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.

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 µg/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

Dietary Sources of Mercury. Galal-Gorchev (1993) analyzed dietary intakes of mercury from 14 countries, including the United States, between 1980 and 1988. This author reported that the contribution of fish to the total intake of mercury varied from a low of 20% in Belgium and the Netherlands to 35% in France, the United Kingdom, and the United States. The highest contribution of fish to mercury intake (85%) was reported for Finland. The author further pointed out (based on information from the Netherlands on levels of mercury contamination in a variety of foods) that although mercury was found at higher concentrations in fish (0.1 mg/kg [ppm]) than in other foods (0.01 mg/kg or less), higher consumption of cereals and meats render the contributions of these food groups to the total mercury intake about the same as that from fish. Therefore, the general assumption that fish is the main contributor to the intake of mercury may, at times, not be justified because of dietary habits of a given population (Galal-Gorchev 1993).

The FDA's Total Diet Study (April 1982–April 1984) estimated an average daily intake of mercury (total) based on measured levels and assumed trace amounts in foods to be representative of the "total diet" of the U.S. population (Gunderson 1988). Estimated daily exposures for mercury were 0.49 µg/day for infants ages 6–11 months, 1.3 µg/d for 2-year-old children, 2.9 µg/day for females ages 25–30, and 3.9 µg/day for males 25–30 years of age. Expressed on a per body weight basis, the intake for all age groups, except 2-year-old children, was approximately 50 ng/kg/day (Clarkson 1990; Gunderson 1988). For 2-year-old children, the intake was estimated to be approximately 100 ng/kg/day (assuming 50% of the fish intake was due to fish caught locally). More recently, MacIntosh et al. (1996) calculated average daily dietary exposure to mercury and 10 other contaminants for approximately 120,000 U.S. adults by combining data on annual diet, as measured by a food frequency questionnaire, with contaminant residue data for table-ready foods that were collected as part of the annual FDA Total Diet Study (1986–1991). The estimated mean dietary exposure (µg/day) for 78,882 adult females and 38,075 adult males in 1990 was 8.2 µg/day (range, 0.37–203.5 µg/day) for females and 8.6 µg/day (range, 0.22–165.7 µg/day) for males. Assuming a body weight of 65 kg for women and 70 kg for men, the daily intakes of mercury would be 126 ng/kg/day (range, 5.7–3,131 ng/kg/day) for women and 123 ng/kg/day (range, 3.1–2,367 ng/kg/day) for men respectively. These authors found that the coefficient of variation was 44% for mercury, indicating that the exposures to this chemical estimated for a given individual may be accurate to within approximately a factor of 2. Lack of data about the actual amount of food consumed accounted for 95% of the total uncertainty for mercury. Individual food items contributing most to the uncertainty of mercury measurements were canned tuna and other fish (MacIntosh et al. 1996).

The FDA currently has advice for consumers posted on the Internet that recommends that pregnant women and women of childbearing age, who may become pregnant, limit their consumption of shark and swordfish to no more that one meal per month (FDA 1998). This advice is given because methylmercury levels are much higher in these fish species than in the more commonly consumed species. Dietary practices immediately before pregnancy could also have a direct bearing on fetal exposure, particularly during pregnancy. The FDA states that nursing mothers who follow this advice, do not expose their infants to increased health risks from methylmercury (FDA 1998). The FDA further advises that persons other than pregnant women and women of child-bearing age limit their regular consumption of shark and swordfish (which typically contain methylmercury at approximately 1 ppm) to about 7 ounces per week (about one serving) to stay below the recommended maximum daily intake for methylmercury. For fish species with methylmercury levels averaging 0.5 ppm, regular consumption should be limited to 14 ounces (about 2 servings) per week. A summary of mercury concentrations in the top 10 types of fish consumed by the general U.S. population is presented in Table 5-13. There is a wide degree of variability in the amount of fish consumed in the diet by various subpopulations within the United States. Various ethnic groups, as well as recreational and subsistence fishers often eat larger amounts of fish than the general population and may routinely fish the same waterbodies (EPA 1995k). If these waterbodies are contaminated, these populations may consume a larger dose of mercury by virtue of the fact that they consume larger amounts of fish (from >30 g/day for recreational fishers to >100 g/day for subsistence fishers) with higher concentrations of mercury in their tissues than individuals in the general population that tend to consume smaller amounts (6.5 g/day) of supermarket-purchased fish that come from a variety of sources. Table 5-14 provides a summary of the amount of fish consumed daily by the general population, as compared to recreational and subsistence fishers, including some Native American tribal groups. Those individuals that consume greater than 100 g of fish per day are considered high-end consumers; they consume more than 10 times the amount of fish estimated to be consumed by members of the general population (6.5 g/day) (EPA 1995k).

Table 5-15 provides an summary of the estimated total number of persons in the U.S. population (excluding Alaska and Hawaii), the total female population of reproductive age (ages 15–44 years), and the total population of children (<15 years). Based on the percentage of people that reported eating fish during a 3-day dietary survey conducted from 1989 to 1991 as part of the Continuing Survey of Food Intake by Individuals (CSFII), the number of persons estimated to consume fish can be calculated. Using this method, more than 76 million people in the U.S. population eat fish; of these, more than 17 million females of reproductive age (15–44 years old) consume fish, and more than 13 million children (<15 years of age) eat

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)

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 *(C. opilio)* (0.088 ppm) Tanner *(C. bairdl)* (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

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

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

fish. In addition, estimates of the total number of persons in the high-end fish consumer group (subsistence fishers) have been calculated, as were estimates of the total number of adult women of reproductive age (15 to 44 years old) and children (<15 years old) in the high-end consumer group, i.e., those potentially at greatest risk of exposure (EPA 1996e). With respect to fish consumers, more than 3.8 million are high-end consumers (>100 grams of fish/day), and of these, it is estimated that more than 887,000 are women of reproductive age (15–44 years), and 665,000 are children (<15 years old). It was also estimated that of the fish consuming females of reproductive age, more than 84,000 are pregnant in any given year.

Fish is generally considered an excellent source of protein in the diet and the health benefit of fish consumption, including the reduction in the incidence of coronary heart disease, is well recognized (Salonen et al. 1995). However, Salonen et al. (1995) studied 1,833 eastern Finnish men ages 42–60 and related high dietary intake of freshwater fish containing mercury residues, as well as elevated hair content and urinary excretion of mercury, to a risk of acute myocardial infarction and death from coronary heart disease and cardiovascular disease. Men with the highest tertile of hair mercury had a 2-fold age-specific risk and a 2.9-fold adjusted risk of acute myocardial infarction and cardiovascular death, compared to men with lower mercury hair levels. Egeland and Middaugh (1997) and Clarkson et al. (1998) contend that the Seychelles population is a more appropriate sentinel population for fish consumers in the United States because: (1) the major source of methylmercury is from open ocean fish; (2) the mercury concentrations in hair are 10–20 times the average found in the United States; and (3) because the Seychellois consistently consume about 12 fish meals per week. These authors feels that the potential adverse effects of methylmercury in fish would be detected in the Seychelles Island population, long before such effects are observed in the United States. The Finnish study (Salonen et al. 1995), however, suggests that freshwater fish, low in selenium and omega-3 polyunsaturated fatty acids, may not protect against cardiovascular risks from methylmercury. The human health benefit/cost tradeoff between fish consumption and mercury exposure varies by species and mercury dose.

Dental Amalgams. Recent animal and human studies have also identified the uptake, distribution, and rate of excretion of elemental mercury from dental amalgams as another significant contributing source to mercury body burden in humans (Björkman et al. 1997; Lorscheider et al. 1995). A summary of contributing sources of mercury to the human body burden is presented in Table 5-12. Because of the wide range of potential exposures and the high retention rate for elemental mercury, dental amalgams potentially represent the largest single contributing source of mercury exposure in some individuals with large numbers (>8) of amalgam fillings.

Dental amalgams may contain 43–54% elemental mercury (DHHS 1993). A single amalgam filling with an average surface area of 0.4 cm^2 has been estimated to release as much as 15 µg mercury/day, primarily through mechanical wear and evaporation, but also through dissolution into saliva (Lorscheider et al. 1995). The rate of release is influenced by chewing, bruxism (grinding of teeth) food consumption, tooth brushing, and the intake of hot beverages (Weiner and Nylander 1995). For the average individual with eight occlusal amalgam fillings, 120 µg of mercury could be released daily into the mouth, and a portion of that swallowed or inhaled (Lorscheider et al. 1995). Experimental results regarding estimated daily dose of inhaled mercury vapor released from dental amalgam restorations are few and contradictory (Berglund 1990). More recently, Björkman et al. (1997) reported that approximately 80% of inhaled mercury from dental amalgams is absorbed (Björkman et al. 1997). Various laboratories have estimated the average daily absorption of amalgam mercury ranging from 1 to 27 µg, with levels for some individuals being as high as 100 µg/day (Björkman et al. 1997; Lorscheider et al. 1995; Weiner and Nylander 1995). Estimates of mean daily elemental mercury uptake from dental amalgams from these and earlier studies are summarized in Table 5-16. A report from the Committee to Coordinate Environmental Health and Related Programs (CCEHRP) of the Department of Health and Human Services determined that "measurement of mercury in blood among subjects with and without amalgam restorations . . . and subjects before and after amalgams were removed . . . provide the best estimates of daily intake from amalgam dental restorations. These values are in the range of 1–5 µg/day" (DHHS 1993). Another source indicates that the consensus average estimate is 10 µg amalgam Hg/day (range, 3–17 µg/day) (WHO 1991). However, Halbach (1994) examined the data from 14 independent studies and concluded that the probable mercury dose from amalgam is less than 10 µg/day. Most recently, Richardson (1995) computed a release rate per filled tooth surface as 0.73 µg/day-surface, with a standard deviation of 0.3 µg/day-surface and a “stimulation magnification factor” of 5.3, based on a weighted average enhancement of mercury vapor concentration following chewing, eating, or tooth brushing reported in three amalgam studies.

By comparison to the estimated daily absorbance of mercury from dental amalgams (range, 3–17 µg), the estimated daily absorbance from all forms of mercury from fish and seafood is 2.31 µg and from other foods, air, and water is 0.3 µg (WHO 1991). These other sources taken together only total 2.61 µg/day, in comparison to estimates of 3–17 µg/day for dental amalgams. Assuming a person has large numbers of amalgams, this source may account for 17 µg/day out of a total absorbance of 19.61 µg/day, or 87% of the absorbed mercury. In contrast, in individuals with only a few amalgams, mercury from this source may account for only 3 µg mercury/day out of a total absorbance of 5.61 µg/day, or 53% of absorbed mercury. Halbach et al. (1994) concluded that the sum of the mercury uptake from dental amalgam and dietary

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

Number of surfaces	Mercury (µg/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

uptake is still below the dose corresponding to the acceptable daily intake (ADI) of mercury. The ADI of 40 µg total mercury, 30 µg of which are allowed for methylmercury, results in a total dose of approximately 30 µg after accounting for absorption (Halbach 1994; WHO 1976). WHO (1990) estimates a daily absorption of 2.61 µg from background exposure for persons without amalgam exposure.

In a recent study by Schweinsberg (1994), the author monitored mercury in blood, urine, and hair of subjects with amalgam fillings, in subjects who consumed fish, and in mercury-exposed workers. With respect to hair concentrations, the author reported a mean mercury level in hair of 560 µg/kg (ppb), 940 µg/kg, and 1,600 µg/kg in subjects that consumed the following mean amounts of fish per month: 120 g/month (range, 0–<400 g fish/month); 600 g/month (range, 400–<1,000 g/month); and 1,900 g/month (>1,000 g/month), respectively. Mercury concentrations in whole blood (µg/L) were 0.2–0.4 µg/L for individuals with no fish consumption and no dental amalgams, 1.047±0.797 µg/L for persons with no fish consumption and >6 dental amalgams, 2.56±2.123 µg/L for persons with fish consumption >990 g/month and no dental amalgams, and 2.852±2.363 µg/l for persons with fish consumption >990 g/month and >6 dental amalgams. Mercury concentrations in the urine of occupationally exposed thermometer factory workers were higher, by a factor of 100, than in the group with amalgam fillings. The author concluded that both amalgam fillings and the consumption of fish burden individuals with mercury in approximately the same order of magnitude.

In a more recent study of lactating women, Oskarsson et al. (1996) assessed the total and inorganic mercury content in breast milk and blood in relation to fish consumption and amalgam fillings. The total mercury concentrations (mean±standard deviation) in breast milk, blood, and hair samples collected 6 weeks after delivery from 30 Swedish women were 0.6±0.4 ng/g (ppb), 2.3±1.0 ng/g, and 0.28±0.16 µg/g, respectively. In milk, an average of 51% of total mercury was in the inorganic form, whereas in blood an average of only 26% was in the inorganic form. Total and inorganic mercury levels in blood and milk were correlated with the number of amalgam fillings. The concentrations of total mercury and organic mercury in blood and total mercury in hair were correlated with the estimated recent exposure to methylmercury via consumption of fish. There was no significant difference between the milk levels of mercury in any form and the estimated methylmercury intake. A significant correlation was found, however, between the levels of total mercury in blood and in milk, with milk levels being an average of 27% of the blood levels. There was also an association between inorganic mercury in blood and in milk; the average level of inorganic mercury in milk was 55% of the level of inorganic mercury in blood. No significant correlations were found between the levels of any form of mercury in milk and the levels of organic mercury in blood. The results indicated that

there was an efficient transfer of inorganic mercury from blood to milk and that, in the study population, mercury from amalgam fillings was the main source of mercury in breast milk. Exposure of the infant to mercury in breast milk was calculated to range up to 0.3 µg/kg/day, of which approximately one half was inorganic mercury. This exposure corresponds to approximately one-half the tolerable daily intake for adults recommended by the World Health Organization (WHO). The authors concluded that efforts should be made to decrease mercury burden in women of reproductive age.

Blood. (EPA 1996d). Because methylmercury freely distributes throughout the body, blood is a good indicator medium for estimating methylmercury exposure. However, because an individual's intake may fluctuate, blood levels may not reflect mercury intake over time (Sherlock and Quinn 1988; Sherlock et al. 1982). Recent reference values for total mercury levels in blood of non-exposed individuals in the general U.S. population are very limited. The mean concentration of mercury in whole blood based on a review of existing data from other countries, is 8 µg/L (ppb) (WHO 1990). Certain groups with high fish consumption may attain blood methylmercury levels of 200 µg/L (ppb), which is associated with a low (5%) risk of neurological damage to adults (WHO 1990).

Urine. Urine is a common indicator used to assess occupational mercury exposure (EPA 1996d). Urinary mercury is thought to indicate most closely the mercury levels present in the kidneys (Clarkson et al. 1988b). But while urinary mercury has been widely used to estimate occupational exposures, reference values for urinary mercury levels in non-exposed individuals in the general U.S. population are very limited. The mean concentration of urinary mercury, based on a review of existing data from other countries, is about 4 µg/L (ppb) (WHO 1990, 1991). For assessment of long-term inorganic mercury exposure, biological monitoring of the urinary mercury is normally used (Skare 1995). Several authors have related elevated urinary mercury levels to dental amalgams in individuals in the general population (Barregard et al. 1995; Skare 1995) and in dentists and dental personnel receiving occupational exposures (Akesson et al. 1991; Chien et al. 1996; WHO 1991).

Breast Milk. Recent reference values for mercury levels in breast milk in non-exposed individuals in the general U.S. population are very limited. The mean concentration of mercury in breast milk, based on a review of existing data from other countries, is 8 µg/L (ppb) (WHO 1990, 1991). Mean concentrations of mercury in breast milk samples from the United States and other countries are summarized in Table 5-17. Pitkin et al. (1976) reported a mean total mercury concentration of 0.93±0.23 ppb in a midwestern community in the United States. This mean value is only about one-third the mean value reported for Inuit

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	Skerfving 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

women living in interior (3.2±0.8 ppb) or urban areas (3.3±0.5 ppb) of Alaska and less than one-seventh the mean value for coastal Alaskan Inuit women (7.6±2.7 ppb) known to consume seal meat and oil, as well as marine fish (Galster 1976). The latter breast milk total mercury level is comparable to the median (2.45 ppb) and maximum (8.7 ppb) values reported for women in the Faroe Islands that consume large amounts of fish and pilot whale meat (Grandjean et al. 1995a).

Levels of total mercury in breast milk have been monitored in several foreign countries over the past three decades. A mean breast milk mercury concentration of 3.6±2.2 ppb (range, non-detected to 9.8 ppb) was reported for an urban population in Tokyo, Japan (Fujita and Takabatake 1977). In a study of urban women residing in Madrid, Spain, the mean breast milk mercury concentration was 9.5±5.5 ppb (range, 0.9–19 ppb) (Baluja et al. 1982). These authors did not provide any information (i.e., whether females were fish consumers, the number of dental amalgams they had, or their occupations) that would explain the relatively high mercury levels. Skerfving (1988) reported mercury concentrations ranged from 0.2 to 6.3 ppb in breast milk of Swedish women that consumed fish; however, this author did not provide specific information on the fish consumption rate or the number of dental amalgams of the study population. Most recently, Oskarsson et al. (1996) reported a mean total breast milk concentration of 0.6±0.4 ppb (range, 0.1–2.0 ppb) for a group of Swedish women that consumed freshwater fish and had an average of 12 amalgam fillings. This was a smaller range in mercury concentrations than that reported by Skerfving (1988).

All of these general population breast milk mercury concentrations are in sharp contrast to those reported for samples collected from women in Minamata, Japan, where industrial effluents containing methylmercury caused widespread contamination of local seafood. Breast milk total mercury concentrations were on the order of 63 ppb in individuals who lived in the vicinity of Minamata, Japan and had consumed highly mercury-contaminated fish (Fujita and Takabatake 1977). Similarly, in Iraq, where consumption of bread made from seed grain treated with methylmercury as a fungicide caused a similar mercury poisoning outbreak, breast milk concentrations as high as 200 ppb were reported (Bakir et al. 1973). Breast milk containing total mercury levels of >4 ppb would exceed the safe level (2 µg methylmercury/day for an average 5-kg infant) (Wolff 1983). It is important to emphasize, however, that in general, the beneficial effects associated with breast feeding seem to override or at least compensate for any neurotoxic effects on milestone development that could be due to the presence of contaminants, such as mercury, in human milk (Egeland et al. 1997).

Hair. Scalp hair is another primary indicator used to assess methylmercury exposure, because the methylmercury is incorporated into the hair at the hair follicle in proportion to its content in the blood (EPA 1996d). The typical hair-to-blood ratio in humans has been estimated to be about 250:1 expressed as μg Hg/g hair to mg Hg/L blood, but some difficulties in measurements, inter-individual variation in body burden, differences in hair growth rates, and variations in fresh and saltwater fish intake have led to varying estimates (Birke et al. 1972; Skerfving 1974). Once incorporated into the hair strand, the methylmercury is stable and gives a longitudinal history of blood methylmercury levels (WHO 1990). Care must be exercised to ensure that the analysis of methylmercury levels in hair are not confounded by adsorption of mercury vapors or inorganic mercury onto the hair (Francis et al. 1982)

Recent reference values for mercury levels in hair from non-exposed individuals in the general U.S. population are very limited. A summary of mercury concentrations in hair from residents (adults, men, women, and children) of several U.S. communities is presented in Table 5-18. Most of the these studies, however, with the exception of Fleming et al. (1995) were conducted from 7 to 20 years ago. For populations studied in the United States, the range in mean hair concentrations was 0.47–3.8 ppm for adults (maximum value of 15.6 ppm) and 0.46–0.77 ppm for children (maximum value of 11.3 ppm). The mean concentration of mercury in hair based on a review of existing data from other countries is 2 μg/g (ppm) (WHO 1990), and the WHO advisory maximum tolerable level for hair is 6 ppm.

The concentration of total mercury in hair in the general population of Japan was determined by Nakagawa (1995). This author sampled hair from 365 healthy volunteers in Tokyo and the surrounding area from June 1992 to June 1993. The mean concentration of mercury in hair was higher in males (2.98 ppm, 81 individuals sampled) than in females (2.02 ppm, 284 individuals sampled). In both males and females, the mercury concentration in hair increased with age up to the mid-30s, then gradually declined. The authors also looked at dietary preferences and found the mean hair levels in males and females were highest in individuals that had a preference for fish (4.0 and 2.7 ppm, respectively), followed by those with a preference for fish and meat (2.88 and 2.00 ppm, respectively), a preference for meat (2.38 ppm and 1.96 ppm, respectively), and was lowest in those individuals that preferred a predominantly vegetarian diet (2.27 and 1.31 ppm, respectively). In an earlier study, the mercury content in human hair was studied in Japanese couples, with husbands having significantly higher mercury concentrations (4.01 ppm) than wives (1.99 ppm), possibly as a result of greater fish consumption among the men (Chen et al. 1990). This same pattern is also apparent for all but one of the U.S. populations (San Diego, California) studied by Airey (1983b). It is noteworthy that some of the highest mercury concentrations in hair measured in women

Table 5-18. Mercury Concentrations in Hair (µg 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									Creason et al. 1978a
adults n=203	0.77				14.0				
children n=280				0.67				11.3	
4 NJ communities (Ridgewood, Fairlawn, Matawan, and Elizabeth)									Creason et al. 1978b may have to drop
adults n=117	0.78				5.6				
children n=204				0.77				4.4	
Birmingham AL and Charlotte NC									Creason et al. 1978c
adults n=282	0.47				7.5				may have to drop
children n=322				0.46				5.4	
U.S. unidentified community									Airey 1983b
males n=22		2.7				6.2			
females n=16			2.6				5.5		
adults n=24	2.1				5.6				
adults n=31	2.2				6.6				
adults n=24	2.9				7.9				
adults n=79	2.4				7.9				
LaJolla-San Diego CA									Airey 1983b
males n=13		2.4				6.2			
females n=13			2.7				5.5		
adults n=8	2.3				4.5				
adults n=17	2.9				6.6				
adults n=5	2.6				6.2				
adults n=30	2.7				6.6				
Maryland									Airey 1983b
adults n=11	1.8				3.8				
adults n=11	1.5				3.9				
adults n=11	2.3				4.5				
adults n=33	1.9				4.5				

Table 5-18. Mercury Concentrations in Hair (µg 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 females of child-bearing age n=80							15.2		Lasora and Citterman 1991
Seguim WA females of childbearing age n=7			0.70				1.5		Lasora and Citterman 1991
Florida Everglades adults that consumed local wildlife n=330	1.3				15.6				Fleming et al. 1995
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

(15.2 ppm) were from Nome, Alaska where the population consumes large amounts of fish and marine mammals (Lasora and Citterman 1991) and from Florida (15.6 ppm), where measurements were made only in adults that consumed wildlife from the Everglades area, a region where high mercury levels in wildlife have been reported (Fleming et al. 1995). Most recently, Davidson et al. (1998) reported the results of the Seychelles Child Development Study at 66 months (5.5 years) post-parturition. These researchers reported that there were no adverse neurodevelopmental outcomes observed in mother-child pairs, with mean maternal and mean child hair total mercury concentrations of 6.8 ppm and 6.5 ppm, respectively, in the Seychelles Island study.

