2.8 INTERACTIONS WITH OTHER CHEMICALS

As with many other metals, both toxic and nontoxic, interrelationships exist that can influence and alter the absorption, distribution, excretion, and toxicity of one or more of the component metals. For example, the zinc status of an individual can affect mercury toxicity. Pretreatment with zinc provides some protection from the nephrotoxic effects of inorganic mercury in rats (Zalups and Cherian 1992). The data indicate that zinc-induced metallothionein binds mercury in the renal cortex and shifts the distribution of mercury from its site of toxicity at the epithelial cells of the proximal tubules. Thus, the renal content of mercury is increased, yet less is available to cause toxicity. In contrast, the renal toxicity of mercuric chloride is exacerbated in zinc-deficient animals (Fukino et al. 1992). In the zinc-deficient state, less mercury accumulates in the kidneys, but the toxicity is greater. The mechanism of the protection appears to involve more than simply a redistribution of renal mercury, because in the absence of mercury exposure, zinc deficiency increases renal oxidative stress (increased lipid peroxidation, decreased reduced ascorbate). When mercury exposure occurs, the oxidative stress is compounded (increased lipid peroxidation and decreased glutathione and glutathione peroxidase). Thus, zinc appears to affect the biochemical protective mechanisms in the kidneys as well.

Similarly, in most studies, the simultaneous administration of mercury and selenium in equimolar doses to animals has resulted in decreased toxicity of both elements in acute and chronic exposure studies. This effect has been observed with inorganic and organic mercury and with either inorganic or organic selenium compounds, although inorganic forms of selenium appear to be more effective than organic forms (Chang 1983; Skerfving 1978). Selenium protects against the acute nephrotoxicity of the mercuric ion and the methylmercuric ion in rats (Ganther 1980; Ganther et al. 1972; Hansen 1988; Magos et al. 1987; Parizek and Ostadolva 1967) and possibly against acute neurotoxicity of methylmercuric ion in rats (Ohi et al. 1980). The protective effect of selenium has been associated with a higher whole-body retention of mercury rather than with increased mercury excretion (Hansen 1988; Magos et al. 1987). Mercury-selenium complexes are formed when these chemicals are co-administered. Mercuric mercury forms a complex with selenium and a high-molecular weight protein (Naganuma and Imura 1981). Methylmercury forms a bismethyl-mercury selenide complex. Although the specific mechanism for the protection is not well understood, possible mechanisms for selenium's protective effect include redistribution of mercury (Mengel and Karlog 1980), competition by selenium for mercury-binding sites associated with toxicity, formation of a mercury-selenium complex that diverts mercury from sensitive targets (Hansen 1988; Magos et al. 1987; Naganuma and Imura 1981), and prevention of oxidative damage by increasing

selenium available for the selenium-dependent glutathione peroxidase (Cuvin-Aralar and Furness 1991; Imura and Naganuma 1991; Nylander and Weiner 1991). Selenium-treated animals can remain unaffected despite an accumulation of mercury in tissues to levels that are otherwise associated with toxic effects (Skerfving 1978). Support for the proposal that an inert complex is formed comes from the 1:1 ratio of selenium and mercury found in the livers of marine mammals and in the bodies of experimental animals administered compounds of mercury and compounds of selenium, regardless of the ratio of the injected doses (Hansen 1988). Mercuric mercury has been shown to form a complex with selenium and a high-molecular weight protein (Naganuma and Imura 1981). Methylmercury forms a bismethyl-mercury selenide complex.

Although the fetotoxicity of methylmercuric chloride has been shown to be enhanced by the feeding of a selenium-deficient diet in mice (Nishikido et al. 1987), additional selenium administration does not appear to protect against teratogenic effects (i.e., cleft palate) of methylmercuric chloride in mice (Lee et al. 1979). High doses of selenium administered as selenite for 30 days prior to gestation and through Gd 18 to mice fed a diet containing high doses of methylmercuric chloride increased the incidence of cleft palate (Nobunaga et al. 1979). It is possible that cleft palate induction by methylmercury is the result of a suppression of growth rather than a tissue-specific teratogenic action (Lee et al. 1979). If this were the case, high doses of selenium that inhibit growth could potentiate the induction of cleft palate by methyl-mercury administration. Further discussion of selenium-mercury interactions can be found in Section 2.3.1.2.

Ethanol promotes an increase in the respiratory excretion of metallic mercury by inhibiting the enzyme catalase, which is responsible for oxidizing metallic mercury to mercuric mercury. This process was shown in workers who ingested a moderate dose of alcohol and experienced a 50% decrease in mercury retention upon inhalation exposure to metallic mercury vapor (Nielsen-Kudsk 1973). Also, ethanol increased the amount of mercury exhaled by people who inhaled metallic mercury vapor or received trace doses of mercuric chloride (Nielsen-Kudsk 1965). Therefore, less mercury should reach the kidneys and less renal toxicity should be observed (Nielsen-Kudsk 1965). However, ethanol also allows elemental mercury to persist longer in the plasma, resulting in prolonged diffusion of elemental mercury throughout the body (Nielsen-Kudsk 1965). Therefore, ethanol can cause mercury to distribute more easily across the blood-brain barrier and the placenta, thereby increasing the risk of mercury toxicity to the brain and the developing fetus. In addition, the oxidation of ethanol with concurrent NADPH generation enhances

the reduction of the mercuric ion to metallic mercury, thereby making it more favorable for permeating the placenta (Khayat and Dencker 1982).

Ethanol also potentiates the toxicity of methylmercury (Rumbeiha et al. 1992; Tamashiro et al. 1986; Turner et al. 1981). Studies in animals have shown increased mortality (Tamashiro et al. 1986), increased severity and decreased time to onset of neurotoxicity (hind-limb ataxia) (Tamashiro et al. 1986; Turner et al. 1981), and increased renal toxicity (increased hematuria, renal weight, blood urea nitrogen, and oliguria) (Rumbeiha et al. 1992; Tamashiro et al. 1986) when methylmercury exposure occurred concomitant with ethanol ingestion. Although increased mercury concentrations were observed in the brain and kidneys, the changes in mercury content were insufficient to fully explain the observed potentiation of toxicity (Tamashiro et al. 1986), suggesting that ethanol may enhance the toxic mechanisms of methylmercury. The mechanism for this enhancement is unknown.

Atrazine and potassium dichromate have also been demonstrated to enhance the toxicity of inorganic mercury. Administration of atrazine, a widely used herbicide, with methylmercury in the diet resulted in a higher deposition of mercury in the liver and an earlier onset of neurotoxicity (Meydani and Hathcock 1984). The mechanism underlying this interaction was unclear. Parenteral administration of minimally toxic doses of potassium dichromate and mercuric chloride resulted in a synergistic inhibition of the renal transport of organic ions *p*-aminohippurate and tetraethylammonium (Baggett and Berndt 1984). Although the mechanism underlying this interaction was not examined, it may be associated with the fact that both mercury and potassium dichromate are both toxic to the renal proximal tubule (Biber et al. 1968).

Agents that deplete nonprotein sulfhydryls may increase the toxicity of mercury. Depletion of glutathione levels with diethylmaleate in rats resulted in greatly increased renal toxicity of mercury chloride (Girardi and Elias 1991). Greater decreases in glomerular filtration and increases in fractional excretion of sodium and lithium, urinary γ-glutamyltransferase, and lipid peroxidation were observed. Conversely, chemicals that protect against oxidative damage may decrease the toxic effects of mercury. Increased survival and decreased toxicity were observed in rats given vitamin E (α-tocopherol) during treatment with methylmercury (Welsh 1979). It is probable that the mechanism for the protection involved the antioxidant properties of vitamin E.

The exogenous application of the monothiols glutathione or its, precursor N-acetyl-DL-homocysteine thiolactone (NAHT), or B-complex and E vitamins to mice exposed to methylmercuric chloride injected

at dosages of 1 mg/kg/day was reported by Bapu et al. (1994). Therapy with both B-complex vitamins and vitamin E was found to mobilize a significant amount of mercury from all tissues examined (brain, spinal cord, liver, and kidneys), with the maximum mobilization (about 63%, compared with controls) being recorded in the spinal cord following vitamin E treatment. NAHT treatment also produced significant mobilization of mercury from nervous tissue but caused an increase in mercury concentration in non-nervous tissue.

Another group of compounds that combines with mercury (and other divalent cation species) is comprised by those used in chelation therapy to reduce the body burden of mercury by enhancing its elimination from the body. Such chelators include: ethylenediaminetetraacetic acid (EDTA); ethylene glycol bis(beta-aminoethyl ether)N,N,N',N'-tetraacetic acid (EGTA); 2,3-dimercaptopropane-1-sulphonate (DMPS); 2,3-dimercaptosuccoinic acid (DMSA); 2,3-dimercaptopropanol (British anti-Lewisite [BAL]; sometimes called dimercaprol); and N-acetylpenicillamine (NAP). While these chelating agents have a very high affinity for Hg^{++}, which makes them effective mercury chelators, they also have an affinity for other divalent cations, many of which are essential for normal physiological function.

BAL was the first chelating agent used for mercury toxicity, and it is still widely used today for inorganic mercury poisoning (ATSDR 1992). BAL is also believed to be effective in treating phenylmercury poisoning, because of the rapid *in vivo* oxidization of phenylmercuric acetate to Hg^{++}, thereby rendering phenylmercury similar in behavior to inorganic mercury. BAL is <u>contraindicated</u> for cases of methyl-mercury poisoning, however, because it has been demonstrated to increase the concentration of methyl-mercury in the brain. Possible side effects of BAL include nausea, vomiting, headache, tachycardia, fever, conjunctivitis, blepharospasm, and lacrimation. As an adjunct or alternative to parenterally administered BAL, oral NAP may be used (ATSDR 1992). Side effects of NAP may include fever, rash, leukopenia, eosinophilia, and thrombocytopenia.

DMPS and DMSA are derivatives of BAL, but they have been found to be more effective than BAL in experimental studies. Although still considered an investigational drug, DMPS decreased the mercury excretion half-life from 33.1 to 11.2 days in 2 workers exposed to high levels of mercury vapor (ATSDR 1992). In a study of the influence of DMPS and DMSA on renal deposition of mercury in rats, both chelating agents were found to cause a significantly increased urinary excretion of mercury (Zalups 1993), although significant differences in the extrarenal handling of these two chelators were found. DMPS was also shown to increase the urinary excretion of mercury 7.6-fold in a group of former chloralkali workers

3 years after cessation of occupational exposure (Sallsten et al. 1994), probably reflecting the excretion of mercury stored in the kidneys. In a case report of two human mercury vapor intoxication incidents, treatment with BAL followed by DMSA was found to decrease plasma inorganic mercury uptake at concentrations <50 µg/L. However, relatively high concentrations of mercury remained in the plasma for a very long time, possibly due to the progressive release of mercury from red blood cells and tissues after oxidation.

EDTA and EGTA also effectively form complexes with Hg^{++}, and enhance its excretion from the body, in what is typically considered a relatively benign or biologically inert fashion. In a study using human brain homogenates from autopsy samples from apparently healthy brains, Duhr et al. (1993) demonstrated that not only is the inhibition of microtubule polymerization and the disruption of already-formed microtubules not prevented by the addition of EDTA and EGTA (which bind Hg^{++} with very high affinity) but, to the contrary, these two chelating agents potentiate the Hg^{++}-induced inhibition of tubulin polymerization. Duhr et al. (1993) further reported that the mercury-EDTA and mercury-EGTA complexes cause the inhibition of tubulin polymerization by disrupting the interaction of GTP with the E-site of brain beta-tubulin, an obligatory step in the polymerization of tubulin.

2.9 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to mercury than will most people exposed to the same level of mercury in the environment. Reasons include genetic makeup, developmental stage, age, health and nutritional status (including dietary habits that may increase susceptibility, such as inconsistent diets or nutritional deficiencies), and substance exposure history (including smoking). These parameters result in decreased function of the detoxification and excretory processes (mainly hepatic, renal, and respiratory) or the pre-existing compromised function of target organs (including effects on clearance rates and any resulting end-product metabolites). Populations more susceptible to the toxic effects of mercury than a healthy young adult include: the elderly because of declining organ function, higher levels of persistent heavy metals (e.g., cadmium) that also accumulate in the kidney, and potentially higher brain to liver or kidney mercury concentrations; people with pre-existing disease (e.g., renal or neurological disease); and the youngest of the population because of their immature and developing organs. Populations at greater risk due to unusually high exposure are discussed in Section 5.7 (Populations With Potentially High Exposure).

Probably the most widely recognized form of hypersensitivity to mercury is the occurrence of acrodynia, or pink disease, in persons exposed to mercury. Acrodynia is characterized by itching, flushing, swelling, and/or desquamation of the palms of the hands or soles of the feet, morbilliform rashes, excessive sweating and/or salivation, tachycardia, elevated blood pressure, insomnia, weakness, irritability, fretfulness, and peripheral sensory disturbances (Warkany and Hubbard 1953). The occurrence of acrodynia was determined to be an idiosyncratic reaction to mercury exposure. Despite widespread exposure of children to mercury-containing laxatives, antiascariasis medications, and teething powders in the 1940s and 1950s, only a few children developed acrodynia. The affected population was not the most highly exposed; numerous reports identified higher exposures in others with no evidence of the disease. The physiological basis for this hypersensitivity is unknown, but patch testing indicated that it is not an allergic response to mercury exposure.

Animal studies (Aten et al. 1992; Druet et al. 1978; Hirszel et al. 1985; Hultman and Enestrom 1992; Matsuo et al. 1989; Michaelson et al. 1985; Pelletier et al. 1990; Pusey et al. 1990; Roman-Franco et al. 1978; van der Meide et al. 1993) and limited human data (Lindqvist et al. 1974; Tubbs et al. 1982) also indicate that there may be persons with a genetic predisposition to develop an autoimmune glomerulo-nephritis upon exposure to mercury. In this form of renal toxicity, proteinuria is observed following the reaction of autoantibodies with renal tissues and deposition of immune material (i.e., IgG and complement C3) in the renal mesangium and glomerular blood vessels. Both susceptible and resistant mouse and rat strains have been identified, and susceptibility appears to be governed by both MHC genes and nonMHC genes (Aten et al. 1991; Druet et al. 1978; Hultman and Enestrom 1992; Hultman et al. 1992; Michaelson et al. 1985; Sapin et al. 1984).

Unborn children are another known susceptible population to the toxic effects of mercury (see Section 2.2.2.4). Data from large-scale poisonings in Japan (Harada 1978) and Iraq (Marsh et al. 1987) indicate that infants exposed *in utero* to alkyl mercury compounds developed severe neurological toxicity whereas their mothers may have experienced no or only mild toxicity. This difference may be due to methylmercury binding to tubulin (Vogel et al. 1985, 1989) and the role of microtubules in neuronal cell division and migration in the developing nervous system (Sager et al. 1982). There is evidence indicating that the developing male fetus may be more susceptible to methylmercury exposure than the female fetus (Buelke-Sam et al. 1985; Grant-Webster et al. 1992; Sager et al. 1984).

Neonates may also be especially susceptible to mercury toxicity. Both inorganic and organic forms of mercury are excreted in the milk (Sundberg and Oskarsson 1992; Yoshida et al. 1992). Furthermore, suckling rats exhibit a very high absorption of inorganic mercury as a percentage of the diet (30–40%) compared to adult rats, which absorb approximately 1% of the inorganic mercury from the diet (Kostial et al. 1978). The highest oral toxicity to inorganic mercury as expressed by the LD_{50} was for 2-week-old rats; by 3–6 weeks of age, rats showed a dramatic drop in sensitivity to inorganic mercury poisoning (Kostial et al. 1978). The transfer of mercury to suckling rats through milk was found to result in greater concentrations of the metal in the brains of the offspring than in the mother (Yang et al. 1973). Developmental neurotoxicity, similar to that seen with *in utero* exposure, has been observed in an infant exposed to alkyl mercury only after birth (Engleson and Herner 1952).

Individuals with diseases of the liver, kidneys, lungs, and nerves are considered to be at greater risk of suffering from the toxic effects of both organic and inorganic mercury. Individuals with a dietary insufficiency of zinc, glutathione, antioxidants, or selenium or those who are malnourished may be more susceptible to the toxic effects of mercury poisoning because of the diminished ability of these substances to protect against mercury toxicity (see Section 2.8).

2.10 METHODS FOR REDUCING TOXIC EFFECTS

This section describes clinical practice and research concerning methods for reducing toxic effects of exposure to mercury. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for the treatment of exposures to mercury. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice.

Although there are a number of treatments currently available, none are completely satisfactory and additional development of treatment drugs and protocols is needed. The recent death of a researcher poisoned with dimethylmercury is a case in point (Nierenberg et al 1998; Toribara et al. 1997). In spite of prompt action and excellent medical care and monitoring, the clinical course in this patient continued to decline, and ultimately ended in death.

In general, even the inorganic mercurials, that are considered to be more easily chelated, are difficult to remove from the body and are not treated without some side effects. Infants and young children are

particularly difficult to treat, sometimes requiring exchange transfusion or other more elaborate measures. Reducing the body burden or toxic effects of mercury in pregnant women presents an even greater challenge (i.e., treatment must be effective for both the mother and the developing child), and specific treatment protocols are needed.

2.10.1 Reducing Peak Absorption Following Exposure

Strategies used to reduce absorption of mercury may differ depending on the route of exposure and the specific chemical to which one is exposed. Elemental mercury and certain organic forms of mercury have high vapor pressures and are readily absorbed by the lungs; inhalation of these chemicals may be the major exposure of concern. Because ingestion of most chemical forms of mercury is possible, strategies for limiting absorption from the gastrointestinal tract would be of utmost concern in such situations. The organic mercury compounds have greater absorption from the gut than elemental and inorganic mercury; thus, strategies differ depending on the form of mercury ingested. Dermal absorption of the various forms of mercury is also possible, so strategies should also consider limiting dermal absorption.

The first step in mitigating the toxic effects of inhalation and dermal exposures to mercury or its compounds is removal from the contaminated area or source (Bronstein and Currance 1988; Gossel and Bricker 1984; Haddad and Winchester 1990; Stutz and Janusz 1988). Since continued exposure may occur when clothing is contaminated, clothing may be removed as well (Bronstein and Currance 1988; Stutz and Janusz 1988). If dermal or ocular exposure has occurred, thoroughly washing the exposed areas with water has been suggested; treatment protocols recommend the use of Tincture of Green® soap a disinfectant) for the skin and normal saline for the eyes (Bronstein and Currance 1988; Stutz and Janusz 1988).

Several treatments have been suggested to reduce absorption of mercury from the gastrointestinal tract; however, most refer to the inorganic forms of mercury. It is likely that strategies that are effective in reducing the absorption of inorganic forms may also have some efficacy for organic forms. Several procedures that have been recommended for trapping mercury in the gastrointestinal tract are based on the mercury's affinity for binding to sulfhydryl groups. For example, oral administration of a protein solution (e.g., milk or egg whites) has been suggested to reduce absorption (Gossel and Bricker 1984; Haddad and Winchester 1990; Stutz and Janusz 1988). Salt-poor albumin administration has also been suggested (Haddad and Winchester 1990). Nonabsorbable agents (e.g., polystyrene resins containing sulfhydryl

groups) have been used to decrease the absorption rate of methylmercury (Clarkson et al. 1973). The oral administration of activated charcoal has also been suggested (Gossel and Bricker 1984; Stutz and Janusz 1988). Rapid removal of mercury from the gastrointestinal tract may be indicated in some acute, high-dose situations. In such situations, immediate emesis or gastric lavage has been suggested (Goldfrank et al. 1990; Haddad and Winchester 1990). Inclusion of salt-poor albumin or sodium formaldehyde sulfoxylate in the lavage fluid to convert the mercuric ion into the less soluble mercurous ion in the stomach has also been recommended (Haddad and Winchester 1990). Emesis is contraindicated following the ingestion of mercuric oxide, presumably because of the risk of damage to the esophagus as the potentially caustic compound is ejected. A saline cathartic, such as magnesium sulfate, to speed removal from the gastrointestinal tract has also been recommended unless diarrhea has already begun (Haddad and Winchester 1990; Stutz and Janusz 1988). Giving CaNa2-EDTA is contraindicated because it binds poorly to mercury, may be toxic to the kidneys, chelates other essential minerals, and may cause redistribution of mercury in the body (Gossel and Bricker 1984).

2.10.2 Reducing Body Burden

Since the main source of mercury exposure for the general public is organic mercury in the diet, minimizing the consumption of mercury-laden fish and shellfish is an effective means of reducing the body burden. The amount of inhaled mercury vapor from accidental spills of metallic mercury (e.g., from broken thermometers or electrical switches) can be minimized by informing the general public of the potential dangers and volatility of liquid mercury, and by prompt and thorough clean-up of liquid mercury spills.

Following exposure and absorption, metallic mercury is distributed primarily to the kidneys. Elemental mercury is highly soluble in lipids and easily crosses cell membranes (Gossel and Bricker 1984), particularly those of the alveoli (Florentine and Sanfilippo 1991). Once in the blood, this form of mercury can distribute throughout the body, as well as penetrate the blood-brain barrier, thus accumulating in the brain (Berlin et al. 1969). The body burden half-life of metallic mercury is about 1–2 months (Clarkson 1989). The kidney is also the primary organ of accumulation for compounds of inorganic mercury, but the liver, spleen, bone marrow, red blood cells, intestine, and respiratory mucosa are target tissues as well (Haddad and Winchester 1990; Rothstein and Hayes 1964). Inorganic mercury is excreted primarily through the kidneys; its half-life ranges from 42–60 days (Hursh et al. 1976; Rahola et al. 1973). As with elemental mercury, organic mercury compounds accumulate throughout the body (Aberg et al. 1969; Miettinen 1973). Accumulation of organic mercury also occurs in the liver, where it

is metabolized, excreted through the bile, and often reabsorbed in the gastrointestinal tract (Florentine and Sanfilippo 1991; Haddad and Winchester 1990). The half-life of lower alkyl mercurials is about 70–79 days (Aberg et al. 1969; Miettinen 1973).

For several years, diaphoresis (excretion through perspiration) was used to lower the body burden of mercury in miners exposed to mercury vapors (Sunderman 1978). Recently, this method of therapy has also been used to lower tissue levels of mercury in a patient exposed to metallic mercury in the manufacture of thermometers (Sunderman 1978).

Chelation therapy is presently the treatment of choice for reducing the body burden of mercury. There are currently a number of chelators that are either in practical use or under investigation in *in vivo* and *in vitro* studies (Florentine and Sanfilippo 1991; Gossel and Bricker 1984; Haddad and Winchester 1990). These chelators differ in their efficacy for various forms of mercury, routes of administration, side effects, and routes of excretion. Depending on the chemical to which one has been exposed and the health status of the individual, different chelators may be indicated. One popularly used chelator, dimercaprol or BAL, has two sulfhydryl groups that can bind mercury and compete with its binding to sulfhydryl groups in body tissues (Florentine and Sanfilippo 1991; Haddad and Winchester 1990). BAL is one of the more effective chelators for inorganic mercury salts. BAL is administered intramuscularly and is the preferred chelator when oral dosing is impractical (Florentine and Sanfilippo 1991; Gossel and Bricker 1984; Haddad and Winchester 1990). Approximately 50% of the dimercaprol-mercury complex is excreted through the kidneys, while the remainder is eliminated in the bile and feces. Thus, this chelator is preferred when renal impairment has occurred. BAL therapy, however, has several limitations. Significant reabsorption of mercury from the bile occurs (Shimada et al. 1993). Also, multiple toxic side effects including urticaria, elevated blood pressure and heart rate, nausea and vomiting, headache, conjunctivitis, lacrimation, and paresthesias have been reported (Goldfrank et al. 1990). Children may develop fevers, and individuals with a glucose-6-phosphatase deficiency may develop hemolysis. BAL treatment is contraindicated for elemental and organic mercury compounds because it has been shown to increase brain levels of mercury in animal studies when used to treat exposures to phenylmercury or methoxyethylmercury compounds (Berlin 1986; Berlin and Rylander 1964) or elemental mercury vapor (Goldfrank et al. 1990), indicating the possibility of increased neurotoxicity.

Another currently used mercury chelator is D-penicillamine. This drug has been used somewhat effectively to reduce the toxicity of elemental and inorganic mercury exposures. It can be taken orally, and its

metabolism is slight in humans. Penicillamine is removed though the kidneys (Florentine and Sanfilippo 1991). However, acute allergic reactions to penicillamine may occur (Goldfrank et al. 1990). An experimental drug, *N*-acetyl-D,L-penicillamine (NAP), is very similar to its analog, penicillamine, in its properties of absorption, metabolism, and excretion; however, it may be more mercury-specific in its chelating abilities and less toxic (Goldfrank et al. 1990; Haddad and Winchester 1990). A high success rate (88%) has been reported by investigators using NAP to treat victims of mercury inhalation (Florentine and Sanfilippo 1991).

2,3-Dimercaptosuccoinic acid (DMSA), an analogue of BAL, is another experimental chelating agent. DMSA can be given orally and is primarily excreted through the kidneys (Aposhian et al. 1992b). It has been shown to be an effective chelator for both inorganic and methylmercury (Magos 1967). Comparative studies have demonstrated that DMSA is as effective, if not more so, as dimercaprol, penicillamine, and NAP. Data also suggest that this chelating drug produces fewer adverse effects than NAP (Florentine and Sanfilippo 1991). 2,3-Dimercaptopropane-1-sulfonate (DMPS) is another BAL analogue that is an orally effective chelator for mercury. Reports differ with respect to which of these analogues is less toxic (Ellenhorn and Barceloux 1988; Goldfrank et al. 1990; Jones 1991; Karagacin and Kostial 1991). Better results were obtained in rats with DMPS than with DMSA when the chelating agent was administered at least 24 hours following exposure to mercuric chloride. However, early oral administration of DMPS (within 24 hours) resulted in increased mercury retention (Karagacin and Kostial 1991). In contrast, DMSA resulted in decreased mercury retention irrespective of when it was administered.

Hemodialysis with infusion of a chelator (cysteine, *N*-acetylcysteine, NAP) has been reported to be effective in some severe cases of poisoning where renal failure is a complication (Berlin 1986; Goldfrank et al. 1990; Haddad and Winchester 1990). It has been reported to be advantageous to begin the hemodialysis before substantial renal damage has occurred (Haddad and Winchester 1990).

Because methylmercury undergoes enterohepatic recirculation, nonabsorbable agents have been used to "trap" methylmercury excreted into the bile (Lund et al. 1984). A polystyrene resin containing sulfhydryl groups added to food at a concentration of 1% doubled the elimination rate of methylmercuric chloride when administered to mice. The elimination half-life decreased from 65 to 20 days (Clarkson et al. 1973). Excretion of methylmercury may also be enhanced by bile drainage either through catheterization and drainage of the choledochal duct or by surgical establishment of gallbladder drainage (Berlin 1986). However, this method has not been used therapeutically.

2.10.3 Interfering with the Mechanism of Action for Toxic Effects

The majority of metallic mercury vapor and organic mercury absorbed by the body is rapidly oxidized to the more toxic and soluble mercuric ion in the blood and tissues through a hydrogen peroxide catalase pathway (Clarkson 1989; Halbach and Clarkson 1978). It is believed that the high affinity of the cation for protein-containing sulfhydryl or thiol groups is the underlying mechanism for the biological activity of mercury (Clarkson 1972a; Hughes 1957; Passow et al. 1961). In a process that is not yet completely understood, mercury disrupts the intracellular sulfhydryl status, resulting in oxidative stress, followed by activation of catabolic enzymes (i.e., proteases, endonucleases), and ultimately in cellular injury (Verity and Sarafian 1991). Treatment with agents that reduce oxygen radical-producing reactions may be effective in reducing mercury-induced oxidative cell damage. For example, pretreatment of rats with deferoxamine, a potent iron chelator and inhibitor of iron-catalyzed oxygen radical-producing reactions, reduced the increase in reactive oxygen species seen in the cerebellum after methylmercury exposure (LeBel et al. 1992; Sarafian and Verity 1991). Similarly, treatment with *N*-acetylcysteine, an antioxidant, resulted in increased survival time and less severe lung lesions in rats following exposure to mercury vapor (Livardjani et al. 1991b). Vitamin E (alpha tocopherol) and *N,N*'-diphenyl-*p*-phenylenediamine therapy have antioxidant effects and have been shown to be effective in protecting against methylmercury-induced toxicity (Ganther 1980; Welsh 1979).

Strategies to block the oxidation of elemental mercury to mercuric ion through the hydrogen peroxide catalase pathway do not appear to be a viable method for mitigating the effects of mercury exposure because treatment with chemicals (e.g., ethanol) that have been shown to block this reaction (Nielsen-Kudsk 1965) result in higher levels of blood mercury and increased renal toxicity (Rumbeiha et al. 1992). Another option would be to reduce the oxidized mercury ions to the monovalent mercurous form. A treatment of this nature has been suggested for ingested inorganic mercury.

Metals and chemicals shown to be antagonistic to the toxic effects of mercury may offer a possible method of interfering with the mercury's mechanism of action. Selenium, as sodium selenite, has been used in counteracting mercury poisoning, although the specific mechanism is not understood (Mengel and Karlog 1980; Naganuma and Imura 1981). The efficacy of selenium administration also appears to be dependent on the form of mercury to which one is exposed. Co-administration of sodium selenite with mercuric chloride resulted in decreased renal toxicity, whereas co-administration with methylmercuric chloride had no effect on renal toxicity (Yasutake et al. 1991b). The nephrotoxic effects of inorganic mercury may be

protected against by pretreatment with zinc (Zalups and Cherian 1992). Data in rats suggest that zinc can induce metallothionein in the renal cortex and cause mercury accumulation in the kidneys to shift from the outer medulla to the cortex, where a greater percentage is bound to the induced metallothionein. However, despite its potential use for interfering with the mercury-induced renal effects, zinc also prolongs retention in the body.

