmental Health Sciences
Schell, LM	State University of New York at Albany Rensselaer, NY	PCBs and well being of Mohawk children and youth--growth, development, cognition	National Institute of Environmental Health Sciences
Woods, JS	University of Washington Seattle, WA	Porphyrin profiles as biomarkers of trace metal exposure and toxicity	National Institute of Environmental Health Sciences
Janoff, EE	University of Washington Seattle, WA	Influence of dental amalgams on mercury and antibiotic resistant bacteria	National Institute of Dental Research
Owens, M	Sciences International Corporation McLean, VA	Dental amalgam study	National Institute of Dental Research
De Rouen, A	University of Washington Seattle, WA	Casa Pia study of dental amalgams in children	National Institute of Dental Research
Crawford, SL	New England Research Institutes, Inc. Watertown, MA	Health effects of dental amalgams in children	National Institute of Dental Research

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Table 5-22. Ongoing Research on Environmental Exposure to Mercury (cont.)

Investigator	Affiliation	Research description	Sponsor
Echeverria, D	Battelle Centers Public Health Research and Evaluation Seattle, WA	Neurologic effects of metallic mercury exposure in dental personnel	National Institute of Dental Research
Factor-Litvak, P	Columbia University New York, NY	Dental amalgams and neuropsychological function	National Institute of Dental Research
Barkay, T	Environmental Protection Agency Gulf Breeze, FL	Bioremediation of mercury in aquatic systems	US Department of Energy, Environmental Restoration and Waste Management
Lindberg, SE	Oak Ridge National Laboratory Environmental Sciences Division. Oak Ridge, TN	Atmosphere canopy interactions	US Department of Energy, Energy Research
Miller, JR Douglas, J	Indiana University Indianapolis, IN	Collaborative research: Transport and fate of mercury within the Carson River Valley	National Science Foundation, Division of Earth Sciences
Warwick, JJ Lechler, P Douglas, J	University of Nevada Reno, NV	Collaborative research: Transport and fate of mercury within the Carson River Valley	National Science Foundation, Division of Earth Sciences
Mason, RP Baier, RW	University of Maryland Solomons, MD	Abiotic and biotic mechanisms for mercury reduction in marine waters	National Science Foundation, Division of Ocean Sciences
Mason, RP Baier, RW	University of Maryland Solomons, MD	Biogeochemical cycling and air-sea exchange of mercury in the south Atlantic	National Science Foundation, Division of Ocean Sciences
Fitzgerald, WF Baier, RW	University of Connecticut Marine Sciences Storrs, CT	Biogeochemical cycling and air-sea exchange of mercury in the equatorial and south Atlantic	National Science Foundation, Division of Ocean Sciences
Lyons, WB Cameron, M	University of Alabama Tuscaloosa, AL	Collaborative research: Mercury biogeochemistry in a semi-arid aquatic ecosystem: Processes controlling	National Science Foundation, Division of Earth Sciences

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Table 5-22. Ongoing Research on Environmental Exposure to Mercury (cont.)

Investigator	Affiliation	Research description	Sponsor
Warwick, JJ Cameron, M	University of Nevada Reno, NV	Collaborative research: Mercury biogeochemistry in a semi-arid aquatic ecosystem: Processes controlling	National Science Foundation, Division of Earth Sciences
Hines, ME Cameron, M	University of New Hampshire Dept. of Biological Sciences Anchorage, AK	Collaborative research: Mercury biogeochemistry in a semi-arid aquatic ecosystem- Processes controlling	National Science Foundation, Division of Earth Sciences
Krabbenhoft, DP		Mercury accumulation, pathways, and processes	Department of Interior, US Geological Survey Water Resources Division.
Lent, RM		Chronology of mercury loading to Devils Lake, North Dakota, inferred from sediment core data	Department. of Interior, US Geological Survey Water Resources Division. North Dakota
Bianco, V Wendroff, A	Puerto Rican Family Institute, Queens, NY	Magico-religious mercury use in Hispanic communities	EPA/Office of Environmental Justice
Markowitz, M Ozuah, P	Montefiore Medical Center, Bronx, NY	Urinary levels of mercury in children exposed to mercury (background levels and from magico-religious exposures)	New York City Department of Health/ New York City Department of Mental Health
Moomey, M Hryhorczuk, D	Illinois Department of Public Health and Great Lakes Center for Occupational and Environmental Safety and Health	Identification of which ritual mercury uses result in the greatest exposure	EPA/Office of Environmental Justice

Source: FEDRIP 1998