Oral Tissues. Mercury concentrations as high as 380 µg/g (ppm) have been found in oral tissues in contact with amalgam fillings. In individuals with more than six amalgam fillings, a mean value of 2.3 µg/g (ppm) was found in tissue without direct contact with amalgam fillings (Björkman et al. 1997). In some European countries, health authorities recommend that sensitive or susceptible individuals in higher risk groups (i.e., pregnant women and individuals with kidney disease) avoid treatment with dental amalgam (Björkman et al. 1997).

Occupational Exposure. Workplace environments presenting the largest potential sources of occupational exposure to mercury include chloralkali production facilities, cinnabar mining and processing operations, and industrial facilities involved in the manufacture and/or use of instruments containing liquid mercury (Stokinger 1981). According to NIOSH (1973), the principal route of occupational exposure to mercury is vapor phase inhalation from workplace atmospheres. Studies by Barregard et al. (1992) and by Langworth et al. (1992b) revealed increased total mercury levels in blood and urine of exposed chloralkali workers. These results are summarized in Table 5-19. Personal air sampling of workers in a mercury recycling plant in Germany showed mercury levels ranging from 115 to 454 µg/m^3 (Schaller et al. 1991).

Human tissues that are routinely monitored as evidence of exposure to mercury are urine, blood, and hair. Urine is most frequently monitored as an indicator of human body burden following chronic exposure to mercury vapor, particularly in occupational settings; approximately 95% of all urine samples contain less than 20 µg /L (ppb) (EPA 1984b). A comparison of mercury content in the urine of Swedish workers exposed to high levels of mercury, dentists, occupationally unexposed workers, and unexposed workers without dental amalgams gave values of 15, 1.7, 0.8, and 0.3 µmol/mol creatinine, respectively (corresponding mercury plasma levels were 35, 9.4, 5.3, and 2.8 nmol/L [7.19, 1.89, 1.06, and 0.56 ppt], respectively) (Molin et al. 1991). Blood and urine monitoring may be useful for groups of workers subject

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

Number of subjects	Exposure group	Air ($\mu g/m^3$)	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	

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 μg/m^3 (ATSDR 1990).

The use of fluorescent tube compactors by industrial facilities may also expose those operating the compactors and workers in adjacent areas to increased levels of mercury vapor if proper filters, scrubbing devices, and ventilation are not used (Kirschner et al. 1988).

Dentists and other dental professionals may have greater exposure to mercury as a result of preparing and applying dental amalgams (Ayyadurai and Krishnashamy 1988; Skare et al. 1990). Nylander et al. (1989) sampled pituitary gland tissue from autopsies of 8 dental staff and 27 control individuals in Sweden. These authors reported median mercury concentrations of 815 μg/kg (ppb) wet weight (range, 135–4,040 μg/kg) in pituitary tissue of dental staff (7 dentists and 1 dental assistant), as compared to a median of 23 μg/kg (wet weight) in 27 individuals from the general population. None of the dental staff had been working immediately prior to their deaths, and in several cases, more than a decade had passed since the cessation of their clinical work. The number of amalgams did not correlate to pituitary gland concentrations in the controls. However, if two of the controls with the highest mercury concentrations were excluded (there was some evidence that these individuals had received occupational exposures), then the correlation was significant ($p<0.01$). In another study, Nylander and Weiner (1991) also reported high mercury concentrations in the thyroid and pituitary glands, with a median of 1.1 μmol/kg (221 ppb) wet weight (range, 0.7–28 μmol/kg [140–5,617 ppb]) in the pituitary. the median mercury concentration in the pituitary of the controls was 0.11 μmol/kg (22 ppb) (range, 0.03–5.83 μmol/kg [6–1,170 ppb]).

Naleway et al. (1991) reported results of a screening study conducted in 1985 and 1986 by the American Dental Association to analyze urinary mercury concentrations in dentists and identify those individuals with elevated urinary mercury levels. In 1985, 1,042 U.S. dentists were screened, and a mean urinary mercury level of 5.8 µg/L (ppb) (maximum 84 µg/L) was reported. In 1986, 772 dentists screened had a mean urinary level of 7.6 µg/L (ppb) (maximum 115 µg/L). Their mean urinary mercury levels were substantially lower than pooled data (mean, 14.2 µg/L) from dentists participating in the screening program from 1975–1983 (Naleway et al. 1985). The authors noted a substantial decline, particularly during the last 5 years (1982–1986), which was attributed to better mercury hygiene and the reduced use of amalgam restorations. This study also evaluated responses from a questionnaire survey of 480 dentists. The results indicated that those dentists reporting skin contact with mercury amalgam had mean urinary mercury levels of 10.4 µg/L (ppb), compared to 6.3 µg/L (ppb) in dentists reporting no skin contact; this difference was found to be statistically significant. Similarly, the mean urinary mercury level in dentists reporting mercury spills in the office was 7.8 µg/L (ppb), compared to 6.0 µg/L (ppb) for those reporting no mercury spills. Again, the difference was significant. Additionally, the number of hours practiced per week was found to weakly correlate with urinary mercury concentrations (Naleway et al. 1991).

Painters are another group that may be occupationally exposed to mercury vapors from volatilization of mercury during application of paint containing phenylmercuric acetate. Hefflin et al. (1993) studied the extent of mercury exposure from the application of exterior latex paints. These authors compared the air and urinary mercury concentrations of 13 professional male painters with those of 29 men having other occupations (nonpainters). The painters applied 2 brands of exterior latex paint that contained mercury; the median concentration was 570 mg/L (ppm). The median air mercury concentration was higher for painters (1.0 µg/m^3; range, non-detectable to 4 µg/m^3) than for nonpainters (non-detected; range, not detected to 3 µg/m^3). The median urinary mercury concentration was nearly twice as high for painters (9.7 µg/L [ppb]; range, 5.9–20.4 µg/L) as for nonpainters (5.0 µg/L [ppb]); range, 2.6–11.6 µg/L [ppb]) (p=0.0001). The normal range of urinary mercury is <20 µg/L (ppb) (EPA 1984b). Among the professional painters, urinary mercury concentrations increased with the percentage of time spent applying the exterior paint. Tichenor and Guo (1991) also studied the amount of mercury emitted from latex paints containing mercury compounds. The concentrations of mercury in the 5 types of paint tested ranged from 93 ppm to 1,060 ppm. These authors also reported that from 12 to 57% of the mercury in the paint was emitted upon application as elemental mercury, with the highest emission rate within the first few hours after paint application.

Commercial artists and crafts people are another group that is also at risk of mercury exposure from a variety of professional arts and crafts materials and techniques (Grabo 1997). This author reported that mercury was a hazard to commercial artists using mercury-based pigments in airbrush painting, brush paintings, and in pastels via pigment in chalk dusts. The author concluded that occupational health professions should be aware of toxic nature of the materials used by artists, whether they are employed in industry, self-employed, or are hobbyists.

Chemists are another group at risk of occupational exposure as a result of activities involving the synthesis of mercury compounds or the analysis of environmental or biological samples containing mercury residues. Methylmercury compounds are still used in laboratory-based research, and so the possibility of occupational exposure remains. Junghans (1983) reviewed the toxicity of methylmercury compounds associated with occupational exposures attributable to laboratory use. Most recently, a poisoning incident was reported from a single acute exposure to dimethylmercury (Blayney et al. 1997). The analytical chemist involved was exposed to approximately 0.1–0.5 mL of dimethylmercury spilled on disposable platex gloves during a transfer procedure in a fume hood, while preparing a mercury nuclear magnetic resonance standard. Blood analyses 5 months after the exposure incident revealed a whole blood mercury concentration of 4,000 µg/L (ppb), which is 80 times the usual toxic threshold (50 µg/L) and 400 times the normal mercury blood range (<10 µg/L) (Blayney et al. 1997). These authors caution that highly resistant laminate gloves should be worn under a pair of long-cuffed unsupported neoprene, nitrile, or similar heavy duty gloves rather than latex or polyvinyl chloride (PVC) gloves. Another group of analytical chemists (Toribara et al. 1997) reported that during the calibration of a mass spectrometer, an operator used a pipette with a plastic tip to transfer dimethylmercury into a Pyrex glass vial equipped with a crimp top for a Teflon-lined silicone stopper in a fume hood. After transfer, the plastic tip was disposed of in a nearby wastebasket and, in a short time, the instrument (which can detect nanogram quantities of mercury) showed measurable quantities in the workplace air around the instrument and operator. Toribara et al. (1997) also cites three other historic incidents where laboratory staff and non-laboratory staff (secretaries) working in proximity to a dimethylmercury spill were poisoned. These authors caution colleagues about the hazards involved in shipping dimethylmercury, if the packaging and container is physically damaged during transport.

The National Occupational Exposure Survey (NOES) conducted by NIOSH from 1980 to 1983 estimated that 67,551 workers, including 21,153 women in 2,877 workplaces were potentially exposed to mercury in the workplace (NIOSH 1984b). Most of these workers were employed in the health services, business services, special trade contractor, and chemical and allied products industries as chemical technicians,

science technicians, registered nurses, and machine operators. These estimates were derived from observations of the actual use of mercury (97% of total estimate) and the use of trade-name products known to contain mercury (3%). It is unknown how many of the potentially exposed workers were actually exposed. Data from the NOES conducted by NIOSH from 1983 to 1986 was broken out by exposure to a variety of mercury compounds (RTECS 1998). Estimates of the total numbers of all workers and women workers potentially exposed are presented in Table 5-20. A total of 151,947 workers were potentially exposed to mercury or various mercury compounds; 33% (50,468) of these workers were women. Table 5-21 summarizes the calculated mercury absorption from air at various occupational exposure guideline concentrations.

5.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans and briefly considers potential pre-conception exposure to germ cells. Differences from adults in susceptibility to hazardous substances are discussed in Section 2.6, Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, and breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults. The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor; they put things in their mouths; they may ingest inappropriate things such as dirt or paint chips; they spend more time outdoors. Children also are closer to the ground, and they do not have the judgement of adults in avoiding hazards (NRC 1993).

Significant health risks, including numerous neuropathological and neurobehavioral effects, are associated with prenatal exposure to methylmercury (Zelikoff et al. 1995). Fetuses and breast-fed infants may be exposed to higher than background concentrations of mercury via maternal consumption of large amounts of fish or marine mammals contaminated with mercury, via maternal exposure to mercury through dental amalgams, via maternal use of consumer products containing mercury or various mercury compounds, and via occupational exposure of the mother (Zelikoff et al. 1995). Fetuses can be exposed to mercury via exposures of their mothers either before or during pregnancy; nursing infants can be exposed via consumption of contaminated breast milk from mothers exposed via medical, domestic, or occupational

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

Table 5-21. Calculated Mercury Absorption from Air

Route (source of exposure)	Guideline	Air concentration ($\mu g/m^3$)	Exposure (μg/week)	Exposure (μg/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 m^3 and no other mercury exposure

[b] Home exposure to ambient air assumes 24 hours per day, 7 days per week, and ventilation rate 20 m^3/day

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

exposures (see Section 5.7). Children can be exposed to various forms of mercury in a variety of ways, including playing with unsecured elemental mercury, inhalation of mercury vapors via the religious or ethnic practices of their parents or unintentional spills of elemental mercury, oral ingestion of herbal or ethnic remedies or mercury-containing consumer products, consumption of methylmercury-contaminated fish and wildlife, and dermal or oral exposure to contaminated soils and sediments.

Mercury concentrations have been measured in cord blood in one study in the United States with levels that suggest prenatal exposure. Pitkin et al. (1976) measured concentrations of total mercury in cord blood samples from 100 maternal cord blood pairs from a population in rural Iowa. The mean cord blood total mercury concentration was 1.24 ppb, while the mean of the paired maternal blood samples was 1.01 ppb. More recently, Wheatley and Paradis (1995a, 1995b) reported on the analysis of 2,405 cord blood samples collected from Canadian aboriginal peoples over the last 20 years. Of these cord blood samples, 523 (21.8%) were found to have total mercury levels greater than 20 ppb, with the highest cord blood sample containing 224 ppb. These latter samples were from populations that routinely consumed fish and marine mammal tissues. Grandjean et al. (1997b) measured cord blood samples from 894 Faroe Islands children whose mothers consumed large amounts of fish and pilot whale meat. The methylmercury exposure in the Faroe Island population is mainly from eating pilot whale meat. The geometric mean concentration of total mercury in these cord blood samples was 22.9 ppb.

Concentrations of mercury have also been measured in breast milk from several populations in the United States as well as other countries (see Table 5-17). Breast milk concentrations have been reported for two U.S. populations; one in rural Iowa (Pitkin et al. 1976) and the other from Alaska (Galster 1976). Pitkin et al. (1976) reported a total mean mercury concentration in breast milk of 0.9±0.23 ppb (range, 0.8–1.6 ppb). The mean total mercury concentrations in the Alaskan populations were 3.3± 0.5 ppb for the urban population, 3.2±0.8 ppb for the interior population, and 7.6±2.7 ppb for the coastal population that consumed fish and marine mammals.

Total mercury concentrations in breast milk from other countries and exposure scenarios were 3.6±2.2 ppb for an urban population in Tokyo, Japan (Fujita and Takabatake 1977), 0.6±0.4 ppb for Swedish women that were fish consumers with 12 dental amalgams (Oskarsson et al. 1996), 0.2–6.3 ppb (range) for Swedish women that consumed fish (Skerfving 1988), and 9.5±5.5 ppb for an urban population of women in Madrid, Spain (Baluja et al. 1982) (Table 5-17). Some of the highest levels were reported in fish eaters, and about 20% of the total mercury content of the milk was methylmercury. The median and maximum mercury

concentrations in breast milk from women in the Faroe Islands, a population that consumes large quantities of fish and marine mammal tissue, were 2.45 and 8.7 ppb, respectively (Grandjean et al. 1995a). Breast milk mercury concentrations reported by these authors were significantly associated with mercury concentrations in cord blood and with the frequency of pilot whale dinners during pregnancy. These are relatively low values in contrast to the values reported in Minamata, Japan, for women who ate contaminated seafood in the Minamata episode, which resulted in total mercury concentrations in breast milk of 63 ppb (Fujita and Takabatake 1977), and in Iraq, where consumption of homemade bread prepared from methylmercury-contaminated wheat occurred, resulted in breast milk concentrations of up to 200 ppb (about 60%) methylmercury (Amin-Zaki et al. 1976; Bakir et al. 1973).

Children can be exposed to mercury by many of the same pathways as adults as discussed in Sections 5.4.4., 5.5, and 5.7. Children can receive mercury exposures from oral or dermal contact with mercury-contaminated soils and sediments or mercury-contaminated objects. Exposure analysis of individuals living near an abandoned mercury-contaminated industrial site suggested that children were exposed primarily via soil ingestion (Nublein et al. 1995). Little experimental information on the bioavailability of mercury via oral or dermal exposure was found relative to mercury or mercury compounds sorbed to contaminated soils and sediments (De Rosa et al. 1996). Paustenbach et al. (1997) noted that, due to the presence of mercury at a number of major contaminated sites in the United States, the bioavailability of inorganic mercury following ingestion has emerged as an important public health issue. Although precise estimates are not available, *in vivo* and *in vitro* estimates of the bioavailability of different inorganic mercury species in different matrices suggest that the bioavailability of these mercury species in soil is likely to be significantly less (on the order of 3 to 10 fold), than the bioavailability of mercuric chloride, the mercury species used to derive the toxicity criteria for inorganic mercury (Paustenbach et al. 1997). These authors suggest that site specific estimates of bioavailability be conducted of various mercury compounds because bioavailability can vary significantly with soil type, soil aging, the presence of co-contaminants and other factors. Canady et al. (1997) concluded that the "100% bioavailability assumption" for mercury-contaminated soils is excessively conservative. These authors note that various mercury compounds have distinctly different bioavailability. For example, mercuric chloride has been reported to be approximately 20–25% bioavailable in adult animals (Nielsen and Andersen 1990; Schoof and Nielsen 1997). Methylmercury is thought to be nearly completely absorbed (Aberg et al. 1969; Miettinen et al. 1971; Rice 1989a, 1989b). Mercuric nitrate was reported to be only 15% bioavailable in humans (Rahola et al. 1973) and elemental mercury is thought to be very poorly absorbed, although experimental evidence is lacking for the latter. Recently, Barnett et al. (1997) reported that analysis of mercury contaminated soil from the flood

plain of East Fork Poplar Creek in Oak Ridge, Tennessee, revealed the presence of submicron, crystalline mercuric sulfide (HgS) in the form of metacinnabar. The HgS formed in place after the deposition and burial of mercury-contaminated soils. The formation of HgS is significant for remediation efforts at the site because the toxicity, leachability, and volatility of mercury in soils are dependent on the solid phase speciation. Because local hydrogeochemical conditions are not unique, the formation of HgS at this site has implications to other environments and contaminated sites as well.

Children may be exposed to mercury vapors when they play with metallic mercury. Metallic mercury is a heavy, shiny, silver liquid and when spilled, forms little balls or beads which fascinate children. Children come in contact with metallic mercury when they trespass in abandoned warehouses, closed factories, or hazardous waste sites (ATSDR 1997; George et al. 1996). Children also have taken metallic mercury from school chemistry and physics laboratories and abandoned warehouses (ATSDR 1997). Broken thermometers and other mercury-containing instruments or equipment (fluorescent light bulbs, barometers, blood pressure measurement equipment, and light switches) used in the home and in some children's sneakers that light up are other sources of metallic mercury. Muhlendahl (1990) reported a case of chronic mercury intoxication in three children who were exposed to vapors from a broken thermometer. The maximum urinary concentrations reported by this author (8 months after the broken thermometer incident) were 250.5 µg/L for a 33-month-old girl, 266.3 µg/L for a 20-month-old girl, and 137.4 ppm for the 7-year-old brother 2 days after each patient received chelation therapy with DMPS (2,3-dimercaptopropan-1-sulphonate). Sometimes children find containers of metallic mercury which were disposed of improperly (ATSDR 1997), or adults intentionally or unintentionally bring home metallic mercury from work (Ehrenberg et al. 1991; Wendroff 1990). Metallic mercury evaporates to a greater extent as the air temperature increases; when it is not stored in a closed container, children may be exposed to mercury vapors (ATSDR 1997; Wendroff 1991).

Metallic mercury is traditionally used in some religious rituals or remedies, including religions such as Santeria (a Cuban-based religion that worships both African deities and Catholic saints), voodoo (a Haitian-based set of beliefs and secret rites), Palo Mayombe (a secret form of ancestor worship practiced mainly in the Caribbean), or Espiritismo (a spiritual belief system native to Puerto Rico) (Wendroff 1990). If these rituals or spiritual remedies containing mercury are used in the home, children may be exposed and the house may be contaminated with mercury (ATSDR 1997; Johnson [in press]; Wendroff 1990, 1991; Zayas and Ozuah 1996). Metallic mercury is sold under the name "azogue" (pronounced ah-SEW-gay) in stores (sometimes called botanicas) which specialize in religious items and ethnic remedies (Johnson [in press];

Wendroff 1990; Zayas and Ozuah 1996). Azogue may be recommended by family members, spiritualists, card readers, and santeros. Typically, azogue is carried on one's person in a sealed pouch, or it is ritually sprinkled in the home or car. Some store owners suggest mixing azogue in bath water or perfume. Some people place azogue in devotional candles. Because metallic mercury evaporates into the air, there is a potential health risk from exposure to mercury vapors in a room where the mercury is sprinkled or spilled onto the floor, put in candles, or where open containers of metallic mercury are present (ATSDR 1997; Wendroff 1990, 1991). Young children spend a lot of time crawling on the floor and carpeting, so they may be subject to a higher risk of exposure, especially when mercury is sprinkled on the floors or carpets.

Very small amounts of metallic mercury (i.e., a few drops) may raise air concentrations of mercury to levels that could be harmful to health (ATSDR 1997). Metallic mercury and its vapors are extremely difficult to remove from clothes, furniture, carpet, floors, walls, and other such items. The mercury contamination can remain for months or years, and may pose a significant health risk for people continually exposed (ATSDR 1997; Johnson [in press]; Wendroff 1990, 1991).

Another potential source of children's exposure to metallic mercury is breakage or improper disposal of a variety of household products, including thermostats, fluorescent light bulbs, barometers, glass thermometers, and some blood pressure machines that contain metallic mercury (ATSDR 1997). These devices do not pose a health threat when the mercury is properly contained within the device. Should the mercury be released, however, the potential for mercury vapors to contaminate the air increases. The appropriate method for cleaning up a spill of a small amount of mercury is to clean it up manually, *without using a vacuum cleaner*, which can cause the mercury to evaporate more rapidly into the air, creating a greater risk of exposure (ATSDR 1997; Schwartz et al. 1992; Votaw and Zey 1991). Votaw and Zey 1991 reported mean mercury concentrations in air samples collected in a dental office were 8.5 μ/m^3 when a vacuum cleaner was not in use and concentrations rose to 69 μ/m^3 when a vacuum cleaner was in use. Special techniques are often needed to prevent mercury vapor from being generated in the cleanup process (Votaw and Zey 1991). The first consideration is to remove children from the area of the spill. The beads of metallic mercury should be cleaned up by carefully rolling them onto a sheet of paper or by drawing them up into an eye dropper. After the mercury has been collected, it should be put in a plastic bag or airtight container. The piece of paper or eye dropper used to remove the mercury should also be bagged and disposed of properly, according to guidance provided by the local health department. After the mercury has been removed, the room should be ventilated to the outside and closed off to the rest of the house. Electric fans should be used for a minimum of one hour to speed the ventilation process. If larger quantities of

metallic mercury are found in a container, make sure the container is airtight and call the local health department for disposal instructions. If the container of mercury is open without a lid, a piece of plastic wrap can be used to seal the container. If the larger amount is spilled, leave the area immediately and contact the local health department or fire department. Members of the general public should seek professional guidance on proper disposal procedures of mercury (ATSDR 1997).