2.11 ADEQUACY OF THE DATABASE

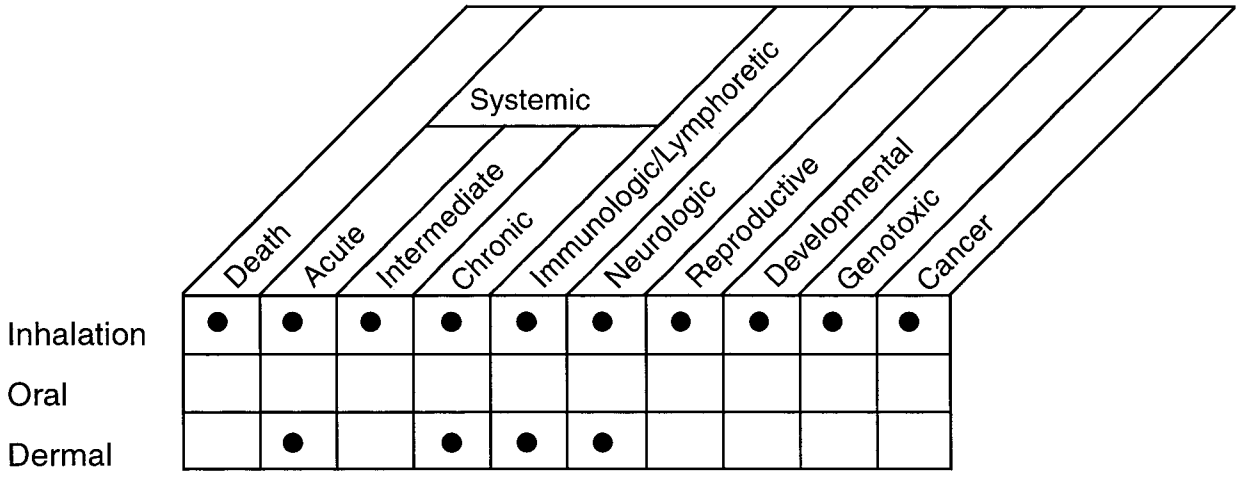
Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of mercury is available. Where adequate information is not available,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

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

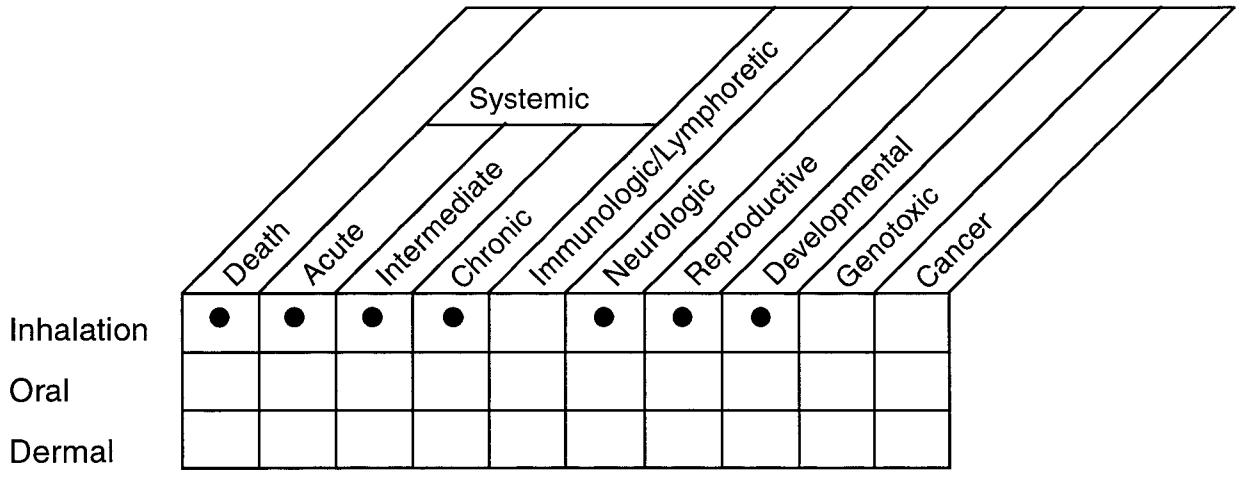
2.11.1 Existing Information on Health Effects of Mercury

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to inorganic and organic mercury are summarized in Figures 2-8 to 2-11. The purpose of these figures is to illustrate the existing information concerning the health effects of inorganic and organic mercury. Each dot in the figures indicates that one or more studies provide information associated with that particular effect. The dot does not imply anything about the quality of the study or studies. Gaps in this figure should not be interpreted as "data needs." A data need, as defined in ATSDR's Decision Guide for Identifying Substance-Specific Data Needs Related to Toxicological Profiles (ATSDR 1989), is substance-specific information necessary to conduct comprehensive public health assessments. Generally, ATSDR defines a data gap more broadly as any substance-specific information missing from the scientific literature.

Figure 2-8. Existing Information on Health Effects of Metallic Mercury



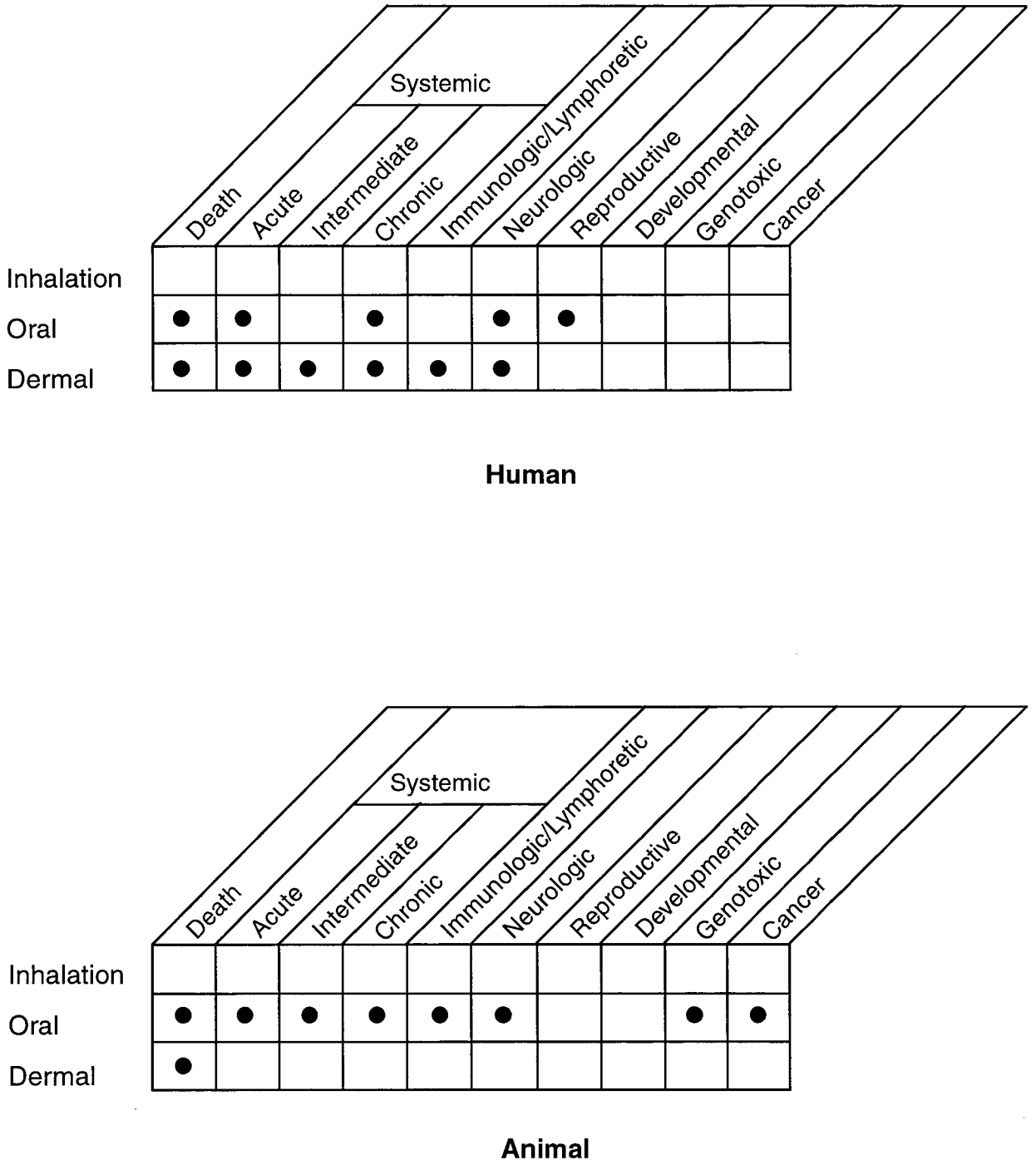
Human



Animal

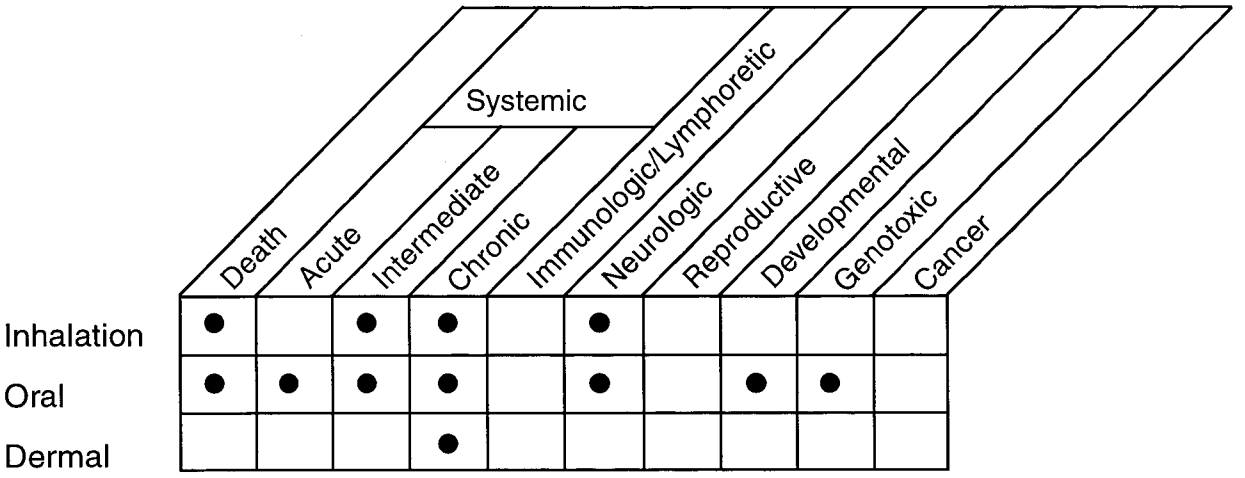
● Existing Studies

Figure 2-9. Existing Information on Health Effects of Inorganic Mercury Salts

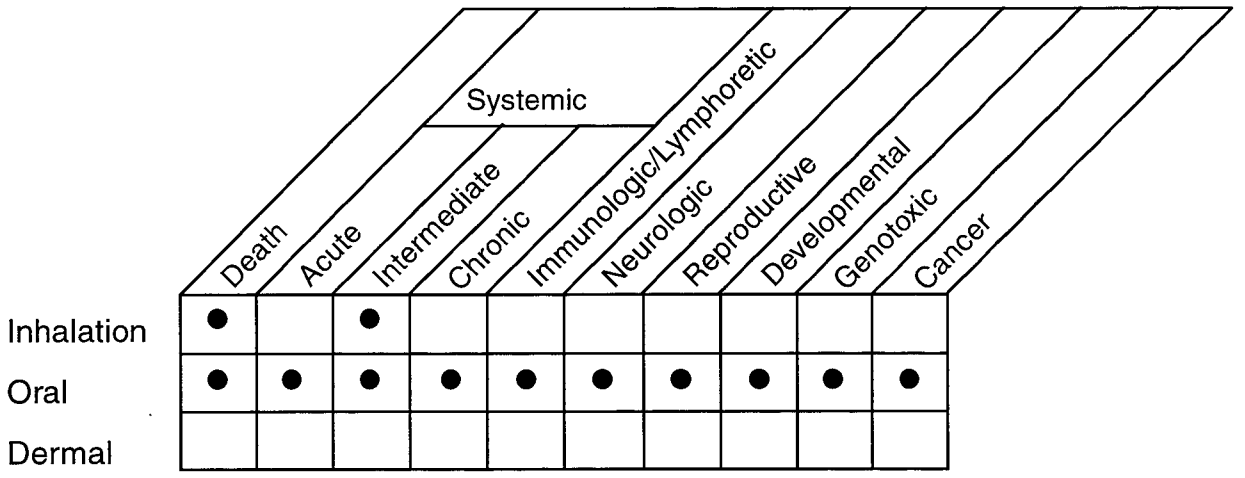


● Existing Studies

Figure 2-10. Existing Information on Health Effects of Methylmercuric Mercury



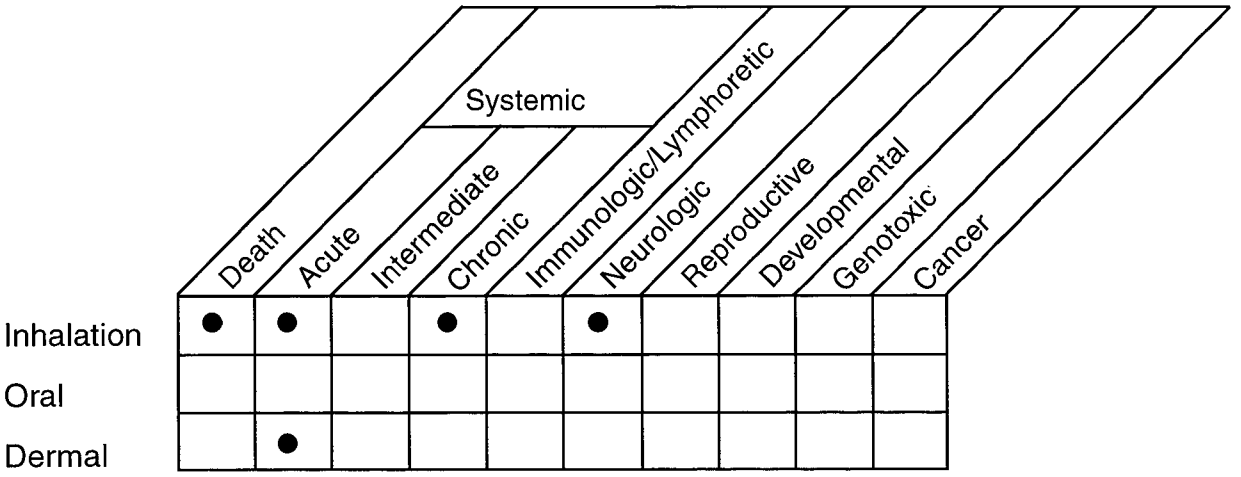
Human



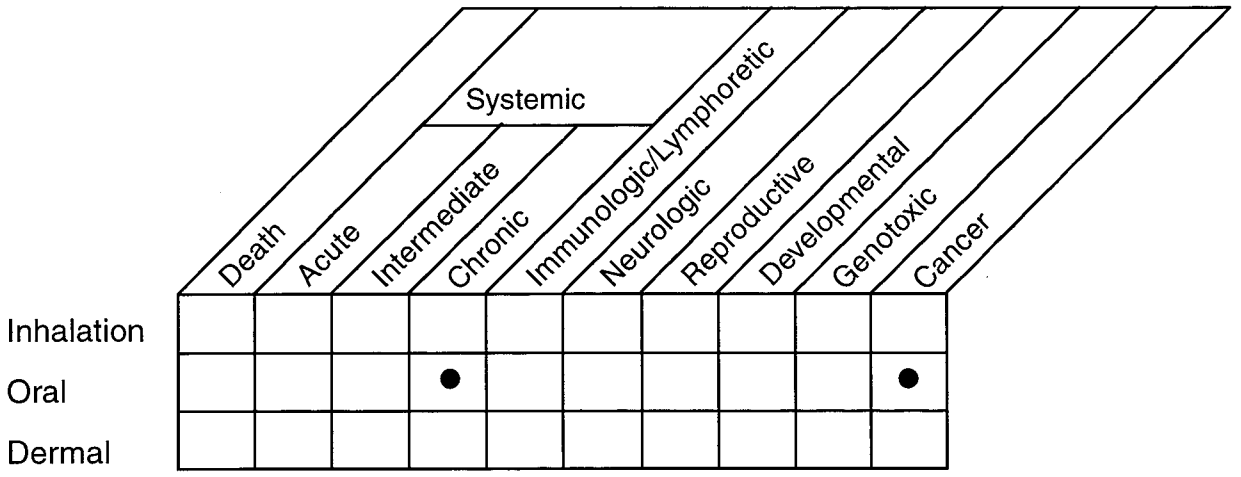
Animal

● Existing Studies

Figure 2-11. Existing Information on Health Effects of Phenylmercuric Mercury



Human



Animal

● Existing Studies

Information concerning metallic mercury exists primarily for the inhalation route of exposure in humans and animals (see Figure 2-8). Human data exist for all categories of effect following inhalation exposure to metallic mercury vapor. The results from inhalation studies in animals have been reported for all end points except immunological and genotoxic effects, and cancer. With the exception of case studies on contact dermatitis and neurological effects after acute and occupational dermal exposure to metallic mercury in humans, no studies were located for either the oral or dermal routes of exposure for either humans or animals.

Existing information on inorganic mercury salts is shown in Figure 2-9. No studies were found on the health effects from inhaled mercury salts in humans or animals. A number of case histories for acute or chronic oral exposure to mercury salts provide information on systemic and neurological effects and death. Some case histories and occupational studies provide information on dermal exposures to mercury salts at acute, intermediate, and chronic exposures leading to death, immunologic, neurologic, and systemic effects. No animal inhalation studies for inorganic mercury salts were identified, and only one acute study provides limited information on death from dermal exposure. A number of animal studies that have investigated the effects from oral exposure to mercury salts provide good information on systemic effects; limited information on cancer, neurologic, immunologic, and genotoxic effects; and no information on reproductive or developmental effects.

Information on methylmercuric and phenylmercuric mercury is presented in Figures 2-10 and 2-11. These two forms of organic mercury were chosen to represent the group of organic mercurials because they have been detected at Superfund sites, and because methylmercury is the predominant form of organic mercury in the environment. There is a paucity of information on phenylmercury. Only a few case histories are available for effects following inhalation exposure (death, acute or chronic systemic effects, and neurologic effects), and the information from these reports is very limited. Only one case history for acute systemic effects following dermal exposure to phenylmercury was identified. One chronic oral study in rats and a cancer study in rats and mice provide the only animal data for phenylmercury. In contrast, there are a number of human studies on systemic, neurologic, and developmental effects resulting from an oral exposure to methylmercury. No human toxicity data were identified for immunologic, reproductive, or genotoxic effect, nor for carcinogenicity. The human data for methylmercury are accompanied by a relatively large number of animal studies representing all three exposure durations and providing some, although often limited, information for all health effects categories. As with phenylmercury, there are only a few case histories for inhalation and dermal exposures, with limited information on neurologic and

systemic effects or death from acute poisonings. The animal data for inhalation exposure to methylmercury is equally scarce and nonexistent for dermal exposures.

2.11.2 Identification of Data Needs

Acute-Duration Exposure. The human toxicity information for acute duration exposures to mercury is limited to qualitative data and case histories following oral, inhalation, and dermal routes of exposure. Several case reports described death due to respiratory impairment from inhaled metallic mercury (Campbell 1948; Kanluen and Gottlieb 1991; Soni et al. 1992; Taueg et al. 1992). Respiratory, cardiovascular, gastrointestinal, hematological, and renal effects have been observed after acute-duration inhalation exposure to metallic mercury vapors (Bluhm et al. 1992a, 1992b; Campbell 1948; Garnier et al. 1981; Haddad and Sternberg 1963; Hallee 1969; Jaffe et al. 1983; Kanluen and Gottlieb 1991; Karpathios et al. 1991; Lilis et al. 1985; McFarland and Reigel 1978; Milne et al. 1970; Snodgrass et al. 1981; Soni et al. 1992; Taueg et al. 1992). Acute exposure to ingested inorganic mercury salts has also resulted in gastrointestinal and renal symptoms (Afonso and deAlvarez 1960; Kang-Yum and Oransky 1992). Tremors, irritability, and decreased motor function and reflexes are common neurological symptoms following high-level acute duration exposures to metallic mercury vapors (Adams et al. 1983; Bluhm et al. 1992a; Hallee 1969; Jaffe et al. 1983; McFarland and Reigel 1978; Snodgrass et al. 1981). Acute exposure to ingested methylmercury has resulted in both neurological and developmental toxicity (Al-Mufti et al. 1976; Amin-Zaki et al. 1974; Bakir et al. 1973; Cox et al. 1989; Engleson and Herner 1952; Harada 1978; Marsh et al. 1980, 1981, 1987; Snyder and Seelinger 1976). Information on short term dermal exposures in humans to inorganic mercury are from case studies, and provide some information on renal, neurological, immunological, and dermatological effects (Bagley et al. 1987; Bourgeois et al. 1986; DeBont et al. 1986; Faria and Freitas 1992; Kawahara et al. 1993; Millar 1916; Pambor and Timmel 1989).

Dermal effects from acute duration dermal exposures to organic mercury compounds have also been reported to a limited extent (Morris 1960). In a highly publicized poisoning, a laboratory researcher was thought to have received a single dermal exposure to the organomercurial, dimethylmercury (estimated at between 0.1 and 0.5 mL at a density of 3 g/mL), that apparently penetrated the researcher's latex safety gloves and resulted in a severe neurotoxicty 5 months later that subsequently ending with death (Blayney et al. 1997; Nierenberg et al. 1998; Toribara et al. 1997). Additional studies on dermal absorption of organic mercury, especially dimethylmercury, are needed to further evaluate the risk to human health.

Acute inhalation exposure to metallic mercury in rats and rabbits have resulted in death, respiratory, gastrointestinal, hepatic, renal, neurological, and/or developmental effects (Ashe et al. 1953; Fredriksson et al. 1992; Livardjani et al. 1991b). Acute oral exposures to inorganic mercury have resulted in renal, gastrointestinal, and thyroid effects in rats and/or mice (Dieter et al. 1992; Nielsen et al. 1991; NTP 1993; Sin et al. 1990) and neurological effects in rats (Chang and Hartmann 1972a, 1972b). An acute oral MRL was derived for inorganic mercury based on renal effects in rats (NTP 1993). Acute oral exposures to organic mercury have resulted in renal, neurological, developmental, and reproductive effects in rats, mice, guinea pigs, and rabbits (Arito and Takahashi 1991; Bornhausen et al. 1980; Cagiano et al. 1990; Chang and Hartmann 1972b; Guidetti et al. 1992; Hughes and Annau 1976; Inouye and Kajiwara 1988; Jacobs et al. 1977; Khera 1973; Khera and Tabacova 1973; Magos et al. 1985; Nolen et al. 1972; Post et al. 1973; Stoltenburg-Didinger and Markwort 1990; Yasutake et al. 1991b). Well conducted animal studies on neurological effects from an acute inhalation exposure to metallic mercury or to an acute dermal exposure to organic mercury are needed because of the potential for these kinds of exposures to populations near hazardous waste sites. The potential for latent or delayed expression of toxicity after an acute exposure to mercury from all the most likely routes and forms (especially for a dermal exposure to dimethylmercury) needs to be addressed.

Intermediate-Duration Exposure. Inhalation data on intermediate-duration exposure to metallic mercury vapors are limited to case reports of individuals exhibiting cardiovascular, gastrointestinal, hematological, renal, dermal, immunological, and neurological effects similar to acute exposures (Anneroth et al. 1992; Barber 1978; Fagala and Wigg 1992; Foulds et al. 1987; Friberg et al. 1953; Schwartz et al. 1992; Sexton et al. 1976; Taueg et al. 1992). Workers inhaling diethylmercury vapors developed gastrointestinal and neurological symptoms prior to death (Hill 1943). No inhalation exposure data are available on intermediate-duration exposure to mercuric mercury. Information on intermediate-duration oral exposure to inorganic mercury is limited to the observation of neurological symptoms in a boy who ingested Chinese medicine containing mercurous mercury for several months (Kang-Yum and Oransky 1992). Intermediate-duration oral exposure to organic mercury has resulted in dermal, neurological, and developmental toxicity (Al-Mufti et al. 1976; Amin-Zaki et al. 1974; Bakir et al. 1973; Cox et al. 1989; Harada 1978; Marsh et al. 1980, 1981, 1987; Snyder and Seelinger 1976). Intermediate-duration dermal exposure to inorganic mercury has resulted in adverse gastrointestinal, renal, and immunological health effects (Anneroth et al. 1992; Kang-Yum and Oransky 1992). No studies were located that examined effects resulting from intermediate-duration dermal exposure to organic mercury.

Inhalation exposure to metallic mercury vapors for an intermediate duration has resulted in renal and/or neurological effects in rabbits (Ashe et al. 1953) and rats (Fukuda 1971; Kishi et al. 1978). No studies were located regarding effects in animals after intermediate-duration inhalation exposure to organic mercury. An intermediate inhalation MRL was not derived for metallic mercury because studies were considered inadequate. Following intermediate-duration oral exposure to inorganic mercury, adverse cardiovascular, hepatic, and renal health effects have were observed in rats and mice exposed to mercuric chloride (Andres 1984; Carmignani et al. 1992; Dieter et al. 1992; Hultman and Enestrom 1992; Jonker et al. 1993a; NTP 1993; Rana and Boora 1992). Immunological and neurological health effects were also observed (Chang and Hartmann 1972a; Dieter et al. 1983; Hultman and Enestrom 1992). An intermediate oral MRL was derived for inorganic mercury based on increased kidney weight in rats (NTP 1993). Intermediate-duration oral exposure to organic mercury has resulted in adverse cardiovascular, renal, immunological, neurological, and developmental health effects in rats, mice, cats, and monkeys (Berthoud et al. 1976; Burbacher et al. 1988; Chang and Hartmann 1972a; Chang et al. 1974; Concas et al. 1983; Elsner 1991; Evans et al. 1977; Fowler 1972; Fowler and Woods 1977; Ganser and Kirschner 1985; Hirano et al. 1986; Ilback 1991; Khera and Tabacova 1973; Leyshon and Morgan 1991; Lindstrom et al. 1991; MacDonald and Harbison 1977; Magos and Butler 1972; Mitsumori et al. 1981; Olson and Boush 1975; Sharma et al. 1982; Tsuzuki 1981; Wakita 1987; Yip and Chang 1981). The data were insufficient to derive an intermediate-duration MRL for oral exposure to organic mercury because serious adverse health effects (e.g., neurological degeneration, behavioral changes) were observed at the lowest doses (Burbacher et al. 1988; Chang et al. 1974; Chang and Hartmann 1972a). No studies were located regarding intermediate-duration dermal exposure in animals. Because populations surrounding hazardous waste sites might be exposed to higher-than-normal levels of mercury for an intermediate duration, more quantitative information on metallic and organic mercury toxicity, specifically neurotoxicity, following inhalation and oral exposure in humans and animals is needed. The potential for latent or delayed expression of toxicity after an exposure of intermediate duration needs to be addressed.

Chronic-Duration Exposure and Cancer. Occupational exposure to metallic mercury vapors has been reported to result in adverse cardiovascular, gastrointestinal, renal, ocular, immunological, and reproductive health effects (Barregard et al. 1988, 1990; Bencko et al. 1990; Bidstrup et al. 1951; Buchet et al. 1980; Cardenas et al. 1993; Cordier et al. 1991; Danziger and Possick 1973; Ehrenberg et al. 1991; Kazantzis et al. 1962; Langworth et al. 1992b; Lille et al. 1988; Moszczynski et al. 1990b; Piikivi 1989; Piikivi and Hanninen 1989; Roels et al. 1982; Schuckmann 1979; Siblerud 1990; Smith et al. 1970; Stewart et al. 1977; Tubbs et al. 1982; Vroom and Greer 1972). Substantial evidence indicates that

chronic inhalation of metallic mercury vapors results in neurotoxicity (Albers et al. 1988; Bidstrup et al. 1951; Chapman et al. 1990; Discalzi et al. 1993; Ehrenberg et al. 1991; Fawer et al. 1983; Langauer-Lewowicka and Kazibutowska 1989; Langworth et al. 1992a; Levine et al. 1982; Melkonian and Baker 1988; Ngim et al. 1992; Piikivi and Hanninen 1989; Piikivi and Tolonen 1989; Piikivi et al. 1984; Shapiro et al. 1982; Smith et al. 1970; Verberk et al. 1986; Vroom and Greer 1972; Williamson et al. 1982). A chronic inhalation MRL was derived for neurological effects observed in workers chronically exposed to metallic mercury (Fawer et al. 1983). Very limited information is available indicating that chronic-duration inhalation of organic mercury (sometimes unspecified) causes adverse cardiovascular, gastrointestinal, renal, and neurological health effects (Brown 1954; Hook et al. 1954; Hunter et al. 1940; Williamson et al. 1982). Chronic-duration ingestion of mercurous chloride resulted in dementia and irritability (Davis et al. 1974). Qualitative and quantitative data on organic mercury exposure are provided by the neurological disorders associated with ingestion of methylmercury-contaminated fish, but the length of exposure is unknown (Kutsuna 1968). Chronic occupational exposure to alkyl mercury compounds caused neurological changes in humans (Lundgren and Swensson 1949). The available evidence indicates that the differences in toxicity between inorganic and organic mercury forms are largely the result of the differences in their distribution in the body. Information concerning methyl-mercury is much more extensive than that for phenylmercury, especially considering the outbreaks of methylmercury poisoning that have occurred in Japan and Iraq.

Cardiovascular and renal health effects in rats and mice after chronic-duration ingestion of inorganic mercury have been reported (Carmignani et al. 1989; Fitzhugh et al. 1950; NTP 1993). An intermediate oral MRL based on renal effects was derived for intermediate oral exposure to inorganic mercury (NTP 1993). Chronic-duration oral exposure to organic mercury has resulted in adverse gastrointestinal, renal, developmental, neurological, and reproductive health effects in rats, mice, cats, and monkeys (Charbonneau et al. 1976; Fitzhugh et al. 1950; Hirano et al. 1986; Mitsumori et al. 1981, 1990; Rice 1989c, 1992; Rice and Gilbert 1982, 1990, 1992; Solecki et al. 1991). A chronic MRL for oral exposure to organic mercury was derived based on a study of prenatal exposures in a fish-consuming population on the Seychelles Islands (Davidson et al. 1998). Additional chronic-duration data on neurological disorders following metallic and organic mercury exposure are needed because they are a sensitive end point. Furthermore, there is a potential for chronic exposure to higher-than-normal levels of mercury in populations living in the vicinity of hazardous waste sites.

Additional chronic-duration oral exposure information in animals concerning renal effects following inorganic mercury exposure is needed to evaluate the threshold of this effect in humans following chronic exposure. The data would be useful if populations living near hazardous waste sites were to be exposed chronically to inorganic mercury that leached into near-by wells or water supplies.

