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MERCURY HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

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SUMMARY

Available literature on the ecological and toxicological aspects of mercury (Hg) in the environment, with special reference to fish and wildlife resources, is reviewed and summarized. Subdivisions include sources, chemical properties, background concentrations, acute and chronic toxicity, sublethal effects, and proposed criteria to protect sensitive resources.

Mercury has been used by man for at least 2,300 years, most recently as a fungicide in agriculture, in the manufacture of chlorine and sodium hydroxide, as a slime control agent in the pulp and paper industry, in the production of plastics and electrical apparatus, and in mining and smelting operations. Mercury burdens in some environmental compartments are estimated to have increased up to 5X precultural levels, primarily as a result of man's activities. The construction of artificial reservoirs, for example, which releases Hg from flooded soils, has contributed to the observed elevation of Hg concentrations in fish tissues from these localities. Elevated levels of Hg in living organisms in Hg-contaminated areas may persist for as long as 100 years after the source of pollution has been discontinued. One major consequence of increased mercury use, coupled with careless waste disposal practices, has been a sharp increase in the number of epidemics of fatal mercury poisonings in humans, wildlife, and aquatic organisms.

Most authorities agree on six points: (1) mercury and its compounds have no known biological function, and the presence of the metal in the cells of living organisms is undesirable and potentially hazardous; (2) forms of mercury with relatively low toxicity can be transformed into forms of very high toxicity, such as methylmercury, through biological and other processes; (3) mercury can be bioconcentrated in organisms and biomagnified through food chains; (4) mercury is a mutagen, teratogen, and carcinogen, and causes embryocidal, cytochemical, and histopathological effects; (5) some species of fish and wildlife contain high concentrations of Hg that are not attributable to human activities; (6) anthropogenic use of Hg should be curtailed, as the difference between tolerable natural background levels of Hg and harmful effects in the environment is exceptionally small.

Concentrations of total Hg lethal to sensitive, representative, nonhuman species range from 0.1 to 2.0 ug/l (ppb) of medium for aquatic organisms; from 2,200 to 31,000 ug/kg body weight (acute oral) and 4,000 to 40,000 ug/kg (dietary) for birds; and from 100 to 500 ug/kg body weight (daily dose) and 1,000 to 5,000 ug/kg diet for mammals. Organomercury compounds, especially methylmercury, are always more toxic than inorganic Hg compounds. Numerous biological and abiotic factors modify the toxicity of Hg compounds--sometimes by an order of magnitude or more--but the mechanisms of action are not clear. Significant adverse sublethal effects were observed among selected aquatic species at water concentrations of 0.03 to 0.1 ug Hg/l. For some birds, adverse effects--predominantly on reproduction--have been associated with total Hg concentrations (in ug/kg fresh weight) of 5,000 in feather, 900 in egg, and 50 to 100 in diet; and with daily intakes of 640 ug/kg body weight. Sensitive nonhuman mammals showed significant adverse effects of Hg when daily intakes were 250 ug/kg body weight, when dietary levels were 1,100 ug/kg, or when tissue concentrations exceeded 1,100 ug/kg.

The most recent mercury criteria proposed by the U.S. Environmental Protection Agency for protection of freshwater aquatic life are 0.012 ug/l medium (4-day average), not to exceed 2.4 ug/l on an hourly average; however, these criteria offer only limited protection to freshwater ecosystems. The saltwater criteria of 0.025 ug Hg/l medium (4-day average), not to exceed 2.1 ug/l hourly, are unsatisfactory for the protection of marine life. For the protection of sensitive species of mammals and birds that regularly consume fish and other aquatic organisms, total Hg concentrations in these prey items should probably not exceed 100 ug/kg fresh weight for birds, and 1,100 ug/kg for small mammals. The significance of elevated Hg levels in tissues of fish and wildlife is not fully understood; some species of marine pinnipeds, for example, normally contain high concentrations of Hg in various tissues without apparent adverse effects. Usually, however, concentrations in excess of 1,100 ug/kg fresh weight of tissue (liver, kidney, blood, brain, hair) should be considered as presumptive evidence of an environmental mercury problem.

Four courses of action now seem warranted. First, toxic mercurials in agriculture and industry should be replaced by less toxic substitutes. Second, controls should be applied at the point of origin to prevent the discharge of potentially harmful Hg wastes. Third, continued periodic monitoring of Hg in fish and wildlife is needed for identification of potential problem areas., and for evaluation of ongoing mercury curtailment programs. And fourth, additional research is merited on mechanisms of mercury accumulation and detoxication in comparatively pristine ecosystems.

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TABLES

FIGURES

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INTRODUCTION

The element mercury (Hg) and its compounds have no known normal metabolic function. Their presence in the cells of living organisms represents contamination from natural and anthropogenic sources; all such contamination must be regarded as undesirable and potentially hazardous (NAS 1978).

The most important ore of mercury, cinnabar (mercuric sulfide), has been mined continuously since 415 BC (Clarkson and Marsh 1982). In the period before the industrial revolution, Hg was used extensively in gold extraction and in the manufacture of felt hats and mirrors; in the 1800's, it was used in the chloralkali industry, in the manufacture of electrical instruments, and as a medical antiseptic; and since 1900, it has been used in pharmaceuticals, in agricultural fungicides, in the pulp and paper industry as a slimicide, and in the production of plastics (Clarkson and Marsh 1982). Current world use of mercury is estimated at 10,000 to 15,000 metric tons annually (Boudou and Ribeyre 1983), of which the United States accounts for about 18% (Clarkson and Marsh 1982).