Metallic mercury vapors are very toxic and are virtually odorless. Inhalation of mercury-laden dust, vapor, or mist should be avoided. Metallic mercury not should not come in contact with eyes, skin, or clothing. If children are exposed directly to metallic mercury, the contaminated body area should be thoroughly washed, and contaminated clothing should be removed and disposed of in a sealed plastic bag (ATSDR 1997). ATSDR and EPA recommend very strongly against the use of any uncontained metallic (liquid) mercury in homes, automobiles, day care centers, schools, offices, and other public buildings. If a child has metallic mercury on his or her clothing, skin, or hair, the fire department should be advised and the child should be properly decontaminated (ATSDR 1997).

Some Chinese herbal remedies for stomach disorders contain mercury (probably as mercury sulfide). If these herbal remedies are made into teas and are given to children, they increase the risk of harmful effects (Espinoza et al. 1995, 1996). Some remedies are in the form of herbal balls, which are aromatic, malleable, earth-toned, roughly spherical, hand-rolled mixtures of primarily herbs and honey. These herbal balls are used as a self-medication for a wide variety of conditions, including fever, rheumatism, apoplexy, and cataracts. Herbal balls similar to those analyzed by Espinoza et al. (1995, 1996) are readily available in specialty markets throughout the United States. Ingesting two herbal balls (the recommended adult dose per day) could theoretically provide a dose of up to 1,200 mg mercury; even if the mercury is in the form of mercuric sulfide, a relatively less bioavailable form, there is an increased risk of mercury entering the body. If a pregnant woman or nursing mother uses mercury-containing herbal remedies, she may also pass the mercury to her unborn child or nursing infant via breast milk. Herbal remedies that contain mercury should be stored so that children can not reach them to prevent accidental poisoning.

Consumers should check the ingredients of any prescription or non-prescription medicine. Hoet and Lison (1997) recently reported an unusual non-occupational source of mercury exposure in a woman who used prescription nasal drops over a long period of time that contained 300 mg/L (ppm) borate phenylmercury. Prescription medicines that contain mercury should be stored out of children’s reach to avoid accidental poisoning.

Children may be exposed to mercury during play at home or in school when using art supplies that contain colors from mercury compounds. Rastogi and Pritzi (1996) reported the migration of several toxic metals including mercury from crayons and artist watercolor paints (see Section 5.4). Migration of mercury from these art supply products occurred in 57% of the samples tested. The authors believe that children might be exposed not only to mercury, but to several other metals that can migrate from the paints. Grabo (1997) also reported that artists may be exposed to mercury because it is a main component in airbrush and brush painting pigments as well as a component of pastel chalks. Artist supplies that contain mercury should be stored out of children's reach to avoid accidental poisoning.

Infants and developing fetuses may be exposed to methylmercury if their mothers consume certain methylmercury-contaminated fish, shellfish, or wildlife species from contaminated waters prior to their pregnancy, during their pregnancy, or while nursing. Older children also may be exposed to methylmercury by eating contaminated fish and wildlife species. Certain states, Native American tribes, and U.S. Territories have issued fish and wildlife advisories for mercury in fresh water, estuarine, and saltwater fish and in freshwater turtles (see Section 5.7).

In a study of lactating women, Oskarsson et al. (1996) assessed the total and inorganic mercury content in breast milk and blood in relation to fish consumption and amalgam fillings (see Section 5.5). In breast milk samples collected 6 weeks after delivery, about half of the total mercury was inorganic and half was methylmercury, whereas in blood samples only 26% was inorganic and 74% was methylmercury. Exposure of the infant to mercury from breast milk was calculated to range up to 0.3 µg/kg/day, of which approximately one-half was inorganic mercury. This exposure corresponds to approximately one-half the tolerable daily intake of total mercury for adults recommended by WHO. The authors concluded that efforts should be made to decrease total mercury burden in women of reproductive age (Oskarsson et al. 1996).

Two-year-old children seem to be different in their weight-adjusted intake of methylmercury as shown by the results of the FDA Total Diet Study. Expressed on a per weight basis, methylmercury intake for all age groups except 2-year-old children was approximately 50 ng/kg/day (Clarkson 1990; Gunderson 1988). For 2-year-old children, the intake was estimated to be approximately 100 ng/kg/day (assuming 50% of the fish intake was due to fish caught locally) or about twice as much methylmercury intake per body weight as for other age groups. For additional details, see Section 5.5, General Population and Occupational Exposure.

Parental exposure can result in subsequent exposure to the developing child or embryo. Anttila and Sallmen (1995) report some epidemiologic data suggesting that paternal exposure to mercury is associated with an increase in spontaneous abortions. These authors also report that maternal exposure to mercury has not been has not been associated with an increased risk of abortion. Lauwerys et al. (1987) reported a case of mercury poisoning in a 3-month-old infant whose mother frequently used a skin lightening cream and soap containing inorganic mercury during pregnancy and the 1-month lactation period following birth. Prenatal and early postnatal exposure of infants to mercury from maternal use of these products is a source of particular concern (Lauwerys et al. 1987).

Data from the National Occupational Exposure Survey (NOES) conducted by NIOSH from 1983 to 1986, provides information on exposure to a variety of mercury compounds, with estimates of the total numbers of workers and the total number of female workers potentially exposed. As presented in Table 5-19, an estimated 50,468 women (33% of workers) were potentially exposed to mercury and various mercury compounds in occupational settings during 1983–1986 (RTECS 1998). More current estimates are not available for the number of women occupationally exposed to mercury in the United States or the percentage of women of reproductive age that may become pregnant or may breast-feed their infants while continuing to work in these occupational settings.

Mercury exposure also may result from the transport of mercury to a workers' home on contaminated clothing and shoes (ATSDR 1990; Hudson et al. 1987; Zirschky 1990). Increased exposure to mercury has been reported in children of workers who are occupationally exposed to the compound (Hudson et al. 1987). Hudson et al. 1987 investigated the exposure to mercury of children of workers in a thermometer manufacturing plant. These investigators reported that the median mercury concentrations in the homes was 0.25 μg/m^3 (range, 0.02–10 μg/m^3), and the levels of mercury in the urine of the children averaged 25 μg/L (ppb), about five times higher than that reported for the controls. While measurements of clothing contamination were not made, the authors noted that elevated mercury concentrations were found in places where work clothes were located and in some washing machines. The children at the highest risk are those whose parents work in facilities that use mercury, but where no protective uniforms or footgear are used. The mercury from these settings is thought to be transferred to the workers' homes on their clothing and shoes. Danzinger and Possick (1973) reported that mercury particles became embedded in the clothing of workers at a scientific glassware plant, especially in knitted fabrics. In an exposure study of families of workers at a chloralkali plant in Charleston, Tennessee, mercury levels in the air of the workers' homes averaged 0.92 μg/m^3 (ATSDR 1990). Although protective clothing was used, work gloves, clothes, and

boots which were soaked with mercury were taken home, exposing family members. Cases of mine workers' homes being contaminated have also been reported, although the authors did not address the impact of this contamination on the health of the family members (West and Lim 1968). Although prevention of this kind of employee transport of mercury to homes is preferred, cleaning homes of workers occupationally exposed to mercury can be effective in reducing exposure for family members (Zirschky 1990).

5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to individuals who are occupationally exposed to mercury (Section 5.5), there are several groups within the general population with potentially high exposures (i.e., higher than background levels) to metallic mercury and various mercury compounds. Historically, populations that have been exposed to higher-than-normal background levels of mercury in the air, water, soil, and/or food have included populations near industrial discharges (e.g., Minamata and Niigata, Japan) and those who inadvertently consumed methylmercury-contaminated food (e.g., grain in Iraq) (WHO 1990, 1991). People living in proximity to former mercury production facilities or mines, secondary mercury production (recycling) facilities, chloralkali facilities, municipal and medical waste incinerators, other mercury-disposal or recycling facilities, or the 714 current or former NPL hazardous waste sites where mercury has been detected (HazDat 1998) are at risk of receiving potentially higher-than-normal background levels of exposure.

Populations with potentially high exposure include recreational and subsistence fishers and hunters, Native American populations who routinely consume larger amounts of locally caught fish than the general population or who consume marine mammals in their diet. Other populations with potential for higher than average exposures are individuals with large numbers of dental amalgams, those who use various consumer products containing mercury (i.e., skin lightening creams and soaps, ethnic remedies, or fingerpaints and make-up paints containing mercury or mercury compounds), and those living or working in buildings recently painted with mercury-containing latex paints or buildings where mercury has been intentionally or unintentionally spilled.

Individuals Living Near Mercury Production, Use, and Disposal Sites. Individuals in the general population living in the vicinity of former primary production or mining sites or current secondary production sites, chloralkali plants, pulp and paper mills, coal-fired power plants, facilities where mercury is released (e.g., municipal waste or medical waste incinerators or other waste disposal facilities), or hazardous

waste sites may be exposed to mercury through several exposure pathways, including inhalation, dermal, and oral exposures. For example, numerous studies have reported increased levels of mercury in air, water, soil, plants, and fish in areas surrounding industrial facilities involved in production or use of mercury (Harnly et al. 1997; Lodenius and Tulisalo 1984; Shaw et al. 1986; Yamaguchi et al. 1971). Significant concentrations of mercury have been detected in sewer overflows and urban runoff (Murphy and Carleo 1977). Thus, general population exposure to mercury may be higher in both industrial and urban areas. Mercury has been detected in various environmental media (air, surface water, groundwater, soil, sediment, and fish and wildlife samples) collected at some of the 714 NPL sites where it has been detected in some environmental media (HazDat 1998). Populations living near hazardous waste sites may be at risk for exposure to high levels of mercury as a result of mercury contamination of surface waters, groundwater, soils, or fish. However, the available data are insufficient to allow for the characterization of the sizes of these populations or the intake levels of mercury to which they are exposed. In 1996, however, De Rosa et al. (1996) reported than in terms of populations at risk, an estimated 41 million people in the United States live within a 4-mile radius of at least one of the 1,134 NPL sites, and 3,300 people live within a 1-mile radius of an NPL site. These authors also reported that metallic mercury was ranked third on the top 10 priority list of hazardous substances found at these NPL sites.

Adults may receive higher mercury exposures from dermal contact if they work with mercury-contaminated soils. Mercury has been detected in soil and sediment at 350 and 208 sites, respectively, of the 714 NPL sites where it has been detected in some environmental media (HazDat 1998). No experimental information on dermal exposure related to the bioavailability of mercury or mercury compounds sorbed to soils was found. However, Hursh et al. (1989) conducted a study to determine the role of dermal exposure in the uptake of mercury vapor from air. These authors estimated that during an 8-hour day, a person would absorb through the skin only 2.6% of the mercury vapor retained by the lungs exposed to the same atmosphere. These authors also noted that half of the dermal uptake is lost through normal shedding of the stratum corneum. Therefore, dermal uptake of mercury adsorbed to soil is likely to be minor compared to other exposure pathways. Recent information from Harnly et al. (1997) showed that urine mercury levels in a Native American population living near an inactive mercury mine in Clear Lake, California were comparable to background levels, indicating that soil and dust exposures were not substantially elevated in the resident population near the inactive site. However, the mean blood methylmercury level in residents of this same community that consumed fish from Clear Lake was 15.6±8.8 µg/L (ppb), which was more than 7 times higher than the mean blood level in individuals that did not consume fish from the lake (2 ppb).

In addition, adults may receive potentially higher oral exposures from ingestion of mercury-contaminated soils from their unwashed hands while working in mercury-contaminated areas. Bioavailability is an integral factor in the estimation of the internal dose (or dose at the target tissue) of the chemical. Like dermal absorption, gastrointestinal absorption of various forms of mercury is highly variable (see Section 2.3.1). The more lipid soluble organic mercury compounds (e.g., methylmercury) are almost completely absorbed, while the extremely insoluble metallic mercury is poorly absorbed through the gut. The bioavailability of mercury from soil is likely to vary, since mercury binds tightly to soil, especially to soils with high organic content. Therefore, the mercury soil concentration alone may not be indicative of the potential for human health hazard from contaminated soils, and site-specific evaluation of the bioavailability of the various forms of mercury at the site is essential. However, unless toxicokinetic studies that use soil samples from the specific site are available, it is difficult to speculate on how much mercury will be bioavailable at any particular site. Adults may also receive higher doses from routine consumption of mercury-contaminated home grown fruits and vegetables (Nublein et al. 1995), and from consumption of fish from local waters receiving runoff or leachate from a waste site. Harnly et al. (1997) studied the impact of inorganic mercury in soil and dust and organic mercury in fish on a Native American population living near an inactive mercury mine near Clear Lake, California. These authors reported average methylmercury blood levels of 15.6±7 µg/L (ppb) in individuals that consumed fish from Clear Lake, which was higher than blood levels reported for individuals that did not consume fish (2 ppb). A significant correlation of methylmercury blood levels and fish consumption was observed. Mercury has been detected in fish collected at 56 of the 714 NPL sites where it has been detected in some environmental media (HazDat 1998). Adults may also receive higher mercury exposures from routine consumption of mercury-contaminated groundwater if this is the primary drinking water supply. Mercury has been detected in groundwater samples collected at 395 of the 714 NPL sites where mercury has been detected in some environmental media (HazDat 1998).

Individuals living near municipal and medical waste incinerators, power plants fired by fossil fuels (particularly coal fired plants), or hazardous waste sites may inhale vapors or particulates contaminated with mercury from ambient outdoor air. Lipfert et al. (1996) evaluated the health risks of methylmercury from burning coal using a Monte Carlo model to simulate a “baseline” and a “worst case” scenario in which a population of 5,000 fish eaters in the upper midwestern United States derived the freshwater fish portion of their diet from local waters near a large, hypothetical coal-fired power plant. The population was characterized by distributions of body mass, half-life of methylmercury, and the ratios of blood to body burden and hair to blood methylmercury. Each person’s diet consisted of varying amounts of tuna fish,

freshwater sportfish, and marine fish and shellfish, the methylmercury content of which were characterized by national distribution statistics, as were the consumption rates for marine fish. The consumption rates for freshwater fish were specific to the region. The fish portion size was linked to body mass by a variable correlation. Each meal was assumed to be an independent sample, so that as metabolic equilibrium was approached, each person's body burden of methylmercury tended to approach the value corresponding to the mean methylmercury intake for the population. Predictions of methylmercury levels in hair by this model compared well with an observed distribution in 1,437 women. Two neurological end points were examined: adult paresthesia as related to methylmercury body burden and congenital neurological effects as associated with average concentrations of methylmercury in maternal hair during pregnancy. In the baseline exposure scenario, the source of the mercury in fish was background atmospheric deposition. In the worst-case scenario, local mercury deposition and concentrations in fish were roughly doubled to represent additional deposition from the hypothetical power plant. For both scenarios, the 99th percentile of methylmercury body burden was more than an order of magnitude below the lowest level at which increased transient paresthesia in adults was experienced in an acute methylmercury poisoning incident in Iraq. The authors concluded that neurological risks to adults from methylmercury resulting from atmospheric deposition are negligible. Based on three epidemiological studies of congenital neurological risks, they found that fetal effects appeared to be more critical, and that there is a smaller margin of safety for pregnant consumers of freshwater sportfish. However, there is still a considerable margin of safety, and uncertainties in the relationships between maternal hair mercury and actual fetal exposures may have overstated the fetal risk (Lipfert et al. 1996).

Recreational and Subsistence Fishers. Methylmercury concentrations in sport fish can be at least an order of magnitude higher than in commercial fish purchased in a supermarket (see Section 5.4.4). Therefore, recreational and subsistence fishers, including some Native American peoples who consume locally caught fish from mercury-contaminated waterbodies or consume long-lived predatory oceanic species such as shark and swordfish, can be exposed to higher mercury concentrations than individuals who consume similar amounts of commercially marketed fish from a variety of sources (Ebert et al. 1996; EPA 1995k). The exposure to mercury will also be higher among people who regularly eat fish and other seafood products, compared to those who only occasionally or never eat fish or other seafood products. This increased exposure has been demonstrated by blood mercury levels several times higher in people who regularly eat fish, compared to those who occasionally or never eat fish (Buzina et al. 1989; Cappon and Smith 1982; Oskarsson et al. 1996; Phelps et al. 1980; Svensson et al. 1995). In addition, the consumption of certain species of fish (e.g., shark and swordfish) is likely to contribute disproportionately to the observed

methylmercury body burden. Because mercury is associated primarily with muscle tissue in the body of a fish, rather than with fatty deposits, trimming and skinning of mercury-contaminated fish does not reduce the mercury content of the fillet portion, as is the case for PCBs, dioxins, and other organochlorine pesticides (Armbuster et al. 1988; Gutenmann and Lisk 1991).

Several recent studies have documented higher fish consumption rates among subsistence fishers, some of which are Native American populations. In 1990, there were an estimated 1,959,234 Native Americans in the United States, including 1,878,285 American Indians, 57,152 Eskimos, and 23,797 Aleuts (Paisano 1998). Approximately 218,320 Native Americans were living on ten reservations and tribal lands, and these people accounted for half of all Native Americans living on reservations. Therefore, approximately 440,000 Native Americans live on reservations. The median family income in 1990 for Native Americans was $21,750, about 65% of the $35,225 median income of all U.S. families. In addition 27% of all Native Americans are living in poverty, compared with 10% of the general population. In a study of 11 Alaskan communities, Nobmann et al. (1992) reported an average daily fish consumption rate of 109 g/day. This average consumption rate for subsistence fishers is more than 16.8 times the mean fish consumption rate of 6.5 g/day estimated for the general population (EPA 1995k). A recent study of fish consumption patterns among the Umatilla, Nez Perce, Yakama, and Warm Springs tribes of the Columbia River Basin in Washington and Oregon (CRITFC 1994) found that adults in these tribes consume an average of 59 g/day and that the 95th percentile of fishers consume 170 g/day of fish. The mean consumption rate for the four tribes is more than nine times the mean fish consumption rate estimated for the general population (EPA 1995k). Furthermore, the consumption rate for Native American children (5 years and younger) from these four tribes was 20 g/day (a rate over 3 times that for adults in the general population) (see Section 5.6).

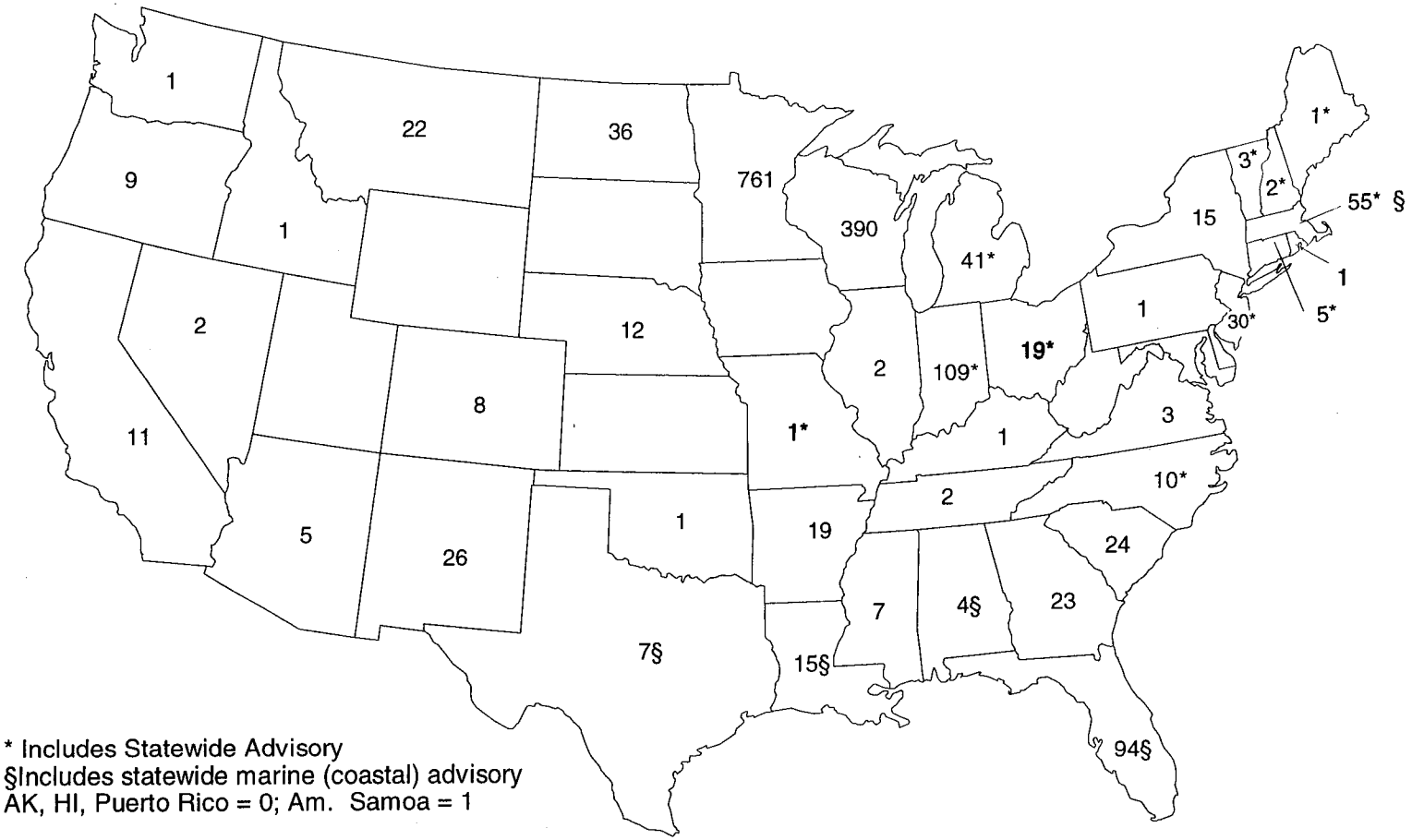
In order to reduce methylmercury exposure from consumption of mercury-contaminated fish and shellfish, consumption advisories are issued by states recommending that individuals restrict their consumption of specific fish and shellfish species from certain waterbodies where mercury concentrations in fish and shellfish tissues exceed the human health level of concern. This level of concern is set by individual state agencies, but several states use the FDA action level of 1 ppm to issue advisories recommending no consumption or restricting consumption of contaminated fish and shellfish from certain waterbody types (e.g., lakes and/or rivers). The FDA value was designed to protect consumers from the health risks associated with consumption of fish and shellfish that are shipped in interstate commerce and that are purchased in commercial markets. The FDA action level was not intended to be used as a criterion for the

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

To address this concern, the EPA Office of Water issued guidance to states on sampling and analysis procedures to use in assessing the health risks from consuming locally caught fish and shellfish. The risk assessment method proposed by EPA was designed to assist states in developing fish consumption advisories for recreational and subsistence fishers, including pregnant women, nursing mothers, and children in these high-end consumption populations (EPA 1995k). Recreational and subsistence fishers consume larger quantities of fish and shellfish than the general population and frequently fish the same waterbodies routinely. Because of this, these populations are at greater risk of exposure to mercury and other chemical contaminants, if the waters they fish are contaminated. The EPA's Office of Water advises states to use a screening value of 0.6 ppm mercury (wet weight) in fillets for the general population as a criterion to evaluate their fishable waters (EPA 1995k). Currently, 1,782 advisories restricting the consumption of mercury-contaminated fish and shellfish are in effect in 41 states and one U.S. Territory (American Samoa) (EPA 1998b). The number of mercury advisories currently in effect in each state is shown in Figure 5-7. It should be noted that mercury is the chemical pollutant responsible in part for over 77% of the fish advisories issued in the United States (EPA 1998a). It is important to note that 11 states (Connecticut, Indiana, Maine, Massachusetts, Michigan, Missouri, New Hampshire, New Jersey, North Carolina, Ohio, and Vermont) currently have state-wide mercury advisories recommending that residents restrict consumption of locally caught freshwater fish. In addition, 5 states (Alabama, Florida, Louisiana, Massachusetts, and Texas) have issued statewide coastal mercury advisories for specific marine fish and shellfish species. In two states (Arizona and Minnesota), wildlife advisories recommending that residents restrict their consumption of freshwater turtles have been issued.