Forestomach squamous cell papillomas and thyroid follicular cell carcinomas have been observed in rats and renal tubule tumors have been observed in mice following oral exposure to mercuric chloride (NTP 1993). Renal tumors have also been observed in rats and mice after oral exposure to organic mercury (Hirano et al. 1986; Mitsumori et al. 1981, 1990; Solecki et al. 1991). These results suggest the potential carcinogenicity of mercury to humans. Therefore, additional chronic-duration animal studies on metallic, inorganic, and organic mercury are needed to confirm the findings of the NTP study. Additional long-term follow-up studies examining carcinogenicity in highly exposed populations (i.e., those involved in mercury mining, or the exposed Iraqi or Japanese populations) are needed to evaluate the likelihood of tumors appearing in humans.

Genotoxicity. Although there are data from several *in vivo* studies on rats (oral exposure) and mice (intraperitoneal) indicating that inorganic and organic mercury compounds can cause clastogenic effects in mammalian germinal cells, the differences in species sensitivity, and in some cases strain sensitivity, do not permit the use of these findings for predicting a potential hazard to human genetic material (Suter 1975; Zasukhina et al. 1983). Epidemiological studies of humans occupationally or accidentally exposed to mercurials were inconclusive, but the combined results from these studies did not suggest that metallic mercury and organic mercury are clastogens for human somatic cells (Anwar and Gabal 1991; Barregard et al. 1991; Mabille et al. 1984; Popescu et al. 1979; Verschaeve et al. 1976, 1979; Wulf et al. 1986). There is, however, convincing evidence that inorganic and organic mercury compounds can interact with and damage DNA *in vitro* (Williams et al. 1987). The outcome of this damage has not been characterized, but there is some indication that mercury compounds are weak mutagens for cultured mammalian cells. In addition, *in vitro* results with human cells (Betti et al. 1992) and animal cells (Howard et al. 1991) and *in vivo* data in mice (Ghosh et al. 1991) suggest that mercury compounds can cause clastogenic effects in somatic cells. Considering the problems stated above in using the whole animal data, and the apparent species- and strain-specific responses noted in the DNA damage tests with cultured mammalian cells, the *in vitro* data, while of interest, are probably not reliable indicators of potential adverse effects in humans exposed to mercury. Well controlled human epidemiological studies are needed to determine the genetic hazard of mercury compounds to humans.

Reproductive Toxicity. Occupational exposure to metallic mercury has not been shown to result in statistically significant effects on male fertility (Alcser et al. 1989; Lauwerys et al. 1985). However, an increase in the rate of spontaneous abortions may occur (Cordier et al. 1991). A spontaneous abortion occurred in a female after ingesting an acute dose of mercuric chloride (Afonso and deAlvarez 1960). There were no studies available on dermal exposure to metallic, inorganic, or organic mercury. Additional epidemiological studies on inhalation and dermal exposure to mercury are needed to evaluate the threshold of reproductive effects in workers (including dentists and dental assistants).

Inorganic mercury exposure caused a significant increase in the incidence of resorptions in hamsters (Gale 1974). Abortions and decreased mean litter size have been observed in rats, mice, guinea pigs, and monkeys following oral exposure to organic mercury (Burbacher et al. 1988; Hughes and Annau 1976; Inouye and Kajiwara 1988; Khera 1973). There was a decrease in conceptions and an increase in early abortions and stillbirths in female monkeys exposed orally to methylmercury for 4 months, but the menstrual cycle length was not affected (Burbacher et al. 1988). However, prolonged estrous cycles were found in rats inhaling metallic mercury (Baranski and Szymczyk 1973). Adverse effects on spermatogenesis and on histopathology of the testes have been reported in several studies in animals exposed to methylmercury (Hirano et al. 1986; Mitsumori et al. 1990; Mohamed et al. 1987). There was no information on reproductive effects following dermal exposure to mercury in animals. A 90-day study is needed to provide reproductive organ pathology data on male and female animals. Multigenerational studies for inorganic and organic mercury are also needed. Additional reproductive studies are needed because reproductive-aged populations near hazardous waste sites might be exposed to mercury.

Developmental Toxicity. Occupational exposure to metallic mercury in males did not result in statistically significant effects on malformations or the number of children born (Alcser et al. 1989; Lauwerys et al. 1985). The results from an inhalation developmental rat study (Baranski and Szymczyk 1973) suggest that metallic mercury vapors may cause a higher incidence of fetal malformations, resorptions, and deaths. Dermal studies on metallic mercury in humans and animals were not available. Additional well-conducted inhalation and dermal studies on metallic mercury in animals are needed to evaluate the potential for adverse developmental effects to humans from mercury.

Inorganic mercury exposure caused a significant increase in the incidence of resorptions in hamsters (Gale 1974). No other human or animal studies were available on developmental effects following inorganic mercury exposure. Therefore, additional studies for inhalation, oral, and dermal exposures are

needed to evaluate the potential developmental toxicity of inorganic mercury to populations, specifically young children, living near hazardous waste sites. Longitudinal studies for higher dose level acute and intermediate exposures are needed to determine the potential delayed expression of toxicity.

Prenatal exposure to methylmercury from contaminated food during the early stages of pregnancy has caused neurological damage in humans (Amin-Zaki et al. 1974; Bakir et al. 1973; Choi et al. 1978; Cox et al. 1989; Engleson and Herner 1952; Harada 1978; Marsh et al. 1980, 1981, 1987; Matsumoto et al. 1965; McKeown-Eyssen et al. 1983; Snyder and Seelinger 1976). Severe neurological impairment developed in a child exposed *in utero* to methylmercury, and effects were still present at 6 years of age (Snyder and Seelinger 1976). In animals, numerous oral exposure studies on the developmental effects of organic mercury have been conducted. Disruptions in the development of the nervous system in rats, mice, hamsters, and guinea pigs (Chang et al. 1977; Inouye and Kajiwara 1988; Khera and Tabacova 1973; Reuhl et al. 1981a, 1981b) and in the immune system in rats (Ilback et al. 1991) have been reported. Behavioral changes were also observed in rats and mice (Bornhausen et al. 1980; Hughes and Annau 1976; Olson and Boush 1975). Additional long-term inhalation, oral, and dermal studies for inorganic and organic mercury are needed to evaluate the threshold of developmental effects in workers chronically exposed to mercury or in populations living near hazardous waste sites.

Immunotoxicity. The results from two occupational studies indicate a decreased serum IgG levels in workers to inhaled metallic mercury vapors (Bencko et al. 1990; Moszczynski et al. 1990b), but these studies are limited and did not evaluate potential confounders (smoking and alcohol). Other studies in similarly exposed populations did not observe an increases in serum immunoglobulins (IgA, IgG, IgE, or IgM) and autoantibody titres (antilaminin or antiglomerular basement membrane antibodies) (Bernard et al. 1987; Cardenas et al. 1993; Langworth et al. 1992b). There is limited information in humans that suggests that certain individuals may develop an autoimmune response (Tubbs et al. 1982; Moszczynski et al. 1995). Data on immunological effects following oral exposure to organic mercury compounds in humans are not available. Oral exposures to inorganic and organic mercury in animals indicate that the immune system may be a target organ for mercury. Immune deposits were observed in the intestines and kidneys of rats exposed to mercuric chloride for 2 months, but no functional changes were evident in these tissues (Andres 1984). Suppression of the lymphoproliferative response occurred at a higher dose of mercury in mice exposed to mercuric chloride for 7 weeks (Dieter et al. 1983). Reduced natural killer cell activity in spleen and blood was exhibited in mice administered a diet containing methylmercury for 12 weeks (Ilback 1991). It is unknown how an adverse effect on the immune system from exposure to one form of mercury

might affect the response to other forms or other routes of exposure (e.g., how an adverse immune effect induced by inhalation of mercury vapor from dental amalgam might effect the dose-response from exposure to ingested methylmercury). Therefore, the potential for immunotoxic effects from exposure to mercury vapor, mercury salts, or methylmercury separately or in combination is of considerable importance and warrants further research, especially from low level chronic exposures.

Neurotoxicity. The nervous system is the major target organ for metallic and organic mercury through inhalation and oral routes, respectively. In humans, the neurological effects of metallic mercury have been observed primarily after acute high-concentration exposures (accidental) to intermediate and chronic low-concentration exposures (occupational). Tremors and irritability are the most prominent symptoms of inhaled metallic mercury in humans (Albers et al. 1988; Bidstrup et al. 1951; Fawer et al. 1983; Piikivi et al. 1984). Information on effects in humans from oral exposure includes case histories, for example, a chronic oral exposure to a laxative containing mercurous chloride (Davis et al. 1974), acute to intermediate duration ingestion of high levels of methylmercury-contaminated food (Bakir et al. 1973; Kutsuna 1968), or to chronic low-level exposures from fish or marine mammals containing methylmercury (Davidson et al. 1995aa, 1995b; Grandjean et al. 1997b, 1998). Case histories of dermal exposure to inorganic mercury cite similar neurological effects from acute (Bourgeois et al. 1986; DeBont et al. 1986) or chronic exposures (Dyall-Smith and Scurry 1990).

The neurotoxicity of inhaled metallic mercury has been studied in animals for acute and intermediate exposures (Ashe et al. 1953; Ganser and Kirschner 1985; Kishi et al. 1978). Behavioral, motor, and cognitive effects, as well as histopathological changes in the brain, were reported in rats, rabbits, and mice. Neurological disturbances in rats and mice resulted from acute, intermediate, and chronic oral exposures to mercuric mercury (Chang and Hartmann 1972b; Ganser and Kirschner 1985). Oral exposure to organic mercury in animals produced a range of neurological changes (Charbonneau et al. 1976; Evans et al. 1977; Magos and Butler 1972; Rice and Gilbert 1982; Sharma et al. 1982; Tsuzuki 1981). A chronic inhalation MRL was derived for metallic mercury. Additional animal studies are needed, however, to evaluate the neurotoxicity of inorganic mercuric salts to resolve some of the conflicting findings from pervious work (Chang and Hartmann 1972b; Ganser and Kirschner 1985; Goldman and Blackburn 1979; NTP 1993). *In vivo* studies are needed to evaluate the mechanisms of neurotoxic effects seen in *in vitro* studies, i.e., the lipoperoxidation and cell injury in methylmercury-exposed cerebellar granule cells (Sarafian and Verity 1991). Further evaluation is needed in humans and animal models of the potential for neurological effects and delayed neurotoxicity from chronic low level exposures to organic and inorganic mercury, especially

from multiple sources (i.e., organic mercury from fish consumption in conjunction with metallic mercury released from dental amalgam).

Epidemiological and Human Dosimetry Studies. There have been a number of occupational studies on workers chronically exposed to metallic mercury vapors. Mercury exposure (as measured by urine or blood mercury levels) and neurological effects have been evaluated (Adams et al. 1983; Miller et al. 1975; Roels et al. 1982; Smith et al. 1970). The most obvious deficiency in these epidemiological studies is the absence of good measures of exposure. Additional data are needed on the potential health effects for populations near hazardous waste sites based upon specific identification of the form of mercury and the pathways of exposure (i.e., the levels of exposure that populations near waste sites may actually experience from inorganic mercury in the air, water, and soil, or methylmercury in contaminated food). An area of considerable controversy, which is in need of good epidemiological data, is the potential for adverse effects from the mercury released from dental amalgam. Although this is not an exposure pathway associated with hazardous waste sites, mercury from amalgam represents a major contributor to the total body burden for a large percentage of the population, and thus must be factored into an assessment of the toxicokinetic behavior and toxic effects of mercury originating from a waste site. Long term longitudinal studies are needed for all dose durations and forms to evaluate delayed or persistent expression of mercury toxicity.

Biomarkers of Exposure and Effect

Exposure. Blood and urine mercury levels have been used as biomarkers of high level exposure in acute and chronic studies for both inorganic and organic mercury (Akesson et al. 1991; Naleway et al. 1991; Verschoor et al. 1988). Hair has been used as a biomarker for chronic low level organic mercury exposure (Nielsen and Andersen 1991a, 1991b; Oskarsson et al. 1990), with an awareness of the potential for external contamination (Clarkson et al. 1983). Further development of more sensitive tests to measure mercury in expired air and retention in hair are needed for monitoring short- and long-term exposures, respectively, for populations at risk.

As seen in other studies comparing European to Japanese hair mercury levels, the hair levels reported by Nakagawa (1995) of 2–4 ppm for a Japanese population are 10–20 times higher than levels observed in the Drasch et al. (1997) study (median, 0.247 µg/g in hair; range, 0.43–2.5 µg/g). These differences in the mercury exposure may affect not only the mercury hair levels but also the mercury hair-to-tissue

correlations. Further study is needed on the effects that the exposure level of methylmercury (as well as other forms of mercury) has on tissue distributions and the correlation to biomarkers of exposure.

There are potential confounding factors and other factors to consider when assessing mercury exposure based upon mercury hair levels. Mercury may be deposited to hair from the air when significant sources of mercury are present in the air or when certain hair treatments are used (Hac and Krechniak 1993; WHO 1991). Potential sources of external mercury exposure should, therefore, be evaluated as part of an exposure assessment. Some studies also report a sex related difference in mercury tissue levels. Nielson et al. (1994) observed a significant sex-related differences in the toxicokinetics of methylmercury in mice following administration of a single radiolabeled dose. Drasch et al. (1997) reported that mercury levels in all tissues assayed in their human cadaver study had higher levels compared to male tissues. The difference was significant for the kidney (median female kidney mercury level=92.0 ng/g, males=40.8 ng/g; p=0.002). In blood and urine there was a similar trend. In contrast, the authors report that mercury hair levels in females were significantly lower than in males (median females=205 ng/g, males 285 ng/g; p=0.02). Nakagawa (1995) also report higher mean mercury hair levels in males (2.98 µg/g) compared with females (2.02 µg/g) in a Japanese population. Further research is, therefore, needed to characterize potential sex related difference in the toxicokinetics of mercury under different exposure scenarios.

Further research on other biomarkers of mercury does not warrant a high priority.

Of particular importance is the collection of pharmacokinetic data showing the relationship between low-level exposure (acute, intermediate, and chronic) and blood and urine levels throughout the study .duration. Also tissue levels at necropsy should be taken immediately after cessation of dosing. In animal studies, a similar group of animals should be followed for urine (and blood, but not as important here) mercury levels for periods of 30, 60, 90, and 120 days postdosing to examine whole-body excretion, and necropsy tissue samples should also be taken from several animals at 30, 60, 90, and 120 days postdosing. Primates would be the best animal model, but rodent models could suffice.

A needed study is a longitudinal epidemiology study that tracked daily individual exposure levels in chloralkali industry workers, fluorescent lightbulb manufacturers, or other mercury utilizing industries, and associated these exposure levels with weekly urine and blood samples for a period of 1–2 years. Neurobehavioral testing (using tests from ATSDR's recommended test battery for adults) should be used

conducted at 6-month intervals. Workers new to these industries would be the best subjects, since their pre-exposure blood and urine levels could be used as reference values.

A biomarker/exposure could also be conducted in persons with dental amalgam fillings. Urine levels should be tracked in those with fillings and in those with removed or replaced amalgam fillings. There are a number of confounding factors and logistical difficulties in conducting such studies, and new study protocols should be developed to address the problems encountered in previous studies.

Effect. Potential biomarkers of effect for mercury-induced renal toxicity have been well described (Cardenas et al. 1993; Lauwerys et al. 1983; Rosenman et al. 1986; Verschoor et al. 1988). Biomarkers for neurological changes (e.g., paresthesia, decreased motor function, and impaired nerve conduction) have also been described (Clarkson et al. 1976; Shapiro et al. 1982). There is long history of evaluation of the neurophysiological and neuropsychological effects associated with mercury levels in blood, urine, and (Levine et al. 1982; Vroom and Greer 1972; Williamson et al. 1982). More recently, studies are evaluating cognitive and neurobehavioral effects with increasing sophistication in the assays and analyses that are used (Davidson et al. 1998; Grandjean et al. 1997b, 1998). Additional biomarkers are needed in this continuing effort to resolve subtle cognitive or neurobehavioral effects, and immune system effects from chronic low level exposures to methylmercury in food or metallic mercury released from dental amalgam, especially in sensitive populations.

Absorption, Distribution, Metabolism, and Excretion. Limited data are available to assess the relative rate and extent of absorption in humans following inhalation exposure to metallic mercury (Barregard et al. 1992; Berlin et al. 1969; Friberg and Vostal 1972; Hursh et al. 1976; Teisinger and Fiserova-Bergerova 1965) and in humans and animals following oral exposure to both inorganic salts and organic mercury (Aberg et al. 1969; Clarkson 1971, 1972a, 1989; Endo et al. 1989, 1990; Fitzhugh et al. 1950; Friberg and Nordberg 1973; Kostial et al. 1978; Miettinen 1973; Nielsen 1992; Nielsen and Andersen 1992; Rice 1989b; Suzuki et al. 1992; Urano et al. 1990; Weiss et al. 1973; Yeoh et al. 1989). Indirect evidence of absorption following inhalation exposure in humans and animals is reported for inorganic and organic mercury (Clarkson 1989; Ostlund 1969; Warfvinge et al. 1992; Yoshida et al. 1990, 1992). Only limited quantitative data were located regarding dermal uptake of metallic mercury in humans (Hursh et al. 1989). Information is needed regarding the rate and extent of dermal absorption of inorganic and organic mercury in humans and animals. Quantitative information concerning the inhalation and oral absorption of mercury (all forms)are needed.

In general, quantitative data are available to evaluate the rate and extent of distribution, metabolism, and elimination of mercury in humans and animals following inhalation and oral exposure. Data on distribution, metabolism, and excretion following dermal exposure are lacking for all forms of mercury. The distribution data for metallic, inorganic and organic mercury are similar in humans and animals (Aschner and Aschner 1990; Berlin 1963; Cherian and Clarkson 1976; Cherian et al. 1978; Clarkson 1989; Clarkson and Magos 1978; Danscher et al. 1990; Grandjean et al. 1992; Nielsen and Andersen 1990, 1991a, 1991b; Nordberg 1976; Schionning et al. 1991; Sin et al. 1983; Suzuki et al. 1992; Warfvinge et al. 1992; Yeoh et al. 1989; Yoshida et al. 1990, 1992). No quantitative distribution data were located for organic mercury compounds following inhalation exposure. The oxidation and reduction reactions that control the disposition of elemental mercury were identified in both animals and humans (Clarkson 1989; Halbach and Clarkson 1978; Nielsen-Kudsk 1973). Quantitative data on the biotransformation of organic mercury are limited (Norseth and Clarkson 1970). Reliable quantitative evidence on excretion of metallic and inorganic mercury in humans and animals following inhalation exposure is available (Cherian et al. 1978; Hursh et al. 1976; Joselow et al. 1968b; Lovejoy et al. 1974).

As discussed in the section on data needs for biomarkers, further study is needed on the effects that the exposure level of methylmercury (as well as other forms of mercury) has on tissue distributions and the correlation to biomarkers of exposure. Age appears to be a factor in the elimination of mercury in rats following inorganic and organic mercury exposures (Daston et al. 1986; Thomas et al. 1982). Elimination of methylmercury in rats may also be sex-related (Ballatori and Clarkson 1982). Nielson et al. (1994) observed a significant sex-related differences in the toxicokinetics of methylmercury in mice following administration of a single radiolabeled dose. Drasch et al. (1997) reported that mercury levels in all tissues assayed in their human cadaver study had higher levels compared to male tissues. Nakagawa (1995) also report higher mean mercury hair levels in males (2.98 µg/g) compared with females (2.02 µg/g) in a Japanese population. Further research is, therefore, needed to characterize potential sex related difference in the toxicokinetics of mercury under different exposure scenarios.

Insufficient data are available to assess whether or not there are any differences in absorption, distribution, metabolism, and excretion of mercury with respect to time or dose (i.e., if saturation phenomena occur). The majority of the available toxicokinetic data involve acute exposures to single doses. For all three routes, studies are needed that compare various dose levels and durations in order to determine if there are any differences in the toxicokinetics of mercury. Little is known about how mercurials are eliminated from specific organs. In particular, the mechanism by which mercury is eliminated from the brain is

unknown. This information is needed to design better treatment drugs and protocols. Mechanistic studies are needed on how mercury (in its various forms) is excreted and how such activities can be enhanced.

An important priority research and data need is a study of the effects of dietary selenium on the absorption and toxicity of methylmercury. Primates would be the most appropriate species for such a study. Oral dosage levels (in food) should cover an sufficient dose range to provide useful information for high fish consuming populations. Mercury excretion should also be measured and compared with controls at least weekly, with the entire study length being not less than 6 months, and preferably one to two years in duration. Concurrent neurobehavioral testing should be included, if possible, and be conducted at fixed intervals depending upon the duration of the study.

Comparative Toxicokinetics. There is only limited data available on species differences in absorption rates following oral exposures to all forms of mercury, and the results are negative (i.e., no differences) (Clarkson 1971, 1972a; Friberg and Nordberg 1973; Nielsen and Andersen 1990; Rice 1989b). There are data concerning inhalation absorption of metallic and inorganic mercury (Berlin et al. 1969; Cherian et al. 1978; Clarkson 1989; Hursh et al. 1976); however, the data are insufficient to allow for interspecies comparisons (Ostlund 1969). Studies comparing the inhalation absorption of all forms of mercury in humans and animals are needed to improve the utility of animal data in assessing human risk. The limited information available on dermal exposure suggests that dermal absorption of both inorganic and organic mercury compounds occurs in humans and animals, although no comparison of the rate or extent of absorption can be made between species (Gotelli et al. 1985; Hursh et al. 1989; Laug and Kunze 1949; Schamberg et al. 1918). As with inhalation exposure, studies comparing the dermal absorption of all forms of mercury in humans and animals are needed to improve the utility of animal data for assessing human risk.

The distribution of mercury in humans and animals appears to be similar. The lipophilic nature of metallic mercury results in its distribution throughout the body in humans (Takahata et al. 1970) and in animals (Berlin and Johansson 1964; Berlin et al. 1966). Distribution of inorganic mercury compounds resembles that of metallic mercury; however, human distribution is preferentially to the kidneys, liver, and intestines. Also, levels in the brain are substantially lower, as these compounds have a lower lipophilicity. Distribution of organic mercury compounds is also similar to that of metallic mercury. The ability of methylmercuric compounds to cross the blood-brain and placental barriers enables ready distribution to all tissues, although, again, the highest levels are found in the kidneys. Phenylmercuric compounds are

initially distributed in a similar manner to methylmercury; however, the distribution eventually resembles that of inorganic mercury.

The available evidence suggests that feces and urine constitute the main excretory pathways of metallic mercury and inorganic mercury compounds in both humans and animals. Additional excretory routes following metallic and inorganic mercury exposure include exhalation and secretion in saliva, sweat, bile, and breast milk (Joselow et al. 1968b; Lovejoy et al. 1974; Rothstein and Hayes 1964; Sundberg and Oskarsson 1992; Yoshida et al. 1992). Excretion following exposure to organic mercury is considered to be predominantly through the fecal route in humans. Evidence from studies in humans and animals (mice, rats) suggests that exposure to methylmercury leads primarily to biliary secretion, while excretion is initially through the bile; it then shifts to the urine following phenylmercury exposure (Berlin and Ullberg 1963; Berlin et al. 1975; Gotelli et al. 1985; Norseth and Clarkson 1971). No further comparative studies on excretion are warranted because there is no apparent difference in the excretion of mercury in any form in humans and animals.

Two PBPK models have recently been published on the pharmacokinetics of methylmercury in rats (Farris et al. 1993; Gray 1995). Additional PBPK studies are needed to support species and dose extrapolations, and a better understanding of the underlying toxic and kinetic mechanisms is needed in support of human risk assessments.

Validation of *in vitro* data is a major need. Much of the data from *in vitro* experimentation is based on unrealistic concentrations of the toxicant or is derived from studies using non-physiological designs. In particular, more validation is needed for immunotoxicity studies and biochemical studies.

Methods for Reducing Toxic Effects. Nonspecific methods or treatments for reducing absorption following mercury exposure include the administration of chelators or protein solutions to neutralize and bind to inorganic mercury compounds (Bronstein and Currance 1988; Florentine and Sanfilippo 1991; Gossel and Bricker 1984). The use of a particular chelator is dependent upon the type of mercury exposure (Gossel and Bricker 1984). Chelation therapy is the treatment of choice for reducing the body burden of mercury (Florentine and Sanfilippo 1991; Gossel and Bricker 1984; Haddad and Winchester 1990). However, chelation releases mercury from soft tissues that can then be redistributed to the brain. Additional research is needed to elucidate the mechanisms of absorption and distribution of inorganic and organic mercury. Animal studies suggest that antioxidants may be useful for decreasing the toxicity of

mercury. Additional work studying the effectiveness of prophylactic administration of vitamin E (or other antioxidants) and of proper diet are needed. Improved chelation and drug therapies for treating acute and chronic mercury poisonings are greatly needed.

Children's Susceptibility. The systemic health effects from different forms of mercury and exposure routes have been fairly well characterized (EPA 1997; Sue 1994; WHO 1990). There is generally sufficient information on the symptoms to resolve the form and route of exposure when children are exposed to high levels of mercury. There is less information to assist the physician or public health official in recognizing the symptoms that might arise from lower level exposure to multiple forms of mercury (e.g., dental amalgam and fish) and multiple pathways (inhalation and ingestion). Whether concurrent exposures would result in a different presentation of symptoms would be important information in determining the best therapeutic treatment. Some health effects categories are not well defined (e.g., immune responses). Earlier identification of immunotoxicity is of concern for children because of the progressive nature of hypersensitization to environmental pollutants, and the burden that a compromised immune system can place on a person's long-term health.

There are not presently adequate measures for neurologic development. Delayed developmental effects are of grave concern for children exposed to mercury; methods for early determination and detection of progressively worsening changes in a child's behavioral or cognitive function are needed. For the measures to be truly useful they should in some way be integrated into a more directed exposure assessment and body burden analysis and to resolve the contribution from other influences on cognitive abilities and behaviors. Other data needs related to developmental effects are discusses above under Developmental Toxicity.

Pharmacokinetics are different for children, and more data are needed to improve chelation therapies for both acute high-level poisoning and for chronic low-level exposures. This is perhaps the area that deserves the most attention because accidental poisonings continue to occur and there are virtually no therapies to ameliorate the inevitable progression of mercury intoxication. Since environmental levels of mercury are also continuing to rise, and levels in food will concurrently rise, strategies to boost the body's ability to eliminate absorbed mercury are going to become increasingly important (i.e., the alternative is to change dietary patterns, i.e., eat less fish, and the risk/benefits of doing that are already being hotly debated).

There appears to be adequate information on the metabolism of mercury, and there are no special metabolites or metabolic pathways that are unique to children and require further evaluation.

The mechanism of mercury toxicity is still largely unknown. It is not known whether there are unique mechanisms of action for the toxic effects in children that would require special consideration for treatment modalities, but at present it appears that target site is determined more by the pharmacokinetics (i.e., which tissues end up with the highest levels) than by a specific mechanism of action (e.g., a receptor binding-process initiating type of mechanism).

The results of a number of accidental food poisonings indicate that children are more vulnerable, and this vulnerability may be a function of easier access of mercury to the systemic circulation and brain, or it may be because disruption of cell growth and organization is more critical for children in developmental stages of growth. More data are needed to determine if the vulnerability of children is due to less plasticity to insult of analogous target tissue in adults, or because target tissues actually receive more toxic agent.

There are not adequate biomarkers of exposure nor adequate access to biomarkers of exposure. Hair, urine, and blood levels are gross measures of body burden and do not provide the essential information about levels of mercury at target tissues. Research is needed into better (preferably noninvasive) monitoring tools. Research is also needed on how to make monitoring tests readily and inexpensively available to the general public. Mercury is one of the top ten most hazardous substances, and its levels are increasing in the environment. There is considerable anxiety present in the general population about potential mercury toxicity from dental amalgam, but this occurs in the absence of good information on actual body burdens. The general public and health officials would benefit from readily available ways for individuals to measure personal and family member mercury body burdens.

The interactions of immediate interest are those that either affect absorption from the gastrointestinal tract or that prevent or reduce mercury toxicity. No information was identified to indicate that mercury interacts differently with iron or zinc, for example, in a child's body then it would in an adult, although the difference in children's physiology and morphology may result in a different response to that interaction. Except for the latter, which is again a toxicokinetic question, chemical interactions do not appear to be a data need.

There is a data need to develop better chelation therapies, better ways to prevent absorption of mercury into the body of children, and better ways to interfere with the mechanism of action, especially for damage to the nervous system. The current literature continues to grow with case histories of poisonings where

supportive therapy and passive observation of a progressively deteriorating health status are the best that can be done.

No information was found that parental exposure to mercury results in heritable defects or deficits in germ cell function that would be translated to the offspring. There is considerable information on the transfer of mercury from the mother to the developing child, both during the prenatal period via the placenta and during postnatal nursing; both inorganic mercury and organic mercury pass from mother to child. This is an area of active research primarily to characterize the dose, duration, and form of mercury to which the child is being exposed. Further work in this area is needed.

Child health data needs related to exposure are discussed in Section 5.8.1, Data Needs: Exposure of Children.

2.11.3 Ongoing Studies

Ongoing studies regarding mercury's health effects and mechanisms of action were reported in the Federal Research In Progress (FEDRIP 1998) database. Table 2-14 lists these studies.

Table 2-14. Ongoing Studies on Health Effects of Mercury

Author	Affiliation	Title	Sponsor
Tan, KH	Winston-Salem State University Winston-Salem, NC	Pilot study--astrocyte gene expression and methyl mercury neurotoxicity.	NCRR
Opella, SJ	University of Pennsylvania Philadelphia, PA	Structural studies of mercury transport protein.	NCRR
Prusiner, SB	University of California-SF San Francisco, CA	Nmr structures of recombinant prps.	National Institute of Neurological Disorders
Agre, PC	Johns Hopkins University Baltimore, MD	Red cell aquaporin-1 water transport protein.	National Heart, Lung, and Blood Institute
Rajanna, B	Alcorn State University Lorman, MS	Developmental neurotoxicity of lead and methyl nercury.	NIGMS
Mitra, AK	Scripps Research Institute San Diego, CA	Structure and function of the chip28 water channel.	NIGMS
Miller, S	University of California-SF San Francisco, CA	Cause and effect of dimer asymmetry in mercuric reductase.	NIGMS
Jensen, JL	California State University-LB Long Beach, CA	Reactivity of heavy metal ions with organosulfur moieties.	NIGMS
Rowland, AS		Effects of dental treatment during pregnancy on childhood development.	NIEHS
Rowland, AS		Chronic disease risks associated with mercury vapor exposure.	NIEHS
Longnecker, MP		Validity of toenail element levels as a surrogate measure of exposure.	NIEHS
Kamel, F		Lead and other neurotoxins as risk factors for amyotrophic lateral sclerosis.	NIEHS
Baird, DD		Environmental effects on fertility.	NIEHS
Morgan, DL		Prenatal effects of chemicals on the respiratory tract.	NIEHS
Knudsen,	Thomas Jefferson University Philadelphia, PA	Environmental impact on the embryonic mtdna genome.	NIEHS
Kono, DH	Scripps Research Institute San Diego, CA	Heavy metal induced autoimmunity.	NIEHS

Table 2-14. Ongoing Studies on Health Effects of Mercury (continued)

Author	Affiliation	Title	Sponsor
Frumkin, H	Emory University Atlanta, GA	Cohort study of former employees of a chloralkali plant.	NIEHS
Nelson, LM	Stanford University Stanford, CA	Exogenous toxicants and genetic susceptibility in als.	NIEHS
Weiss, B	University of Rochester Rochester, NY	Developmental neurotoxicity of metallic mercury.	NIEHS
Pollard, KM	Scripps Research Institute San Diego, CA	Immunotoxicology of a heavy metal.	NIEHS
Pollard, KM	Scripps Research Institute San Diego, CA	Mercury induced autoimmunity.	NIEHS
Aschner, M	Wake Forest University Winston-Salem, NC	Mechanisms of methylmercury induced neuronal toxicity.	NIEHS
Ballatori, N	University of Rochester Rochester, NY	Methylmercury transport across cell membranes.	NIEHS
Newland, MC	Auburn University at Auburn Auburn, AL	Behavioral teratology of methylmercury.	NIEHS
Kane, AS	University of Maryland Baltimore Baltimore, MD	Mechanisms underlying segment-specific nephrotoxicity.	NIEHS
Zalups, RK	Mercer University Macon Macon, GA	Transport and toxicity of mercury in the nephron.	NIEHS
Korrish, SM	Harvard University Boston, MA	*In utero* PCB and metal exposure and infant development.	NIEHS
Clarkson, TW	University of Rochester Rochester, NY	Dosimetry.	NIEHS
Myers, GI	University of Rochester Rochester, NY	Child development following prenatal methyl mercury exposure via fish diet.	NIEHS
Clarkson, TW	University of Rochester Rochester, NY	Health hazards of methylmercury.	NIEHS
Zalups, RK	Mercer University Macon Macon, GA	Mercury nephrotoxicity after a reduction of renal mass.	NIEHS
Reuhl, KR	Rutgers The State Univ New Brunswick New Brunswick, NJ	Mechanisms of mehg neurotoxicity during development.	NIEHS

Table 2-14. Ongoing Studies on Health Effects of Mercury (continued)

Author	Affiliation	Title	Sponsor
Gandolfi, AJ	University of Arizona Tucson, AZ	Metal-metal interactions in the kidney.	NIEHS
Schell, LM	State University of New York a Albany, NY	PCBs and well being of Mohawk children and youth--growth, development, cognition.	NIEHS
Checkoway, H	University of Washington Seattle, WA	Environmental and biochemical risk factors for Parkinson's disease.	NIEHS
Woods, JS	University of Washington Seattle, WA	Porphyrin profiles as biomarkers of trace metal exposure and toxicity.	NIEHS
Silva, P	Mount Desert Island Biological Salsbury Cove, ME	Mercury in chloride transport in shark rectal gland and rabbit thick ascending limb.	NIEHS
Preston, RL	Mount Desert Island Biological Salsbury Cove, ME	Mercury interaction with the taurine transport system of red blood cells.	NIEHS
Kinne, R	Mount Desert Island Biological Salsbury Cove, ME	Effects of cadmium and mercury on na-k-cl cotransporter in shark rectal gland.	NIEHS
Dawson, DC	Mount Desert Island Biological Salsbury Cove, ME	Effect of mercury on thiazide-sensitive sodium chloride cotransporter in flounder.	NIEHS
Boyer, J	Mount Desert Island Biological Salsbury Cove, ME	Mercury impairment of cell volume registration in skate hepatocytes.	NIEHS
Burbacher, TM	University of Washington Seattle, WA	Developmental effects of methylmercury.	NIEHS
Atchison, WD	Michigan State University East Lansing, MI	Neurotoxic mechanisms of methylmercury poisoning.	NIEHS
Bigazzi, PE	University of Connecticut Farmington, CT	Immune effects of metals--mercury-induced autoimmune disease.	NIEHS
Lawrence, DA	Wadsworth Center Albany, NY	Immunotoxicity of heavy metals.	NIEHS
Crawford, S	New England Research Institute Watertown, MA	Contribution of amalgam restoration to total body burden.	NIDCR
Janoff, EE	University of Washington Seattle, WA	Influence of dental amalgams on mercury and antibiotic resistant bacteria.	NIDCR

Table 2-14. Ongoing Studies on Health Effects of Mercury (continued)

Author	Affiliation	Title	Sponsor
De Rouen, TA	University of Washington Seattle, WA	Casa Pia study of dental amalgams in children.	NIDCR
Crawford, SL	New England Research Institute Watertown, MA	Health effects of dental amalgams in children.	NIDCR
Echeverria, D	Battelle Memorial Institute Columbus, OH	Neurologic effects of HgO exposure in dental personnel.	NIDCR
Factor-litvak, P	Columbia University Health Sci New York, NY	Dental amalgams and neuropsychological function.	NIDCR
Shenker, BJ	University of Pennsylvania Philadelphia, PA	Immunotoxic properties of mercuric compounds.	NIDCR
Kingman, A		Correlations between amalgam exposure and mercury levels in urine and blood.	NIDCR
Kingman, A		The NIDR amalgam study and health effects protocol.	NIDCR
Winn, DM		Occupation and reproductive health of women dentists.	NIDCR
Barron, DJ	University of Rochester Rochester, NY	Mercury toxicity and the blood-brain factor.	NIDCR
Sobel, ES	University of Florida Gainesville, FL	Hgc12 induction of systemic autoimmune disease in mice.	National Instute of Arthritis and Musculoskeletal and Skin
Casiano, CA	Loma Linda University Loma Linda, CA	Autoantigen cleavage during apoptosis and necrosis.	National Institute of Allergy and Infectious Diseases
Markesbery, WR	University of Kentucky Lexington, KY	Oxidative, antioxidant and trace element studies in the alzheimer's brain.	National Institute on Aging
Smith DE	North Carolina State University Raleigh, NC	Effects of metal ions on in vitro estrogen action in the rat uterus.	U. S. Department of Agriculture

NCRR = National Center for Research Resources; NIDCR = National Instute of Arthritis and Musculoskeletal Research; NIEHS = National Institute of Environmental Health Science; NIGMS = National Institute of General Medical Sciences

3. CHEMICAL AND PHYSICAL INFORMATION

3.1 CHEMICAL IDENTITY

Information regarding the chemical identity of mercury compounds is located in Table 3-1.

3.2 PHYSICAL AND CHEMICAL PROPERTIES

Information regarding the physical and chemical properties of mercury compounds is located in Table 3-2. Mercuric acetate has been included as an organic form of mercury. However, the bonds of the salt are not covalent and, in aqueous solution, the mercury behaves like an inorganic form.

Table 3-1. Chemical Identity of Selected Inorganic and Organic Mercury Compounds[a]

		Inorganic	
Characteristic	Mercury	Mercuric (II) chloride	Mercuric (II) sulfide
Chemical name	Mercury	Mercuric (II) chloride	Mercuric (II) sulfide
Synonym(s)	Colloidal mercury; liquid silver; mercury, metallic (DOT); quicksilver; metallic mercury[b]; hydrargyrum[c]	Bichloride of mercury; mercury bichloride[d]; mercury chloride[d]; mercury dichloride; mercury perchloride; mercury (II) chloride; perchloride of mercury; corrosive sublimate[d]; corrosive mercury chloride; dichloromercury	Etiops mineral[c]; mercury sulfide, black[d]; vermilion; chinese red; C.I. Pigment Red 106; C.I.77766[c]; quicksilver vermilion; chinese vermilion; red mercury sulfide; artificial cinnabar; red mercury sulfuret[d]
Registered trade name(s)	No data	Calochlor; Fungchex; TL 898	No data
Chemical formula	Hg[c]	$HgCl_2$[c]	HgS[c]
Chemical structure	Hg^c	Hg^{++} Cl^- Cl^-	Hg ═ S
Identification numbers:			
CAS registry	7439-97-6[c]	7487-94-7[c]	1344-48-5[c]
NIOSH RTECS	OVA4550000	OV9100000	No data
EPA hazardous waste	U151;D009	D009	No data
OHM/TADS	7216782	No data	No data
DOT/UN/NA/IMCO shipping	UN 2024 (mercury compounds, liquid); UN 2025 (mercury compounds, solid); IMO 6.1 (mercury compounds, liquid or solid); UN 2809 (DOT)[b]	UN 1624 (mercuric chloride) IMO 6.1 (mercuric chloride)	No data
HSDB	1208	33	No data
NCI	C60399[b]	C60173	No data
STCC	49 232 69 (mercury compound, solid), 49 443 25 (mercury, metallic)	49 232 45 (mercuric chloride) 49 232 71 (mercuric chloride, solid)	No data

Table 3-1. Chemical Identity of Selected Inorganic and Organic Mercury Compounds[a] (continued)

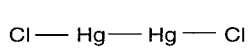
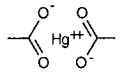
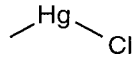
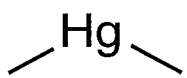
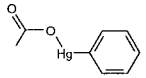
	Inorganic (continued)	Organic	
Characteristic	Mercurous (I) chloride	Mercuric (II) acetate[f]	Methylmercuric chloride
Chemical name	Mercurous (I) chloride	Mercuric (II) acetate	Methylmercuric chloride
Synonym(s)	Calomel; mild mercury chloride; mercury monochloride; mercury protochloride; mercury subchloride; calogreen; cyclosan[c]; mercury chloride[d]	Acetic acid, mercury (2+) salt; bis(acetyloxy) mercury; diacetocymercury; mercury diacetate; mercuriacetate; mercury (II) acetate; mercury (2+) acetate; mercury acetate[d]	Chloromethylmercury; monomethyl mercury chloride; methylmercury chloride; methylmercury monochloride[b]
Registered trade name(s)	Calogreen; Calomel Calotab; Cylcosan	No data	Caspan
Chemical formula	Hg_2Cl_2[b]	$HgC_4H_6O_4$[b]	CH_3HgCl[e]
Chemical structure			$(CH_3)^-Hg^{2+}Cl^-$ 
Identification numbers:			
CAS registry	10112-91-1[c]	1600-27-7	115-09-3[e]
NIOSH RTECS	OV8750000[b]	A18575000	OW1225000
EPA hazardous waste	No data	D009	No data
OHM/TADS	No data	No data	No data
DOT/UN/NA/IMCO shipping	No data	UN 1629 (mercury acetate); IMO 6.1 (mercury acetate)	No data
HSDB	No data	1244	No data
NCI	77764[b]	No data	No data
STCC	No data	49 232 41	No data

Table 3-1. Chemical Identity of Selected Inorganic and Organic Mercury Compounds[a] (continued)

	Organic (continued)	
Characteristic	Dimethyl mercury	Phenylmercuric acetate
Chemical name	Dimethyl mercury	Phenylmercuric acetate
Synonym(s)	Mercury, dimethyl; methyl mercury[c]	(Acetato)phenylmercury; acetoxyphenylmercury; phenylmercury acetate[c]; acetoxyphenylmercury; mercury (II) acetate, phenyl-; mercury, (acetato)phenyl-; phenylmercury acetate; phenylmercuriacetate
Registered trade name(s)	No data	PMA; PMAC; Pmacetate;Cerasan Slaked Lime; Gollitox; liquiphene; Mersolite; Tag Fungicide; Tag HL-331; Nylmerate; Scutl; Riogen; PMAS
Chemical formula	C_2H_6Hg[c]	$C_8H_8HgO_2$[c]
Chemical structure		
Identification numbers:		
CAS registry	593-74-8[c]	62-38-4[c]
NIOSH RTECS	No data	OV6475000
EPA hazardous waste	No data	PO92
OHM/TADS	No data	7216544
DOT/UN/NA/IMCO shipping	No data	UN 1674 (phenylmercuric acetate); IMO 6.1 (phenylmercuric acetate)
HSDB	No data	1670
NCI	No data	No data
STCC	No data	29 216 53

[a] All information obtained from HSDB 1997, except where noted. [b]RTECS 1997 [c]Merck 1989 [d]Lewis 1993 [e]ASTER 1997 [f] Although organic moieties are associated with the Hg atom, the mercury-carbon bonds are ionic, not covalent, in nature and in aqueous solution, Hg^{2+} is released.

CAS = Chemical Abstracts Service; DOT/UN/NA/IMO = Dept. of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data System; RTECS = Registry of Toxic Effects of Chemical Substances

Table 3-2. Physical and Chemical Properties of Selected Inorganic and Organic Mercury Compounds[a]

		Inorganic		
Property	Mercury	Mercuric (II) chloride	Mercuric (II) sulfide	Mercurous (I) chloride
Molecular weight	200.59	271.52	232.68	472.09
Color	Silver-white (liquid metal); tin-white (solid mercury)	White	Black or grayish-black (mercuric sulfide, black); bright scarlet-red blackens on exposure to light (mercuric sulfide, red)	White
Physical state	Heavy, mobile, liquid metal; Solid mercury is ductile, maleable mass which may be cut with a knife	Crystals, granules or powder; rhombic crystals, crystalline solid[c]	Heavy amorphous powder, also occurs as black cubic crystals (mercuric sulfide, black); powder, lumps, hexagonal crystals (mercuric sulfide, red)	Heavy powder; rhombic crystals or crystalline powder[b]
Melting point	-38.87 °C	277 °C	Transition temp (red to black) 386 °C; 583 , sublimes at 446 °C (mercuric sulfide, black)[b]; sublimes at 583 °C (mercuric sulfide, red)	Sublimes at 400–500 °C without melting; 302 °C[b]
Boiling point	356.72 °C	302 °C	No data	384 °C[b]
Density at °C	13.534 g/cm^3 at 25 °C	5.4 g/cm^3 at 25 °C	7.55-7.70 (mercuric sulfide, black), 8.06-8.12 g/cc (mercuric sulfide, red)[b]	7.15 g/cc; 6.993 g/cc[b]
Odor	Odorless[c]	Odorless[b]	Odorless	Odorless
Odor threshold:				
Water	No data	No data	No data	No data
Air	No data	No data	No data	No data
Solubility:				
Water	0.28 µmoles/L at 25 °C	1 g/35 mL, 1 g/2.1 mL boiling H_2O; 6.9 g/100 cc H_2O at 20 °C[c], 48 g/100 cc at 100 °C[c]	Insoluble (mercuric sulfide, black), soluble in aqua regia with separation of S, in warm hydriodic acid with evolution of H_2S (mercuric sulfide, red)	2.0x10^{-4} g/100mL at 25 °C

Table 3-2. Physical and Chemical Properties of Selected Inorganic and Organic Mercury Compounds[a] (continued)

		Inorganic		
Property	Mercury	Mercuric (II) chloride	Mercuric (II) sulfide	Mercurous (I) chloride
Solubility:				
Organic solvents	Soluble in H_2SO_4 upon boiling, in lipids, readily soluble in HNO_3, insoluble in HCL[b]; soluble in 2.7 mg/L pentane[c]	1 g/3.8 mL alcohol, 1 g/200 mL C_6H_6, 22 mL ether, 12 mL glycerol, 40 mL CH_3COOH, acetone, CH_3OH, ethyl acetate; 33 g/100 cc alcohol at 25 °C, slightly soluble in carbon disulfide, pyridine[c]	Insoluble in alcohol, dilute mineral acids	Insoluble in alcohol, ether
Partition coefficients:				
Log K_{ow}	5.95[g]	No data	No data	No data
Log K_{oc}	No data	No data	No data	No data
Vapor pressure	$2x10^{-3}$ mm Hg at 25 °C	1 mm Hg at 136.2 °C	No data	No data
Henry's law constant at 24.8 °C	No data	No data	No data	No data
Degradation reaction rate constant	Gas-phase reaction with O_3 = $1.7x10^{-18}$ $cm^3/mol/s$[j]; $8 x10^{-19}$ $cm^3/mol/s$[k]	No data	No data	No data
Autoignition temperature	Not flammable[c]	No data	No data	No data
Flashpoint	Not flammable[c]	Not flammable[c]	No data	No data
Flammability limits in air	Not flammable[c]	Not flammable[c]	No data	No data
Conversion factors: ppm (v/v) to mg/m^3 in air at 25 °C	1 ppm = 8.18 mg/m^3	No data	No data	No data
mg/m^3 to ppm (v/v) in air at 25 °C	1 mg/m^3 = 0.122 ppm	No data	No data	No data
Explosive limits	Non-combustible[c]	Non-combustible[c]	No data	No data
Valence states	+1, +2	+2	+2	+2

Table 3-2. Physical and Chemical Properties of Selected Inorganic and Organic Mercury Compounds[a] (continued)

	Organic			
Property	Mercuric (II) acetate	Methylmercuric chloride[l]	Dimethyl mercury	Phenylmercuric acetate
Molecular weight	318.70	251.1[d]	230.66	336.75
Color	White[b]	White[f]	Colorless	White to cream[b]
Physical state	Crystals or crystalline powder; Solid at 25 °C and 1 atm[c]	Crystals[f]	Liquid	Small lustrous prisms; crystalline powder, small prisms or leaflets[c]
Melting point	178–180 °C	170 °C[d]	No data	149 °C; 148-150 °C[b]
Boiling point	No data	No data	92 °C	No data
Density at °C	3.28 g/cm^3	4.06 g/mL at 25 °C[f]	3.1874 g/mL at 20 °C	No data
Odor	Slight acetic odor	No data	No data	Odorless[e]
Odor threshold:				
Water	No data	No data	No data	No data
Air	No data	No data	No data	No data
Solubility:				
Water at 25 °C	1 g in 2.5 mL cold, 1 mL boiling H_2O; 25 g/100 mL at 10 °C, 100 g/100 mL at 100 °C[c]	<0.1 mg/mL at 21 °C[f]	Insoluble; 1.00x10^3 mg/L[d]	Soluble in about 600 parts H_2O; 1 g/180 mL[c]
Organic solvents	Soluble in alcohol; acetic acid[c]	DMSO >=100 mg/mL at 27°C, 95% C_2H_5OH 10–50 mg/mL at 27 °C; acetone >= 100 mg/mL at 27 °C[f]	Easily soluble in ether, alcohol	Soluble in alcohol, benzene, acetone; 6.8 mL $CHCl_3$, 200 mL ether[c]
Partition coefficients:				
Log K_{ow}	No data	No data	2.28[h]	No data
Log K_{oc}	No data	No data	2.73[d]	1.72[d]
Vapor pressure at 25 °C	No data	0.0085 mm Hg at 25 °C[f]	No data	9x10^{-4} mm Hg at 35 °C[f]; 1.20x10^{-4} mm Hg at 25 °C[c]; <1mm Hg at 35 °C[e]
Henry's law constant °C	No data	No data	No data	1.22x10^{-8} atm m^3/mol[c]
Degradation reaction rate constant	No data	No data	Volatilizes to air where it photolyzes to CH_4 and Hg or is oxidized by the OH radical[i]	No data
Autoignition temperature	No data	probably nonflammable[f]	Easily inflammable	No data

Table 3-2. Physical and Chemical Properties of Selected Inorganic and Organic Mercury Compounds[a] (continued)

	Organic			
Property	Mercuric (II) acetate	Methylmercuric chloride[l]	Dimethyl mercury	Phenylmercuric acetate
Flashpoint	Not flammable[c]	probably nonflammable[f]	Easily inflammable	>38 °C[e]
Flammability limits in air	Not flammable[c]	probably nonflammable[f]	Easily inflammable	No data
Conversion factors: ppm (v/v) to mg/m^3 in air at 25 °C	No data	1 ppm = 10.27 mg/m^3	1 ppm = 9.43 mg/m^3	No data
mg/m^3 to ppm (v/v) in air at 25 °C	No data	1 mg/m^3 = 0.0974 ppm	1 mg/m^3 = 0.106 ppm	No data
Explosive limits	Non-combustible[c]	No data	No data	Probably combustible[f]
Valence state	+2	+2	+2	+2

[a] All information obtained from Merck 1989 except where noted.
[b] All information obtained from Lewis 1993
[c] HSDB 1997
[d] Aster 1997
[e] NFPA 1994
[f] NTP Chemical Repository 1997 (Radian Corporation)
[g] Stein et al. 1996
[h] Wasik 1978
[i] Bodek et al. 1988 (to be verified)
[j] Schroeder et al. 1991
[k] Signeur et al. 1994
[l] Commonly occuring form of methyl mercury; proprietary names include bis-methylmercuric sulfate (cerewet), methylmercury cyanoguanidine or methylmercury dicyanodiamide (agrosol, morsodren, panogen, panospray), methylmercury nitrile (chipcote) and methylmercury propionate (metasol MP)
[m] Iverfeldt and Lindquist 1984
[n] Although organic moieties are associated with the Hg atom, the bonds are ionic, not covalent, in nature. In aqueous solution, Hg^{2+} is released.

4. PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL

4.1 PRODUCTION

Mercury is a naturally occurring element that is usually found as mercuric sulfide (cinnabar), an insoluble, stable compound. It occurs in the earth's crust at levels averaging 0.5 ppm, but the actual concentration varies considerably depending on location (Merck 1989; Sidle 1993). Mercury is mined using both open pit (10% of production) and underground mining techniques (90%) (Drake 1981).

Mercury ores are processed inexpensively to produce metallic mercury. Due to the low boiling point of elemental mercury, mercury can be refined by heating the ore and condensing the vapor to form metallic mercury. This method is 95% efficient and yields mercury that is 99.9% pure. The methods used to refine mercury ores are uncomplicated. Smaller refineries use simple firing and condensing equipment, while larger operations use continuous rotary kilns or mechanically feeding and discharging multiple-hearth furnaces (Carrico 1985).

Table 4-1 lists the facilities in each state that manufacture or process mercury, the intended use, and the range of maximum amounts of mercury that are stored on site. There are currently 34 facilities that produce or process mercury in the United States. The data listed in Table 4-1 are derived from the Toxics Release Inventory (TRI96 1998). Since only certain types of facilities are required to report (EPA 1996d), this is not an exhaustive list.

With the closure of the McDermitt Mine in Nevada in 1990, mercury ceased to be a principal product of U.S. industry (USGS 1997). The figures for the total output of this mine have been withheld by the Bureau of Mines to avoid disclosure of company proprietary data (see Table 4-2). As of 1995, eight mines in California, Nevada, and Utah produced mercury as a by-product from gold mining operations. Metals in the gold ores are extracted with an aqueous cyanide solution, with typical mercury recoveries of between 10 and 20% (Jasinski 1993; USGS 1997). Approximately 58 metric tons of mercury were produced as a by-product from 8 mines in 1991 and 64 metric tons were produced as a by-product from 9 mines in 1992. Since then, production volumes have been withheld to avoid disclosing company proprietary data.

Although most of the world production of mercury is generated by mercury mines, most of the mercury produced in the United States comes from secondary production sources (recycling) (EPA 1997).

Table 4-1. Facilities That Manufacture or Process Mercury

FACILITY	LOCATION [a]	RANGE OF MAXIMUM AMOUNTS ON SITE IN POUNDS	ACTIVITIES AND USES
OCCIDENTAL CHEMICAL CORP.	MUSCLE SHOALS , AL	100,000 - 999,999	CHEMICAL PROCESSING AID
TUSCALOOSA STEEL CORP.	TUSCALOOSA , AL	0 - 99	ARTICLE COMPONENT
OCCIDENTAL CHEMICAL CORP.	NEW CASTLE , DE	100,000 - 999,999	CHEMICAL PROCESSING AID
OLIN CHLOR-ALKALI PRODS.	AUGUSTA , GA	100,000 - 999,999	CHEMICAL PROCESSING AID
ALEXANDER MFG. CO.	MASON CITY , IA	0 - 99	IMPORT , ON-SITE USE/PROCESSING , ARTICLE COMPONENT
MICRO SWITCH	FREEPORT , IL	10,000 - 99,999	ARTICLE COMPONENT
VALSPAR CORP.	ROCKFORD , IL	10,000 - 99,999	FORMULATION COMPONENT
DURAKOOL INC.	ELKHART , IN	10,000 - 99,999	ARTICLE COMPONENT
HERMASEAL CO.	ELKHART , IN	10,000 - 99,999	ARTICLE COMPONENT
U.S. STEEL	GARY , IN	10,000 - 99,999	PRODUCE , BYPRODUCT
UNITED TECHS. AUTOMOTIVE INC.	EDINBURGH , IN	10,000 - 99,999	ARTICLE COMPONENT
KOCH SULFUR PRODS. CO.	DE SOTO , KS	0 - 99	ANCILLARY/OTHER USE
BF GOODRICH CO.	CALVERT CITY , KY	100,000 - 999,999	CHEMICAL PROCESSING AID
DU PONT	LOUISVILLE , KY		
BORDEN CHEMICALS & PLASTICS	GEISMAR , LA	100,000 - 999,999	IMPORT , ON-SITE USE/PROCESSING , CHEMICAL PROCESSING AID
DOW CHEMICAL CO.	PLAQUEMINE , LA	1,000 - 9,999	PRODUCE , BYPRODUCT
PIONEER CHLOR ALKALI CO. INC.	SAINT GABRIEL , LA	100,000 - 999,999	CHEMICAL PROCESSING AID
PPG IND. INC.	LAKE CHARLES , LA	100,000 - 999,999	CHEMICAL PROCESSING AID
HOLTRACHEM MFG.	ORRINGTON , ME	100,000 - 999,999	CHEMICAL PROCESSING AID
ELM PLATING CO.	JACKSON , MI	0 - 99	ARTICLE COMPONENT
KERR CORP.	ROMULUS , MI	1,000 - 9,999	REPACKAGING
HOLTRACHEM MFG. CO. L.L.C.	RIEGELWOOD , NC	100,000 - 999,999	CHEMICAL PROCESSING AID
MERCURY REFINING CO. INC.	ALBANY , NY	10,000 - 99,999	PRODUCE , SALE/DISTRIBUTION , REPACKAGING , ANCILLARY/OTHER USE
ASHTA CHEMICALS INC.	ASHTABULA , OH	10,000 - 99,999	CHEMICAL PROCESSING AID
COMPONENT REPAIR TECHS.	MENTOR , OH		
SINCLAIR OIL CORP.	TULSA , OK	100 - 999	PRODUCE , BYPRODUCT
ADVANCED ENVIRONMENTAL	ALLENTOWN , PA	10,000 - 99,999	PRODUCE , SALE/DISTRIBUTION
BETHLEHEM APPARATUS CO. INC.	HELLERTOWN , PA	100,000 - 999,999	PRODUCE , IMPORT , ON-SITE USE/PROCESSING , SALE/DISTRIBUTION , REPACKAGING
ZINC CORP. OF AMERICA	MONACA , PA	10,000 - 99,999	PRODUCE , IMPURITY
OLIN CORP.	CHARLESTON , TN	100,000 - 999,999	CHEMICAL PROCESSING AID
OCCIDENTAL CHEMICAL CORP.	DEER PARK , TX	100,000 - 999,999	CHEMICAL PROCESSING AID
GEORGIA-PACIFIC WEST INC.	BELLINGHAM , WA	100,000 - 999,999	CHEMICAL PROCESSING AID
VULCAN MATERIALS CO.	PORT EDWARDS , WI	100,000 - 999,999	CHEMICAL PROCESSING AID
PPG IND. INC.	NEW MARTINSVILLE , WV	100,000 - 999,999	CHEMICAL PROCESSING AID

Source: TRI96 1998

[a] Post Office state abbreviations used

blank = not available

Table 4-2. U.S. Mercury Supply Demand, Imports, and Exports (metric tons)

Category	1987	1988	1989	1990	1991	1992	1993	1994	1995
Supply									
Mine production[a]		379	414	448	0	0	0		
By-product production[b]		W[c]	W	114	58	64	W		
Industrial recovery		278	137	108	165	176	350	466[e]	534[e]
DLA sales		52	170	52	103	267	543		
DOE sales		214	180	193	215	103	0		
Imports	636[e]	329	131	15	56	92	40		
Total supply	**NA[d]**	**1,252**	**1032**	**930**	**597**	**702**	**933**	**NA[d]**	**NA[d]**
Subtotal: federal sales	NA[d]	266	350	245	318	370	543	NA[d]	NA[d]
Federal sales as % of total supply	NA[d]	21.2%	33.9%	26.3%	53.3%	52.7%	58.1%	NA[d]	NA[d]
Demand	**NA[d]**	**1,503**	**1,212**	**720**	**554**	**621**	**558**	**NA[d]**	**NA[d]**
Federal sales as % of demand	NA[d]	17.6%	0.29%	34%	57.4%	59.6%	0.97%	NA[d]	NA[d]
Imports	**636[e]**	**329**	**131**	**15**	**56**	**92**	**40**	**129[e]**	**277[e]**
Exports	**NA[d]**	**N/A[d]**	**221**	**311**	**786**	**977**	**389**	**316[e]**	**179[e]**

[a] Mercury production from McDermitt mine; closed November 1990
[b] Mercury by-product from 9 gold mining firms
[c] Withheld for proprietary reasons
[d] Not available
[e] Information from USGS 1997

Source: EPA 1996b

Secondary production of mercury includes the processing of scrapped mercury-containing products, and industrial waste and scrap (EPA 1997). As a result of the increasingly stricter regulations that have been placed on the disposal of mercury-containing products, secondary production using recycled mercury has increased from 165 metric tons in 1991 to 176 metric tons in 1992, 350 metric tons in 1993, 466 metric tons in 1994, and 534 metric tons in 1995. Mercury was recovered from various waste materials, including mercury batteries, dental amalgams, switches (including thermostats), manometers, chloralkali wastewater sludges, chemical solutions, and fluorescent light tubes. Refining of the recycled mercury was dominated by three companies: Bethlehem Apparatus Co., Hellertown, Pennsylvania; D.F. Goldsmith Co., Evanston, Illinois; and Mercury Refining Co., Albany, New York (USGS 1997).