Subsistence Hunters. Native American populations, such as the Inuit of Alaska and other subsistence hunters (particularly those living in high latitude areas of the United States), may be exposed to mercury in wild game (e.g., seals, narwhal, walrus, and other game species or marine mammals). Mercury has been detected in liver, kidney, and muscle tissues of pilot whales, harp seals, narwhal, and walrus (Meador et al. 1993; Wagemann et al. 1995). Mean total mercury concentrations and methylmercury concentrations were highest in pilot whale liver tissue: 176 ppm (dry weight) and 8 ppm (dry weight), respectively. In fish, almost all of the mercury (>95%) body burden is methylmercury (Bloom 1992), but in marine mammals, the percentage of inorganic mercury is much higher, at least in liver tissue. For example, in Alaskan beluga whales, mean methylmercury levels were 0.788 ppm (μg/g wet weight), but mean total mercury levels were

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

28 ppm (wet weight), in liver tissue (Becker et al. 1995). Similarly, in Alaskan ringed seal, mean methylmercury levels were 0.410 ppm (wet weight) and mean total mercury levels were 1.970 ppm (wet weight) in liver tissue. However, no information was available for methylmercury levels in muscle tissue from Alaskan mammals. An older report by Smith and Armstrong (1975) also examined total mercury and methylmercury levels in marine mammal livers eaten by native Inuit in the Northwest Territory of Canada. Smith and Armstrong (1975) reported total mercury concentrations of 143 and 26.2 ppm (wet weight) and mean methylmercury levels of 0.300 and 0.120 ppm (wet weight) in liver tissue of bearded seals sampled in 1973 and 1974, respectively. Smith and Armstrong (1975) also reported total mercury concentrations of 27.5 ppm (wet weight) (maximum, 184 ppm), and 0.72 ppm in liver and muscle tissue, respectively, and mean methylmercury levels of 0.96 and 0.83 ppm in liver and muscle tissue, respectively, of ringed seals sampled near Victoria Island in Canada's Northwest Territory. These authors also reported a mean total mercury concentration of 143 ppm and a mean methylmercury concentration of 0.30 ppm in liver tissue of bearded seals. The mean total mercury concentration in the muscle tissue of the bearded seals was 0.53 ppm (no methylmercury concentrations in muscle tissue were available for this species).

In Greenland, the percentage of total mercury that was methylmercury in seal muscle tissue was 57–86%; however, the concentration of total mercury was very low. Mercury concentrations in the blood of mothers and infants in Greenland were closely correlated with the amount of marine mammal meat the mothers consumed. Mercury concentrations in the blood of mothers eating primarily imported food ranged from 11.0 to 32.7 µg/L (ppb) and concentrations in the blood of their children ranged from 15.0 to 51.4 µg/L (ppb). In contrast, mercury concentrations in the blood of mothers who consumed primarily a local diet heavy in marine animals ranged from 16.4 to 44.6 µg/L (ppb) and concentrations in the blood of their children ranged from 27.5 to 140.0 µg/L (ppb) (Hansen 1991).

Native American populations that depend heavily on marine mammals are considered to be at higher risk than the general population. Wheatley and Paradis (1995a, 1995b) reported blood mercury levels in native peoples from 514 communities across Canada. Of these individuals, 23% had methylmercury blood levels >20 µg/L (the WHO assessment level), while 1.6% of these individuals had blood levels >100 µg/L (the WHO benchmark for at-risk populations). Native American populations in the western Arctic (Alaska) may be at similar risk as a result of their consumption of marine mammals, although no recent information on methylmercury concentrations in blood, hair or urine for these populations was located. In Alaskan Inuit women that consume marine mammal tissue, Galster (1976) reported higher total mercury levels in breast

milk of women living in coastal areas (7.6±2.7 ppb) than in breast milk of Inuit women living in the interior (3.2±0.8 ppb) or in urban areas (3.3±0.5 ppb). In addition, mercury red blood cell concentrations were also higher in Inuit women living in coastal areas (33.5±5.1 ppb), as compared to those living in the interior (22.6±3.0 ppb) or in urban areas (8.9±0.9 ppb). Higher mercury levels in coastal populations were attributed to higher consumption of seal meat and oil and marine fish (Galster 1976). By analogy to the Canadian populations of native peoples (Wheatley and Paradis 1995a, 1995b), it is anticipated that methylmercury concentrations in these tissues are likely to be higher among individuals who consume large quantities of marine mammal species with high concentrations of methylmercury (as well as inorganic mercury) in their tissues than among members of the general population. In a study of subsistence economies in the State of Alaska, Wolfe and Walker (1987) reported that total annual per capita harvest of wild game species (including land mammals, marine mammals, and fish) ranged from 10 to 1,498 pounds (median harvest of 252 pounds), compared to 222 pounds of meat, fish, and poultry (combined) consumed each year per individual in the western United States. The wild game harvest in 84% of the 98 Alaskan subsistence communities surveyed was at least half or greater than the 222 pounds consumed in the western United States. Because hunters often share wild game they harvest with other family members, the amount harvested may not represent the actual amount consumed (Egeland et al. 1998). The average daily per capita consumption was estimated to be 0.67 pounds of fish and 0.23 pounds of land mammals based on all 98 communities, and 0.2 pounds of marine mammals based on the 41 coastal communities surveyed. Marine mammals consumed in these communities included seal, walrus, and whales. Subsistence hunters and their families are a population at potentially higher risk of mercury exposure, if the wild game species they consume are contaminated with high concentrations of inorganic and methylmercury. Although the existence of larger amounts of mercury in subsistence diets does give cause for concern, the available Alaskan data do not support the conclusion that current exposures are a serious problem for Alaskan subsistence hunters (Egeland et al. 1998).

Individuals with Large Numbers of Dental Amalgams. Individuals with dental amalgams have greater exposure to elemental mercury than members of the general population that do not have dental amalgams. Richardson (1995) computed a release rate per filled tooth surface of 0.73 µg/day-surface, with a standard deviation of 0.3 µg/day-surface and a “stimulation magnification factor” of 5.3, based on a weighed average enhancement of mercury vapor concentration following chewing, eating, or tooth brushing

reported in three amalgam studies. Patterson et al. (1985) measured elemental mercury in exhaled breath, and levels of mercury ranging from 0.0001 to 62 ng/L (ppb) (mean, 0.0082 µg/L [ppb]) were detected in 167 persons with dental restorations, compared to 0.000008–0.0001 µg/L (ppb) (mean, 0.00006 µg/L [ppb]) in 5 persons with no amalgams; however, these values were measured after the people had brushed their teeth. Jokstad et al. 1992 reported that mercury urine concentrations increased with increasing number of amalgams. Individuals with 36 to 39 dental amalgams had mercury urine levels of 6 ppb compared to 1.2 ppb in individuals without amalgams. Mercury concentrations in whole blood were also higher in persons who ate no fish, but had >6 dental amalgam fillings (mean, 1.047±0.797 µg/L [ppb] as compared to persons who did not eat fish and had no dental amalgams (0.2±0.4 µg/L [ppb]) (Schweinberg 1994). Individuals who have large numbers of dental amalgams installed or replaced at one time are likely to exhibit transient elevated blood and urine mercury levels (PHS 1995).

Individuals Exposed to Consumer Products and Medicinal Products Containing Mercury. Individual who use various consumer products containing mercury (i.e., medicinal herbal remedies, skin lightening creams and soaps, laxatives, tattoo dyes, fingerpaints, and make-up paints) are also exposed to higher mercury levels than the general population (Barr et al. 1973; Dyall-Smith and Scurry 1990; Espinoza et al. 1995; Geffner and Sandler 1980; Lauwerys et al. 1987; Rastogi 1992; Wendroff 1990). Metallic mercury has been used by Mexican American and Asian populations in traditional remedies for a variety of medical conditions, including chronic stomach disorders. Several papers have been published related to the use of metallic mercury as a folk remedy (ATSDR 1992, 1997; Department of Health 1997; Geffner and Sandler 1980; Hartman 1995; Johnson [in press]; Trotter 1985; Wendroff 1990, 1991; Zayas and Ozuah 1996). Some Mexican-Americans believe that disorders of the alimentary tract may be caused by a bolus of food adhering to the stomach wall, a condition known as *empacho*. Geffner and Sandler (1980) reported cases of two young patients with acute gastroenteritis who received traditional remedies of oral administration of metallic mercury, presumably to dislodge the bolus. Both patients were successfully treated and released from the hospital after 2 and 10 days of treatment, respectively. Trotter (1985) reported that metallic mercury known as *azogue* is in common use in New Mexico and the bordering areas for treating this gastrointestinal condition, *empacho*. Metallic mercury was also implicated in two cases of mercury poisoning caused by the dermal application of an over-the-counter antilice product (Bourgeois et al. 1986). Wands et al. (1974) reported the deaths of two individuals due to the excessive use of a laxative

preparation containing mercurous chloride (calomel). Espinoza et al. (1995) reported that while examining imported Chinese herbal balls for the presence of products from endangered species, the authors detected potentially toxic levels of mercury and arsenic in certain herbal ball preparations. Herbal balls are aromatic, malleable, earth-toned, roughly spherical, hand-rolled mixtures of primarily herbs and honey. These herbal balls are used as a self-medication for a wide variety of conditions, including fever, rheumatism, apoplexy, and cataracts. Herbal balls similar to those analyzed are readily available in specialty markets throughout the United States. Mercury (probably mercury sulfide) was detected in 8 of the 9 herbal balls tested. The recommended adult dose for the herbal balls is two per day. Ingesting two herbal balls could theoretically provide a dose of up to 1,200 mg of mercury. Perharic et al. (1994) reported poisonings resulting from exposure to traditional remedies and food supplements reported to the National Poisons Unit in London, England. From 1989 to 1991, metallic mercury was implicated in several poisonings following exposure to Asian medicines. The issuance of informational notices by health departments cautioning members of these subpopulation about the toxic properties of mercury may be appropriate.

Mercuric sulfide, or cinnabar, was reported to be used in tattooing dyes to produce a red pigmentation (Bagley et al. 1987; Biro and Klein 1967). An analysis of finger paints and make-up paints manufactured in Europe showed that they all contained less than 1 ppm mercury (Rastogi 1992). The author did not discuss whether these products are available in the United States. While some of medicinal and pharmaceutical uses of mercury compounds have been replaced in recent years, individuals in some ethnic or religious groups may still use mercury in various traditional remedies, ceremonies, and rituals.

Individuals that Use Mercury in Religious Ceremonies and/or Ethnic Practices or Live in Dwellings where Intentional or Unintentional Elemental Mercury Spills have Occurred.

Metallic mercury has been used in Latin American and Caribbean communities as part of certain religious practices (e.g., Voodoo, Santeria, and Espiritismo) predominantly in domestic settings (Wendroff 1990). Metallic mercury is sold in shops called botanicas (sometimes under the name *azogue)* which stock medicinal plants, magical medicines, incense, candles, and perfumes. Botanicas typically dispense mercury in gelatin capsules or, sometimes, in small glass vials. Some practices involve sprinkling metallic mercury on the floor of the dwelling or of a car, mixing elemental mercury with soap and water to wash the floor, or placing it in an open container to rid the house of evil spirits. Other practices involve carrying a small amount of mercury in a vial on the person or mixing mercury in bath water or perfumed soaps, devotional

candles, ammonia, or camphor. Any of these practices can liberate mercury vapor into the room air exposing the occupants to unnecessarily elevated levels of mercury vapors (ATSDR 1997; Wendroff 1990, 1991). The issuance of cautionary notices by health departments to members of these user populations may be appropriate. While some medicinal and pharmaceutical uses of mercury compounds have been replaced in recent years, individuals in some religious and ethnic groups may still use mercury in various rituals. This use of mercury can contaminate the dwelling if the mercury is not removed from flooring, carpeting, and woodwork in an appropriate manner.

Individuals Living in Homes Where Mercury-containing Latex Paints Have Been Used. Prior to 1991, phenylmercuric compounds were used as biocides in 25–30% of interior and exterior latex paints; however, this use of mercury was voluntarily discontinued for interior paint in 1990 and for exterior paint in 1991 (Hefflin et al. 1993; Reese 1990). This use of phenylmercury resulted in the exposure of house painters and residents to elemental mercury vapors in homes where interior or exterior latex paint was applied. The concentration of mercury in interior paints was less than 200 ppm; however, the atmospheric concentrations of elemental mercury vapor were found to be as high as 200 $\mu g/m^3$ less than 6 hours after painting, 10 $\mu g/m^3$ at 24 hours, and 6 $\mu g/m^3$ after 1 month. Although the use of mercury biocides in latex paint has been discontinued, it is possible that people who use old latex paint in their homes will be exposed to mercury for a considerable time (Blondell and Knott 1993). Furthermore, although phenylmercury use in exterior latex paints was discontinued in 1991, paint companies were allowed to continue to produce and sell paint containing phenylmercury until the existing stocks of phenylmercury were exhausted. Paint produced after 1990 containing phenylmercury must be so labeled. Exterior latex paints may have contained phenylmercury at concentrations of up to 1,500 ppm, and their use has been shown to result in elevated mercury levels in painters (see Section 5.5) (Hefflin et al. 1993). However, each year many homeowners (66%) repaint their own homes, rather than employing professional painters; therefore, these individuals may also be exposed (Hefflin et al. 1993). In addition, consumers can mistakenly use exterior paints indoors, which may produce higher exposures to mercury than when the paints are used outdoors. Blondell and Knott (1993) estimated that approximately 13 million people could be exposed to mercury through painting, assuming the interior of houses were painted once every 5 years, that 78% of the interior paint used is latex, and that one-third of the interior latex paint contained mercury. These authors emphasize that key populations at risk include the painters, residents in the painted homes and children living in those homes.

5.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of mercury is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of mercury.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

5.8.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of metallic mercury and its inorganic and organic compounds have been well characterized to permit estimation of their environmental fate (Lewis 1993; Merck 1989; NFPA 1994; Osol 1980; Spencer and Voigt 1968; Verschueren 1983; Weast 1988; Weiss 1986). Most values are available for the log K_{ow}, log K_{oc}, Henry's law constant, vapor pressure, and solubility in water. Experimental data exist that allow characterization of the environmental fate of metallic mercury and inorganic and organic mercury compounds in a variety of environmental media.

Production, Import/Export, Use, Release, and Disposal. Information on mercury production, import/export, and use are well documented (Blayney et al. 1997; Drake 1981; EPA 1997a; Hefflin et al. 1993; IARC 1993; Jasinski 1993; Reese 1990; Reiber and Harris 1994; Toribara et al. 1997; USGS 1997).

Information on disposal methods and recycling of mercury and mercury containing wastes are available (Carrico 1985; DOI 1989; Jasinski 1993; TRI96 1998).

One area that requires additional study is the use of elemental mercury by members of specific religious or ethnic groups in their ceremonies, rituals, and practices so an assessment of the magnitude of these activities can be made. In addition, information on how mercury is used in these ceremonies and rituals, as well as the methods of mercury disposal used, would be helpful in assessing the potential pathways for human exposure and environmental releases.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI), which contains this information for 1996, became available in May 1998. This database will be updated yearly and should provide a list of industrial production facilities and emissions.

Environmental Fate. Mercury released to the atmosphere may be transported long distances before being removed by wet or dry deposition. Residence time in the atmosphere has been estimated to range from 60–90 days to 0.3–2 years (EPA 1984b; Glass et al. 1991). Volatile forms of mercury released in water or soil can enter the atmosphere, but most mercury is adsorbed to soil and sediment (EPA 1984b; Meili et al. 1991). Sorbed mercury may be reduced to elemental mercury or bioconverted to volatile organic forms (EPA 1984b). The major transport and transformation processes involved in the environmental fate of mercury have been fairly well defined; the most important fate process for human exposure, bioaccumulation of methylmercury in aquatic food chains is also well defined (Callahan et al. 1979; EPA 1984b; Stein et al. 1996). Additional information on mercury transport and flux in waterbodies would be helpful.

Bioavailability from Environmental Media. Metallic mercury vapors in the air are readily absorbed through the lungs following inhalation exposure, while inorganic and organic mercury compounds are poorly absorbed via this route (Berlin et al. 1969). Gastrointestinal (GI) absorption of methylmercury is nearly complete, while GI absorption of inorganic mercury is low (typically <10%) (Clarkson 1989; Friberg and Nordberg 1973). Metallic mercury vapor can be absorbed following dermal exposure; however, dermal absorption of the vapor accounts for a much smaller percentage (2.6% of the total absorbed through the lungs) than absorption through the inhalation route (Hursh et al. 1989). Inorganic mercury salts and organomercury compounds can also be dermally absorbed to some extent (Blayney et al. 1997; Junghaus

1983; Schamberg et al. 1918; Toribara et al. 1997). Data are needed regarding the bioavailability of elemental, inorganic, and organic mercury forms from contaminated surface water, groundwater, soil, or plant material. Data are also needed regarding the bioavailability of mercuric chloride in air because of the possibility of inhalation of volatilized mercuric chloride near emission sources. Additional data on the bioavailability of elemental mercury, inorganic mercury compounds, and organic mercury compounds (specifically, methylmercury) in soil would also be useful in assessing the risks from dermal and oral exposures at mining, industrial, or hazardous waste sites.

Food Chain Bioaccumulation. Mercury is known to bioconcentrate in aquatic organisms and biomagnify in aquatic food chains (ASTER 1997; EPA 1984b; Jackson 1991; Kohler et al. 1990; Mason et al. 1995, 1996; Porcella 1994; Watras and Bloom 1992). While bioconcentration in the aquatic food chain is well studied, little is known about the bioaccumulation potential for terrestrial food chains, although it appears to be smaller than in aquatic systems (Lindqvist 1991a). Additional information on the potential for terrestrial food chain biomagnification would be useful in light of the binding of mercury to organic matter in soils and sediment. Information on foliar uptake of mercury and of plant/mercury chemistry is needed to determine whether plants convert elemental or divalent mercury into other forms of mercury that are more readily bioaccumulated and whether plants are able to emit these different forms to the air. Additional information is also needed to improve biotransfer factors for mercury from soil to plants to animals.

Exposure Levels in Environmental Media. Environmental monitoring data are available for mercury in ambient air, surface water, groundwater, drinking water, soils, sediments, and foodstuffs (EPA 1984b, 1985; Glass et al. 1990; Lindqvist 1994); however, additional monitoring data on mercury levels in all environmental media, particularly drinking water, would be helpful in determining current exposure levels. Estimates of human intake from inhalation of ambient air and ingestion of contaminated foods and drinking water are available (Burger et al. 1992), although the estimates may be based on specific intake scenarios (e.g., information is most extensive for fish and other seafood products). Better estimates of fish consumption rates for high-end consumers (subsistence fishers) and recreational fishers is needed, as is information on fish-specific consumption rates by these populations. Additional information on the levels of mercury in foods other than fish and seafood would be very useful in determining total dietary intakes. Additional research is needed to characterize mercury exposures via consumption of marine mammal species. Available data indicate that the ratio of methylmercury to total mercury varies within tissues, and that only a small portion of mercury is methylated in the marine mammal's liver. Also, other trace metal constituents of marine mammal tissues such as selenium, cadmium, and other metals may interact with and

influence the bioavailability of mercury. Additional studies are needed in order to understand why the relatively high concentrations of mercury measured in marine mammal tissues do not appear to result in elevation of hair mercury levels among Alaskan natives that consume marine mammal tissues.

Reliable monitoring data for the levels of mercury in contaminated media at hazardous waste sites are needed so that the information obtained on levels of mercury in the environment can be used in combination with the known body burden of mercury to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Exposure Levels in Humans. Mercury has been measured in human blood, hair, breast milk, urine, feces, and saliva (Bakir et al. 1973; EPA 1984b; Fujita and Takabatake 1977; Galster 1976; Oskarsson et al. 1996; Pitkin et al. 1976; Wheatley and Paradis 1995a, 1995b; WHO 1990). However, current information on mercury levels in blood, hair, breast milk, and urine of members of the general U.S. population are almost entirely lacking. Data are needed for the general population that measure the levels of mercury in blood, hair, breast milk, and urine derived from dietary exposures (such as fish consumption) versus mercury derived from dental amalgams in order to obtain additional information about the importance of each of these exposure pathways to resulting mercury body burden. Additional information on mercury levels in urine of persons with varying numbers of amalgam surfaces as well as in persons that have had amalgam fillings removed or replaced would be useful in evaluating mercury exposure form this source. Data are available for some Native American populations (Galster 1976) and several foreign populations that consume large amounts of locally caught fish and wildlife (Airey 1983b; Fleming et al. 1995; Lasora and Citterman 1991). The most common method of assessing human exposure in the workplace involves the measurement of mercury in urine (Baser and Marion 1990; Bell et al. 1973; Lindstedt et al. 1979; Roels et al. 1987; Rosenman et al. 1986). Urine mercury levels have been correlated with ambient air exposure levels, particularly to mercury vapor. A longitudinal epidemiological study that tracks individual exposure levels to metallic mercury vapors in occupational settings (chloralkali industry workers, fluorescent lightbulb manufacturers, or other mercury utilizing industries) on a daily basis and associated these exposure levels with weekly urine and blood samples for a period of 1–2 years is needed. Neurobehavioral testing should also be conducted of these workers at 6-month intervals. Workers new to these industries would make the best subjects since they could provide pre-exposure blood and urine levels as a point of reference. Information is available on populations living near former production sites or hazardous waste sites (Harnly et al. 1997; Nublein et al. 1995; Reif et al. 1993; Shaw et al. 1986). Additional information on the biological monitoring of populations living in the vicinity of hazardous waste

sites would be helpful in estimating exposure of these populations to mercury compounds. This information is useful for assessing the need to conduct health studies on these populations.

Exposures of Children. Children are exposed to mercury by a variety of exposure pathways depending on their age. The most important pathways appear to be via inhalation of metallic mercury vapors, intake of inorganic mercury associated with dental amalgams in children up to 18 years old, and ingestion of methylmercury in foods primarily fish and shellfish. These are the same important pathways of exposure for adults as well. Infants can also be exposed to mercury from mother's milk. More data are needed on the levels of mercury exposure in nursing women from inhalation of metallic mercury in occupational or domestic situations, including religious and ethnic uses (ATSDR 1997; Johnson [in press]; Wendroff 1990, 1991; Zayas and Ozuah 1996); from use of commercial or hobby arts and crafts (Grabo 1997; Rastogi and Pritzi 1996); from mercury-containing herbal remedies, cosmetics, and prescription drugs (Al-Saleh and Al-Doush 1997; Barr et al. 1973; Dyall-Smith and Scurry 1990; Espinoza 1995, 1996; Lauwerys et al. 1987; Perharic et al. 1994); and from consumption of mercury-contaminated fish and wildlife, including marine mammals (CRITFC 1994; Egeland et al. 1998; Oskarsson et al. 1996). Exposure and body burden studies especially related to consumption of freshwater fish in the U.S. populations are needed to determine exposure levels, particularly in the children of recreational and subsistence fishers. Individual members of freshwater sport fish species in the Northeastern United States have been found to have tissue concentrations as high as 8.94 ppm mercury, while some species have mean tissue concentrations as high as 0.77 ppm (NESCAUM 1998). Exposure and body burden studies are also needed in Alaskan populations of subsistence hunters that consume large amounts of marine mammal tissues. Existing data on levels of mercury in breast milk in Alaskan women (Galster 1976) are dated and may not reflect either current levels of mercury contamination in fish and wildlife or dietary habits of Inuit or other subsistence fishing/hunting populations.

A unique exposure pathway that has received little research attention is the exposure to children from religious and ethnic uses in homes and cars or in remedies containing metallic mercury (ATSDR 1997; Johnson [in press]; Wendroff 1990, 1991). In some religious practices of Latin American or Caribbean origin, there are traditional rituals or remedies that involve mercury. These include intentional sprinkling of liquid elemental mercury on the floor, burning candles made with mercury, using mercury in baths, adding it to perfume, or wearing small containers of mercury around the neck for good luck. There is an urgent need to obtain information on the levels of exposure from these practices to determine if children or adults are at risk. Mercury vapor concentrations may be much higher after use during the winter months when the heat is

turned on and the windows are closed, so data that reflect a variety of possible exposure scenarios are also needed.

Results of the Total Diet Study conducted by the FDA suggest that two-year-old children differ in their weight-adjusted intake of mercury, based on the assumption that 50% of the fish consumed were locally caught species (Clarkson 1990; Gunderson 1988). Additional information on weight adjusted intakes would be helpful for the general population, and particularly in determining the health risks for young children in Native American populations. Children in these populations may consume relatively large quantities of locally caught fish as part of their traditional ceremonial practices (CRITFC 1994) or may consume large quantities of marine mammal tissues (blubber, muscle, and organ meats) if they are in subsistence fishing or hunting populations.

One childhood-specific means of decreasing exposure scenarios for children is through better education of school age children and their parents on the health risks particularly of metallic mercury exposure from accidental spillage, intentional uses, or from improper industrial exposures.

Exposure Registries. New York State has instituted a Heavy Metals Registry that monitors occupational exposure to heavy metals, including mercury. Cases are reported when mercury exposure is equal to or exceeds 50 µg/L (ppb) in blood or 20 µg/L (ppb) in urine. Between 1982 and 1986, 1,000 cases of mercury exposure were reported and linked to 47 companies. Most exposures (494 cases) occurred in workers in the alkali and chlorine industry, where mercury is used as a cathode because exposure occurs when the cells are opened; the median blood mercury concentration was 76 µg/L (ppb) (maximum concentration 916 µg/L [ppb]). The second most frequent exposure category (213 cases) was the manufacture of industrial instruments, such as the manual assembly and fabrication of thermometers; median blood mercury concentration was 145 µg/L (ppb) and the maximum concentration was 889 µg/L (ppb) (Baser and Marion 1990).

This substance is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to this substance.

5.8.2 Ongoing Studies

A search of Federal Research in Progress (FEDRIP 1998) identified numerous research studies that are currently being conducted that may fill some of the data needs discussed in Section 5.8.1. Ongoing studies and long-term research concerning occupational or general population exposures to mercury and studies that address the issue of the religious and ethnic uses of elemental mercury are presented in Table 5-22.

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

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

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

6. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting, and/or measuring, and/or monitoring mercury, its metabolites, and other biomarkers of exposure to and effects of mercury. The intent is not to provide an exhaustive list of analytical methods. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used for environmental samples are the methods approved by federal agencies and organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that modify previously used methods to obtain lower detection limits, and/or to improve accuracy and precision.

The analysis of metals in biological and environmental samples is complicated by the different organic and inorganic forms of the metal that may be present. For mercury, this complication is usually overcome by reducing all the mercury in the sample to its elemental state prior to analysis; this solution is not appropriate when information about the individual mercury species is desired. Mercury has an additional problem of being relatively volatile and, therefore, easily lost during sample preparation and analysis. In spite of these complications, several methods have been developed for determining trace amounts of mercury in biological and environmental samples, even in complex media. Careful attention must be paid to inadvertent contamination of the sample with mercury, especially when determining trace concentrations. Labware (glass or Teflon) should be thoroughly cleaned and acid-leached before being used for trace-level analysis. It has been shown that final soaking of laboratory ware, particularly Teflon, in hot (70 EC) 1% HCL removes any traces of oxidizing compounds (e.g., chlorine) that may subsequently destroy methylmercury in solution (Horvat 1996). Appropriate method blanks must be included.

Attention must be paid also to sample preservation to avoid perturbing the distribution of mercury compounds in the sample (Horvat 1996). The preservation of aqueous samples is often accomplished using acidification. However, suspended matter must be removed prior to acidification and dimethylmercury and Hg(0) have to be removed or else conversion of these species into methylmercury and mercury(II) can occur (Horvat 1996). For solid matrices, the preservation method of choice is freezing (Bloom 1993). Freezing preserves all major mercury species indefinitely, although coagulation will occur for sediments thus making it difficult to obtain representative subsamples of the sediment for analysis. For most metals, such storage

issues would be solved by drying the samples first, but for mercury, especially methylmercury, there is a risk of losses from volatilization. Tissue samples may be freeze-dried without loss of methylmercury. Repeated freezing and thawing of wet, biological samples can also cause loss of methylmercury (Horvat and Byrne 1992) but such degradations are dependant on the matrix.

Numerous standard or certified reference materials exist for verifying the reliability of new or modified methods, especially for total mercury; standard reference materials for individual organomercury species can be more difficult to obtain. The existing methods for determining mercury in biological and environmental matrices are described more fully in the following sections.

6.1 BIOLOGICAL MATERIALS

Many researchers have attempted to determine mercury levels in the blood, urine, tissues, and hair of humans and animals. Most methods have used atomic absorption spectrometry (AAS), atomic fluorescence spectrometry (AFS), or neutron activation analysis (NAA). In addition, methods based on mass spectrometry (MS), spectrophotometry, and anodic stripping voltametry (ASV) have also been tested. Of the available methods, cold vapor (CV) AAS is the most widely used. In most methods, mercury in the sample is reduced to the elemental state. Some methods require predigestion of the sample prior to reduction. At all phases of sample preparation and analysis, the possibility of contamination from mercury found naturally in the environment must be considered. Rigorous standards to prevent mercury contamination must be followed. Table 6-1 presents details of selected methods used to determine mercury in biological samples. Methods have been developed for the analysis of mercury in breath samples. These are based on AAS with either flameless (NIOSH 1994) or cold vapor release of the sample to the detection chamber (Rathje et al. 1974). Flameless AAS is the NIOSH-recommended method of determining levels of mercury in expired air (NIOSH 1994). No other current methods for analyzing breath were located.

In recent years, increasing attention has been paid to human exposure to mercury via dental amalgams (Skare 1995). Exposure results from elemental mercury vapor released from amalgams that is either inhaled directly or swallowed after dissolution in saliva. A Jerome 511 Gold Film Mercury Vapor Analyzer (Arizona Instrument Corp., Jerome, AZ) has been used to measure mercury vapor released from amalgam during routine dental procedures (Engle et al. 1992) or at other times to establish baseline exposure data (Halbach 1995). Accuracy and precision data were not reported. Although the detection limit for this method was not reported, mercury concentrations at μg concentrations are detectable. A similar instrument

Table 6-1. Analytical Methods for Determining Mercury in Biological Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Breath	Personal sampler (collection of an aliquot of air); analysis of sample at 253.7 nm.	AAS (flameless)	1 ng/sample	No data	NIOSH 1984 (method 6000)
Breath	Fasten a hopcalite sampling tube to a temple of the worker's safety glasses; draw air through the sampler.	CVAAS	No data	No data	Rathje et al. 1974
Human whole blood	Treatment of sample with dilute hydrochloric acid; addition of a pH buffer and a complexing agent (diethyldithiocarbamate); extraction of mercury species into toluene	ETAAS	2 µg/dm^3	>94%	Emteborg et al. 1992
Blood	Cleavage of both organic and inorganic mercury from blood protein thiol groups using hydrochloric acid, extraction of mercury species into toluene as their diethyldithiocarbamate (DDTC) complexes; addition of Grignard reagent to toluene phase to form butyl derivatives of the mercury species	GC/MPD	0.4 µg/L	>100%	Bulska et al. 1992
Blood and erythrocytes (inorganic, total)	Digestion of sample with H_2SO_4 (mixture of nitric and perchloric for total) overnight, reduction with $SnCl_2$, purging onto gold wire to form amalgam (preconcentration) followed by thermal release of elemental mercury.	CVAAS	0.06 ng/g (0.06 ppb) for total; 0.04 ng/g for inorganic.	75–114%	Bergdahl et al. 1995
Blood and urine	Dilution of sample in ammonia buffer; reduction with sodium borohydride	ICPAES	0.5 µg/L	100	Buneaux et al. 1992
Blood and urine	Total mercury: precipitation-extraction with 50% volume/volume hydrochloric acid containing EDTA and cysteine; centrifugation; filtration through screening column. Methyl mercury: extraction of the methyl mercury into benzene or toluene; back extract into aqueous cysteine solution	ICP-MS	0.2 µg/L	91.6–110.2	Kalamegham and Ash 1992
Blood, plasma, urine (total)	Digestion of blood and plasma samples overnight in a mixture of nitric acid and perchloric acid	CVAAS	5 nmol/L	93.4–103	Vesterberg 1991
Blood, urine, tissues (inorganic)	Dilution of blood or urine sample with water; homogenization of tissue samples with water; reduction of mercury with $SnCl_2$ followed by purging to detector	CVAAS	≈6 µg/L	77–110	Friese et al. 1990

Table 6-1. Analytical Methods for Determining Mercury in Biological Samples (continued)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Blood, breast milk (total, inorganic)	Digestion of sample with nitric/perchloric acid overnight for total, and with H_2SO_4 overnight for inorganic; reduction and purging	CVAAS	0.1 ng/g (blood); 0.04 ng/g (milk)	97%	Oskarsson et al. 1996
Blood (total)	Irradiation of sample followed by treatment with permanganate, sulfuric acid, distilled water, ammonia, and hydroxylamine hydrochloride; treatment with ion exchange.	NAA	0.3 ng/mL	100%	Fung et al. 1995
Serum, bovine liver (total)	Digestion of sample with HNO_3 and heat in closed container in microwave oven; reduction with $SnCl_2$ and TBP; purging to gold-coated sand adsorber to preconcentrate (amalgamation); thermally desorb to detector	CVAAS	0.84 ng/g	93–111	Vermeir et al. 1989
Urine (total)	Digestion with $HNO_3/HClO_3$ and heat; evaporation; addition of NH_4Cl/ammonium solution; dilution with water	ASV	NR	100–105	Liu et al. 1990
Urine (total)	Addition of HCl to sample followed by bromate/bromide solution and equilibration for 15 minutes; decomposition of excess bromine by addition of hydroxylamine hydrochloride.	AFS	1 ng/L	95-98% (methyl mercury, phenyl mercury)	Corns et al. 1994
Urine, tissue, hair (total)	Digestion of sample with HNO_3 in closed vessel in microwave; cooling and dilution with water; reduction with $SnCl_2$; purging to detector	AFS	0.9 ng/L	94–102	Vermeir et al. 1991a, 1991b
Blood, urine, hair, fish (total, methyl Hg)	Total: digestion of sample with nitric, perchloric, and sulfuric acids; Methyl mercury in hair: digestion with HCl and extraction into benzene. Methyl mercury in blood, fish, and urine: digestion with KOH and extraction into dithizone solution, cleaned up via extractions.	Total: CVAAS, methyl mercury: GC/ECD	0.5 ng	No data	Akagi et al. 1995

Table 6-1. Analytical Methods for Determining Mercury in Biological Samples (continued)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Tissue, hair	Washing of hair sample with acetone and water; homogenization of hair or tissue sample in micro dismembrator; irradiation; addition of carriers; digestion with concentrated HNO_3/ H_2SO_4 solution and heat in a closed Teflon bomb; extraction of digest with $CHCl_3$ to remove bromide ion, extraction of aqueous phase with Zn-$(DDC)_2$/$CHCl_3$; counting of ^{197}Hg in organic phase	NAA	0.36 ng/g (tissue) 3.6 ng/g (hair)	85–110	Zhuang et al. 1989
Liver tissue (methyl mercury)	Extraction of sample with toluene; concentration of methylmercury in aqueous phase; mixing with bacterial cells and incubation in microreaction vessel; injection of headspace gas containing methane	GC/FID	15 ng	NR	Baldi and Filippelli 1991
Hair	Washing of samples with acetone and water; digestion with HNO_3 and heat; oxidation with permanganate solution and heat; cooling and addition of hydroxylamine hydrochloride; reduction of mercury with $SnCl_2$; purging to detector	CVAAS	NR	100–101	Pineau et al. 1990
Wrist and temporal areas	None	XRF	20 µg/g	No data	Bloch and Shapiro 1986

AAS = atomic absorption spectrometry; AFS = atomic fluorescence spectrometry; ASV = anodic stripping voltametry; $CHCl_3$ = trichloromethane; CVAAS = cold vapor atomic absorption spectrometry; ECD = electron capture detection; ETAAS = electrothermal atomic absorption spectrometry; FID = flame ionization detector; GC = gas chromatography; $HClO_3$ = perchlorous acid; Hg = mercury; HNO_3 = nitric acid; H_2SO_4 = sulfuric acid; ICPAES = inductively coupled plasma atomic emission spectroscopy; ICP-MS = inductively coupled plasma-mass spectrometry; MPD = microwave-induced plasma emission; NAA = neutron-activation analysis; NH_4Cl = ammonium chloride; NR = not reported; $SnCl_2$ = tin(II) chloride; TBP = tri-*n*-butyl-phosphate; XRF = X-ray fluorescence; Zn-$(DCC)_2$ = zinc diethyldithiocarbamate

(Jerome 431X Mercury Vapor Analyzer) was used by Chien et al. (1996) to measure elemental mercury vapor released from dental amalgams in the oral cavity and was reported to have a sensitivity of 0.003 mg/m^3. Absorbed mercury can be measured using blood and urine measurements as described below.

CVAAS is the primary method that is used to determine mercury in blood and serum (Friese et al. 1990; Ngim et al. 1988; Vermeir et al. 1988, 1989; Vesterberg 1991). Using CVAAS, concentrations in the sub- to low-ppb can be reliably measured. Both direct reduction of sample (Friese et al. 1990; Ngim et al. 1988) and predigestion followed by reduction (Oskarsson et al. 1996; Vermeir et al. 1988, 1989) produced good accuracy and precision. However, with predigestion techniques, best results were obtained on samples that were heated in a closed teflon container in a microwave oven and preconcentrated on gold-coated sand (Vermeir et al. 1989). A complimentary method to CVAAS for total mercury determination in blood is electrothermal atomic absorption (ETAAS) (Emteborg et al. 1992). Recoveries are excellent and sensitivity is 2 µg/dm^3. GC/microwave-induced plasma atomic emission detection (MPD) can also be used to measure both organic and inorganic mercury in blood samples (Bulska et al. 1992). Sensitivity is in the sub-ppb range, and recovery is excellent (100%).

Methylmercury and inorganic mercury were extracted from human whole blood samples, as their diethyldithiocarbamate complexes, into toluene and butylated them by using a Grignard Reagent (Bulska et al. 1992). The mercury species were then detected by a microwave-induced plasma atomic emission spectrometric system (GC/MPD). The absolute detection limit was calculated to be 1 pg of mercury in either the methylmercury or inorganic mercury form. This corresponds to a detection limit of about 0.4 µg/L. The method is reproducible. Methods for inorganic mercury and organic mercury (mostly methylmercury) have been reported for blood, urine, hair, and breast milk (Akagi et al. 1995; Bergdahl et al. 1995; Oskarsson et al. 1996). Total mercury is typically determined using CVAAS after complete conversion of all mercury to the volatile elemental form using harsh (nitric acid/perchloric acid, bromate/bromide) digestions followed by reduction of ionic mercury to the elemental form. Inorganic mercury can be determined after milder digestions (HCl, sulfuric acid) and reduction. The organic form is determined by the difference between total and inorganic. Sub-ng/g (ppb) detection limits are routine. Methylmercury is also determined using GC with electron capture detection (ECD) (Akagi et al. 1995).

There is evidence to suggest that urinary mercury levels are good measures of exposure to inorganic mercury in the environment (Ikingura and Akagi 1996). The primary method used to analyze urine for mercury is CVAAS (Akagi et al. 1995; Friese et al. 1990; Ngim et al. 1988; Oskarsson et al. 1996; Ping

and Dasgupta 1989, 1990; Vesterberg 1991). Methods using AFS (Corns et al. 1994; Vermeir et al. 1991a, 1991b), ASV (Liu et al. 1990), and isotope-dilution spark source (IDSS) MS have also been developed. CVAAS is sensitive (low-ppt), reliable (recovery is >76% and precision is generally <10% relative standard deviation [RSD]), and may be used on either digested or undigested samples (Friese et al. 1990; Ngim et al. 1988; Ping and Dasgupta 1989, 1990). Improved sensitivity (sub-ppt), accuracy (>90% recovery), and precision (7% RSD or better) were obtained with AFS when samples were digested in a closed container in a microwave (Vermeir et al. 1991a, 1991b). Good results have also been achieved with ASV (Liu et al. 1990) and IDSSMS (Moody and Paulsen 1988). The precision of these methods is especially high (<5% RSD), and recoveries with ASV are >90%. Both these methods require predigestion of the sample. As an alternative to CVAAS, total mercury determination in blood and urine can be performed by inductively coupled plasma-atomic emission spectroscopy (ICP-AES) or ICP-mass spectrometry (Buneaux et al. 1992; Kalamegham and Ash 1992). These methods are sensitive, with detection limits in the sub-ppb range. Recoveries (>90%) and precision (<17% coefficient of variation [CV]) are good.

AAS-based methods and NAA have been used to measure mercury in tissues. The AAS methods differ in the way the sample is released for detection. CVAAS is the best-defined of the AAS techniques. Mercury concentrations in the sub- to low-ppb have been reliably determined in tissue samples (Friese et al. 1990; Vermeir et al. 1988, 1989). Best results were obtained when the sample was digested in a closed container in a microwave oven, and the vaporized mercury was preconcentrated on gold-coated sand (Vermeir et al. 1989). Flameless AAS, which uses an electric furnace to atomize the mercury, has yielded high recoveries, but no data are available on the sensitivity or precision of the technique (Ichinose and Miyazawa 1989). Separative column atomizer AAS (SCA-AAS) introduces the mercury to the detector by running the sample through a heat-activated charcoal column (Yanagisawa et al. 1989). Little sample preparation is required, but high background interference is a problem with this method. Good results were reported for tissue samples with sub-ppm mercury concentrations (from control rats), but decreased accuracy and precision occurred in samples containing higher levels (from dosed rats). AFS offers a good alternative to CVAAS. Sensitivity was in the sub-ppt range, and recovery and precision were excellent (Vermeir et al. 1991a, 1991b). In addition, sample preparation is relatively simple and rapid. NAA permits determination of mercury in tissue samples at the sub- to low-ppb level, but erratic accuracy and precision make the method less reliable (Taskaev et al. 1988; Zhuang et al. 1989). An extraction method using zinc diethyldithiocarbamate produced good results with NAA (Zhuang et al. 1989). GC equipped with a flame ionization detector (FID) has also been used to detect methylmercury in tissues at ng levels (Baldi and Filippelli 1991). Recovery and precision data were not reported.