4.2 IMPORT/EXPORT

Until 1989, the United States was a net importer of mercury. After that, market values of mercury fluctuated and consumption diminished, leading to a decreased need for imported mercury (Carrico 1985; Drake 1981). U.S. imports of mercury fell sharply between 1987 and 1990 (Jasinski 1993; Reese 1990). The import volumes decreased drastically during the period from 1987 to 1990: 636 metric tons in 1987, 329 metric tons in 1988, 131 metric tons in 1989, and 15 metric tons in 1990 (see Table 4-2). However, import figures generally have increased substantially since 1990: 56 metric tons in 1991, 92 metric tons in 1992, 40 metric tons in 1993, 129 metric tons in 1994, and 277 metric tons in 1995 (USGS 1997). The major reason for the recent escalation in mercury imports is the suspension of mercury sales from the National Defense Stockpile (NDS) in 1994, which had been the major supplier of mercury to the domestic market in recent years. The suspension was imposed by Congress after the EPA raised questions about potential problems associated with the release of mercury. Also, there was concern about the export of NDS mercury for uses banned in the United States (USGS 1997).

From 1978 to 1988, figures were unavailable for the amount of mercury exported by the United States. The U.S. export figures for mercury from 1989 to 1992 are: 221 metric tons in 1989, 311 metric tons in 1990, 786 metric tons in 1991 (Jasinski 1993; Reese 1990), 977 metric tons in 1992, 389 metric tons in 1993, 316 metric tons in 1994, and 179 metric tons in 1995 (USGS 1997) (see Table 4-2). General trends in exportation of mercury are difficult to characterize because the data are unavailable for the 11 years prior to 1989. However, the decline of exports in 1995 is largely due to the suspension of sales from the NDS (USGS 1997).

Major mercury producing countries (primary production from mining operations) in the world currently include Algeria, China, Czechoslovakia, Finland, Kyrgyzstan, Mexico, Morocco, Russia, Slovakia,

Slovenia, Spain, Turkey, and the Ukraine (USGS 1997). The world reserves of mercury are estimated to be sufficient to supply enough product for 100 years, given current production and consumption estimates (Jasinski 1993).

4.3 USE

Mercury has many applications in industry due to its unique properties, such as its fluidity, its uniform volume expansion over the entire liquid temperature range, its high surface tension, and its ability to alloy with other metals. However, domestic consumption of mercury has shown a downward trend since the early 1970s. In 1995, consumption was 463 metric tons, down 10% from 1994. The largest commercial use of mercury in the United States was for electrolytic production of chlorine and caustic soda in mercury cells, accounting for 35% of domestic consumption. Manufacture of wiring devices and switches accounted for 19%, measuring and control instruments for 9%, dental equipment and supplies used 7%, electric lighting used 7%, and other uses used 21% (EPA 1997; USGS 1997). Due to the high toxicity of mercury in most of its forms, many applications have been canceled as a result of attempts to limit the amount of exposure to mercury waste.

Electrical applications. Mercury is a critical element in alkaline batteries. In the past, excess amounts of mercury were used in batteries; however, alkaline battery manufacturers in Europe, Japan, and the United States are now reducing the mercury load from 0.1% to 0.025% of battery content. This reduction will ultimately limit the amount of mercury needed in the battery industry to below 4 metric tons per year (Cole et al. 1992; Reese 1990). Mercuric oxide has become increasingly important commercially in the production of galvanic cells with mercuric oxide anodes in combination with zinc or cadmium cathodes. The voltage for these small, button-shaped batteries remains constant during discharge. The batteries are used in hearing devices, digital watches, exposure meters, pocket calculators, and security installations (IARC 1993), but their use has been declining as non-mercury replacement battery production has increased. Some electrical lamps use mercury vapors in discharge tubes. These lamps are efficient, long-lasting, and produce more lumens per watt than most other industrial lamps (Drake 1981). Wiring and switching devices, such as thermostats and cathode tubes, use mercury because of its predictable contact resistance, thermal conductivity, and quiet operation (Carrico 1985; Drake 1981). In 1985, 64% of the mercury used in the United States was for electrical applications. This use declined to 29% in 1992 (IARC 1993).

Medical applications. Metallic mercury is used in dental restorations because of its ability to alloy with other metals. The World Health Organization (WHO 1991) estimated that, in industrialized countries, about 3% of the total mercury consumption is for dental amalgams. Based on 1992 dental manufacturer specifications, amalgam (at mixing) contains approximately 50% metallic mercury, 35% silver, 9% tin, 6% copper, and trace amounts of zinc. Estimates of annual mercury usage by United States dentists range from approximately 100,000 kg in the 1970s to 70,000 kg in 1995. More than 100 million fillings are replaced each year in the United States (Lorscheider et al. 1995). Until 30 years ago, mercury compounds were used extensively in pharmaceuticals. Mercury salts were components of antiseptics (e.g., merthiolate, mercurochrome), diuretics, skin lightening creams, and laxatives (calomel). Organic mercury compounds were employed in antisyphilitic drugs and some laxatives. Phenylmercury acetate was used in contraceptive gels and foams and as a disinfectant (IARC 1993). Since then, more effective and less toxic alternatives have replaced most pharmaceutical uses of mercury. Medical equipment, such as thermometers and manometers, use metallic mercury to measure temperature and pressure (Carrico 1985).

Chemical/mining applications. Mercury is a catalyst in reactions to form polymers, such as vinyl chloride and urethane foams. The preparation of chlorine and caustic soda (NaOH) from brines also uses mercury as a catalyst. In this process, mercury is used as a moving cathode to separate sodium and chlorine (Rieber and Harris 1994). This mercury can be recycled with 95% efficiency (Drake 1981). Consumption occurs as mercury is lost in wastewater treatment, is recaptured, reprocessed, and sent to landfills (Rieber and Harris 1994). Mercuric oxide and mercuric sulfide are used as pigments in paints (Winship 1985). Gold mining operations use mercury to extract gold from ores through amalgamation (Carrico 1985).

Other applications. Phenylmercuric acetate has been used in aqueous preparations such as inks, adhesives, and caulking compounds, as a catalyst for the manufacture of certain polyurethanes, and as a fungicide in seed dressings and interior and exterior paints (IARC 1993; Reese 1990). Dimethylmercury is used to prepare mercury nuclear magnetic resonance standards (Blayney et al. 1997) and mass spectrometer mercury calibration standards (Toribara et al. 1997).

Discontinued applications. The use of phenylmercuric acetate as a fungicide in interior latex paints was banned in 1990 (Reese 1990), and its use in exterior paint was banned in 1991 (Hefflin et al. 1993). Both of these bans were prompted because of releases of mercury vapors as the paint degraded. Alkyl mercurial

compounds were used until the mid-1970s as a treatment to disinfect grain seeds. Most other agricultural applications of mercury compounds in bactericides and fungicides have been banned due to the toxicity of mercury. Mercuric nitrate was used in the production of felt hats to hydrolyze rabbit fur. The use of mercury as a wood preservative has ceased due to the use of polyurethane (Drake 1981).

4.4 DISPOSAL

Mercury is an element, and therefore its chemical structure cannot be further broken down. In its elemental form, mercury is highly toxic when inhaled. Therefore, incineration of mercury is not recommended as a disposal method. Mercury-containing waste products include waste effluents from chloralkali plants and discarded mercury-containing mechanical and electrical devices (Carrico 1985). Under current federal guidelines, mercury and its compounds are considered hazardous substances, and various regulations are in effect to control the emission of mercury into the environment (especially organic compounds) (Carrico 1985). Emissions from mercury ore processing facilities and mercury cell chloralkali plants are limited to 2.3 kg/day/facility. Emissions of mercury from the incineration or drying of wastewater sludges is limited to 3.2 kg/day/facility (EPA 1975a, 1975b). In addition, dumping wastes containing more than trace amounts of mercury is prohibited.

Recycling of mercury-containing compounds is an important method of disposal. Recycling (retorting) is a treatment for five categories of mercury wastes including: (D009) characteristic mercury; (K106) chlor-alkali waste; (P065) mercury fulminate; (P092) phenylmercuric acetate; and (U151) elemental mercury (see Table 7-1). From 1987 to 1991, annual production of mercury from old scrap averaged nearly 180 metric tons, equivalent to 16% of the average reported consumption during that period (Jasinski 1993). Virtually all mercury can be reclaimed from mercury cell chloralkali plants, electrical apparati, and control instruments when plants are dismantled or scrapped (Carrico 1985). Increased recycling would decrease the mercury load from waste sites and treatment plants. As environmental concerns increase with respect to the disposal of mercury, the recovery by recycling and industrial processes will become a more significant source of domestic supply (Carrico 1985).

Of the estimated 646,896 pounds of mercury reported in the Toxics Release Inventory (TRI) in 1991 to have been released to the environment, the largest percentage (96%, or 619,310 pounds) was transferred off-site from 51 industrial processing facilities, and another 314 pounds were transferred to publicly owned

treatment works (POTWs) (TRI91 1993) (see Section 5-2 for additional information). By comparison, in 1994, only 83,064 pounds of mercury (less than 14% of the total reported in 1991) were released to the environment; and of this amount, 81% (67,480 pounds) was transferred off-site from 29 large processing facilities (TRI94 1996) and an estimated 15 pounds of mercury were released to POTWs (TRI94 1996). Again, by comparison, in 1996, only 84,772 pounds of mercury (less than 14% of the total reported in 1991) were released to the environment and of this amount, 78% (66,573 pounds) was transferred off-site from 34 large processing facilities and an estimated 15 pounds of mercury were released to POTWs (TRI96 1998). Releases of mercury to each of these compartments—the total environment, POTWs, and the volume transferred off-site—decreased dramatically (approximately 90%) in only 5 years. The data listed in the TRI should be used with caution, because only certain types of facilities are required to report (EPA 1996d). This is not an exhaustive list. A facility is required to report information to the Toxics Release Inventory only if the facility is a general manufacturing or processing facility with 10 or more full-time employees that produces, imports, or processes 75,000 or more pounds of any TRI chemical or that uses more than 10,000 pounds of a TRI chemical in a calendar year. No additional information on trends in disposal volume or on specific methods of disposal was located.

In addition, unknown quantities of metallic mercury used in religious or ethnic ceremonies, rituals, and practices (see Sections 5.4.4, 5.6, and 5.7) may reach municipal landfill sites by being improperly disposed of in domestic garbage, or may reach POTWs by being improperly discarded into domestic toilets or sink drains (Johnson [in press]). A survey was conducted to determine the use patterns of elemental mercury in the Latin American and Caribbean communities in New York City (Johnson [in press]). In a survey of 203 adults, about 54% used elemental mercury in various religious and ethnic practices. Of these users, 64% disposed of the mercury in household garbage, 27% flushed the mercury down the toilet, and 9% disposed of the mercury outdoors. It is commonly thought that the high mercury load found in sewage and garbage in New York City comes from dental clinics; however, improper disposal of mercury by religious practitioners in the Latin American and Caribbean communities may also contribute to this load (Johnson [in press]).

5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Mercury occurs naturally as a mineral and is distributed throughout the environment by both natural and anthropogenic processes. The natural global bio-geochemical cycling of mercury is characterized by degassing of the element from soils and surface waters, followed by atmospheric transport, deposition of mercury back to land and surface water, and sorption of the compound to soil or sediment particulates. Mercury deposited on land and open water is in part revolatilized back into the atmosphere. This emission, deposition, and revolatilization creates difficulties in tracing the movement of mercury to its sources. Major anthropogenic sources of mercury releases to the environment include mining and smelting; industrial processes involving the use of mercury, including chlor-alkali production facilities; combustion of fossil fuels, primarily coal; production of cement; and medical and municipal waste incinerators and industrial/commercial boilers (EPA 1996b).

The element has three valence states and is found in the environment in the metallic form and in the form of various inorganic and organic complexes. The major features of the bio-geochemical cycle of mercury include degassing of mineral mercury from the lithosphere and hydrosphere, long-range transport in the atmosphere, wet and dry deposition to land and surface water, sorption to soil and sediment particulates, revolatilization from land and surface water, and bioaccumulation in both terrestrial and aquatic food chains.

Potential sources of general population exposure to mercury include inhalation of mercury vapors in ambient air, ingestion of drinking water and foodstuffs contaminated with mercury, and exposure to mercury through dental and medical treatments. Dietary intake is the most important source of nonoccupational exposure to mercury, with fish and other seafood products being the dominant source of mercury in the diet. Most of the mercury consumed in fish or other seafood is the highly absorbable methylmercury form. Intake of elemental mercury from dental amalgams is another major contributing source to the total mercury body burden in humans in the general population (WHO 1990, 1991).

Because the two major sources of mercury body burden include dietary intake and intake from dental amalgams, mercury is present at low concentrations in a variety of human tissues. Mercury has been detected in blood, urine, human milk, and hair in individuals in the general population. Inhalation of

mercury vapor in workplace atmospheres is the main route of occupational exposure to the compound. The most recent estimate (1983–1986) indicates that about 152,000 people, including over 50,000 women, are potentially exposed to mercury in workplace environments in the United States (RTECS 1998). Occupational exposure to mercury is highest in industries processing or using the element (e.g., chloralkali workers and individuals involved in the manufacturing of industrial instruments, thermometers, and fluorescent lights). Dentists and dental staff, house painters, chemists involved in the synthesis or analysis of environmental samples containing mercury, and individuals involved in disposal or recycling of mercury-contaminated wastes are also at risk of exposure.

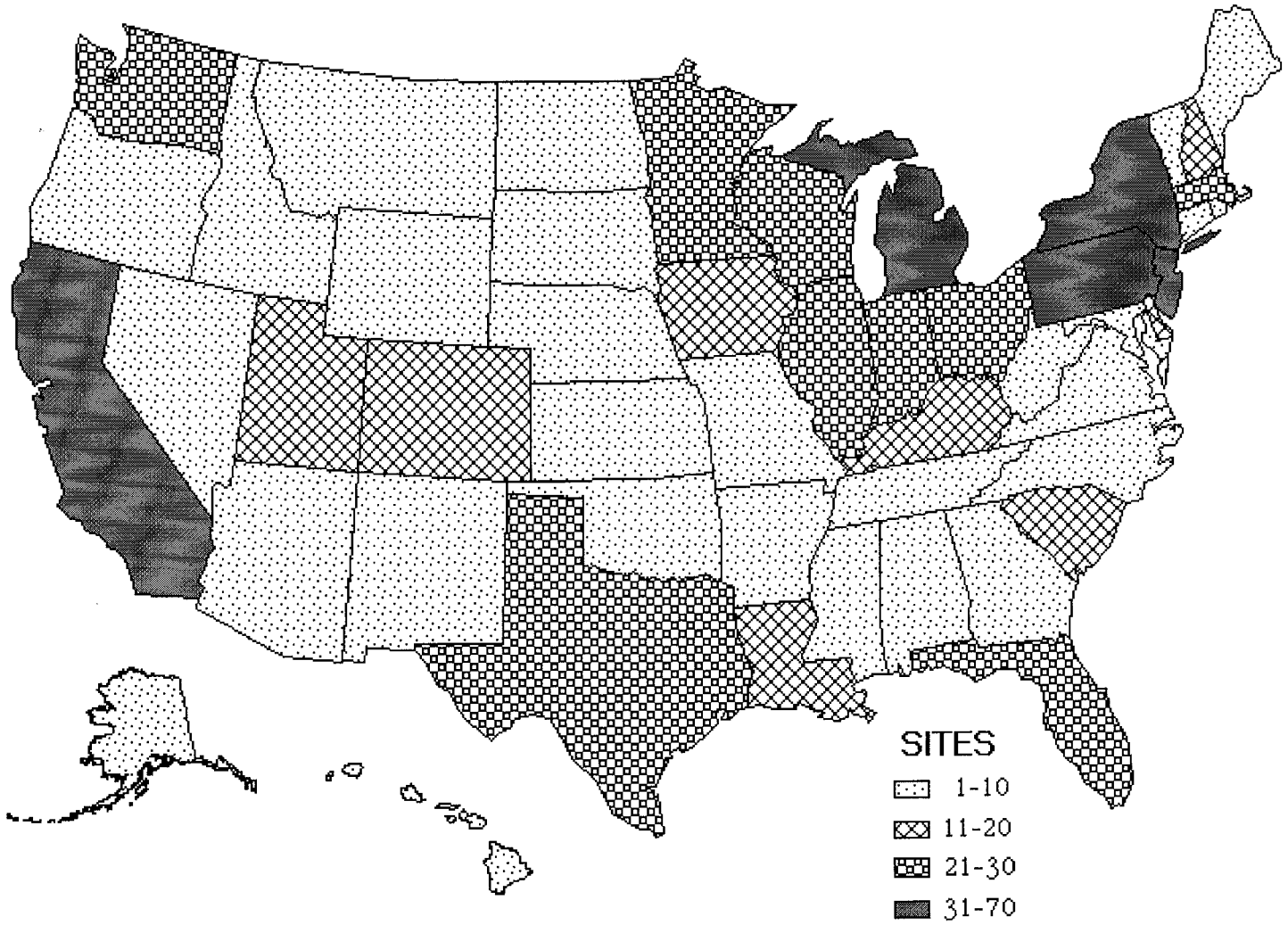
Members of the general public with potentially high exposures include individuals who live in proximity to former mercury mining or production sites, secondary production (recycling) facilities, municipal or medical incinerators, or coal-fired power plants. Other populations at risk of exposure include recreational and subsistence fishers who routinely consume meals of fish that may be contaminated; subsistence hunters who routinely consume the meat and organ tissues of marine mammals or other feral wildlife species; individuals with a large number of dental amalgams; pregnant women and nursing mothers (including their developing fetuses and breast-fed infants) who are exposed to mercury from dietary, medical, or occupational sources, or from mercury spills; individuals who use consumer products containing mercury (e.g., traditional or herbal remedies, or cosmetics, including skin lightening creams); and individuals living or working in buildings where mercury-containing latex paints were used, or where intentional (religious or ethnic use) or unintentional mercury spills have occurred.

Mercury (elemental) has been identified in 714 of the 1,467 hazardous waste sites on the NPL (HazDat 1998). The frequency of these sites can be seen in Figure 5-1. Of these sites, 705 are located in the contiguous United States, 6 are located in the Commonwealth of Puerto Rico (not shown), 2 are located in the U.S. Virgin Islands (not shown), and 1 is located in Guam (not shown). Mercuric acetate, mercuric chloride, mercurous chloride, and dimethylmercury have been identified in 2, 3, 1, and 2 sites, respectively, of the 1,467 hazardous waste sites on the NPL (HazDat 1998). The frequency of these sites can be seen in Figures 5-2 through 5-5. All of these latter sites are located in the contiguous United States.

5.2 RELEASES TO THE ENVIRONMENT

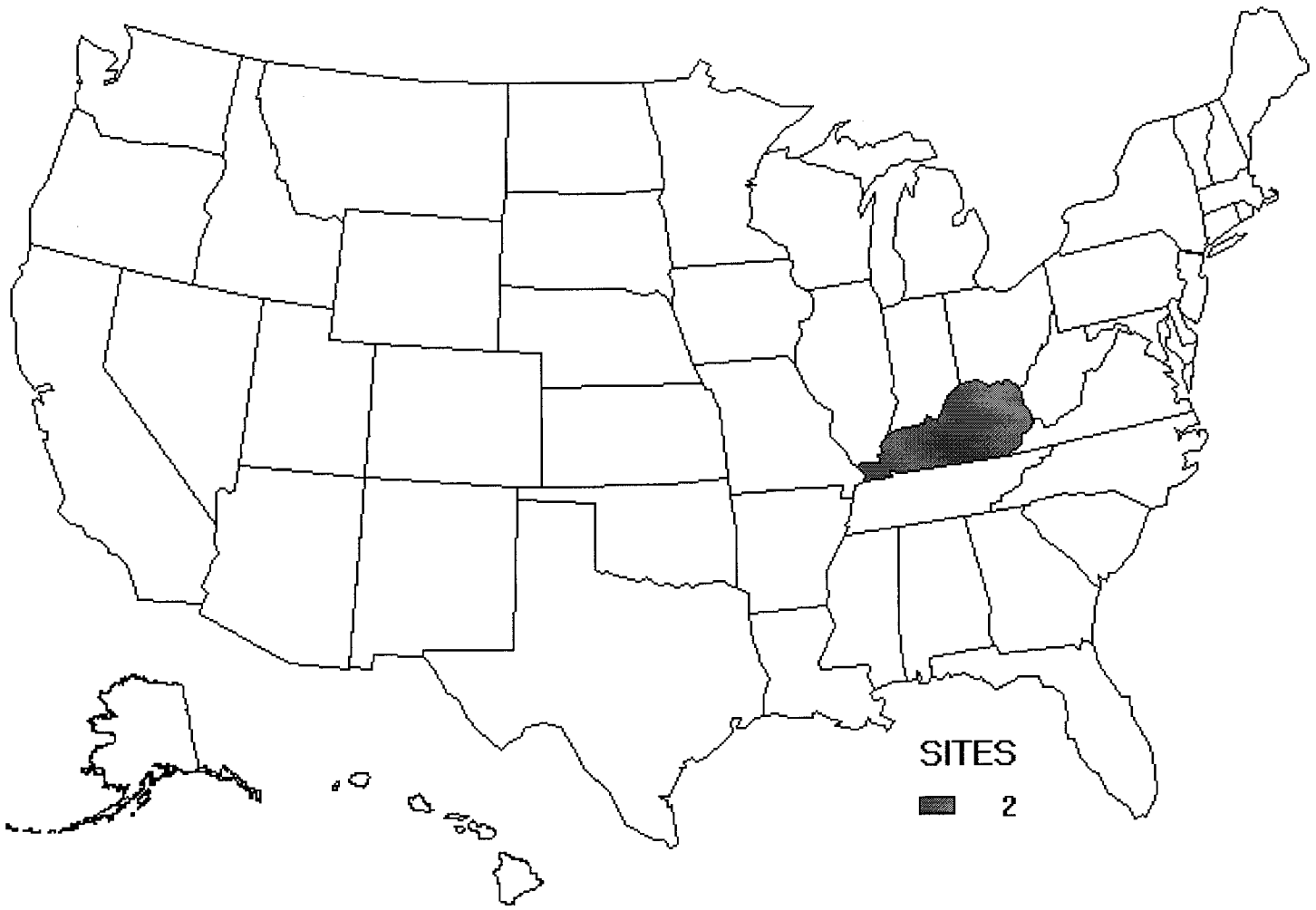
Mercury is released to the environment by both natural processes (e.g., volcanic activity and weathering of mercury-containing rocks) and anthropogenic sources. Anthropogenic releases are primarily to the

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



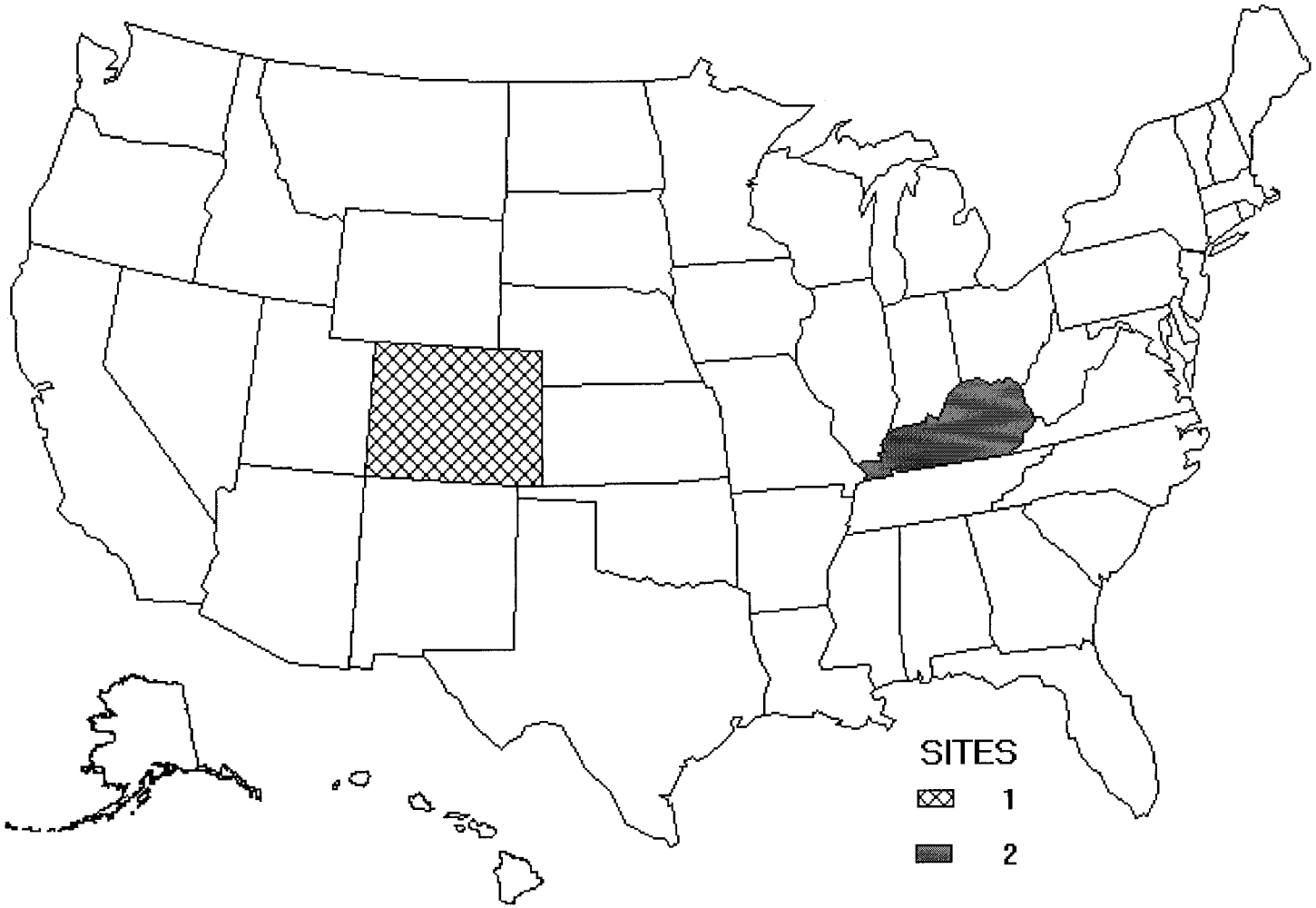
Derived from HazDat 1998

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



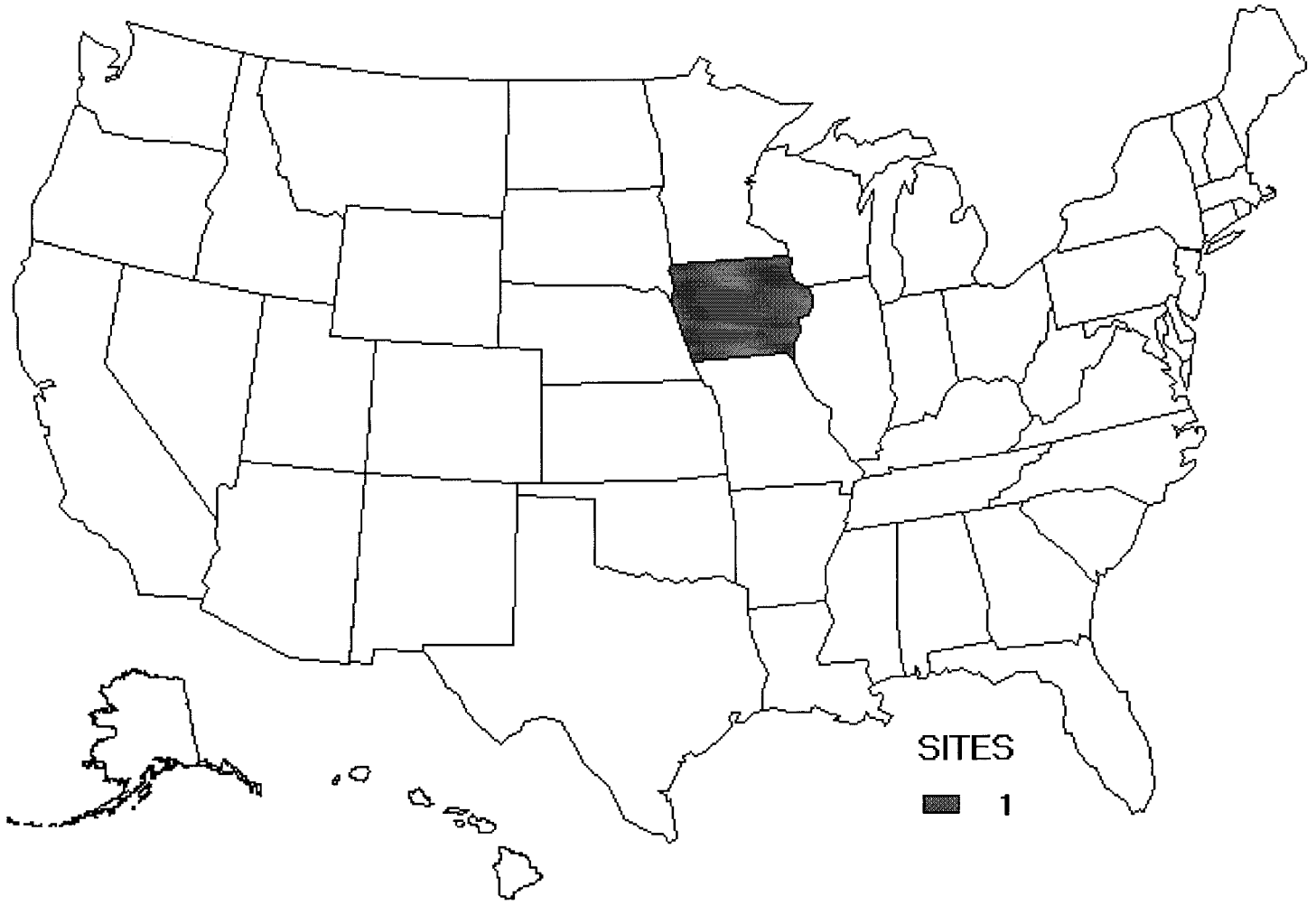
Derived from HazDat 1998

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



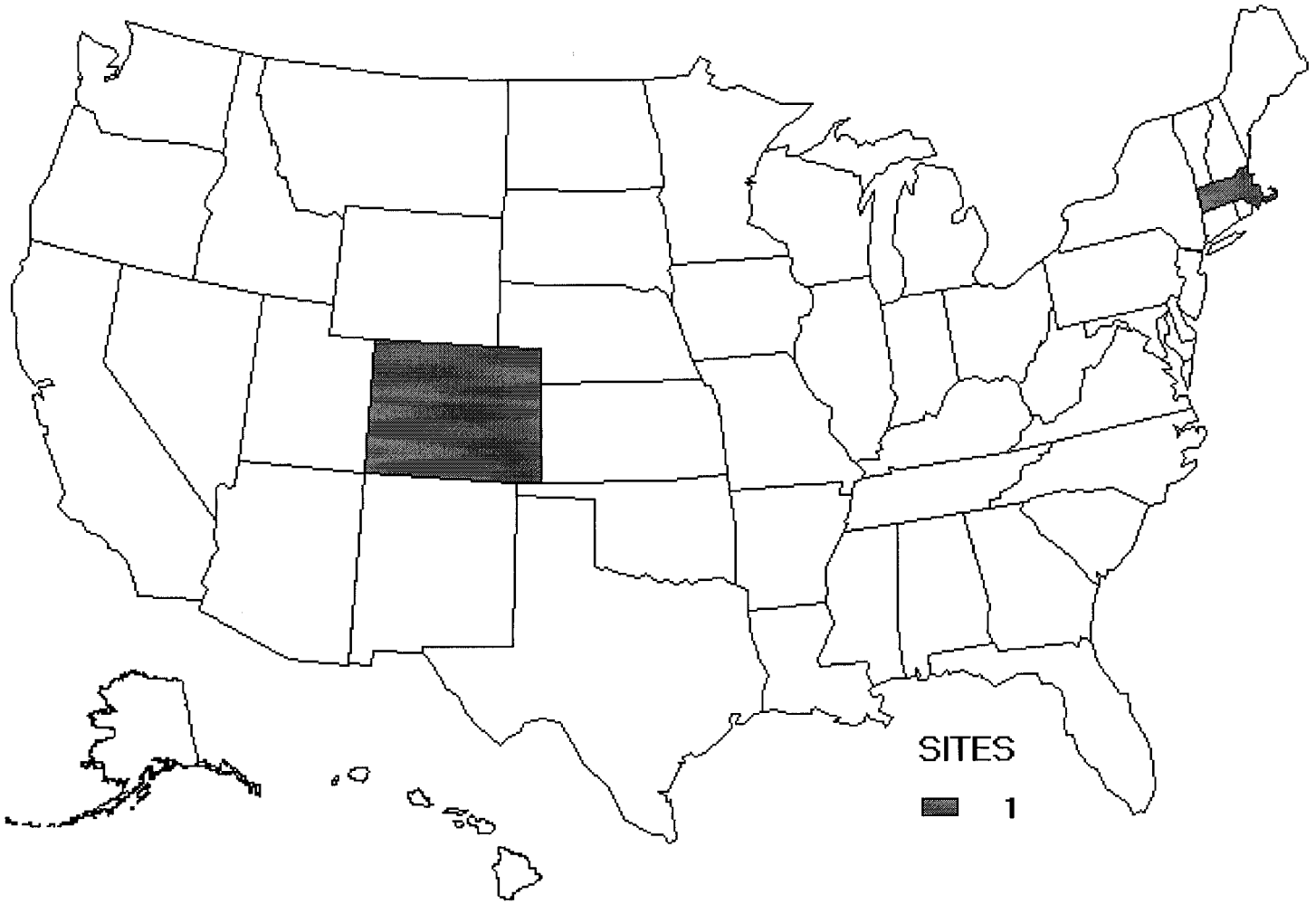
Derived from HazDat 1998

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



Derived from HazDat 1998

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



Derived from HazDat 1998

atmosphere. According to the Toxic Chemical Release Inventory (TRI), in 1996, a total of 84,772 pounds of mercury were released to the environment (air, water, soil, underground injection, and off-site transfer) from 31 large processing facilities (TRI96 1998). Table 5-1 lists the amounts released from these facilities. The amounts of mercury released to the various environmental compartments in 1996, 1994, and 1991 are also compared in Table 5-2. It is noteworthy that the total environmental releases of mercury have decreased by about 90% from 1991 to 1996 from those production and processing facilities that are required to report their releases to TRI. The individual quantities of mercury released to land, publicly owned treatment works (POTWs), and via off-site waste transfer have decreased most substantially since 1991 by 90%, 95%, and 89% respectively. In contrast, releases to air, water, and underground injection have fluctuated over the past few years, but overall have remained relatively unchanged or declined slightly. The data listed in the TRI should be used with caution because only certain types of facilities are required to report (EPA 1996f). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the Toxics Release Inventory only if they employ 10 or more full-time employees; if their facility is classified under Standard Industrial Classification (SIC) codes 20 through 39; and if their facility produces, imports, or processes 25,000 or more pounds of any TRI chemical or otherwise uses more than 10,000 pounds of a TRI chemical in a calendar year (EPA 1996f). Nationwide mercury emissions from a variety of emission sources are discussed in detail in Sections 5.2.1 through 5.2.3.