Studies have indicated that the mercury concentration in the hair correlates well with dietary mercury exposure (Inasmasu et al. 1986; Wilhelm and Idel 1996). Methylmercury is the primary dietary mercury contaminant and is present in large amounts in seafood (Ikingura and Akagi 1996). Most of the mercury measured in hair is methylmercury; hair is a good matrix for assessing exposure to methylmercury (Wilhelm and Idel 1996). Hair analysis has been conducted using CVAAS, AFS, and NAA (Grandjean et al. 1992; Ngim et al. 1988; Pineau et al. 1990; Suo et al. 1992; Suzuki et al. 1992; Taskaev et al. 1988; Vermeir et al. 1991a, 1991b; Zhuang et al. 1989). Segmental hair analysis is commonly used as a means of determining an historical record of exposure or uptake of mercury (Grandjean et al. 1992; Suzuki et al. 1992). The method involves cutting the hair strands into smaller segments, usually 1 cm each, and analyzing each segment separately. Detection limits for hair using CVAAS were not reported but are expected to be similar to those for tissue (sub- to low-ppb). The sensitivity of NAA is similar to that of CVAAS, but variable recoveries and precision make NAA less reliable. Good results were reported for one NAA method (Zhuang et al. 1989). Results from studies using AFS suggest this method may be the most sensitive and reliable technique (Suo et al. 1992; Vermeir et al. 1991a, 1991b). A detection limit in the sub-ppt range was obtained, and precision and accuracy were both excellent.

An X-ray fluorescence (XRF) technique has been used to measure mercury in the wrist and temporal areas of dentists exposed to various heavy metals in the work place (Bloch and Shapiro 1986). This technique allows simultaneous evaluation of the tissue burden of a number of different metals. Bone levels may be more closely related to long-term exposure than levels in blood, urine, and hair. The detection limit for XRF is in the low ppm.

A method for detecting methylmercury in biological samples by its enzymatic conversion to methane is an alternative biological technique for methylmercury or other organomercurial analyses (Baldi and Filippelli 1991). *Pseudomonas putida* strain FB1, a broad spectrum mercury-resistant strain, is able to enzymatically convert methylmercury to Hg^0 and methane either in whole cell or in cell-free extracts. GC/FID was used to determine methane produced by the biological derivatization of methylmercury. The detection limit was 15 ng of methylmercury extracted from 1 g of biological tissue. The coefficient of variation was 1.9%. Chemical interferences are negligible in the enzymatic determination of methyl-mercury. The specificity of this determination places the method among the most reliable ones. Recovery was not reported.

6.2 ENVIRONMENTAL SAMPLES

Mercury levels have been determined in numerous environmental matrices, including air, water (surface water, drinking water, groundwater, sea water, and industrial effluents), soils and sediments, fish and shellfish, foods, pharmaceuticals, and pesticides. The sample preparation varies with the complexity of the matrix, but most complex samples require decomposition of the matrix and reduction of the mercury to its elemental form. As described Section 6.1 for biological samples, special sample preparation methods need to be employed if inorganic and organic mercury are to be determined separately, or if the individual species of the organic mercury fraction are to be determined. More detailed information on selected methods in various environmental samples is given in Table 6-2.

Both CVAAS and CVAFS have been used to monitor air and suspended particulates in air for mercury (Baeyens and Leermakers 1989; Bloom and Fitzgerald 1988; Friese et al. 1990; NIOSH 1994; Paudyn and Van Loon 1986; Sengar et al. 1990; Stockwell et al. 1991; Temmerman et al. 1990). Both methods are sensitive, accurate, and precise, although slightly greater sensitivity was reported with AFS (low ppt) than with AAS (mid ppt); AFS is becoming a more common method of analysis (Horvat 1996). When AAS or AFS was combined with gas chromatography (GC), the different mercury species (inorganic mercury, dimethylmercury, diethylmercury, and methylmercury chloride) present in the air could be separated (Bloom and Fitzgerald 1988; Paudyn and Van Loon 1986). A colorimetric method, based on the formation of a colored complex formed in the presence of mercury, has been used as a quick and simple field test that can detect mercury present at the mid-ppb level (Cherian and Gupta 1990).

Numerous methods, including CVAAS, ASV, inductively coupled plasma (ICP) MS, ICP atomic emission spectrometry (AES), microwave-induced plasma (MIP) AES, NAA, GC/AFS, high-performance liquid chromatography (HPLC)/UV, HPLC/ECD, and spectrophotometry, have been used to determine mercury levels in aqueous media. Mercury has been measured in drinking water, surface water, groundwater, snow, waste water effluents, and sea water. Of the available methods, CVAAS is the method of choice (Baxter and Frech 1989, 1990; Birnie 1988; Eaton et al. 1995; Goto et al. 1988; Lee et al. 1989; Mateo et al. 1988; Munaf et al. 1991; Paudyn and Van Loon 1986; Ping and Dasgupta 1989; Robinson and Schuman 1989; Schintu et al. 1989; Shkinev et al. 1989) and the method recommended by EPA and AOAC (AOAC 1984; Beckert et al. 1990; EPA 1994f, 1994g). This method is very sensitive for mercury in water (sub- to low-ppt) and has been proven to be reliable. Water samples generally do not require digestion, but mercury in the samples is usually reduced to the elemental state and preconcentrated prior to analysis. When combined

Table 6-2. Analytical Methods for Determining Mercury in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air (elemental)	Drawing of air through Hopcalite sorbent tube, dissolution of sorbent with HNO_3 then HCl, dilution, addition of stannous chloride	CVAAS	3 µg/m^3	100 (4.6% RSD at 0.9 µg)	NIOSH 1994 (Method 6009)
Air (methyl mercury)	Passage of air through a Tenax column, thermal desportion.	GC/AAS	0.1 ng/m^3 (methyl mercury)	No data	Paudyn and Van Loon 1986
Air (elemental mercury)	Preconcentration of mercury in sample by collection onto gold-coated sand absorber; thermal desorption and collection onto second absorber; desorb to detector	CVAFS	<1 ng/m^3	105–111	Temmerman et al. 1990
Air (methyl, dimethyl mercury)	Preconcentration of sample onto graphitized carbon substrate; separation by cryogenic gas chromatography	GC/CVAFS	0.3 pg (mercury, dimethyl mercury); 0.4 pg (diethyl mercury); 2 pg (methyl mercury chloride)	91–105	Bloom and Fitzgerald 1988
Air	Collection of sample onto gold-coated quartz wool; thermal desorption	CVAAS	0.08 ng	97–101	Friese et al. 1990
Water (total)	Addition of H_2SO_4/HNO_3 and $KMNO_4$, equilibrate, addition of $K_2S_2O_8$ and heating; addition of hydroxylamine, reduction to elemental mercury using stannous chloride, purging of sample	CVAAS	<1 µg/L (1 ppb)	79–92 (9–23% RSD)	Eaton et al. 1995 (Standard Method 3112B/3500B)
Water	Addition of permanganate and sulfuric acid and heating; addition of $K_2S_2O_8$ and hydroxylamine; extraction with dithizone	Spectro-photometry at 492 nm	2 µg/L (2 ppb)	95 at 250 µg/L	Eaton et al. 1995 (Standard Method 3500C)
Water (inorganic mercury)	Reduction of sample with $SnCl_2$ in HNO_3; purging of mercury to detector	CVAAS	0.1 ng/L	99	Lee et al. 1989
Water, sea water (inorganic)	Reduction of mercury in sample with $SnCl_2$; preconcentration onto platinum-lined graphite tube	GFAAS	<2 ng/L	94–102	Baxter and French 1989

Table 6-2. Analytical Methods for Determining Mercury in Environmental Samples (continued)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Water, waste water	Oxidation of organic Hg to inorganic Hg by $KMnO_4$, $K_2S_2O_8$ and heat; reduction to elemental state with stannous ion	AAS (flameless)	No data	No data	AOAC 1984 (methods 32.095 to 33.099)
Water (total)	Digestion of sample with HNO_3/H_2SO_4 plus permanganate and persulfate solutions; reduction with hydroxylamine; purging to detector	CVAAS	0.1 mg/L	101–112	Beckert et al. 1990; EPA 1994f (method 7470a)
Water and snow	Extraction of organic mercury immediately after sampling; addition of organic mercury standards; addition of KB and a benzene/ toluene mixture; isolation and volume reduction of organic layer	GC/AAS	4 ng (dimethyl mercury); 5 ng (ethyl mercury)	No data	Paudyn and Van Loon 1986
Drinking water	Collection of sample in quartz ampoule and evaporation followed by irradiation; precipitation of Hg as sulfide; isolation of precipitate and dissolution in aqua regia; counting of ^{203}Hg	NAA	45 µg/L	95–107	Itawi et al. 1990
Water (total)	Digestion with $HNO_3/HClO_4$ and heat; volume reduction; addition of NH_4Cl/ ammonium solution; dilution with water	ASV	No data	100–105	Liu et al. 1990
Surface water	Acidification of sample with HNO_3; addition of ^{199}Hg; oxidization with potassium permanganate solution; reduction with sodium borohydride; purging to plasma	ICP/IDMS	ng/L	86–98	Haraldsson et al. 1989
Drinking water	Direct injection	DIN-ICPMS PN-ICPMS	30–40 ng/L	No data	Powell et al. 1992
Drinking water and groundwater	Separation of mercury species in sample on HPLC column using buffered methanol as eluent	HPLC/ECD	≈1.8 µg/L	77–104	Evans and McKee 1988

Table 6-2. Analytical Methods for Determining Mercury in Environmental Samples (continued)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Drinking water, sea water	Reduction of sample with $SnCl_2$ in H_2SO_4; oxidation with $KMnO_4$; addition of Cd(IV), sodium arsenite, iodide, and H_2SO_4 to thermometric cell; inject sample	Kinetic thermometry	≈2 µg/L	No data	Mateo et al. 1988
Waste water (total)	Digestion of sample with HNO_3/H_2O_2 and heat; cooling and adjustment of pH to 6; equilibration with polyacrylamidoxime; stripping of Hg from resin by equilibration with HNO_3; filtration	ICP/AES	1.15 µg/L	96–98	Mahanti 1990
Waste water	Addition of HNO_3 to sample and evaporation; redissolution in water (pH should be ≈2); separation on ion chromatography column using $TPPS_4$/PAR/NaCl/$Na_2B_4O_7$-NaOH as post-column derivatization agent	Spectro-photometry	50 µg/L	No data	Yan et al. 1989
Industrial effluents	Concentration of sample by heating; digestion with concentrated H_2SO_4; dilution; addition of sodium salicylate to aqueous sample; adjustment of pH to 5.5–5.8; extraction with triphenylphosphine oxide solution; for high levels of Hg (mg), back-extraction with acetate buffer and analysis; for low levels of Hg (µg), addition of sodium hydrogen carbonate buffer and PAN; isolation of organic layer; dilution with toluene and analysis	Spectro-photometry	No data	99–119	Raman and Shinde 1990
Soil (total)	None	XRF	10 µg/g	No data	Grupp et al. 1989
Soil (total)	Digestion of sample with HCl/HNO_3 with heat in closed Teflon vessel in microwave; dilution; reduction with $SnCl_2$ and hydroxylammonium chloride in H_2SO_4; purging to detector	CVAAS	No data	90–110	Van Delft and Vos 1988

Table 6-2. Analytical Methods for Determining Mercury in Environmental Samples (continued)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Soil, sediment, sludge (total)	Digestion of sample with aqua regia and permanganate in steam bath or with HNO_3/H_2SO_4 and permanganate in autoclave; reduction with hydroxylamine; purging to detector	CVAAS	0.1 mg/L	84–101	Beckert et al. 1990; EPA 1994g (method 7471a)
Soil, sediment (methyl Hg, phenyl Hg)	SFE of spiked sample using CO_2methanol containing diethylammonium diethyldithio-carbamate; dilution with octane, addition of pentylMgBr to form pentyl derivatives, addition of H_2SO_4, extraction of organic phase with water, treatment with anhydrous magnesium sulfate	GC/AED	2.5 ng/mL in extract	106 (methyl) 6.3% RSD; 59 (phenyl) 12% RSD)	Liu et al. 1994
Sediment, mussel (total)	Digestion of sample with concentrated acid; evaporation; redissolution in HNO_3 and dilution with water; reduction of sample with $SnCl_2$ in HNO_3; purging to detector	CVAAS	0.1 ng/L	111 (sediment); 60 (mussel)	Lee et al. 1989
Sediment (total)	Digestion of sample with HCl/HNO_3 and heat in Teflon bomb; oxidation with potassium permanganate solution; reduction with sodium borohydride; purging to plasma	ICP/MS	≈2 ng/g	96	Haraldsson et al. 1989
Solid samples (total)	Introduction of a slurry of sample in nitric acid into FIA system using on-line microwave digestion, mix with tin(II) chloride to form elemental mercury	CV-AFS	0.09 ng/g	84–108 (2.9–4% RSD)	Morales-Rubio et al. 1995
Fish (methyl mercury)	Homogenization of sample; extraction with HCl/KBr/ $CuSO_4$/toluene solution; centrifugation; mixing of organic phase with cysteine and centrifugation; mixing of aqueous phase with HCl/KBr/ $CuSO_4$/toluene solution; centrifugation; drying of organic phase over anhydrous Na_2SO_4	GC/ECD	50 ng/g (methyl mercury)	89–111	Ahmed et al. 1988

Table 6-2. Analytical Methods for Determining Mercury in Environmental Samples (continued)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Fish	Drying of visceral parts of fish in oven; digestion of sample with concentrated H_2SO_4; dilution and filtration of digest; addition of sodium salicylate to aqueous sample; adjustment of pH to 5.5–5.8; extraction with triphenylphosphine oxide solution; for high levels of Hg (mg), back-extraction with acetate buffer and analysis; for low levels of Hg (µg), addition sodium hydrogen carbonate buffer and PAN; isolation of organic layer; dilution with toluene and analysis	Spectro-photometry	No data	99–119	Raman and Shinde 1990
Fish and plant materials	Preparation of sample condensate; dilution with HNO_3; addition of Ag and co-precipi-tatation of Ag and Hg with H_2S; preparation of sample electrode	IDSSMS	0.1 µg/g	95	Moody and Paulsen 1988
Fish and shell fish (methyl mercury)	Homogenization of sample; removal of organics by washing with acetone and benzene; addition of HCl to release protein-bound methyl mercury and extraction into benzene; analysis for methyl mercury chloride	GC/ECD	No data	No data	AOAC 1984 (methods 25.146–25.157)
Fish (total)	Digestion of samples with nitric acid in a microwave acid digestion bomb, reduction to elemental mercury	CVAAS	0.195 ng/mL	>95	Navarro et al. 1992
Fish muscle (total)	Digestion of sample with H_2SO_4-HNO_3	AAS (flameless)	No data	No data	AOAC 1984 (methods 25.134–25.137)
Oyster tissue, milk powder, wheat flour (total)	Digestion of sample with HNO_3 and heat in closed container in microwave over; reduction with $SnCl_2$ and TBP; purging of mercury to gold-coated sand absorber to preconcentrate; desorption to detector	CVAAS	0.84 ng/g	93–111	Vermeir et al. 1989

Table 6-2. Analytical Methods for Determining Mercury in Environmental Samples (continued)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Wheat flour, citrus leaves, pine needles (total)	Digestion of sample using $K_2Cr_2O_7$ and H_2SO_4, heating and dilution	CVAAS	6 ng/g	91–108	Landi et al. 1990
Milk powder, oyster tissue (total)	Digestion of sample with HNO_3 in closed vessel in microwave; dilution with water; reduction with $SnCl_2$; purging to detector	AFS	0.9 ng/L	94–102	Vermeir et al. 1991a, 1991b
Food (total)	Digestion of 5 g of sample with HNO_3-$HClO_4$	AAS (flameless)	No data	No data	AOAC 1984 (methods 25.131–25.133)
Food (total)	Digestion of sample with HNO_3 and H_2SO_4 under reflux; isolation of mercury by dithizone extraction	Colorimetric dithizone method	No data	No data	AOAC 1984 (methods 25.138–25.145)
Wine (total)	Digestion of sample with concentrated HNO_3 and chromic acid; addition of hydroxylamine chlorhydrate to cold flask; transferring to mercury/hydride generator; addition of $SnCl_2$ in H_2SO_4 to reduce; purging to detector	AAS (flameless)	6 µg/L	95–107	Cacho and Castells 1989
Pharmaceuticals	Extraction of mercury with *N*-phenylcinnamohydroxamic acid; measurement of absorbance at 390 nm	Spectrophotometry	No data	No data	Agrawal and Desai 1985
Pharmaceuticals	Removal of lipids from greasy or soapy samples with diethyl ether; digestion of sample with $KMnO_4/HNO_3$; removal of excess permanganate with sodium oxalate; adjustment of pH to alkaline with ammonium chloride/ ammonia buffer and H_2O_2 ; filtration; heating and dilution; titration of mercury with 4,4'-dihydroxybenzophenone	Spectrophotometry	2 µg/L	95–111	Marquez et al. 1988

Table 6-2. Analytical Methods for Determining Mercury in Environmental Samples (continued)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Cigarettes	Digestion by heating with concentrated H_2SO_4; dilutition and filtration of digest; addition of sodium salicylate to sample; adjustment of pH to 5.5–5.8; extraction with triphenylphosphine oxide solution; for high mercury (mg), back-extraction with acetate buffer and analysis; for low mercury (µg), addition of sodium hydrogen carbonate buffer and PAN; isolation of organic layer; dilution with toluene	Spectro-photometry	No data	99–119	Raman and Shinde 1990

AAS = atomic absorption spectrometry; AED = atomic emission detection; AES = atomic emission spectrometry; AFS = atomic fluorescence spectrometry; Ag = silver; ASV = anodic stripping voltammetry; Cd = cadmium; $CuSO_4$ = copper sulfate; CVAAS = cold vapor atomic absorption spectrometry; CVAFS = cold-vapor atomic fluorescence spectrometry; DIN-ICPMS = direct injection nebulizer inductively coupled plasma mass spectrometry; ECD = electron capture detection or electrochemical detector; GC = gas chromatography; GFAAS = graphite furnace atomic absorption spectrometry; HCl = hydrochloric acid; $HClO_3$ = perchlorous acid; $HClO_4$ = perchloric acid; Hg = mercury; HNO_3 = nitric acid; HPLC = high-performance liquid chromatography; H_2O_2 = hydrogen peroxide; H_2S = hydrogen sulfide; H_2SO_4 = sulfuric acid; ICP = inductively coupled plasma; IDMS = isotope-dilution mass spectrometry; IDSSMS = isotope-dilution spark-source mass spectrometry; KB = potassium boride; KBr = potassium bromide; $K_2Cr_2O_7$ = potassium chromate; $KMnO_4$ = potassium permanganate; $K_2S_2O_8$ = potassium sulfhydrate; MS = mass spectrometry; NAA = neutron-activation analysis; $Na_2B_4O_7$ = sodium borohydrate; NaOH = sodium hydroxide; Na_2SO_4 = sodium sulfate; NaCl = sodium chloride; NH_4Cl = ammonium chloride; PAN = 1-(2-pyridylazo)-2-naphthol; PAR = 4-(2-pyridylazo)resorcinol; PN-ICPMS = pneumatic nebulizer inductively coupled plasma mass spectrometry; $SnCl_2$ = tin(II) chloride; TBP = tri-*n*-butyl-phosphate; $TPPS_4$ = meso-tetra(4-sulfonatophenyl)porphyrin; XRF = x-ray fluorescence

with GC, CVAAS has been used to separate and determine individual mercury species in aqueous samples (Paudyn and Van Loon 1986). Spectrophotometry has often been used to determine mercury in aqueous matrices (Abbas et al. 1989; Ajmal et al. 1989; Eaton et al. 1995; Raman and Shinde 1990; Singh et al. 1989). Sample preparation methods vary and have included separation by thin-layer chromatography (TLC) (Ajmal et al. 1989) or column chromatography (Yan et al. 1989), selective extraction (Abbas et al. 1989), and ligand formation (Raman and Shinde 1990; Singh et al. 1989). While recoveries were good, spectrophotometry is not as sensitive a technique as CVAAS. Tests of additional methods, including ASV (Liu et al. 1990), ICP/MS (Haraldsson et al. 1989), NAA (Itawi et al. 1990), AES-based techniques (Kitagawa and Nishimoto 1989; Mahanti 1990; Nakahara et al. 1988), HPLC-based techniques (Evans and McKee 1988; Shofstahl and Hardy 1990), and graphite-furnace (GF) AAS (LeBihan and Cabon 1990) indicate that these methods may also be useful for determining mercury in water samples. One of the most promising methods is GC/AFS, which has the advantages of increased sensitivity and precision compared to CVAAS and can also be used to isolate individual mercury species (Bloom 1989). A colorimetric assay has also been developed that is useful for rapid preliminary screening of field samples (Cherian and Gupta 1990).

CVAAS is the most commonly used technique for determining the mercury concentration of sediments, soils, and sludge (Bandyopadhyay and Das 1989; Beckert et al. 1990; EPA 1994g; Van Delft and Vos 1988). As with other matrices, it is sensitive, reliable, and requires little sample preparation beyond digestion of the matrix and reduction of the mercury to its elemental form. It is the method recommended by EPA for solid matrices (Beckert et al. 1990; EPA 1994g). A method based on CVAFS that uses flow injection analysis with on-line microwave digestion for the determination of total mercury has been described recently (Morales-Rubio et al. 1995). Good sensitivity (90 ppt) and precision (4% RSD) was demonstrated. Gas chromatography in conjunction with atomic emission detection (GC/AED) has been used to determine organomercury species in soils and sediments (Liu et al. 1994). Direct current ASV (DCASV) has been tested for use in determining mercury levels in river sediment (Lexa and Stulik 1989). The accuracy and sensitivity of this method are good, but it is less precise than CVAAS. A field method using XRF has been developed to monitor soil contamination (Grupp et al. 1989). This method is rapid and portable, but its high detection limit (low-ppm) makes it useful only for on-site screening.

Methods have been developed for the determination of mercury in fish, shellfish, foods, food sources, and pharmaceuticals. AAS, usually with cold vapor generation (CVAAS), is one of the primary methods used to measure mercury in these complex matrices (Carrillo et al. 1986; Friese et al. 1990; Landi et al. 1990;

Navarro et al. 1992; Odukoya 1990; Vermeir et al. 1988, 1989), because of its sensitivity and reliability. Although the sensitivity (sub- to low-ppb), accuracy, and precision are not as good as with less complex gaseous and aqueous media, it is still one of the best methods available for analysis of mercury in any matrix. Flameless AAS without cold vapor generation has also produced good results when used to determine ppb levels of mercury in wine (Cacho and Castells 1989) and fish (Filippelli 1987); it is also one of the methods recommended by AOAC for fish and food (AOAC 1984). When combined with high resolution GC (HRGC), the individual organic mercury species in fish could be determined (Jiang et al. 1989). Sub-ppt levels of mercury in powdered milk and oyster tissue were reliably determined using AFS (Vermeir et al. 1991a, 1991b). NAA was used to measure mercury levels in copepod homogenate and tomato leaves, but the sensitivity (mid- to low-ppb) and reliability were not as good as that of CVAAS or AFS (Taskaev et al. 1988; Zhuang et al. 1989). Several other methods, including IDSSMS (Moody and Paulsen 1988), HPLC/ICP/MS (Bushee 1988), square-wave voltametry (ASV) (Mannino et al. 1990), ASV (Golimowski and Gustavsson 1983), MIP/AES (Natajaran 1988), GC/ECD (Ahmed et al. 1988; AOAC 1984), and spectrophotometry (Agrawal and Desai 1985; Marquez et al. 1988) have also been used to analyze fish, plant material, and pharmaceuticals for mercury. HPLC/ICP/MS has the additional advantage of permitting separation and quantitation of individual mercury species (Bushee 1988). An AOAC-recommended colorimetric method is available for screening food samples (AOAC 1984).

Several other environmental matrices have been analyzed for mercury content. These include coal fly ash (Horvat and Lupsina 1991; Lexa and Stulik 1989), coal dust (Wankhade and Garg 1989), minerals (Bichler 1991), pesticides (Sharma and Singh 1989), gasoline (Costanzo and Barry 1988), and oily waste (Campbell and Kanert 1992). The methods used include CVAAS, DCASV, NAA, spectrophotometry, and GC/alternating current plasma detection (ACPD). The data on each method for each matrix were insufficient for making comparisons.

6.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of mercury is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of mercury.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. There are reliable methods for detecting and quantifying elemental mercury in human breath, blood, urine, milk, tissues, hair, and bones. The method of choice is CVAAS (Akagi et al. 1995; Friese et al. 1990; Pineau et al. 1990; Ping and Dasgupta 1989, 1990; Rathje et al. 1974; Vermeir et al. 1988, 1989; Vesterberg 1991). Other methods that have produced good results include ETAAS (Emteborg et al. 1992), AFS (Corns et al. 1994; Vermeir et al. 1991a, 1991b; Suo et al. 1992), flameless AAS (NIOSH 1994), IDSSMS (Moody and Paulsen 1988), XRF (Bloch and Shapiro 1986), NAA (Fung et al. 1995; Zhuang et al. 1989), GC/MPD (Bulska et al. 1992), ICP-AES (Buneaux et al. 1992), and ICP-MS (Kalamegham and Ash 1992). Using these methods, mercury levels at μg to pg concentrations are detectable. This makes them useful for measuring background and higher levels (Ikingura and Agaki 1996). Many of the methods can also distinguish between organic and inorganic mercury. No further methods for analysis of elemental mercury in biological fluids and tissues are needed. Additional research will be needed to validate the determination of individual mercury species (i.e., methylmercury, phenyl mercury, mercury acetate, etc.) in matrices determined to be important. Methods exist for the separation and detection of these species, but few standard reference materials exist for comparative studies.

Biochemical indicators of possible renal dysfunction (increased urinary NAG levels, and elevated porphyrins) have been associated with increased urinary levels of mercury (Rosenman et al. 1986; Wada et al. 1969; Woods 1996). Functional indicators of adverse neurological effects (reduced nerve conduction velocity, prolonged nerve latency, increased tremor frequency, increased reaction time, reduced hand-eye coordination, and performance on memory and verbal intelligence tests) have also been correlated with increased urinary levels of mercury (Levine et al. 1982; Piikivi et al. 1984; Smith et al. 1970, 1983; Verberk et al. 1986; Vroom and Greer 1972; Williamson et al. 1982). Decreased nerve conduction velocity has been correlated with increased tissue levels of mercury (Shapiro et al. 1982). These biomarkers are not specific for mercury and may be induced by exposure to other metals and chemicals or to disease conditions. Other

nonspecific indicators of possible mercury exposure (insomnia, emotional instability, paresthesia, and abnormal EEG) that have been observed in exposed individuals cannot be quantified, but an increased incidence in specific populations may be correlated with increased urinary levels of mercury in the population (Davis et al. 1974; Jaffe et al. 1983; McFarland and Reigel 1978). The existing analytical methods that have been discussed for exposure can reliably measure the levels in blood, urine, and tissue at which these effects occur. Standard methods exist to measure the effects that can be quantified. No further methods need to be developed.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. There are analytical methods to detect and measure elemental and organic mercury in air, water, sediment, soil, sludge, foods, plant materials, and other environmental matrices. The methods used include CVAAS (the most commonly used and recommended method) (AOAC 1984; Baxter and Frech 1989; Eaton et al. 1995; EPA 1994f, 1994g; Munaf et al. 1991; Navarro et al. 1992; Paudyn and Van Loon 1986; Ping and Dasgupta 1989), AFS (Bloom 1989; Bloom and Fitzgerald 1988; Morales-Rubio et al. 1995; Vermeir et al. 1991a, 1991b), IDSSMS (Moody and Paulsen 1988), flameless AAS (Cacho and Castells 1989; Filippelli 1987; NIOSH 1994), and several other methods. Several of the methods have been proven reliable and are sensitive enough to measure background levels. Methods also exist to determine individual mercury species (Bloom and Fitzgerald 1988; Liu et al. 1994; Paudyn and Van Loon 1986). No further methods are needed for mercury analysis in environmental samples. Additional work would be required to validate methods for individual organomercury species in particular matrices.

6.3.2 Ongoing Studies

Ongoing studies concerning the detection and measurement of mercury in biological or environmental samples identified through a search of Federal Research in Progress (FEDRIP 1998) are shown in Table 6-3.

Table 6-3. Research on New Methods for the Detection of Mercury

Investigator	Sponsor	Research
RJ Schlager ADA Technologies Englewood, CA	USDoE, Energy Research	Developing a continuous emission monitor for total and organic mercury in stack gases
PC Efthimion EEI Pluckemin, NJ	EPA	Developing a continuous emission monitor for flue gas based on plasma emission using a microwave-powered source
CW Brown Brooks Rand, Ltd. Seattle, WA	DoE	Developing a monitor for methyl mercury based on luminescence
D Mcallister Biode, Inc. Cape Elizabeth, ME	DoE	Developing a simple sensor for use in waste, surface, and groundwater using a shear horizontal acoustic plate mode (SHAPM) sensor, a form of piezoelectric sensor
LG Piper Physical Sciences, Inc., Andover, MA	DoE	Developing a sensor for mercury in exhaust stack effluents from coal burning power plants based on the fluoerescence of merucry excited by active nitrogen
JCMay	FDA	Developing methods for the determination of mercury and trace metals in injectable products based on high performance liquid chromatography in conjunction with ICP-MS

Source: FEDRIP 1998

7. REGULATIONS AND ADVISORIES

The international, national, and state regulations and guidelines regarding mercury and mercury compounds in air, water, and other media are summarized in Table 7-1. Unless otherwise indicated, the listings in the table refer to mercury.

An MRL of 0.0002 mg/m^3 has been derived for chronic-duration inhalation exposure (365 days or more) to metallic mercury vapor in a group of 26 mercury-exposed workers from three industries exposed to low levels of mercury for an average of 15.3 years (range, 1–41 years) (Fawer et al. 1983).

An MRL of 0.007 mg mercury/kg/day has been derived for acute-duration oral exposure (14 days or less) to inorganic mercury based on a NOAEL of 0.93 mg mercury/kg for renal effects (increased absolute and relative kidney weights) in rats exposed to gavage doses of mercuric chloride for 14 days (NTP 1993).

An MRL of 0.002 mg mercury/kg/day has been derived for intermediate-duration (15–364 days) oral exposure to inorganic mercury based on a NOAEL of 0.23 mg mercury/kg for renal effects (increased absolute and relative kidney weights) in rats (Dieter et al. 1992; NTP 1993).

An MRL of 0.0003 mg mercury/kg/day has been derived for chronic-duration (365 days or more) oral exposure to methylmercury, based on neurodevelopmental outcomes in a study by Davidson et al. (1998) of children exposed *in utero* to methylmercury from maternal fish ingestion.

EPA has derived an oral RfD of 8×10^{-5} mg/kg/day (0.08 µg/kg/day) for phenylmercuric acetate as mercury (IRIS 1997). The RfD is based on a LOAEL of 0.5 ppm mercury or 0.042 mg/kg/day phenyl mercuric acetate for detectable kidney damage in female rats after 2 years (Fitzhugh et al. 1950). EPA has derived an oral RfD of 3×10^{-4} mg/kg/day (0.3 µg/kg/day) for mercuric chloride. The RfD is based on LOAELs of 0.226, 0.317, and 0.633 mg/kg/day of mercuric chloride. Although no one study was found adequate for deriving an oral RfD, EPA's mercury workgroup derived an oral RfD of high confidence using the weight of evidence from three studies (Andres 1984; Bernaudin et al.; Druet et al. 1978) which used Brown-Norway rats, and an intensive review and discussion of the entire inorganic mercury data base (IRIS 1997). EPA has derived an oral RfD of 1×10^{-4} mg/kg/day (0.1 µg/kg/day) for methylmercury based on developmental neurological abnormalities in human infants (IRIS 1997). EPA has not derived an RfD value for elemental mercury. The EPA inhalation reference concentration (RfC) for elemental mercury is $3x10^{-4}$ mg/m^3

(0.3 µg/m^3). The RfC is based on a LOAEL of 0.025 ppm for human occupational exposure studies. Critical effects seen during these studies included hand tremors, increases in memory disturbances, and slight subjective and objective evidence of autonomic dysfunction (IRIS 1997). No RfC was reported for other mercury compounds.

The American Conference of Governmental Industrial Hygienists (ACGIH) and the EPA have determined that inorganic forms of mercury, including metallic mercury, are not classifiable as to their human carcinogenicity. These agencies have assigned mercury and its inorganic compounds the weight-of-evidence classifications of A4 and D, respectively (ACGIH 1996; IRIS 1997). Mercuric chloride and methylmercury have been assigned EPA's weight-of evidence classification of C, which indicates that they are possible human carcinogens (IRIS 1997).

OSHA requires employers of workers who could be occupationally exposed to mercury to institute engineering controls and work practices which ensure that during any part of the workday, mercury concentrations do not exceed the ceiling value of 1 mg/10 m^3 (0.1 mg/m^3) (OSHA 1974).

Mercuric cyanide, mercuric nitrate, mercuric sulfate, mercuric thiocyanate, mercurous nitrate, mercury, and mercury fulminate have been designated as hazardous substances pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980 (EPA 1995i). Mercuric acetate, mercuric chloride, and mercuric oxide are mercury compounds that have been individually designated as extremely hazardous substances under Section 313 of Title III of the Superfund Amendments and Reauthorization Act (SARA) of 1986 (EPA 1995j). Phenylmercury acetate is consider both a hazardous substance and an extremely hazardous substances. The statutory sources designating mercury and regulated mercury compounds as CERCLA hazardous substances are section 307(a) of the Clean Water Act (CWA), section 112 of the Clean Air Act (CAA), and section 3001 of the Resource Conservation and Recovery Act (RCRA) (EPA 1995i). The owner and operator of facilities using these substance on their sites are required to immediately report releases to any environmental media, if the amount released exceeds the established "reportable quantity" (EPA 1995i). The statutory and final reportable quantities for mercury and regulated mercury compounds as established by Section 102 of CERCLA are given in Table 7-1 (EPA 1995i). Although mercury compounds are listed generically as CERCLA hazardous substances no reportable quantity has been established for them as a broad class (EPA 1995i). Title III of SARA is also known as "The Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986." As chemicals subject to the emergency planning and release reporting requirements of EPCRA, owners and operators of

facilities that have mercuric acetate, mercuric chloride, and mercuric oxide on their sites in amounts exceeding the "threshold planning quantity" established for these substances must develop a program that addresses implementing emergency response plans and for notifying the public of accidental releases (EPA 1987a, 1995j). When extremely hazardous substances are formulated as a solids they are subject to either of two threshold planning quantities (EPA 1995j). If the solid exits in powdered form and has a particle size less than 100 microns, it is subject to the lower number. If the solid does not meet this criteria, it is subject to the higher number. The threshold planning quantities for mercuric acetate, mercuric chloride, mercuric oxide, and phenylmercury acetate are given in Table 7-1. It is important to note that reportable quantities for these compounds are the same as their threshold planning quantities.

The EPA regulates mercury under the Clean Air Act (CAA) and has designated it as a hazardous air pollutant (HAP). Emission standards for release of mercury to the atmosphere have been promulgated for mercury cell chloralkali plants, mercury ore processing facilities, major stationary sources, and municipal waste combustors (EPA 1975a, 1975b, 1995a, 1996b).

In accordance with the authority of the Safe Drinking Water Act (SDWA), EPA has established a safe drinking water standard for mercury at 2 μg/L (FSTRAC 1995). Under the Clean Water Act (CWA) EPA provides criterion concentrations for mercury as a priority toxic pollutant (EPA 1992).

Mercury is regulated as a "priority pollutant" in accordance with the Clean Water Act (CWA). The CWA establishes the basic structure for regulating the discharge of pollutants to waterways and is designed to ensure that all waters are sufficiently clean to protect public health and/or the environment. However, if waters and their sediments become contaminated from sources such as atmospheric deposition and discharges from industrial, municipal, or agricultural operations, toxic substances could concentrate in the tissue of fish and wildlife.

Advisories have been developed and issued to warn people about the health risks of consuming methylmercury-contaminated fish, shellfish, or wildlife and provide guidance as to the amount of fish or wildlife that can be safely consumed by each group (adults, pregnant women, nursing mothers, and young children). Each state, Native American tribe, or U.S. Territory establishes its own criteria for issuing fish and wildlife advisories. A fish or wildlife advisory will specify which waters (lake, rivers, estuaries, or coastal areas) or hunting areas have restrictions. The advisory provides information on the species and size range of the fish or wildlife of concern. The advisory may completely ban eating fish, shellfish, or

freshwater turtles, or it may recommend consumption limits (numbers of fish meals per specified time period) considered to be safe to eat. For example, an advisory may recommend that a person eat a certain type of fish no more than once a month. Advisories may specify the tissues of the fish or wildlife that can be safely eaten or proper preparation and cooking practices to help decrease exposure to methylmercury. The fish or wildlife advisory is typically more restrictive to protect pregnant women, nursing mothers, and young children. To reduce children's exposure to methylmercury, state advisory recommendations for fish consumption limits (meals per week or meals per month) should be strictly observed. Published information in the form of brochures on fish and wildlife advisories is available from State Public Health Departments, Natural Resources Departments, or Fish and Game Departments. Signs may be posted in certain fishing and hunting areas frequently used by recreational fishers and hunters to warn them about specific contamination problems (EPA 1995 Fish Sampling analysis and Guidance Document).

Currently, 1,782 advisories are in effect in 41 states and one U.S. Territory (American Samoa) restricting the consumption of mercury-contaminated fish, shellfish, or wildlife (freshwater turtles) (EPA 1998a). Methylmercury is the chemical pollutant responsible, in part, for over 77% of fish advisories issued in the United States (EPA 1998b). Eleven states (Connecticut, Indiana, Maine, Massachusetts, Michigan, Missouri, New Hampshire, New Jersey, North Carolina, Ohio, and Vermont) currently have state-wide mercury advisories recommending that all residents restrict consumption of locally caught freshwater fish. In addition, 5 states (Alabama, Florida, Louisiana, Massachusetts, and Texas) have issued statewide coastal mercury advisories for specific marine fish or shellfish species. In two states (Arizona and Minnesota), wildlife advisories have been issued recommending that residents restrict their consumption of freshwater turtles (EPA 1998a, 1998b).

The FDA currently has advice for consumers (posted on the Internet) recommending that pregnant women, and women of childbearing age who may become pregnant, limit their consumption of shark and swordfish to no more that one meal per month (FDA 1998). Methylmercury levels are much higher in these fish species than in the more commonly consumed species. The FDA advisory covers women of childbearing age who might become pregnant because dietary practices immediately before the pregnancy may have a direct bearing on fetal exposure during pregnancy. The FDA states that nursing mothers who follow this advice, will not expose their infants to increased health risks from methylmercury (FDA 1998). The FDA consumer advice hotline telephone number is **1-800-332-4010** and the FDA Web site is **www.FDA.gov**.

7. REGULATIONS AND ADVISORIES

The Food and Drug Administration (FDA) regulates the use of mercury compounds in the cosmetics industry. The FDA regulations on the use of mercury compounds in cosmetics state that "because of the known hazards of mercury, its questionable efficacy as a skin-bleaching agent, and the availability of effective and less toxic non-mercurial preservatives, there is no justification for the use of mercury in skin-bleaching preparations or its use as a preservative in cosmetics, with the exception of eye-area cosmetics" (FDA 1974). The use of mercury compounds as cosmetic ingredients has primarily been limited to their use as preservatives in eye area cosmetics for which no other effective and safe non-mercurial preservative is available. In other preparations they must contain no more than trace amounts of mercury that are unavoidable under the conditions of good manufacturing practices (FDA 1974). The mercurial concentration in these other preparations must measure less than 1 ppm or 0.0001% mercury metal (FDA 1974).

The FDA has also established an action level of 1 ppm for methylmercury in fish (FDA 1994, 1996). Because of reports that swordfish, shark and other large predatory fish may contain methylmercury levels which exceed the FDA 1 ppm limit, the agency’s advice to consumers warns pregnant women and women of childbearing age to limit their consumption of shark and swordfish to no more than one meal a month (FDA 1996). For others, the agency recommends that regular consumption of fish species with methylmercury levels around 1 ppm be limited to approximately 7 ounces per week; for fish with levels averaging 0.5 ppm, the limit is about 14 ounces per week (FDA 1996). The consumption advice is considered unnecessary for the top 10 species of fish that make up approximately 80% of the seafood market (FDA 1996). Canned tuna, shrimp, pollock, salmon, cod, catfish, clams, flatfish, crabs, and scallops are the top 10 species of fish consumed (FDA 1996). Since methylmercury levels in these species are usually less than 0.2 ppm and because few people eat more than the suggested weekly limit of 2.2 pounds (1 kilogram) for this contamination level, consumption limits are considered unnecessary (FDA 1996).

On May 28, 1998, the Consumer Product Safety Commission (CPSC) issued a guidance statement recommending that manufacturers of liquid-filled consumer products eliminate the use of hazardous chemicals in the liquid portion of their products (CPSC 1998). The guidance statement was issued as an effort to reduce the risk of exposing young children to hazardous chemicals contained in the liquid. The hazardous chemicals found in the liquid include mercury, ethylene glycol, diethylene glycol, methanol, methylene chloride, petroleum distillates, toluene, and xylene. Children’s products identified by the Commission as containing these hazardous chemicals include rolling balls, maze toys, bubble watches, and necklaces. Paperweights, keychains, liquid timers, and pens were household items identified as containing

mercury or other hazardous chemicals (CPSC 1998). In addition to the recommendation that manufacturers eliminate the use of hazardous chemicals in these products, the Commission also recommends that importers, distributors, and retailers who purchase a liquid-filled product for resale, obtain from the manufacturer assurances that their product does not contain hazardous liquid chemicals. Although the guidance is not a rule, it focuses on certain obligations authorized by the Federal Hazardous Substance Act (FHSA). Under the FHSA toys or other articles that contain an accessible and harmful amount of hazardous chemical and are intended for use by children are banned (CPSC 1998). Articles that are not intended for use by children, but create a risk of injury because they contain hazardous chemicals, require precautionary labeling under the FHSA (CPSC 1998).

In 1995, the CPSC assisted in facilitating the recall of necklaces bearing small vials or glass balls containing metal mercury (CPSC 1995). Although the vials and glass balls posed no immediate health threat, the recall noted that exposure to mercury vapor could cause long term health problems, especially for small children and pregnant women, if the vials or balls were broken (CPSC 1995).