5.2.1 Air

Mercury is a naturally occurring metal that is ubiquitous in the environment. Mercury is released to environmental media by both natural processes and anthropogenic sources. Mercury ore is found in all classes of rocks, including limestone, calcareous shales, sandstone, serpentine, chert, andesite, basalt, and rhyolite. The normal concentration of mercury in igneous and sedimentary rocks and minerals appears to be 10–50 ng/g (ppb) (Andersson 1979); however, the mineral cinnabar (mercuric sulfide) contains 86.2% mercury (Stokinger 1981). Currently, the average mercury level in the atmosphere is about 3 to 6 times higher than the estimated level in the preindustrial atmosphere (Mason et al. 1995). Results of several studies suggest increases in anthropogenic mercury emissions over time. Zillioux et al. (1993) used peat cores to estimate that present day deposition of mercury is 2 to 3 times greater than preindustrial levels. Lindqvist (1991c) estimated that sediment concentrations in Swedish lakes are 5 times higher than background levels from precolonial times. Travis and Blaylock (1992) reported that mercury levels in tree

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

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

Source: TRI96 1998

[a] Data in TRI are maximum amounts released by each facility

[b] Post office state abbreviations used

[c] The sum of fugitive and stack releases are included in releases to air by a given facility

[d] The sum of all releases of the chemical to air, land, and water, and underground injection wells; and transfers off-site by a given facility

POTW = publicly owned treatment works

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

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

[a] Source: TRI91 1993
[b] Source: TRI94 1996
[c] Source: TRI96 1998

rings, as well as in soil and sediment cores, suggest that a four- to five-fold increase in mercury levels in air has occurred since the beginning of the industrial revolution.

A degree of uncertainty exists with respect to estimates of the relative contributions of natural and anthropogenic sources of mercury emissions to the environment reported in the scientific literature. Nriagu and Pacnya (1988) estimated anthropogenic emissions to be more than half of the total global emissions of 6,000 tons/year. Nriagu (1989) estimated mercury emissions from natural sources to be 2,500 tons/year. In contrast, WHO (1990, 1991) reported that the major source of atmospheric mercury is global degassing of mineral mercury from the lithosphere and hydrosphere at an estimated rate of 2,700–6,000 metric tons/year, which is approximately 1.3 to 3 times the rate of release from anthropogenic sources. Lindqvist (1991b) estimated world anthropogenic emissions at 4,500 tons with an additional 3,000 tons attributed to natural sources. Most recently, Pirrone et al. (1996) estimated world emissions of mercury at 2,200 metric tons/year and concluded that natural sources, industrial sources, and the recycling of anthropogenic mercury each contribute about one-third of the current mercury burden in the global atmosphere. A major source of the uncertainty is that emissions from terrestrial and marine systems include a "recycled" anthropogenic source component (WHO 1990).

Recent estimates of anthropogenic releases of mercury to the atmosphere range from 2,000–4,500 metric tons/year, mostly from the mining and smelting of mercury and other metal sulfide ores. An estimated 10,000 metric tons of mercury are mined each year, although there is considerable year-to-year variation (WHO 1990). Other anthropogenic sources include: industrial processes involving the use of mercury, including chloralkali manufacturing facilities; combustion of fossil fuels, primarily coal; production of cement; and medical and municipal waste incineration and commercial/ industrial boilers (Bache et al. 1991; EPA 1987f, 1996b; Lindberg 1984; Lindqvist 1991b; Nriagu and Pacyna 1988; WHO 1990, 1991). Stein et al. (1996) estimated that approximately 80% of the anthropogenic sources of mercury are emissions of elemental mercury to the air, primarily from fossil fuel combustion, mining, smelting, and from solid waste incineration. Another 15% of the anthropogenic emissions occur via direct application of fertilizers and fungicides and municipal solid waste (e.g., batteries and thermometers) to the land. Recently, Carpi et al. (1998) studied the contamination of sludge-amended soil with inorganic and methyl-mercury and the subsequent emission of this mercury contamination into the atmosphere. These authors reported the routine application of municipal sewage sludge to crop land significantly increased the concentration of both total mercury and methylmercury in surface soil from 80 to 6,1000 μ/kg (ppb) and 0.3 to 8.3 μ/kg (ppb), respectively. Both inorganic and methylmercury were transported from the

sludge/soil matrix to the environment by emission to the atmosphere. An additional 5% of mercury emissions occur via direct discharge of industrial effluent to bodies of water. Mercury emissions from coal-fired power plants are almost exclusively in the vapor phase (98%) (Germani and Zoller 1988). Brown et al. (1993) reported that 79–87% of mercury contained in coal was released with the flue gas at coal-fired power plants. These authors monitored emissions from plants using sub-bituminous C (low sulfur), lignite (medium sulfur), and bituminous (both low- and high-sulfur) coals. Anthropogenic emissions, mainly from combustion of fossil fuels, account for about 25% of mercury emissions to the atmosphere (WHO 1990). These mercury emissions eventually may be deposited on the surrounding soil, although soil concentrations have not been correlated with distance or direction from such plants (Sato and Sada 1992). Other potential emission sources include copper and zinc smelting operations, paint applications, waste oil combustion (EPA 1987f), geothermal energy plants (Baldi 1988), crematories (Nieschmidt and Kim 1997; WHO 1991), and incineration of agricultural wastes (Mariani et al. 1992). The incineration of medical waste has been found to release up to 12.3 mg/m^3 of mercury (Glasser et al. 1991). Medical wastes may release approximately 110 mercury mg/kg of uncontrolled emissions from medical waste incinerators, compared with 25.5 mercury mg/kg general municipal waste, indicating that medical equipment may be a significant source of atmospheric mercury. The use of scrubbers on the incinerators may remove up to 51% of the mercury emissions (Walker and Cooper 1992). Other potential emission sources of mercury emissions to the air include slag from metal production, fires at waste disposal sites, and diffuse emissions from other anthropogenic sources, such as dentistry and industrial activities. The anthropogenic mercury contributions are greater in the northern hemisphere than in the southern hemisphere, and are greatest in heavily industrialized areas.

Balogh and Liang (1995) conducted a 9-week sampling and analysis program to determine the fate of mercury entering a large municipal wastewater treatment plant. Mercury removal in primary treatment averaged 79%; and the average removal across the entire plant was 96%. Mercury loading on the secondary treatment (activated sludge) process was elevated to near plant influent levels due to recycling of the spent scrubber water from the sewage sludge incinerator control equipment. This internal recycling of the spent incinerator scrubber water resulted in elevated mercury loadings to the incinerator and reduced the mercury control efficiency to near zero. Measurements indicated that publicly owned treatment works (POTWs) can remove mercury from wastewater very effectively; however, approximately 95% of the mercury entering the plant was ultimately discharged to the atmosphere via sludge incineration emissions.

Bullock (1997) used the Regional Lagrangian Model of Air Pollution (RELMAP) to simulate the emission, transport, chemical transformation, and wet and dry deposition of elemental mercury gas, divalent mercury gas, and particulate mercury from various point and area source types to develop an atmospheric mercury emissions inventory by anthropogenic source type. The results of the RELMAP model are shown in Table 5-3. On a percentage basis, various combustion processes (medical waste incinerators, municipal waste incinerators, electric utility power production [fossil fuel burning] and non-utility power and heat generation) account for 83% of all anthropogenic emissions in the United States. Overall, of the emissions produced, 41% were associated with elemental mercury vapor (Hg^{o}), 41% with the mercuric form (Hg^{2+}), and 18% was mercury associated with particulates.

A more detailed estimate of national mercury emission rates for various categories of sources is shown in Table 5-4. As shown in this table, point sources of anthropogenic mercury emissions appear to represent the greatest contribution of mercury releases, with combustion sources representing 85% of all emissions.

According to the most recent Toxics Release Inventory (Table 5-1), in 1996, the estimated releases of 17,097 pounds of mercury to the air from 31 large processing facilities accounted for about 20% of annual environmental releases for this element (TRI96 1998). This is slightly more (13%) than the estimated 13,885 pounds that were released to the air in 1994 (TRI94 1996), but 35% less than the 21,288 pounds released to the air in 1991 (Table 5-2). The TRI data listed in Tables 5-1 and 5-2 should be used with some caution, since only certain types of facilities are required to report (EPA 1996f). This is not an exhaustive list.

Mercury has been identified in air samples collected at 25 of the 714 NPL hazardous waste sites where it has been detected in at least one environmental medium (HazDat 1998).

5.2.2 Water

Natural weathering of mercury-bearing minerals in igneous rocks is estimated to directly release about 800 metric tons of mercury per year to surface waters of the earth (Gavis and Ferguson 1972). Atmospheric deposition of elemental mercury from both natural and anthropogenic sources has been identified as an indirect source of mercury to surface waters (WHO 1991). Mercury associated with soils can be directly washed into surface waters during rain events. Surface runoff is an important mechanism for transporting mercury from soil into surface waters, particularly for soils with high humic content (Meili

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

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

[a] Emission rates are specified in units of megagrams per year ($Mg\ yr^{-1}$)

Hg^0 = elemental mercury vapor; Hg^{2+} = mercuric form; Hg_p = mercury associated with particulates

Source: Bullock 1997

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

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

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

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

[a] Numbers do not add exactly because of rounding.

[b] Insufficient information to estimate 1990 emissions.

[c] Mercury has been phased out of use.

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

[e] Parentheses denote subtotal within a larger point source category.

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

Source: EPA 1996b

1991). Mercury may also be released to surface waters in effluents from a number of industrial processes, including chloralkali production, mining operations and ore processing, metallurgy and electroplating, chemical manufacturing, ink manufacturing, pulp and paper mills, leather tanning, pharmaceutical production, and textile manufacture (Dean et al. 1972; EPA 1971c). Discharges from a regional wastewater treatment facility on the St. Louis River that received primarily municipal wastes contained 0.364 µg/L (ppb) of mercury, resulting in concentrations in the adjacent sediment of up to 5.07 µg/g (ppm) (Glass et al. 1990). Industrial effluents from a chemical manufacturing plant on the NPL (Stauffer Chemical's LeMoyne, Alabama site) contained more than 10 ppm of mercury; these effluents had contaminated an adjacent swamp and watershed with mercury concentrations in the sediments ranging from 4.3 to 316 ppm (Hayes and Rodenbeck 1992). Effluent monitoring data collected under the National Pollutant Discharge Elimination System (NPDES) Program were used to estimate pollutant loadings from effluent discharges to the San Francisco Bay Estuary between 1984 and 1987 (Davis et al. 1992). Of the 1,030 samples of industrial effluents monitored entering the San Francisco Estuary during this period, 39% were found to contain mercury (Davis et al. 1992). Although these authors did not specify the limits of detection for mercury and did not provide quantitative information on the concentrations detected, they did indicate that measurements for most of the priority pollutants including mercury were at or below the detection limit. This precluded quantitative assessment of spatial and temporal trends in calculating loadings to the estuary for all but four metals (Davis et al. 1992).

According to the most recent Toxics Release Inventory, in 1996, the estimated releases of 541 pounds of mercury to water from 31 large processing facilities accounted for about 0.64% of total environmental releases for this element (TRI96 1998). An addition 15 pounds of mercury were released indirectly to POTWs, and some of this volume ultimately may have been released to surface waters. This is approximately 215 pounds more mercury than was released to water directly or indirectly via POTWs in 1994 (TRI94 1996), but 445 pounds less than that released to water either directly (144 pounds) or indirectly via POTWs (301 pounds) in 1991 (TRI91 1993). The TRI data listed in Tables 5-1 and 5-2 should be used with some caution, since only certain types of facilities are required to report (EPA 1996f). This is not an exhaustive list.

Mercury has been identified in surface water, groundwater, and leachate samples collected at 197, 395, and 58 sites, respectively, of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.2.3 Soil

Atmospheric deposition of mercury from both natural and anthropogenic sources has been identified as an indirect source of mercury to soil and sediments (Sato and Sada 1992; WHO 1990, 1991). Mercury is released to cultivated soils through the direct application of inorganic and organic fertilizers (e.g., sewage sludge and compost), lime, and fungicides containing mercury (Andersson 1979). Recent interest in community recycling of sewage sludge and yard compost may result in increased releases of mercury from these wastes. Sewage sludge contained approximately 20 times more mercury than yard compost (2.90 ppm versus 0.15 ppm) (Lisk et al. 1992a); municipal solid waste contained the highest concentration (3.95 ppm) (Lisk et al. 1992b). Recently, Carpi et al. (1998) studied the contamination of sludge-amended soil with inorganic and methylmercury and the emission of this mercury contamination into the atmosphere. These authors reported the routine application of municipal sewage sludge to crop land significantly increased the concentration of both total mercury and methylmercury in surface soil from 80 to 6,1000 µg/kg (ppb) and 0.3–8.3 µg/kg (ppb), respectively. Both the inorganic and methylmercury were transported from the sludge/soil matrix to the environment by emission to the atmosphere.

Additional anthropogenic releases of mercury to soil are expected as a result of the disposal of industrial and domestic solid waste products (e.g., thermometers, electrical switches, and batteries) to landfills (see Table 5-5). Another source of mercury releases to soil is the disposal of municipal incinerator ash in landfills (Mumma et al. 1990). In 1987, nationwide concentrations of mercury present in the ash from municipal waste incineration ranged from 0.03 to 25 ppm (Mumma et al. 1990). Such releases may exhibit a seasonal variability. For example, fly ash collected prior to Christmas contained significantly less mercury (6.5 ppm) than ash collected after Christmas (45–58 ppm), possibly as a result of the increased use and disposal of batteries containing mercury in toys and other equipment during this season (Mumma et al. 1991). Emission sources include stack emissions, ashes collected at the stack, ashes from electrostatic precipitators, and in slags (Morselli et al. 1992). An analysis of mercury concentrations in soil, refuse combustibles, and bottom and fly ash from incinerators showed increasing concentrations of 0, 2, 4, and 100 mg/kg (ppm), respectively (Goldin et al. 1992).

According to the Toxics Release Inventory, in 1996, the estimated releases of 537 pounds of mercury to land from 31 large processing facilities accounted for about 0.63% of the total 1996 environmental releases for this element (TRI96 1998). In addition, an estimated 9 pounds of mercury (<0.01% of total environmental releases) were released via underground injection (see Table 5-1). This is approximately

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

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

[a] EPA (1992a) (except fluorescent lamps estimates)
[b] Discards before recovery, 1 ton equals 2,000 pounds
[c] The estimates for the years 1995 and 2000 do not reflect recent state, Federal, or battery manufacturers' efforts to reduce the mercury content of batteries. Since 1992, several states have restricted mercury use in batteries and/or banned the sale of mercuric oxide batteries. Federal legislation to restrict mercury use in batteries is pending. The battery industry has eliminated mercury as an intentional additive in alkaline batteries, except in button cells.
[d] The estimated contribution of mercury from fluorescent lamps disposal to MSW was calculated based on industry estimates of a 4% growth rate in sales in conjunction with a 53% decrease in mercury content between 1989–1995, and a further 34% decrease in mercury content by the year 2000 (to 15 mg mercury per 4-foot fluorescent lamp (National Electric Manufacturers Association (1995).

Source: EPA 1996b

57% of the mercury that was released to soil in 1994 (TRI94 1996) and is only 10% of the mercury released to soil in 1991 (see Table 5-2). The TRI data listed in Tables 5-1 and 5-2 should be used with some caution, since only certain types of facilities are required to report (EPA 1996f). This is not an exhaustive list.

Mercury has been identified in soil and sediment samples collected at 350 and 208 sites, respectively, of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.3 ENVIRONMENTAL FATE

The natural global bio-geochemical cycling of mercury is characterized by degassing of the element from soils and surface waters, followed by atmospheric transport, deposition of mercury back to land and surface waters, and sorption of the compound to soil or sediment particulates. Mercury deposited on land and open water is in part revolatilized back into the atmosphere. This emission, deposition, and revolatilization creates difficulties in tracing the movement of mercury to its sources (WHO 1990). Particulate-bound mercury can be converted to insoluble mercury sulfide and precipitated or bioconverted into more volatile or soluble forms that re-enter the atmosphere or are bioaccumulated in aquatic and terrestrial food chains (EPA 1984b).

5.3.1 Transport and Partitioning

Mercury has three valence states. The specific state and form in which the compound is found in an environmental medium is dependent upon a number of factors, including the redox potential and pH of the medium. The most reduced form is metallic or elemental mercury, which is a liquid at ambient temperatures, but readily vaporizes. Over 95% of the mercury found in the atmosphere is gaseous mercury (Hg^0), the form involved in long-range (global) transport of the element. Residence time in the atmosphere has been estimated to range from 6 days (Andren and Nriagu 1979) to 2 years (EPA 1984b). Approximately 5% of atmospheric mercury is associated with particulates, which have a shorter atmospheric residence time, are removed by dry or wet deposition, and may show a regional or local distribution pattern (Nater and Grigal 1992). Atmospheric inputs may be more significant in areas where other sources of contamination, such as contaminated rivers, are less important or nonexistent (Kelly et al. 1991). Although local sources are important, a 72-hour travel time trajectory for mercury indicates that some mercury found in rain may originate from sources up to 2,500 km (1,550 miles) away (Glass et al.

1991). Over the last 140 years, the atmospheric mercury concentrations have increased by a factor of 3.7, or approximately 2% per year (Swain et al. 1992).

Metallic mercury released in vapor form to the atmosphere can be transported long distances before it is converted to other forms of mercury, and wet and dry deposition processes return it to land and water surfaces. Dry deposition may account for approximately 70% of the total atmospheric deposition of mercury during the summer, although on an annual basis, wet and dry deposition may be of equal importance (Lindberg et al. 1991). Up to 22% of the annual input of mercury to Lake Erie is from dry deposition of mercury-containing atmospheric particles or from precipitation (Kelly et al. 1991). Wet deposition is the primary method of removal of mercury from the atmosphere (approximately 66%) (Fitzgerald et al. 1991; Lindqvist 1991c) and may account for virtually all of the mercury content in remote lakes that do not receive inputs from other sources (e.g., industrial effluents) (Hurley et al. 1991; Swain et al. 1992). Most inert mercury (Hg^{+2}) in precipitation is bound to aerosol particulates, which are relatively immobile when deposited on soil or water (Meili et al. 1991). Mercury is also present in the atmosphere to a limited extent in unidentified soluble forms associated with particulate matter. In addition to wet and dry deposition processes, mercury may also be removed from the atmosphere by sorption of the vapor form to soil or water surfaces (EPA 1984b).

In soils and surface waters, mercury can exist in the mercuric (Hg^{+2}) and mercurous (Hg^{+1}) states as a number of complex ions with varying water solubilities. Mercuric mercury, present as complexes and chelates with ligands, is probably the predominant form of mercury present in surface waters. The transport and partitioning of mercury in surface waters and soils is influenced by the particular form of the compound. More than 97% of the dissolved gaseous mercury found in water consists of elemental mercury (Vandal et al. 1991). Volatile forms (e.g., metallic mercury and dimethylmercury) are expected to evaporate to the atmosphere, whereas solid forms partition to particulates in the soil or water column and are transported downward in the water column to the sediments (Hurley et al. 1991). Vaporization of mercury from soils may be controlled by temperature, with emissions from contaminated soils being greater in warmer weather when soil microbial reduction of Hg^{+2} to the more volatile elemental mercury is greatest (Lindberg et al. 1991). Vapor-phase mercury volatilized from surface waters has been measured (Schroeder and Fanaki 1988); however, the dominant process controlling the distribution of mercury compounds in the environment appears to be the sorption of nonvolatile forms to soil and sediment particulates, with little resuspension from the sediments back into the water column (Bryan and Langston 1992). Cossa et al. (1988) found that 70% of the dissolved mercury in St. Lawrence River water was

associated with organic matter. The authors reported that the removal mechanism was flocculation of organic mercury colloids in freshwater. Methylmercury and other mercury fractions are strongly bound to organic matter in water and may be transported in runoff water from contaminated lakes to other surface waters and soils (Lee and Iverfeldt 1991). Small amounts (2–4 ng/L [ppt]) of mercury are able to move from contaminated groundwater into overlying lakes, with concentrations reaching a maximum near the sediment/water interface; however, since most of the mercury in the groundwater is derived from atmospheric sources, this low value indicates that most of the mercury deposited on soil (92–96% of the 10.3 μg/m^2/year of mercury deposited) is absorbed to the soil and does not leach down into the groundwater (Krabbenhoft and Babiarz 1992).

The sorption process has been found to be related to the organic matter content of the soil or sediment. Mercury is strongly sorbed to humic materials and sesquioxides in soil at a pH higher than 4 (Blume and Brummer 1991) and to the surface layer of peat (Lodenius and Autio 1989). Mercury has been shown to volatilize from the surface of more acidic soils (i.e., soil pH of less than 3.0) (Warren and Dudas 1992). Adsorption of mercury in soil is decreased with increasing pH and/or chloride ion concentrations (Schuster 1991). Mercury is sorbed to soil with high iron and aluminum content up to a maximum loading capacity of 15 g/kg (15,000 ppm) (Ahmad and Qureshi 1989). Inorganic mercury sorbed to particulate material is not readily desorbed. Thus, freshwater and marine sediments are important repositories for inorganic forms of the element, and leaching is a relatively insignificant transport process in soils. However, surface runoff is an important mechanism for moving mercury from soil to water, particularly for soils with high humic content (Meili 1991). Mobilization of sorbed mercury from particulates can occur through chemical or biological reduction to elemental mercury and bioconversion to volatile organic forms (Andersson 1979; Callahan et al. 1979; EPA 1984b). Metallic mercury may move through the top 3–4 cm of dry soil at atmospheric pressure; however, it is unlikely that further penetration would occur (Eichholz et al. 1988).

The volatilization and leaching of various forms of mercury (elemental, mercuric sulfide, mercuric oxide, and mercurous oxide) from soils or wastes was examined using the headspace method for volatilization and the Resource and Conservation Recovery Act (RCRA) leaching protocols for leaching through soil to determine if the leachates exceeded the RCRA limit of 200 μg/L (ppb) (Willett et al. 1992). With the exception of mercuric sulfide, the other forms of mercury increased in concentrations in the headspace vapor and in the leachate as the soil concentrations increased, although the elemental mercury concentrations never exceeded the RCRA limit, indicating that it was relatively unleachable. Mercuric sulfide also did not exceed the background level for the leachate and was consistently less than

0.001 mg/m^3 for the vapor concentrations, indicating that it was also nonleachable and did not readily volatilize. This study also showed that concentrations of mercury in leachate could not be correlated with the concentration of mercury in the soil or in the headspace vapors (Willett et al. 1992). Mercuric sulfide has been found to strongly adsorb to soil, and even with weathering, any mercury released from the mercuric sulfide is readsorbed by the soil (Harsh and Doner 1981).

The most common organic form of mercury, methylmercury, is soluble, mobile, and quickly enters the aquatic food chain. This form of mercury is accumulated to a greater extent in biological tissue than are inorganic forms of mercury (Riisgard and Hansen 1990). Methylmercury in surface waters is rapidly accumulated by aquatic organisms; concentrations in carnivorous fish (e.g., pike, shark, and swordfish) at the top of both freshwater and marine food chains are biomagnified on the order of 10,000–100,000 times the concentrations found in ambient waters (Callahan et al. 1979; EPA 1984b; WHO 1990, 1991). The range in experimentally determined bioconcentration factor (BCF) values is shown in Table 5-6. The bioaccumulation potential for methylmercury in fish is influenced by the pH of the water, with a greater bioaccumulation seen in waters with lower pH (Ponce and Bloom 1991). Mercury concentrations in fish have also been negatively correlated with other water quality factors, such as alkalinity and dissolved oxygen content (Wren 1992).

The biomagnification of methylmercury has been demonstrated by the elevated levels found in piscivorous fish compared with fish at lower levels of the food chain (Jackson 1991; Kohler et al. 1990; Porcella 1994; Watras and Bloom 1992). Biomagnification factors for methylmercury in the food webs of Lake Ontario were lowest for the transfer of methylmercury from mysids to amphipods (1.1), plankton to amphipods (1.8), and plankton to mysids (2.4); were intermediate for the transfer from mysids to fish (5.1) and amphipods to fish (6.5); and were highest for the transfer from plankton to fish (10.4) (Evans et al. 1991). (The biomagnification of methylmercury from water through several trophic levels is compared to the biomagnification of inorganic mercury in Table 5-7.) Watras and Bloom (1992) reported that biomagnification of methylmercury in Little Rock Lake seems to be a result of two processes: the higher affinity of inorganic mercury in lower trophic level organisms and the high affinity of methylmercury in fish. Fish appear to accumulate methylmercury from both food sources and the water column. However, Hall et al. (1997) found that food was the predominant source of mercury uptake in fish. The biological concentration factor (BCF) of methylmercury in fish in Little Rock Lake was three million (Porcella 1994). Mason et al. (1995) also compared bioaccumulation of inorganic mercury and methylmercury. These authors showed that passive uptake of the mercury complexes ($HgCl_2$ and CH_3HgCl) results in high

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

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

[a] Results are based on the concentration of mercury, not the concentration of the mercury compound to which the animal was exposed.
[b] From concentrations that caused adverse effects in a life-cycle test

Source: ASTER 1997

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

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

Source: Watras and Bloom 1992

concentrations of both the inorganic and methylated mercury in phytoplankton. However, differences in partitioning within phytoplankton cells between inorganic mercury (which is principally membrane-bound) and methylmercury (which accumulated in the cytoplasm) lead to a greater assimilation of methylmercury during zooplankton grazing.

Most of the discrimination between inorganic and methylmercury thus occurs during trophic transfer, while the major enrichment factor is between water and the phytoplankton. This also has been reported for the diatom *Thalassiosura weissflogii* in a marine food chain (Mason et al. 1996). Methylmercury was accumulated in the cell cytoplasm, and its assimilation by copepods was 4 times more efficient than the assimilation of inorganic mercury. Bioaccumulation has been demonstrated for predator fish in both freshwater and marine systems and in marine mammals (see Section 5.4.4). Bioaccumulation of methylmercury in aquatic food chains is of interest, because it is generally the most important source of nonoccupational human exposure to this compound (EPA 1984b; WHO 1990, 1991).

Aquatic macrophytes have been found to bioconcentrate methylmercury in almost direct proportion to the mercury concentration in the water (Ribeyre et al. 1991). Mortimer (1985) reported bioconcentration factors (BCFs) for several species of submerged aquatic plants exposed to inorganic mercury in laboratory aquaria of 3,300, 1.3, 0.9, and 1.3 for *Utricularia*, *Ceratophyllum*, *Najas*, and *Nitella*, respectively. The concentrations factor used by this author was based on $\mu g\ g^{-1}$ dry weight in the plant/$\mu g\ mL^{-1}$ water day^{-1}.

The potential for bioaccumulation in terrestrial food chains is demonstrated by the uptake of mercury by the edible mushroom *Pleurotus ostreatus*, grown on compost containing mercury at concentrations of up to 0.2 mg/kg (ppm). The bioaccumulation factors reported ranged from 65 to 140, indicating that there are potential risks to human health if these mushrooms are eaten in large quantities (Bressa et al. 1988). Elevated concentrations of mercury in 149 samples of mushrooms representing 11 different species were reported by Kalcac et al. (1991). These authors collected mushrooms within 6 km of a lead smelter in Czechoslovakia in operation since 1786. Mercury was accumulated by *Lepista nuda* and *Lepiota rhacodes* at 11.9 mg/kg (ppm) and 6.5 mg/kg (ppm) (dry weight), respectively. The mean concentration of other species ranged from 0.3 to 2.4 mg/kg (ppm). Concentrations of mercury in most of the mushroom species collected in that location were higher than in mushrooms collected in other parts of the country.

Data from higher plants indicate that virtually no mercury is taken up from the soil into the shoots of plants such as peas, although mercury concentrations in the roots may be significantly elevated and reflect the

mercury concentrations of the surrounding soil (Lindqvist 1991e). In a study by Granato et al. (1995), municipal solid waste sludge mercury concentrations from the Metropolitan Water Reclamation District of Greater Chicago were found to range from 1.1 to 8.5 mg/kg (ppm), with a mean concentration of 3.3 mg/kg (ppm). From 1971 to 1995, sludge applications were made to a Fulton County, Illinois sludge utilization site. About 80–100% of the mercury applied to the soils in sewage sludge since 1971 still resided in the top 15 cm of soil. These authors reported that sewage sludge applications did not increase plant tissue mercury concentrations in corn or wheat raised on the sludge utilization site.

Earthworms, *Lumbricus sp*., bioaccumulate mercury under laboratory and field conditions in amounts which are dependent on soil concentrations and exposure duration (Cocking et al. 1994). Maximum mercury tissue concentrations in laboratory cultures were only 20% of the 10–14.8 µg/g (ppm) (dry weight) observed in individual worms collected from contaminated soils (21 µg/g) on the South River flood plain at Waynesboro, Virginia. Bioconcentration occurred under field conditions in uncontaminated control soil (0.2 µg Hg/g); however, total tissue mercury concentrations (0.4–0.8 µg/g dry weight) were only 1–5% of those for earthworms collected on contaminated soils. Uptake by the earthworms appeared to be enhanced in slightly acidic soils (pH 5.9–6.0) in laboratory cultures. Soil and earthworm tissue mercury contents were positively correlated under both field and laboratory conditions. Predation of earthworms contaminated with mercury could pass the contamination to such predators as moles and ground feeding birds, such as robins (Cocking et al. 1994).

5.3.2 Transformation and Degradation

Mercury is transformed in the environment by biotic and abiotic oxidation and reduction, bioconversion of inorganic and organic forms, and photolysis of organomercurials. Inorganic mercury can be methylated by microorganisms indigenous to soils, fresh water, and salt water. This process is mediated by various microbial populations under both aerobic and anaerobic conditions. The most probable mechanism for this reaction involves the nonenzymatic methylation of mercuric mercury ions by methylcobalamine compounds produced as a result of bacterial synthesis. Mercury forms stable complexes with organic compounds. Monoalkyl mercury compounds (e.g., methylmercuric chloride) are relatively soluble; however, the solubility of methylmercury is decreased with increasing dissolved organic carbon content, indicating that it is bound by organic matter in water (Miskimmin 1991). Dialkyl mercury compounds (e.g., dimethyl-mercury) are relatively insoluble (Callahan et al. 1979; EPA 1984b). Dimethylmercury is volatile, although it makes up less than 3% of the dissolved gaseous mercury found in water (Andersson et al. 1990;

Vandal et al. 1991). The major pathways for transformation of mercury and various mercury compounds in air, water, and soil are shown in Figure 5-6.

5.3.2.1 Air

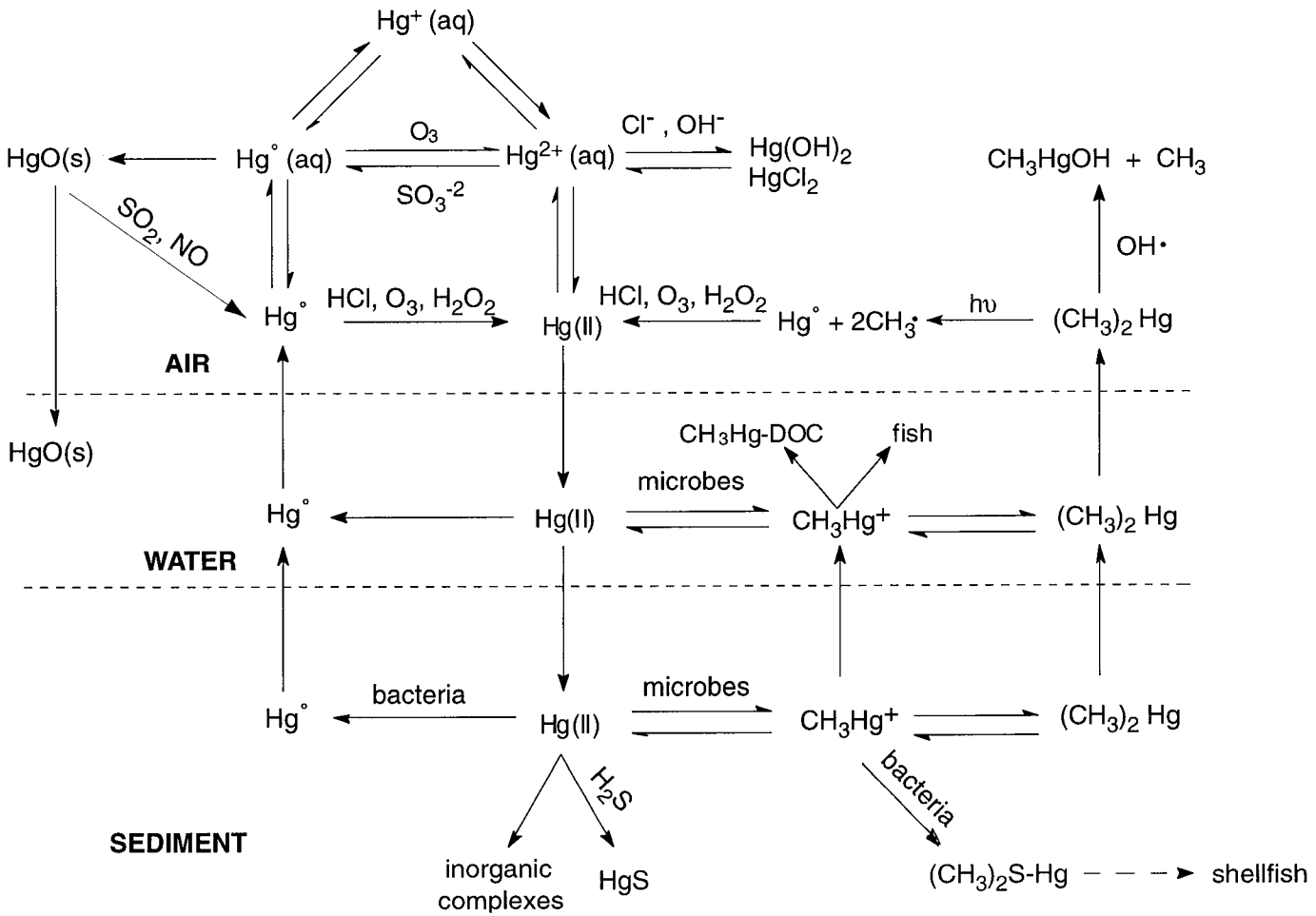
The primary form of atmospheric mercury, metallic mercury vapor (Hg^0), is oxidized by ozone to other forms (e.g., Hg^{+2}) and is removed from the atmosphere by precipitation (Brosset and Lord 1991). The oxidation/reduction of mercury with dissolved ozone, hydrogen peroxide, hypochlorite entities, or organoperoxy compounds or radicals may also occur in the atmosphere (Schroeder et al. 1991). The overall residence time of elemental mercury in the atmosphere has been estimated to be 6 days to 2 years, although in clouds, a fast oxidation reaction on the order of hours may occur between elemental mercury and ozone. Some mercury compounds, such as mercuric sulfide, are quite stable in the atmosphere as a result of their binding to particles in the aerosol phase (Lindqvist 1991b). Other mercury compounds, such as mercuric hydroxide ($Hg[OH]^2$), which may be found in the aqueous phase of the atmosphere (e.g., rain), are rapidly reduced to monovalent mercury in sunlight (Munthe and McElroy 1992). The main atmospheric transformation process for organomercurials appears to be photolysis (EPA 1984b; Johnson and Bramen 1974; Williston 1968).

5.3.2.2 Water

The most important transformation process in the environmental fate of mercury in surface waters is biotransformation. Photolysis of organomercurials may also occur in surface waters, but the significance of this process in relation to biotransformation is not clear (Callahan et al. 1979).

Any form of mercury entering surface waters can be microbially converted to methylmercuric ions, given favorable conditions. Sulfur-reducing bacteria are responsible for most of the mercury methylation in the environment (Gilmour and Henry 1991), with anaerobic conditions favoring their activity (Regnell and Tunlid 1991). Yeasts, such as *Candida albicans* and *Saccharomyces cerevisiae*, whose growth is favored by low pH conditions, are able to methylate mercury and are also able to reduce ionic mercury to elemental mercury (Yannai et al. 1991). Methyl cobalamine compounds produced by bacterial synthesis appear to be involved in the nonenzymatic methylation of inorganic mercury ions (Regnell and Tunlid 1991). The rate of methylmercury formation by this process is largely determined by the concentration of methyl cobalamine compounds, inorganic mercuric ions, and the oxygen concentration of the water, with the rate

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



Dashed lines represent the boundary between environmental compartments.

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

Source: Stem et al. 1996

increasing as the conditions become anaerobic. Volatile elemental mercury may be formed through the demethylation of methylmercury or the reduction of inorganic mercury, with anaerobic conditions again favoring the demethylation of the methylmercury (Barkay et al. 1989; Callahan et al. 1979; Regnell and Tunlid 1991). Increased dissolved organic carbon levels reduce methylation of mercury in the water column (Gilmour and Henry 1991), possibly as a result of the binding of free mercury ions to the dissolved organic carbon at low pH, thus reducing their availability for methylation, or the dissolved organic carbon may inhibit the methylating bacteria (Miskimmin et al. 1992). Alternatively, low pH favors the methylation of mercury in the water column, particularly in acid deposition lakes, while inhibiting its demethylation (Gilmour and Henry 1991). It has also been shown that the methylation rate is not affected by addition of sulfate in softwater lakes (Kerry et al. 1991).

At a pH of 4–9 and a normal sulfide concentration, mercury will form mercuric sulfide. This compound is relatively insoluble in aqueous solution (11×10^{-17} ppb), and therefore it will precipitate out and remove mercury ions from the water, reducing the availability of mercury to fish. Under acidic conditions, however, the activity of the sulfide ion decreases, thus inhibiting the formation of mercuric sulfide and favoring the formation of methylmercury (Bjornberg et al. 1988). Low pH and high mercury sediment concentrations favor the formation of methylmercury, which has greater bioavailability potential for aquatic organisms than inorganic mercury compounds. Methylmercury may be ingested by aquatic organisms lower in the food chain, such as yellow perch, which in turn are consumed by piscivorous fish higher on food chain (Cope et al. 1990; Wiener et al. 1990). Mercury cycling occurs in freshwater lakes, with the concentrations and speciation of the mercury being dependent on limnological features and water stratification. Surface waters may be saturated with volatile elemental mercury, whereas sediments are the primary source of the mercury in surface waters. During the summer months, surface concentrations of methyl and elemental mercury decline as a result of evaporation, although they remain relatively constant in deeper waters (Bloom and Effler 1990).

Abiotic reduction of inorganic mercury to metallic mercury in aqueous systems can also occur, particularly in the presence of soluble humic substances (i.e., acidic waters containing humic and fulvic acids). This reduction process is enhanced by light, occurs under both aerobic and anaerobic conditions, and is inhibited by competition from chloride ions (Allard and Arsenie 1991).

5.3.2.3 Sediment and Soil

Mercury compounds in soils may undergo the same chemical and biological transformations described for surface waters. Mercuric mercury usually forms various complexes with chloride and hydroxide ions in soils; the specific complexes formed depend on the pH, salt content, and composition of the soil solution. Formation and degradation of organic mercurials in soils appear to be mediated by the same types of microbial processes occurring in surface waters and may also occur through abiotic processes (Andersson 1979). Elevated levels of chloride ions reduce methylation of mercury in river sediments, sludge, and soil (Olson et al. 1991), although increased levels of organic carbon and sulfate ions increase methylation in sediments (Gilmour and Henry 1991). In freshwater and estuarine ecosystems, the presence of chloride ions (0.02 M) may accelerate the release of mercury from sediments (Wang et al. 1991).

In the late 1950s, unknown quantities of mercuric nitrate and elemental mercury were released into East Fork Poplar Creek from a government facility in Oak Ridge, Tennessee. Total mercury concentrations in the flood plain soil along the creek ranged from 0.5 to 3,000 ppm (Revis et al. 1989). An estimated 170,000 pounds of that mercury remained in floodplain soil of the creek (DOE 1994). The form of that mercury has been reported to be primarily mercuric sulfide (85–88%), with 6–9% present as elemental mercury (Revis et al. 1989, 1990). A very small amount was detected in the form of methylmercury (less than 0.02%). The reported presence of the mercuric sulfide suggests that the predominant biological reaction in soil for mercury is the reduction of Hg^{+2} to mercuric sulfide by sulfate-reducing bacteria under anaerobic conditions (Revis et al. 1989, 1990). Mercuric sulfide has very limited water solubility (4.5×10^{-24} mol/L), and thus, in the absence of other solvents, is likely to have limited mobility in soil. Aerobic microorganisms can solubilize Hg^{+2} from mercuric sulfide by oxidizing the sulfide through sulfite to sulfate, with the Hg^{+2} being reduced to elemental mercury (Wood 1974). However, examination of the weathering of mercuric sulfide indicated that mercuric sulfide does not undergo significant weathering when bound to riverwash soil with a pH of 6.8, although degradation may be increased in the presence of chloride and iron (Harsh and Doner 1981).

Mercury, frequently present in mine tailings, was toxic to bacteria isolated from a marsh treatment system used to treat municipal waste waters. The minimum concentration that inhibited the bacteria (as determined by intracellular ATP levels) was approximately 0.07±0.15 mg/L (ppm) (Desjardins et al. 1988).

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to mercury and various mercury compounds depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of mercury in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of detection of current analytical methods even for determining total mercury. In reviewing data on mercury levels monitored or estimated in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable. The analytical methods available for monitoring mercury and various inorganic and organic mercury compounds in a variety of environmental media are discussed in Chapter 6.

5.4.1 Air

Indoor air mercury concentrations were determined in 37 houses in Ohio that had been painted with latex paint (Beusterien et al. 1991). Of the 37 homes studied, 21 homes had been painted with interior latex paint containing mercury a median of 86 days earlier, while the 16 control homes had not been recently painted with mercury-containing latex paints. Paint samples from the exposed homes contained a median concentration of 210 mg/L (ppm) (range, 120–610 mg/L). The median air mercury concentration (0.3 $\mu g/m^3$) was found to be significantly higher ($p<0.0001$) in the exposed homes (range, not detectable to 1.5 $\mu g/m^3$) than in the unexposed homes (range, not detectable to 0.3 $\mu g/m^3$). Among the exposed homes, there were 7 in which paint containing <200 mg/L had been applied. In these homes, the median air mercury concentration was 0.2 $\mu g/m^3$ (range, not detectable to 1 $\mu g/m^3$). Six exposed homes had air mercury concentrations >0.5 $\mu g/m^3$. The authors reported that elemental mercury was the form of mercury released to the air and that potentially hazardous mercury exposure could occur in homes recently painted with paint containing <200 mg Hg/L (Beusterien et al. 1991). In an indoor exposure study of families of workers at a chloralkali plant in Charleston, Tennessee, mercury levels in the air of the workers' homes averaged 0.92 $\mu g/m^3$ (ATSDR 1990).

Ambient air concentrations of mercury have been reported to average approximately 10–20 ng/m^3, with higher concentrations in industrialized areas (EPA 1980a). In 1990, metallic mercury concentrations in the gas and aerosol phases of the atmosphere in Sweden were 2–6 ng/m^3 and 0.01–0.1 ng/m^3, respectively (Brosset and Lord 1991). Higher levels (10–15 $\mu g/m^3$) have been detected near point emission sources, such as mercury mines, refineries, and agricultural fields treated with mercury fungicides. Atmospheric

concentrations of mercury over lakes in Wisconsin averaged 2.0 ng/m^3 (Wiener et al. 1990) and ranged from 6.3 ng/m^3 to 16.0 ng/m^3 above the water surface of the mercury-contaminated Wabigoon River in Ontario (Schroeder and Fanaki 1988). Mean vapor concentrations of mercury in air over a forested watershed (Walker Branch Watershed) in Tennessee were 5.5 ng/m^3 in 1988–1989, while particle-associated aerosol mercury concentrations were determined to be 0.03 ng/m^3, or approximately 0.5% of the total atmospheric mercury (Lindberg et al. 1991). Lindberg et al. (1994) measured mercury vapor at concentrations of 2–6 ng/m^3 and particulate mercury at 0.002–0.06 ng/m^3 at Walker Branch Watershed, Tennessee, from August 1991 to April 1992. Particulate mercury concentrations are greater in precipitation than in ambient air. In the St. Louis River estuary, mercury levels in precipitation averaged 22 ng/L (ppt), although ambient air levels averaged 3 ng/m^3 (Glass et al. 1990).

Total gaseous mercury was measured (1992–1993) as part of the Florida Atmospheric Mercury Study (FAMS) (Gill et al. 1995). Average total gaseous mercury concentrations for 3- to 6-day integrated samples ranged from 1.43 to 3.11 ng/m^3 (mean, 1.64 ng/m^3). In the same study, Dvonch et al. (1995) reported that the mean concentrations of total gaseous mercury measured at two inland Florida sites were significantly higher (3.3 and 2.8 ng/m^3) than measurements at an Atlantic coastal site (1.8 ng/m^3). The mean concentrations of particle phase mercury collected at the inland sites (51 and 49 pg/m^3) were 50% higher than those at the coastal site (34 pg/m^3). The mean mercury concentration in rain samples was 44 ng/L (ppt) (range, 14–130 ng/L). Guentzel et al. (1995) also reported results of the FAMS from 1992 to 1994. These authors found that the summer time wet season in south Florida accounted for 80–90% of the annual rainfall mercury deposition. Depositional rates in south Florida are 30 to almost 50% higher than those in central Florida. Particle phase measurements ranged from 2 to 18 pg/m^3 at all sites. Measurement of monomethylmercury in precipitation ranged from <0.005 to 0.020 ng/L (ppt).

Keeler et al. (1995) reported that particulate mercury may contribute a significant portion of the deposition of mercury to natural waters. Mercury can be associated with large particles (>2.5 µm) at concentrations similar to vapor phase mercury. Particulate phase mercury levels in rural areas of the Great Lakes and Vermont ranged from 1 to 86 pg/m^3, whereas particulate mercury levels in urban and industrial areas were in the range of 15–1,200 pg/m^3. Sweet and Vermette (1993) sampled airborne inhalable particulate matter in urban areas (southeast Chicago and East St. Louis) and at a rural site. Mean particulate phase mercury concentrations in particles (<2.5 µm and >2.5 µm) at the rural site were 0.3 ng/m^3 (range, <0.1–0.9 ng/m^3) and 0.2 ng/m^3 (range, <0.1–0.5 ng/m^3), respectively, as compared to 1.0 ng/m^3 (range, <0.1–0.7 ng/m^3) and

0.5 ng/m^3 (range, <0.1–1.5 ng/m^3), respectively, in Chicago and 0.7 ng/m^3 (range, <0.1–20 ng/m^3) and 0.5 ng/m^3 (range, <0.1–1.5 ng/m^3), respectively, in East St. Louis.

In an earlier study, Keeler et al. (1994) measured atmospheric mercury in the Great Lakes Basin. These authors reported that vapor phase mercury levels were four times higher in Chicago, Illinois, than in South Haven, Michigan, (8.7 ng/m^3 versus 2.0 ng/m^3). Furthermore, a diurnal pattern was observed in the vapor phase mercury levels measured at the Chicago site. The average concentration (ng/m^3) was 3.3 times greater for the daytime samples (8 AM to 2 PM) than for the night samples (8 PM to 8 AM), and the average concentration for the afternoon samples (2 PM to 8 PM) was 2.1 times greater than the night samples (average, 3.7 ng/m^3). Particulate phase mercury concentrations were also higher at the Chicago site than at the South Haven site (98 pg/m^3 versus 19 pg/m^3). Burke et al. (1995) reported that the concentration of mercury in vapor phase samples measured over Lake Champlain was consistent with other rural areas (mean, 2.0 ng/m^3; range, 1.2–4.2 ng/m^3), and the concentrations were consistent across all seasons. Particulate phase mercury concentrations averaged 11 pg/m^3, with the highest concentrations detected during the winter.

A monitoring program established at a facility at Oak Ridge National Laboratories found that the major sources of mercury release to the air were vaporization from soil, burning of coal for a steam plant, and fugitive exhaust from a former lithium isotope separation facility contaminated with mercury (Turner et al. 1992). When the monitoring program began in 1986, ambient air mercury vapor concentrations at the facility ranged from 0.011 to 0.108 µg/m^3. These values decreased to 0.006 to 0.071 µg/m^3 by 1990, while background levels near the facility remained at 0.006 µg/m^3. The decrease in mercury vapor concentrations occurred primarily as a result of an 80% reduction in coal burning at the steam plant; however, periods of drought and activities such as moving contaminated soil for construction were found to increase the atmospheric mercury concentrations on a transient basis (Turner et al. 1992). Turner and Bogle (1993) monitored ambient air for mercury around the same industrial complex site at Oak Ridge, Tennessee. Elemental mercury was used in large quantities at the nuclear weapons plant between 1950 and 1963 in a process similar to chloralkali production. Soil and water contamination had been found at the site. The results of weekly ambient monitoring for gaseous mercury from 1986 through 1990 showed that gaseous mercury levels were well below the National Emission Standard for Hazardous Air Pollutants (1.0 mg/m^3) with the exception of one station. Mean mercury levels at the control site ranged from 5 to 6 µg/m^3, while levels at the on-site stations were 6–11, 11–143, 68–174, 71–109, and 4–46 µg/m^3. Mean

particulate mercury levels were 0.00003 μg/m^3 at the control site, compared with mean concentrations at the on-site stations ranging from 0.00006 to 0.00024 μg/m^3 (Turner and Bogle 1993).

Mercury has been identified in air samples collected at 25 sites of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.4.2 Water

Concentrations of mercury in rainwater and fresh snow are generally below 200 ng/L (ppt) (EPA 1984b). Fitzgerald et al. (1991) measured total mercury in rainwater from May through August 1989 at Little Rock Lake, Wisconsin. The total mercury concentrations ranged from 3.2 to 15.2 ng/L (ppt). Mercury concentrations in precipitation collected in Minnesota during 1988 and 1989 averaged 18 ng/L (ppt) for an average annual mercury deposition of 15 μg/m^2 (Glass et al. 1991). Antarctic surface snow contained a mean mercury concentration of less than 1 pg/g (ppt) (Dick et al. 1990). In Ontario, Canada, mercury present in precipitation at an average concentration of 10 ng/L (ppt) accounted for more than half of the mercury inputs to surface waters compared with inputs from stream runoff, suggesting that atmospheric deposition is a significant source of mercury in surface waters (Mierle 1990). Lindberg et al. (1994) measured total mercury in rain collected at Walker Branch Watershed, Tennessee from August 1991 to April 1992. Rain concentrations of total mercury ranged from 7.57 ng/L (ppt) in February 1992 to 17.4 ng/L (ppt) in April 1992. Burke et al. (1995) reported that the average concentration of mercury in precipitation samples measured over Lake Champlain was 8.3 ng/L (ppt) for the sampling year, and the average amount of mercury deposited per precipitation event was 0.069 μg/m^2. The highest concentrations of mercury in precipitation samples occurred during spring and summer months. Guentzel et al. (1995) reported results of the Florida Atmospheric Monitoring Study from 1992 to 1994. These authors found that the summer time wet season in south Florida accounted for 80 to 90% of the annual rainfall mercury deposition. Depositional rates in south Florida are 30–50% higher than those in central Florida. Measurement of monomethylmercury in precipitation samples ranged from <0.005 to 0.020 ng/L (ppt).

The natural occurrence of mercury in the environment means that mercury is likely to occur in surface waters, even when anthropogenic sources of mercury are absent. Freshwaters without known sources of mercury contamination generally contain less than 5 ng/L (ppt) of total mercury in aerobic surface waters (Gilmour and Henry 1991). Mercury levels in water-borne particulates in the St. Louis River estuary ranged from 18 to 500 ng/L (ppt) (Glass et al. 1990). Water samples from lakes and rivers in the Ottawa,

Ontario, region of Canada had total mercury concentrations of 3.5–11.4 ng/L (ppt), with organic mercury constituting 22–37% of the total mercury (Schintu et al. 1989). Mercury was detected in water samples from Crab Orchard Lake, Illinois, at 70–281 ng/L (ppt) (Kohler et al. 1990). Total mercury concentrations in surface waters of California lakes and rivers ranged from 0.5 to 104.3 ng/L (ppt), with the dissolved particulate fraction being dominant (89%; 0.4–12 ng/L [ppt]) (Gill and Bruland 1990).

The baseline concentration of mercury in unpolluted marine waters has been estimated to be less than 2 ng/L (2 ppt) (Fowler 1990). In contrast, the New York Bight, an inshore coastal area near the industrialized areas of New York Harbor and northern New Jersey, contained dissolved mercury concentrations in the range of 10–90 ng/L (ppt) (Fowler 1990).

Near-surface groundwaters in remote areas of Wisconsin were found to contain approximately 2–4 ng/L (ppt) of mercury, of which only a maximum of 0.3 ng/L (ppt) was determined to be methylmercury, indicating that groundwater was not a source of methylmercury in the lake (Krabbenhoft and Babiarz 1992). Mercury was found at levels greater than 0.5 µg/L (ppb) in 15–30% of wells tested in some groundwater surveys (EPA 1985b). Drinking water is generally assumed to contain less than 0.025 µg/L (ppb) (EPA 1984b). A chemical monitoring study of California's public drinking water from groundwater sources was conducted by Storm (1994). This author reported that mercury was analyzed in 6,856 samples, with 225 positive detections and 27 exceedances of the maximum contaminant level (0.002 mg/L [200 ppb]). The mean mercury concentration was 6.5 ppb (median, 0.62 ppb; range, 0.21 to 300 ppb).

Mercury has been identified in surface water, groundwater, and leachate samples collected at 197, 395, and 58 sites, respectively, of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.4.3 Sediment and Soil

In a review of the mercury content of virgin and cultivated surface soils from a number of countries, it was found that the average concentrations ranged from 20 to 625 ng/g (0.020 to 0.625 ppm) (Andersson 1979). The highest concentrations were generally found in soils from urban locations and in organic, versus mineral, soils. The mercury content of most soils varies with depth, with the highest mercury concentrations generally found in the surface layers. Mercury was detected at soil concentrations ranging from 0.01 to 0.55 ppm in orchard soils in New York State (Merwin et al. 1994).

Granato et al. (1995) reported that municipal solid waste sludge mercury concentrations from the Metropolitan Water Reclamation District of Greater Chicago ranged from 1.1 to 8.5 mg/kg (ppm), with a mean concentration of 3.31 mg/kg (ppm). Sludge applications to a sludge utilization site in Fulton County, Illinois, from 1971 to 1995 significantly increased extractable soil mercury concentrations. In addition, 80–100% of the mercury applied to the soils in sewage sludge since 1971 still resided in the top 15 cm of soil.

Facemire et al. (1995) reported industrial contamination of soils and sediment in several states in the southeastern United States. The authors reported soil concentrations up to 141,000 ppm associated with contamination in northeastern Louisiana from mercury-charged manometers used to measure pressure and delivery from natural gas wells. In Tennessee, a maximum mercury concentration of 1,100 ppm (associated with previous operations of the Oak Ridge nuclear facility) was found in wetland soils adjacent to the East Fork Poplar Creek. A pharmaceutical company's effluents enriched sediments in a localized area of Puerto Rico to 88 ppm mercury (Facemire et al. 1995). Rule and Iwashchenko (1998) reported that mean soil mercury concentrations of 1.06 ppm were collected within 2 km of a former chlor-alkali plant in Saltsville, Virginia, and that these concentrations were 17 times higher than regional background soil samples (0.063 ppm). These authors further reported that soil organic content, topographic factors, wind patterns, and elevation were variables significantly related to mercury concentration as determined by regression analysis. Soil mercury levels decreasing with distance from the former plant were indicative of a point source distribution pattern. A made land soil type (Udorthent), which appears to be a by-product of the chlor-alkali manufacturing process, was found proximal to the former plant site and contained about 68 times (4.31 ppm) the regional background concentration.