Table 7-1. Regulations and Guidelines Applicable to Mercury

Agency	Description	Information	References
INTERNATIONAL			
Guidelines:			
WHO	Drinking-water guideline values for health-related organics (applies to all forms of mercury)	0.001 mg/L	WHO 1984
	Permissible tolerable weekly intake	5 µg/kg total 3.3 µg/kg CH_3Hg	WHO 1976
NATIONAL			
Regulations: a. Air:			
OSHA	Air Contaminants permissible exposure limit (PEL) 8-hr. time weighted average (TWA)	0.1 mg/m^3	29 CFR 1910.1000 OSHA 1974[a]
EPA/OAR	Hazardous Air Pollutants	Yes	CAA Amendment Title III, Section 112 (b) U.S. Congress 1990
	Prevention of Significant Deterioration of Air Quality–pollutant emissions rate defined as significant	0.1 tons per year	40 CFR 51.166 EPA 1996h
	pollutant emission rate-exemption of major stationary source	< 0.25 µg/m^3 (24-hour average)	
	Standards of Performance for New Stationary Sources–emissions limits for municipal waste combustors	0.080 mg/m^3 or 15% of the potential mercury emission concentration corrected to 7% oxygen	40 CFR 60, Subpart Cb EPA 1995a
	standards of performance for municipal waste combustors	Yes	40 CFR 60, Subpart Eb EPA 1995b
	National Emission Standards for Hazardous Air Pollutants (NESHAPs)–list of pollutants and applicability	Yes	40 CFR 61.01 EPA 1971a
	standard for mercury ore processing facilities and mercury cell chlor-alkali plants (mercury)	< 2300 g per 24-hour period	40 CFR 61, Subpart E EPA 1975c
	standard for sludge incineration plants, sludge drying plants, or a combination of these that process wastewater treatment plant sludges (mercury)	< 3200 g per 24-hour period	

Table 7-1. Regulations and Guidelines Applicable to Mercury (continued)

Agency	Description	Information	References
NATIONAL (cont.)			
	National Emission Standards for Hazardous Air Pollutants for Source Categories-Regulations Governing Extension for Early Reductions of Hazardous Air Pollutants–list of high-risk pollutants	Yes	40 CFR 63, Subpart D EPA 1994a
b. Water			
EPA-ODW	National Primary Drinking Water Regulations		
	Maximum Contaminant Level (MCL) for inorganic compounds	0.002 mg/L	40 CFR 141, Subpart F EPA 1992b
	BAT for inorganic compounds	coagulation/ filtration[e,f], granular activated charcoal, lime softening[e,f], reverse osmosis[e]	
	Hazardous Waste Injection Restrictions–waste specific prohibitions; California list wastes	Yes	40 CFR 148.12 EPA 1988
EPA-OW	Designation of Hazardous Substances- List of hazardous substances, Table 116.4 (mercuric cyanide, mercuric nitrate, mercuric sulfate, mercuric thiocyanate, mercurous nitrate)	Yes	40 CFR 116.4 EPA 1978a
	Determination of Reportable Quantities for Hazardous Substances-RQ Pursuant to Section 311 CWA–mercuric cyanide	1 pound (0.45 kg)	40 CFR 117.3 EPA 1995c
	mercuric nitrate, mercuric sulfate, mercuric thiocyanate, mercurous nitrate	10 pounds (4.54 kg)	
	EPA Permit Programs: National Pollution Discharge Elimination System (NPDES)–other toxic pollutant (metals and cyanide) and total phenols	Yes	40 CFR 122, App. D EPA 1983
	Criteria and Standards for the NPDES-Instructions for Form 2C, Application for Permit to Discharge Wastewater (mercuric cyanide, mercuric nitrate, mercuric sulfate, mercuric thiocyanate, mercurous nitrate)	Yes	40 CFR 125 EPA 1984a

Table 7-1. Regulations and Guidelines Applicable to Mercury (continued)

Agency	Description	Information		References
NATIONAL (cont.)				
	Toxics Criteria for those States Not Complying with CWA Section 303(c)(2)(B)-criterion concentration for priority toxic pollutants	Maximum[b] (µg/L)	Continous[c] (µg/L)	40 CFR 131.36 EPA 1992a
	freshwater	2.10	0.012[d]	
	saltwater	1.80	0.025	
	human health consumption of:			
	water and organisms	0.14[e]		
	organisms only	0.15[e]		
	Water Quality Guidance for the Great Lakes Systems-protection of aquatic life in ambient water	Maximum[b] (µg/L)	Continous[c] (µg/L)	40 CFR 132 EPA 1995d
	acute water quality criteria for mercury (II) total recoverable	1.694 µg/L	NA	
	chronic water quality criteria for mercury (II) total recoverable	NA	0.908 µg/L	
	water quality criteria for protection of human health (HNV for mercury including methylmercury) drinking water and non-drinking water	$1.8x10^{-3}$ µg/L		
	water quality criteria for protection of human health (mercury including methylmercury)	$1.3x10^{-3}$ µg/L		
	pollutants that are bioaccumulative chemicals of concern	mercury		
	Standards for the Control of Residual Radioactive Materials from Inactive Uranium Processing Sites–maximum concentration of constituents for groundwater (mercury)	0.002 µg/L		40 CFR 192.04 EPA 1995e
	Criteria for the Evaluation of Permit Applications for Ocean Dumping of Materials–constituents prohibited as other than trace contaminants	Yes		40 CFR 227.6 EPA 1978b
c. Food:				
FDA	Action Level for Poisonous or Deleterious Substances in Human Food and Animal Feed			FDA 1994 and FDA 1998
	fish, shellfish, crustaceans, other aquatic animals (fresh, frozen or processed)	1 ppm		
	wheat-pink kernels only; an average of 10 or more pink kernels per 500 grams	1 ppm		
	Bottled water	0.002 µg/L		21 CFR 165.110 FDA 1995

Table 7-1. Regulations and Guidelines Applicable to Mercury (continued)

Agency	Description	Information	References
NATIONAL (cont.)			
d. Other:			
EPA-OERR	List of Hazardous Substances and Reportable Quantities (RQ)	Statutory / Final (pounds)	40 CFR 302.4 EPA 1995i
	mercury	1 / 1	
	mercuric cyanide	1 / 1	
	mercuric nitrate	10 / 10	
	mercuric sulfate	10 / 10	
	mercuric thiocyanate	10 / 10	
	mercurous nitrate	10 / 10	
	mercury fulminate	1 / 10	
	phenylmercury acetate	1 / 100	
EPA-OSW	Criteria for Classification of solid Waste Disposal Facilities and Practices	Yes	40 CFR 257, App I EPA 1991a
	Criteria for Municipal Solid Waste Landfills–MCLs	0.002 µg/L	40 CFR 258.40 and App. II
	list of hazardous constituent	Yes	EPA 1991b
	Identification and Listing of Hazardous Waste–definition of a Hazardous Waste	Yes	40 CFR 261.3 EPA 1992c
	generic exclusion levels for K061 and K062 wastes for nonwastewater HTMR residues	0.009 mg/L (maximum for single composite sample-TCLP)	
	toxicity characteristic–maximum concentration	0.2 mg/L	40 CFR 261.24 EPA 1993a
	hazardous waste from specific sources	K071 K106	40 CFR 261.32 EPA 1992d
	discarded commercial chemical products, off-specification, container residues, and spills	Yes	40 CFR 261.33 EPA 1994b
	mercury	U151	
	mercury fulminate	P065	
	phenylmercuric acetate	P092	
	basis for listing hazardous wastes	K071 K106	40 CFR 261, App. VII EPA 1995g
	hazardous constituents-		40 CFR 261, App. VIII
	mercury	U151	EPA 1994c
	mercury fulminate	P065	
	phenylmercury acetate	P092	
	Standards for Owners and Operators of Hazardous Waste Treatment, Storage, and Disposal Facilities–Releases from Solid Waste Management Units	Yes	40 CFR 264.94 EPA 1995f
	concentration limits for groundwater protection	0.002 µg/L	
	ground-water monitoring list	yes	40 CFR 264, App. IX EPA 1995h

Table 7-1. Regulations and Guidelines Applicable to Mercury (continued)

Agency	Description	Information	References
NATIONAL (cont.)			
	Interim Status Standards for Owners and Operators of Hazardous Waste Treatment, Storage, and Disposal Facilities–thermal treatment, incinerators, and land treatment	Yes	40 CFR 265 EPA 1991c
	Standards for Management of Specific Hazardous Wastes Facilities–hazardous waste burned in boilers and industrial furnaces	Yes	40 CFR 266.100 EPA 1991d
	reference air concentration	0.3 µg/m^3	40 CFR 266 App. IV EPA 1991d
	health-based limits for exclusion of waste-derived residues	$2x10^{-1}$	40 CFR 266 App. VII EPA 1993b
	mercury bearing waste that may be processed in exempt mercury recover units	Yes	40 CFR 266 App. XIII EPA 1994d
	methods Manual for Compliance with BIF Regulations	Yes	40 CFR 266, App. IX EPA 1991e
	Land Disposal Restrictions– treatment Standards	WW NWW (technology code or mg/L TCLP)	40 CFR 268.40 EPA 1997d
	D009	0.20 IMERC, RMERC, AMLGM, 0.20	
	F039	0.15 0.025	
	K001	NA 0.025	
	K071	NA 0.20	
	K084	0.15 0.25 or 0.25	
	K101	0.15 NA	
	K102	0.15 NA	
	K106 (≥260 mg/kg total mercury)	0.15 RMERC, 0.20, 0.025	
	P065 (mercury fulminate)	0.15 IMERC, RMERC, 0.2, 0.025	
	P092 (phenylmercuric acetate)	0.15 IMERC, RMERC, 0.20, 0.025	
	U151	0.15 RMERC, AMLGM, 0.20, 0.025	
	Treatment Standards Expressed as Specified Technologies	AMLGM, IMERC, and RMERC,	40 CFR 268.42 EPA 1994e

Table 7-1. Regulations and Guidelines Applicable to Mercury (continued)

Agency	Description	Information	References
NATIONAL (cont.)			
	Treatment Standards for Hazardous Debris	Yes	40 CFR 268.45 EPA 1992e
	Emergency Planning and Notification-Extremely Hazardous Substances and Their Threshold Planning Quantities		40 CFR 355, App. A EPA 1995j
	mercuric acetate	500/10,000 pounds	
	mercuric chloride	500/10,000 pounds	
	mercuric oxide	500/10,000 pounds	
	phenylmercury acetate	500/10,000 pounds	
	Emergency Planning and Notification-Extremely Hazardous Substances and Their Reportable Quantities		
	mercuric acetate	500 pounds	
	mercuric chloride	500 pounds	
	mercuric oxide	500 pounds	
	phenylmercury acetate	100 pounds	
	Toxic Chemical Release Reporting: Community Right-to-Know--Specific Chemical Listing-Chemicals and Chemical Categories	Yes	40 CFR 372.65 EPA 1987a
FDA	Cosmetics-use of mercury compounds	Yes	21 CFR 700.13 FDA 1974
	eye area cosmetics (mercury calculated as the metal)	< 65 ppm	
NRC	Standards for Protection Against Radiation	Yes	10 CFR 20 DOE 1993
	Rules of General Applicability to Domestic Licensing of Byproduct Material	Yes	10 CFR 30 DOE 1994a
	Domestic Licensing of Source Material	Yes	10 CFR 40 DOE 1994b
	Packaging and Transport of Radioactive Material	Yes	10 CFR 71 DOE 1996a
	Export and Import of Nuclear Equipment and Material	Yes	10 CFR 110 DOE 1996b
Guidelines a: Air:			
ACGIH	STEL/Ceiling-alkyl compounds	0.3 mg/m^3	ACGIH 1996
	TWA-alkyl compounds	0.01 mg/m^3	
	aryl compounds	0.1 mg/m^3	
	inorganic forms including metallic mercury	0.025 mg/m^3	
NIOSH	Recommended Exposure Limit for Occupation Exposure (8-hr TWA)-aryl or inorganic mercury as mercury	0.1 mg/m^3 ceiling (skin)	NIOSH 1992
	mercury (organo) alkyl compounds as mercury	0.01 mg/m^3 TWA 0.03 mg/m^3 STEL (skin)	
	mercury vapor as mercury	0.05 mg/m^3 TWA	

Table 7-1. Regulations and Guidelines Applicable to Mercury (continued)

Agency	Description	Information	References
NATIONAL (cont.)			
b. Water:			
EPA-ODW	National Primary Drinking Water Regulations–MCLGs for inorganic compounds	0.002 mg/L	40 CFR 141, Subpart F EPA 1992b
	Lifetime Health Advisory (adult)-inorganic mercury (final)	0.002 mg/L	EPA 1996g
	Longer-term Health Advisory (adult)-inorganic mercury (final)	0.002 mg/L	
	Drinking Water Equivalent Level	0.002 mg/L	
	Ambient Water Quality Criteria for Human Health–mercury and phenylmercuric acetate		IRIS 1997 IRIS 1997
	water and fish	1.44×10^{-1} µg/L	
	fish only	1.46×10^{-1} µg/L	
	Ambient Water Quality Criteria for Aquatic Organisms–mercury and phenylmercuric acetate as mercury	Marine Freshwater (µg/L)	IRIS 1997 IRIS 1997
	acute(1- hour average)	2.1 2.4	
	chronic (4-day average)	2.5×10^{-2} 1.2×10^{-2}	
c. Food			
FDA	Consumption of shark or swordfish by pregnant or childbearing age women	No more than one meal a month	FDA 1998
	Regular consumption of fish species with methylmercury levels around 1 ppm	7 ounces per week	
	Fish with levels averaging 0.5 ppm	14 ounces per week	
d. Other:			
ACGIH	Cancer Ranking-metallic mercury	A4[g]	ACGIH 1996
EPA	Cancer Classification		IRIS 1997
	elemental (metallic) mercury	D[h]	
	methyl mercury	C[i]	
	mercuric chloride	C[i]	
CPSC	Notice of Availability of Guidance Document on Hazardous Liquid Chemicals in Children's Products	Yes	63 FR 29182 CPSC 1998
EPA	RfC (elemental mercury)	3×10^{-4} mg/m^3 (0.3 µg/m^3)	IRIS 1997
	RfD (mercuric chloride)	3×10^{-4} mg/kg/day (0.3 µg/kg/day)	
	RfD (methyl mercury)	1×10^{-4} mg/kg/day (0.1 µg/kg/day)	
	RfD (phenylmercuric acetate)	8×10^{-5} mg/kg/day (0.08 µg/kg/day)	

Table 7-1. Regulations and Guidelines Applicable to Mercury (continued)

Agency	Description	Information	References
STATE			
Regulations and Guidelines:			
a. Air:	Average Acceptable Ambient Air Concentrations-Mercury		NATICH 1992
AZ	1 hour	1.5 $\mu g/m^3$	
	24 hours	$4.0x10^{-1}$ $\mu g/m^3$	
(phenylmercuric acetate)	1 hour	$2.5x10^{-1}$ $\mu g/m^3$	
	24 hours	$7.9x10^{-2}$ $\mu g/m^3$	
CT	8 hours	1.0 $\mu g/m^3$	
	8 hours	2.0 $\mu g/m^3$	
FL-FtLdle	8 hours	$5.0x10^{-4}$ mg/m^3	
FL-Pinella	8 hours	$1.0x10^{-1}$ $\mu g/m^3$	
	24 hours	$2.4x10^{-2}$ $\mu g/m^3$	
(phenylmercuric acetate)	Annual	$7.5x10^{-1}$ $\mu g/m^3$	
FL-Tampa	8 hours	$5.0x10^{-4}$ mg/m^3	
IN	8 hours	$5.0x10^{-2}$ $\mu g/m^3$	
KS	Annual	$2.4x10^{-2}$ $\mu g/m^3$	
LA	8 hours	1.19 $\mu g/m^3$	
MT	24 hours	$8.0x10^{-2}$ $\mu g/m^3$	
	Annual	$1.0x10^{-2}$ $\mu g/m^3$	
NC	15 minutes	$6.0x10^{-4}$ mg/m^3	
NC-Forco	24 hours	$6.0x10^{-4}$ mg/m^3	
ND	8 hours	$5.0x10^{-4}$ mg/m^3	
NV	Not Indicated	$2.0x10^{-3}$ mg/m^3	
NY	1 year	$1.67x10^{-1}$ $\mu g/m^3$	
OK	24 hours	$5.0x10^{-1}$ $\mu g/m^3$	
(phenylmercuric acetate)	24 hours	$5.0x10^{-1}$ $\mu g/m^3$	
PA-Phil	1 year	$2.4x10^{-1}$ $\mu g/m^3$	
	Annual	$2.4x10^{-1}$ $\mu g/m^3$	
SC	24 hours	$2.5x10^{-1}$ $\mu g/m^3$	
TX	30 minutes	$5.0x10^{-1}$ $\mu g/m^3$	
	Annual	$5.0x10^{-2}$ $\mu g/m^3$	
VA	24 hours	$1.7x10^{4}$ $\mu g/m^3$	
	24 hours	$8.3x10^{1}$ $\mu g/m^3$	
	24 hours	1.7 $\mu g/m^3$	
VT	Annual	$1.2x10^{-1}$ $\mu g/m^3$	
WA-SWEST	Annual	$3.0x10^{-1}$ $\mu g/m^3$	
b. Water	Water Quality Criteria: Human Health		FSTRAC 1995
AL	Drinking water (standard)	2 µg/L	
AZ	Drinking water (standard)	2 µg/L	

Table 7-1. Regulations and Guidelines Applicable to Mercury (continued)

Agency	Description	Information		References
STATE (cont.)				
ME	Drinking water (guideline)	2 µg/L		
MN	Drinking water (guideline)	2 µg/L		
c. Other				
	Fish and Wildlife Consumption Advisories	Number of Advisories Issued for 1997		EPA 1998a
		Fish	Wildlife	
AL[j]	Freshwater; Marine (statewide)	4		EPA 1998a EPA 1998b
AS	Marine	1		EPA 1998a
AZ	Freshwater	2	3 (turtles)	
AR	Freshwater	19		
CA	Freshwater; Estuarine	11		
CO	Freshwater	8		
CT[k]	Freshwater (statewide)	5		EPA 1998a EPA 1998b
FL[j]	Freshwater;Estuarine;Marine (statewide)	96		
GA	Freshwater; Estuarine	23		EPA 1998a
ID[k]	Freshwater	1		EPA 1998a EPA 1998b
IL	Freshwater	2		EPA 1998a
IN	Freshwater (statewide)	109		EPA 1998a EPA 1998b
KY	Freshwater	1		EPA 1998a
LA[j]	Freshwater; Marine (statewide)	15		
MA[j,k]	Freshwater (statewide); Marine (statewide)	55		
ME[k]	Freshwater (statewide)	1		
MI[k]	Freshwater (statewide)	41		
MN	Freshwater	755	6 (turtles)	EPA 1998a
MO[k]	Freshwater (statewide)	1		EPA 1998a EPA 1998b
MS	Freshwater	7		EPA 1998a
MT	Freshwater	22		
NC[k]	Freshwater (statewide)	10		EPA 1998a EPA 1998b
ND	Freshwater	36		EPA 1998a
NE	Freshwater	12		
NH[k]	Freshwater (statewide)	2		EPA 1998a EPA 1998b
NJ[k]	Freshwater (statewide)	30		
NM	Freshwater	26		EPA 1998a
NY	Freshwater	15		
NV	Freshwater	2		EPA 1998a

Table 7-1. Regulations and Guidelines Applicable to Mercury (continued)

Agency	Description	Information	References
STATE (cont.)			
OH[k]	Freshwater (statewide)	19	EPA 1998a EPA 1998b
OK	Freshwater	1	EPA 1998a
OR	Freshwater	9	
PA	Freshwater	1	
RI	Freshwater	1	
SC	Freshwater	24	
TN	Freshwater	2	
TX[j]	Freshwater; Estuarine; Marine (statewide)	7	EPA 1998a EPA 1998b
VT[k]	Freshwater (statewide)	3	
VA	Freshwater	3	EPA 1998a
WA	Estuarine	1	
WI	Freshwater	390	

[a] A U.S. Court of Appeals rescinded the 1989 PELs promulgated by OSHA. Only PELs in place prior to the 1989 rule are currently allowed (58 FR 35338, June 30, 1993).
[b] Criteria maximum concentration (CMC) is the highest concentration of a pollutant to which aquatic life can be exposed for a short period of time (l-hour average) without deleterious effects and is not to be exceeded more than once every three years.
[c] Criteria continuous concentration (CCC) is the highest concentration of a pollutant to which aquatic life can be exposed for a short period of time (4 days) without deleterious effects and is not to be exceeded more than once every three years.
[d] If the CCC for total mercury exceeds 0.012 µg/L more than once in a 3-year period in the ambient water, the edible portion of aquatic species of concern must be analyzed to determine whether the concentration of methyl mercury exceeds the FDA action level of 1.0 mg/kg.
[e] BAT only if influent mercury concentration is less than 10 µg/L.
[f] BAT for systems with less than 500 service connections.
[g] A4 means that the substance in not classifiable as a human carcinogen. There are inadequate data on which to classify the substance for humans and/or animals.
[h] Cancer classification D means that the substance is not classifiable as to its carcinogenicity. There is inadequate or no human and animal evidence of carcinogenicity.
[i] Cancer classification C means that the substance is a possible human carcinogen.
[j] States issuing coastal for mercury in specific marine fish and shellfish species.
[k] State issuing state-wide advisories for mercury recommending that all residents restrict consumption of locally-caught freshwater fish.

AMLGM = Amalgamation of Liquid, Elemental Mercury Contaminated with Radioactive Materials; BAT = Best Available Technology; BIF = Boilers and Industrial Furnaces; CAA = Clean Air Act; CWA = Clean Water Act; CPSC = Consumer Product Safety Commission; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; FSTRAC = Federal State Toxicology and Regulatory Alliance committee; HAP = Hazardous Air Pollutants; HNV = Human Noncancer Value; HTMR = High Temperature Metals Recovery; IARC = International Agency for Research on Cancer; IMERC = Incineration of Wastes containing Organics and Mercury;MCL = Maximum Contaminant Level; MCLG = Maximum Contaminant Level Goal; NAS = National Academy of Sciences; NESHAP= National Emission Standards for Hazardous Air Pollutants; NIOSH = National Institute of Occupational Safety and Health; NPDES = National Pollution Discharge Elimination System; NRC = Nuclear Regulatory Commission; NWW = Nonwastewaters; OAR - Office of Air and Radiation; ODW = Office of Drinking Water; OERR = Office of Emergency and Remedial Response; OSHA = Occupational Safety and Health Administration; OSW = Office of Solid Wastes; OTS = Office of Toxic Substances; PEL = Permissible Exposure Limit; RfD = Reference Dose; RMERC = Retorting or Roasting of Mercury RQ = Reportable Quantities; SOCMI = Synthetic Organic Chemicals Manufacturing Industry; STEL = Short-term exposure Limit; TCLP = Toxicity Characteristic Leaching Procedure; TLV= Threshold Limit Value; TWA = Time-weighted Average; WHO = World Health Organization; WW = Wastewaters