The top 15 cm of sediments in Wisconsin lakes contained higher levels of mercury (0.09–0.24 µg/g [ppm]) than sediments at lower sediment levels (0.04–0.07 µg/g [ppm]). Because the lakes are not known to receive any direct deposition of mercury, it was postulated that the primary mercury source was atmospheric deposition (Rada et al. 1989). Mercury levels in surface sediments of the St. Louis River ranged from 18 to 500 ng/L (ppt) (Glass et al. 1990). Mercury was detected in sediment samples from Crab Orchard Lake in Illinois at concentrations greater than 60 µg/L (ppb) (Kohler et al. 1990). Surficial sediment samples from several sites along the Upper Connecting Channels of the Great Lakes in 1985 had mercury concentrations ranging from below the detection limit to 55.80 µg/g (ppm) (mean concentrations ranged from 0.05 to 1.61 µg/g [ppm] at four sites) (Nichols et al. 1991). Mercury concentrations were correlated with particle size fractions and organic matter content (Mudroch and Hill 1989). Surface

sediment samples from the Lake Roosevelt/Upper Columbia River in Washington State were found to contain up to 2.7 µg/g (ppm) mercury (Johnson et al. 1990). Mercury concentrations in sediments up to 28 cm in depth in lakes adjacent to coal-fired power plants near Houston, Texas ranged from 255 to 360 mg/kg (ppm) in the summer and from 190 to 279 mg/kg (ppm) in the winter (Wilson and Mitchell 1991).

Surface sediments taken from Canadian lakes receiving atmospheric input from smelters contained between 0.03 and 9.22 µg/g (ppm) mercury, with the highest values being found in lakes nearest the smelters. However, sediment concentrations were not correlated with mercury concentrations in fish from the lakes; the fish concentrations ranged from 0.003 to 0.88 µg/g (ppm), with the highest concentration found in fish from one of the least contaminated lakes (Harrison and Klaverkamp 1990).

Estuarine and coastal marine sediment samples analyzed for NOAA's National Status and Trends Program between 1984 and 1987 showed that 38 of 175 sites contained mercury concentrations in excess of 0.41 µg/g (ppm) (dry weight) (O'Connor and Ehler 1991). In addition, mercury sediment concentrations at 6 sites exceeded the NOAA ER-M concentration of 1.3 ppm (dry weight), which is the concentration determined to be equivalent to the median (50th percentile) for all sites monitored. These 6 sites included 5 sites in the Hudson River/Raritan Estuary, New York Bight, and Raritan Bay areas between New York and New Jersey (ranging from 1.6 to 3.3 ppm dry weight) and one site in the Oakland Estuary in California (2.3 ppm dry weight) (NOAA 1990). Sediments taken from coastal areas off British Columbia, Canada contained concentrations of mercury ranging from 0.05 µg/g to 0.20 µg/g (ppm), while mercury concentrations in fish from these waters were only slightly higher; bioconcentration factors ranged from less than 1 to 14 (Harding and Goyette 1989).

Mercury has been identified in soil and sediment samples collected at 350 and 208 sites, respectively, of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

5.4.4 Other Environmental Media

Foods. The U.S. Food and Drug Administration (FDA) conducted a Total Diet Study (April 1982 to April 1984) to determine dietary intakes of selected industrial chemicals (including mercury) from retail purchases of foods representative of the total diet of the U.S. population (Gunderson 1988). The data were collected as part of 8 food collections, termed “market baskets”, collected in regional metropolitan areas

during the 2-year study and involved individual analysis of 234 food items representing the diets of 8 different population groups. Mercury was detected in 129 adult foods; seafood, the major contributing food group, accounted for 77% (3.01 µg of the 3.9 µg of mercury) of the total mercury intake for 25–30 year old males (Gunderson 1988). Minyard and Roberts (1991) reported results of a survey conducted on food samples analyzed at 10 state food laboratories between 1988 and 1989. These laboratories conducted food regulatory programs and analyzed findings of pesticides and related chemical residues for 27,065 food samples. In 1988, these laboratories reported methylmercury residues in 13 (0.09%) of 13,980 samples, with 1 sample exceeding federal or state tolerances. Similarly, in 1989, methylmercury was detected in 25 (0.19%) of 13,085 samples, with 1 sample exceeding federal or state tolerances. A survey of 220 cans of tuna, conducted in 1991 by the FDA, found an average methylmercury content (expressed as mercury) of 0.17 ppm (range, <0.10–0.75 ppm) (Yess 1993). Levels of methylmercury were higher in solid white (0.26 ppm) and chunk white tuna (0.31 ppm) than in chunk light (0.10 ppm) or chunk tuna (0.10 ppm). Previously, the FDA had determined methylmercury concentrations in 42 samples of canned tuna between 1978 and 1990 (Yess 1993) to range from <0.01 to 0.67 ppm methylmercury (expressed as mercury), with an average concentration of 0.14 ppm. These earlier results are similar to those obtained in the 1991 survey (Yess 1993).

The use of fish meal as a food for poultry and other animals used for human consumption may result in increased mercury levels in these animals. In Germany, poultry and eggs were found to contain average mercury concentrations of 0.04 and 0.03 mg/kg (ppm), respectively. Cattle are able to demethylate mercury in the rumen and thus absorb less mercury; therefore, beef (meat) and cow's milk contained only 0.001–0.02 mg/kg (ppm) and 0.01 mg/kg (ppm) of mercury, respectively (Hapke 1991). A survey of raw foods in Germany in 1986 found that grains, potatoes, vegetables, and fruits contained average mercury concentrations of 0.005 to 0.05 mg/kg (ppm fresh weight); however, wild mushrooms contained up to 8.8 mg/kg (ppm) of mercury. Cocoa beans, tea leaves, and coffee beans contained average mercury concentrations of 0.005, 0.025, and 0.04 mg/kg (ppm), respectively. In all cases where the mercury content was high, selenium was also found in measurable, but lower, concentrations (Weigert 1991).

Pedersen et al. (1994) conducted a monitoring study to assess the levels of trace metals, including mercury, in table wine, fortified wine, beer, soft drinks, and various juices. These authors reported that in all samples tested, mercury concentrations were at or below the detection limit (6 µg/L [6 ppb]).

Fish and Shellfish. As part of the National Pesticide Monitoring Program (NPMP), the U.S. Fish and Wildlife Service collected freshwater fish during 1976–1977 from 98 monitoring stations nationwide and analyzed them for mercury and other heavy metals (May and McKinney 1981). As part of this program, duplicate composite samples of a bottom-dwelling species and several representative predatory species were collected. Bottom-dwelling species sampled included common carp, common sucker, and channel catfish or other catfish species. Predatory species sampled were rainbow, brown, brook or lake trout at cold water stations; largemouth bass or other sunfish family members, such as crappie or bluegill, at warm water stations; and walleye or other perch family members at cool water stations. May and McKinney (1981) reported that the mean concentration of mercury was 0.153 ppm (wet weight basis) in the 1972 NPMP survey and that the mean concentration declined significantly to 0.112 ppm (range, 0.01–0.84 ppm) in the 1976–1977 survey. This decline was presumably due to curtailed production, use, and emissions of mercury (Lowe et al. 1985). May and McKinney (1981) identified an arbitrary 85th percentile concentration of 0.19 ppm for mercury to identify monitoring stations having fish with higher than normal concentrations of mercury. Most of these stations were downstream of industrial sites (e.g., chloralkali operations, pulp and paper plants; or pre-1900 gold and silver mining operations), while others were located in areas with major mercury ore (cinnabar) deposits. In a follow-up NPMP study conducted from 1980–1981, Lowe et al. (1985) reported a geometric mean mercury concentration of 0.11 ppm (wet weight) (range, 0.01–1.10 ppm). These authors reported that the downward trend in mercury residues in fish reported by May and McKinney (1981) may have leveled off, since no significant difference in the geometric mean values was detected in the follow-up study conducted in 1984–1985 as part of the National Contaminant Biomonitoring Program (Lowe et al. 1985; Schmitt and Brumbaugh 1990). However, large variations in mercury uptake among the fish species sampled, as well as among size classes of fish within the same species, may mask actual trends.

From 1986 to 1989, the National Study of Chemical Residues in Fish (NSCRF) was conducted by the EPA to assess the concentrations of 60 toxic pollutants (including mercury) in the tissues of benthic and predatory gamefish nationwide (EPA 1992f). Benthic species were analyzed as whole-body samples, while game fish species were analyzed as fillet samples, and all concentrations were reported on a wet weight basis. Mercury was detected at 92% of the 374 sites surveyed nationwide at a mean concentration of 260 ng/g (0.26 ppm) (median concentration of 0.17 ppm and a maximum concentration of 1.8 ppm), and at 2% of the sites, measured mercury concentrations exceeded 1 ppm. Most of the higher mercury concentrations in fish were collected in the Northeast. Ten of the sites in the top 10th percentile for high mercury concentrations were near pulp and paper mills, four were near Superfund sites, and most of the

remaining sites were near industrial areas. However, the mercury sources could not be identified at all of these sites. Five sites were considered to represent background conditions and six U.S. Geological Survey (USGS) National Stream Quality Accounting Network (NASQAN) sites were also among the sites in the top 10th percentile (EPA 1992f).

A recent national survey conducted by the EPA solicited data on mercury concentrations in fish collected by the states as part of their fish contaminant monitoring programs (EPA 1997b). The EPA asked all states to submit mercury residue data collected from their fish sampling programs from 1990 through 1995 to assess whether there were geographic variations or trends in fish tissue concentrations of mercury. Thirty-nine states provided information on the levels of contamination in their fish. The study included the following: information on the tissue concentrations of mercury, including the number of fish sampled (by species); the mean mercury concentration; and the minimum, median, and maximum concentrations reported for each species by state. Residue information for the three most abundant species sampled in each state included such species as the largemouth and smallmouth bass; channel, flathead, and blue catfish; brown and yellow bullhead; rainbow and lake trout; carp; walleye; north pike; and white sucker. The highest mean mercury residue for an edible species was 1.4 ppm, reported by the state of Arizona; the highest maximum mercury concentrations were 7.0 ppm for bowfin in South Carolina, followed by 6.4 ppm for white sucker in Ohio and 5.7 ppm for bowfin in North Carolina. (Note: This EPA report is currently under review by the states; however, the final report should be available by December 1998).

A summary of the mean, minimum, and maximum tissue concentrations of mercury detected for two of the sampled species with the widest geographical distribution; the largemouth bass and the channel catfish are given in Tables 5-8 and 5-9. As Table 5-8 shows, the maximum mercury residues reported for the largemouth bass exceeded the FDA action level (1 ppm) in 15 of the 25 states that collected and analyzed tissue samples for this species. The highest maximum mercury concentration reported for this species was 4.36 ppm, reported by Florida. Table 5-9 shows the maximum mercury residue reported for another widely distributed species, the channel catfish. While the maximum mercury residues reported for this species are not consistently as high as those for the largemouth bass, maximum residues in channel catfish from 6 of the 20 reporting states still exceeded the FDA action level (1 ppm). The highest maximum value reported for the channel catfish was 2.57 ppm, reported by Arkansas. Consumption of large amounts of feral fish containing these high mercury residues exposes high-end fish consuming populations (those that consume >100 grams fish/day) to potentially greater risk of mercury exposure than members of the general population (see Sections 5.5 and 5.7).

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

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

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

Source: EPA 1997b

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

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

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

Source: EPA 1997b

Most recently, the Northeast states and Eastern Canadian provinces issued their own mercury study, including a comprehensive analysis of current mercury concentrations in a variety of fresh water sportfish species (NESCAUM 1998). This study involved a large number of fish sampling sites in each state, many of which were remote lake sites that did not receive point source discharges. Top level piscivores (i.e., predatory fish) such as walleye, chain pickerel, and large and smallmouth bass were typically found to exhibit some of the highest concentrations, with average tissue residues greater than 0.5 ppm and maximum residues exceeding 2 ppm. One largemouth bass sample was found to contain 8.94 ppm of mercury, while one smallmouth bass sampled contained 5.0 ppm. A summary of the mean and minimum–maximum (range) of mercury concentrations in 8 species of fish sampled is shown in Table 5-10. This study also identified a relationship between elevated mercury levels in fish and certain water quality parameters, including low pH, high conductivity, and elevated levels of dissolved organic carbon.

Lake trout taken from Lake Ontario between 1977 and 1988 did show a progressive decline in mercury contamination from 0.24 µg/g (ppm) in 1977 to 0.12 µg/g (ppm) in 1988 (Borgmann and Whittle 1991). Samples of zooplankton taken from an Illinois lake in 1986 contained approximately 10 ng/g (ppb) mercury; however, fish that fed on the zooplankton had whole body mercury concentrations ranging from 11.6 µg/kg (ppb) for inedible shad to 69 µg/kg (ppb) for edible largemouth bass, indicating bioaccumulation was occurring up the aquatic food chain. Older fish generally had higher mercury concentrations (Kohler et al. 1990). Mercury concentrations in crayfish taken from 13 Ontario lakes with no known mercury inputs ranged from 0.02 to 0.64 µg/g (ppm); the concentrations were positively correlated with organism weight and fish mercury concentrations (Allard and Stokes 1989). Brown trout taken from Lake Ontario contained between 0.18–0.21 µg/g (ppm) mercury in unskinned fillets and between 0.24–0.26 µg/g (ppm) mercury in skinned fillets, indicating that methylmercury is associated with the protein fraction of fish tissue (Gutenmann and Lisk 1991).

Methylmercury constitutes over 99% of the total mercury detected in fish muscle tissue, with no detection of inorganic or dimethylmercury (Grieb et al. 1990; Bloom 1992). Mercury levels were examined in aquatic organisms taken from the Calcasieu River/Lake Complex in Louisiana. The order of enrichment was as follows: shrimp (0.2 µg/g [ppm]) <mussel (0.3 µg/g [ppm]) <fish (0.4 µg/g [ppm]) = oyster (0.4 µg/g [ppm]) <zooplankton (1.4 µg/g [ppm]) (Ramelow et al. 1989). Average mercury concentrations for aquatic organisms collected from the Wabigoon/English/Winnipeg River system in Canada were as follows: 0.06–2.2 µg/g (ppm) for crayfish, 0.01–0.55 µg/g (ppm) for perch, and 0.04–1.2 µg/g (ppm) for pike. Methylmercury concentrations were found to increase with distance from the pollutant source,

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

Species	Number of samples[b]	Mean Hg concentration	Minimum–maximum Hg[c] concentration range (ppm)
Largemouth bass	1,019	0.51	0–**8.94**
Smallmouth bass	738	0.53	0.08–**5.0**
Yellow perch	1,346	0.40	0–3.**15**
Eastern chain pickerel	157	0.63	0–**2.81**
Lake trout	877	0.32	0–**2.70**
Walleye[d]	257	0.77	0.10–**2.04**
Brown bullhead	421	0.20	0–**1.10**
Brook trout	200	0.26	0–0.98

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

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

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

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

Source: NESCAUM 1998

possibly as a result of the increased bioavailability of organic mercury produced by aquatic micro-organisms, whereas inorganic mercury was the predominant form at the source (Parks et al. 1991).

Typical mercury concentrations in large carnivorous freshwater fish (e.g., pike) and large marine fish (e.g., swordfish, shark, and tuna) have been found to exceed 1 µg/g (ppm) (EPA 1984b; Fairey et al. 1997; FDA 1998; Hellou et al. 1992; Hueter et al. 1995), with mercury content again being positively correlated with the age of the fish (Gutenmann et al. 1992; Hueter et al. 1995). Methylmercury concentrations in muscle tissue of 9 species of sharks were analyzed from 4 locations off Florida (Hueter et al. 1995). Muscle tissue methylmercury concentration averaged 0.88 µg/g (ppm) (wet weight) and ranged from 0.06 to 2.87 µg/g (ppm), with 33.1% of the samples exceeding the FDA action level (1 ppm). A positive correlation between methylmercury and shark body length (size) also was found, such that sharks larger than 200 cm in total length contained methylmercury concentrations >1 ppm. Sharks collected off the southern and southwestern coastal areas contained significantly higher concentrations than those caught in the northeast coastal region (Cape Canaveral and north).

Methylmercury concentrations were highest in the Caribbean reef shark (*Carcharhinus perezi*). The two most abundant shark species in the U.S. East Coast commercial shark fishery, sandbar *(C. plumbeus*) and blacktip (*C. limbatus*) sharks, are of special concern with respect to human health. Although the mean concentration of methylmercury in the sandbar shark (0.77 µg/g) was below the average for all sharks, sandbar shark tissues contained up to 2.87 ppm methylmercury, and 20.9% of the samples exceeded the FDA action level of 1 ppm. A total of 71.4% of the blacktip shark samples (mean, 1.3 µg/g) exceeded the FDA action level. The authors suggest that continued monitoring of methylmercury concentrations in various sharks species in the commercial marketplace is warranted. In a recent study of sportfish collected in San Francisco Bay, Fairey et al. (1997) reported that the highest concentrations of mercury were detected in leopard shark muscle tissue (1.26 ppm). Bluefin tuna caught in the Northwest Atlantic Ocean in 1990 contained mercury at a mean muscle concentration of 3.41 µg/g (ppm) dry weight (Hellou et al. 1992).

As part of the National Oceanic and Atmospheric Administration (NOAA) Status and Trends Program conducted from 1984 to 1987, mercury concentrations were analyzed in four marine bivalve species in U.S. coastal waters (NOAA 1987). Mercury concentrations in bivalve tissues ranged from 0.01 to 0.48 µg/g (ppm) dry weight in oysters (*Crassostrea virginica*), 0.28 to 0.41 µg/g (ppm) in the Hawaiian oyster (*Ostrea sandwichensis*), 0.05 to 0.47 µg/g (ppm) in the blue mussel (*Mytilus edulis*), and 0.04 to

0.26 µg/g (ppm) in the California mussel (*Mytilus californianus*). Oysters (*Crassostrea virginica*) collected around the Gulf of Mexico between 1986 and 1989 had mercury concentrations ranging from <0.01 to 0.72 µg/g [ppm] (mean, 0.127 µg/g [ppm]) (Presley et al. 1990). Oysters taken from the Mississippi Sound in 1986 generally did not contain mercury at levels exceeding the detection limit (0.02 µg/g [ppm]), although two samples had detectable mercury levels of 0.66 and 6.6 µg/g [ppm] (Lytle and Lytle 1990).

Mercury has been detected in fish samples collected at 56 of the 714 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998).

Marine mammals. Mercury concentrations have been analyzed in various tissues (i.e., muscle, liver, kidneys) from several species of marine mammals, including beluga whales, narwhal, white-toothed dolphins, pilot whales, ringed seals, harp seals, and walruses in the western and eastern Canadian Arctic (Wagemann et al. 1995). The mean mercury concentration (µg/g [ppm] dry weight) in liver tissue was highest in pilot whales (78 ppm), harp seals (36 ppm), Eastern Arctic ringed seals (29 ppm), narwhal (25 ppm), and Eastern Arctic beluga (22 ppm), with lesser amounts in Arctic walrus (5 ppm) and dolphins (4 ppm). Of the three tissues analyzed, mercury was most concentrated in the liver, with successively lower concentrations in the kidney and muscle tissue. This pattern prevails in most marine mammals. The concentration of total mercury is greater by a factor of 3 in the liver than in the kidney, but can be significantly higher in some species (see Table 5-11). Mean tissue residues in ringed seals from the western Arctic had significantly higher concentrations of mercury than those from the eastern Arctic. The authors reported higher mercury levels in sediment (68–243 ng/g [ppb] dry weight) and water (11–29 ng/L [ppt]) from the western Arctic, as compared to sediment (40–60 ng/g [ppb] dry weight) and water (3.7 ng/L [ppb]) from the eastern Arctic. These differences in sediment and water mercury levels may be responsible for some of the observed differences in mercury tissue concentrations in the seals.

Mercury tissue concentrations were detected in 17 adult and 8 fetal pilot whales from two stranding episodes off Cape Cod, Massachusetts (Meador et al. 1993). Total mercury occurred in high concentrations in both the liver and kidney, and liver concentrations were significantly correlated with the animal's length. Methylmercury, as a percentage of total mercury, varied inversely with total mercury, indicating that demethylation was occurring. Mean adult mercury concentrations in µg/g (ppm) dry weight in liver and kidneys were 176 ppm (range, 1.9–626 ppm dry weight) and 27.5 ppm (range, 6.8–49.7 ppm dry weight), respectively. Mean fetal mercury concentrations in µg/g (ppm) dry weight in liver and kidneys

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

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

were 2.3 ppm (range, 0.9–5.4 ppm dry weight) and 1.9 ppm (range, 0.6–3.9 ppm dry weight), respectively. The mean methylmercury concentration in µg/g (ppm) dry weight in adult liver tissue was 8 ppm (range, 5.6–10 ppm). Aguilar and Borrell (1995) studied mercury tissue levels (1970 to 1988) in harbor porpoises in the eastern North Atlantic. These authors reported that in most tissues of harbor porpoises, the mercury was virtually all in the form of methylmercury; however, the fraction of organic mercury in the liver was much lower than in the rest of the body tissues. These authors found that for a given tissue, the concentrations detected were extremely variable between localities and years. Mercury concentrations in harbor porpoises ranged from 0.62 to 70 ppm in liver and from 0.66 to 22 ppm in muscle. The mean mercury concentration in liver for the eastern harbor porpoise population was 11.2 ppm. Mercury tissue levels progressively increased with the age of the animal; no significant differences were found between the sexes (Aguilar and Borrell 1995).

Plants. Although data on mercury distribution among freshwater vascular plant parts is lacking for unpolluted systems, Mortimer (1985) reported that total mercury in the roots of five species of freshwater vascular plants in the polluted Ottawa River was 10–40% higher than in the shoots. Speciation may be important in determining the patterns of mercury uptake, translocation, and excretion in macrophytes. Shoots of *Elodea densa* more readily accumulated methylmercury than inorganic mercury, and also excreted more inorganic mercury than methylmercury (Czuba and Mortimer 1980). Significant translocation of inorganic mercury from shoots to roots occurred in *E. densa* (Czuba and Mortimer 1980). In this species, methyl- and inorganic mercury moved in opposite directions, with methylmercury moving towards the young shoot apex, and inorganic mercury moving towards lower (older) parts of the shoot (Czuba and Mortimer 1980). Dolar et al. (1971) noted the same methylmercury pattern in the water milfoil (*Myriophyllum spicatum*). Using solution culture experiments, these authors showed that mercury accumulation was greater when plants were exposed to inorganic mercury ($HgCl_2$) than organic methylmercury (CH_3HgCl) and that mercury accumulation from the nutrient solution was rapid and approached maximum values in 2 hours. Organomercury compounds (methylmercury chloride, phenylmercuric acetate, phenylmercuric chloride, and phenylmercuric hydroxide) were more available than inorganic compounds (HgF_2 and $HgCl_{2)}$ from lake sediments. The various organomercury and inorganic mercury compounds were added to sediment at concentrations of 0, 46, 230, and 460 ppm prior to rooting water milfoil. After 20 days, concentration of mercury in the plant tissues exposure to 46, 230, and 460 ppm of the inorganic mercury compounds in the sediment ranged from 1.71 to 4.01, 4.81–6.03, and 6.61–10.2, respectively. In contrast, the concentrations of mercury in plant tissues exposed to 46, 230, and 460 ppm of the organic mercury compounds in the sediment ranged from 2.40 to 7.15 ppm, 36–84.5 ppm, and 114.6–243.1 ppm,

respectively. The control plants (no mercury compounds added to the sediments) contained 0.3 ppm mercury. It is clear from this experiment that organomercury compounds may accumulate significantly in the above-ground parts of some macrophytes. Mortimer (1985) found that although *E. densa* shoots had lower total mercury contents than roots, with 32% of the mercury in the shoots in the form of methylmercury, compared to only 10% in the roots.

Grasses sampled downwind of a municipal waste incinerator contained up to 0.20 µg/g (ppm) of mercury, with concentrations decreasing with increasing distance from the facility (Bache et al. 1991). Background mercury levels in vegetation were usually below 0.1 µg/g (ppm) dry weight (Lindqvist 1991e); however, mushrooms collected 1 km from a lead smelter in Czechoslovakia contained between 0.3 and 12 mg/kg (ppm) dry weight (Kalac et al. 1991).

Consumer and Medicinal Products. Various consumer and medicinal products contain mercury or mercury compounds (i.e., skin lightening creams and soaps, herbal remedies, laxatives, tattooing dyes, fingerpaints, artists paints, and make-up paints) (Barr et al. 1973; Dyall-Smith and Scurry 1990; Lauwerys et al. 1987; Rastogi 1992; Wendroff 1990).

Barr et al. (1973) reported elevated mercury levels in the blood of women using skin lightening creams, although the mercury compound and concentrations in the skin cream were not determined. More recently, Dyall-Smith and Scurry (1990) reported that one skin lightening cosmetic cream contained 17.5% mercuric ammonium chloride. Lauwerys et al. (1987) reported a case of mercury poisoning in a 3-month-old infant whose mother frequently used a skin lightening cream and soap containing inorganic mercury during her pregnancy and during the 1-month lactation period following birth. However, the mercury concentration and specific mercury compound in the cream and soap were not determined. Al-Saleh and Al-Doush (1997) analyzed the inorganic mercury content of 38 skin lightening creams in Saudi Arabian markets. The creams were manufactured in a variety countries, including India and Pakistan, other Arab countries, Thailand, Taiwan, Indonesia, England and Germany. Almost 50% of the creams tested exceeded the tolerance limit of 1 ppm. The mean concentration of mercury in the 38 creams was 994 ppm, with a range of 0–5,650 ppm. It is not known whether any of these products are available in the United States.

Metallic mercury was also the source of two cases of mercury poisoning caused by the dermal application of an over-the-counter anti-lice product (Bourgeois et al. 1986). The more severely poisoned individual applied 30 g of ointment containing 9 g of metallic mercury (300,000 ppm) to his entire body. Wands et al.

(1974) also reported the deaths of two individuals due to the excessive use of a laxative preparation containing mercurous chloride (calomel).

Metallic mercury has been used by Mexican American and Asian populations in traditional remedies for chronic stomach disorders (Espinoza et al. 1995; 1996; Geffner and Sandler 1980; Trotter 1985). Most recently, Perharic et al. (1994) reported cases of poisonings resulting from exposure to traditional remedies and food supplements reported to the National Poisons Unit in London, England. From 1989 to 1991, elemental mercury was implicated in several poisonings following exposure to traditional Asian medicines. In one case, the mercury concentration in the medicinal product taken orally was 540 mg/g (540,000 ppm). The mercury was in its elemental or metallic form. Espinoza et al. (1995, 1996) reported that while examining imported Chinese herbal balls for the presence of products from endangered species, the authors detected potentially toxic levels of arsenic and mercury in certain herbal ball preparations. Herbal balls are aromatic, malleable, earth-toned, roughly spherical, hand-rolled mixtures primarily composed of herbs and honey that are used to make medicinal teas. These herbal balls are used as a self-medication for a wide variety of conditions, including fever, rheumatism, apoplexy, and cataracts. Herbal balls similar to those analyzed are readily available in specialty markets throughout the United States. Mercury (probably mercury sulfide) was detected in 8 of the 9 herbal balls tested. The recommended adult dose for the herbal balls is two per day. Ingesting two herbal balls could theoretically provide a dose of up to 1,200 mg of mercury.

Samudralwar and Garg (1996) conducted trace metal analysis on a variety of plants used in Indian herbal remedies and other medicinal preparations. These authors reported mercury concentrations of 139, 180, 27, 12.5, 11.7, and <10 ppb for Bowen's kale, Neem leaves, Gulvei leaves, Kanher bark, Vekhand root, and orange peel, respectively.

Hoet and Lison (1997) reported on an unusual non-occupational source of mercury exposure that resulted in a woman that used prescription nasal drops that contained 300 mg/L (ppm) borate phenylmercury. These authors reported that the woman, who had used the nasal drops over a long period of time, had high urinary levels of mercury (82 µg/g), but that blood levels were not abnormal (5.5 µg/L).

Mercuric sulfide, or cinnabar, was reported to be used in tattooing dyes to produce a red pigmentation (Bagley et al. 1987; Biro and Klein 1967). An analysis of finger paints and make-up paints manufactured in Europe showed that they all contained less than 1 ppm mercury (Rastogi 1992). Rastogi and Pritzi (1996)

conducted another study to assess the migration of several toxic metals from crayons, watercolor paints, and water-based paints. Migration of mercury from the art materials was determined by scraping flakes of the products into dichloromethane for 2 hours at 54E C. The degreased material was then placed in an aqueous HCl solution, shaken, and centrifuged. The supernatant was then filtered through a 0.45 µ membrane filter and was analyzed. These authors reported that the migration of mercury from these art supplies was 0.24–5.98 ppm for red, 0.26–3.63 ppm for blue, 0.20–4.79 ppm for yellow, 0.22–5.68 ppm for green, and 0.17–3.63 ppm for white paint. Migration of mercury from the product occurred in 57% of the samples tested. The migration limit set by European Standard EN71-3 for mercury is 60 ppm. This value was not exceeded in any of the art supplies tested. The authors, however, believe that children might be exposed not only to mercury, but to several other metals that also co-migrated from the paints.

Cigarettes. In a study conducted in West Germany, Pesch et al. (1992) analyzed mercury concentrations in 50 brands of cigarettes manufactured in 2 Western and 6 Eastern European countries. These authors reported that in 1987, the average mercury concentration detected in cigarettes was 0.098 µg/g (ppm) (dry weight) (range, 0.06 to 0.14 ppm dry weight). In 1991, the mean mercury concentrations for cigarettes were 0.034 µg/g (ppm) dry weight (range, 0.007–0.092 ppm dry weight) for Eastern Europe and 0.015 µg/g (ppm) dry weight (range, 0.006–0.037 ppm dry weight) for Western European countries. The authors attributed the decline in mercury content of cigarettes to environmental protection measures instituted in the intervening years (Pesch et al. 1992).

Religious and Ethnic Rituals, Ceremonies, and Practices. While some of medicinal and pharmaceutical uses of mercury compounds have been replaced in recent years, individuals in some ethnic or religious groups may still use mercury in various religious or ethnic rituals, practices, and ceremonies that can expose them to elevated mercury concentrations in room air. Metallic mercury has been used in Latin American and Caribbean communities as part of certain religious practices (e.g., Voodoo, Santeria, and Espiritismo), predominantly in domestic settings (Wendroff 1990). This use of mercury can contaminate a dwelling or automobile if the mercury is not completely removed from flooring, carpeting, and woodwork in an appropriate manner. Metallic mercury (sometimes under the name *azogue)* currently is sold in shops called botanicas which stock medicinal plants, traditional medicines, incense, candles, and perfumes. Botanicas typically dispense mercury in gelatin capsules or sometimes in small glass vials. Some religious practices involve sprinkling metallic mercury on the floor of the dwelling or of a car, mixing metallic mercury with soap and water to wash the floor, or placing it in an open container to rid the house of evil spirits. Other practices involve carrying a small amount of mercury in a vial on the person, or mixing