The first cases of fatal mercury poisoning were reported for two men in a European chemical laboratory in 1865 (Das et al. 1982). The first documented human poisoning from an agricultural exposure to methylmercury occurred in 1940 (Das et al. 1982). As summarized by Elhassani (1983), exposure of humans to mercury compounds may result from dermal application (e.g., 1,600 infants in Argentina showed symptoms of Hg poisoning after a laundry treated their diapers with a Hg disinfectant), from diet (i.e., ingestion of Hg-contaminated fish, pork, seafoods, or grains), and from contact by respiratory routes (e.g., occupational exposure of mercury fungicide applicators in Nicaragua). Sporadic incidences of human poisonings have occurred in the United States, the Soviet Union, and Canada; and major epidemics have been reported in Japan, Pakistan, Guatemala, Ghana, Yugoslavia, and Iraq (Clarkson and Marsh 1982; Das et al. 1982; Elhassani 1983; Greener and Kochen 1983). In 1972, for example, there were 6,530 hospital admissions within 18 months (459 hospital deaths) among Iraqi farmers who ate bread made from seed wheat treated with a methylmercury fungicide. A water soluble red dye was washed off the wheat, with the assumption that the mercury would be equally soluble. Before the wheat was consumed by humans, it was fed (without apparent effect) to chickens and other :livestock for only a few days; it was not realized that a lengthy latency period was involved (Das et al. 1982; Elhassani 1983). There is no effective antidote to counteract the effects of methylmercury on the central nervous system (Elhassani 1983).

Poisoning of game birds and other wildlife in Sweden, apparently by seeds treated with organomercurials, was noticed in 1960 (Das et al 1982). Massive kills of the grey heron (*Ardea cinerea*) in the Netherlands during 1976, were attributed to a combination of low temperatures, undernourishment, and high body burdens of mercury (Van der Molen et al. 1982). Mercury contamination has resulted in the closure of pheasant and partridge hunting areas in Alberta, Canada (Mullins et al. 1977). In 1967, the Swedish medical board banned the sale of fish that contained high concentrations of organomercury salts, originating from about 40 lakes and rivers (Das et al. 1982). In 1970, after the discovery of high levels of mercury in fish from Lake St. Clair, Canada, restrictions on fishing and the sale of fish were imposed in many areas of the United States and Canada (Das et al. 1982). Since 1970, a total of 26 of the 48 States in the conterminous United States have reported mercury pollution in their waters as a direct result of human activities. These States have banned sport or commercial fishing in Hg-contaminated waters, or have issued health warnings about the consequences of eating Hg-contaminated fish or seafood from selected water courses, or have placed restrictions on fish consumption from certain streams, lakes, or rivers polluted with mercury (NAS 1978). In general, the number of Hg-contaminated fish and wildlife habitats has progressively increased--almost all as a direct result of anthropogenic activities (Boudou and Ribeyre 1983).

Most authorities on Hg ecotoxicology agree on six points. First, Hg and its compounds have no known biological function, and its presence in living organisms is undesirable and potentially hazardous. Second, forms of mercury with relatively low toxicity can be transformed into forms with very high toxicity through biological and other processes. Third, methylmercury can be bioconcentrated in organisms and biomagnified through food chains, returning mercury directly to man and other upper trophic level consumers in concentrated form. Fourth, mercury is a mutagen, teratogen, and carcinogen, and causes embryocidal, cytochemical, and histopathological effects. Fifth, high body burdens of mercury normally encountered in some species of fish and wildlife from remote locations emphasize the complexity of natural mercury cycles and human impacts on these cycles. And finally, the anthropogenic use of mercury should be curtailed, because the difference between

tolerable natural background levels of mercury and harmful effects in the environment is exceptionally small. These, and other aspects of mercury and its compounds in the environment as a result of anthropogenic or natural processes, have been the subject of many reviews, including those by Montague and Montague (1971), D'Itri (1972), Friberg and Vostal (1972), Jernelov et al. (1972, 1975), Keckes and Miettinen (1972), Buhler (1973), Holden (1973), D'Itri and D'Itri (1977), Eisler (1978, 1981), NAS (1978), Birge et al. (1979),.Magos and Webb (1979), Nriagu (1979), EPA (1980, 1985), Jenkins (1980), Clarkson and Marsh (1982), Das et al. (1982), Boudou and Ribeyre (1983), Elhassani (1983), Clarkson et al. (1984), Robinson and Touvinen (1984), and Wren (1986).

This report was prepared in response to requests for information from environmental specialists of the U.S. Fish and Wildlife Service. it is part of a continuing series of synoptic reviews on chemical contaminants and natural resources.

SOURCES OF ENVIRONMENTAL MERCURY

As a direct result of human activities, mercury levels in river sediments have increased fourfold since precultural times, and twofold to fivefold in sediment cores from lakes and estuaries (Das et al. 1982). During the past 100 years, it has been estimated that more than 500,000 metric tons of Hg entered the atmosphere, hydrosphere, and surface soils, with eventual deposition in subsurface soils and sediments (Das et al. 1982). Several activities that contribute significantly to the global input of Hg include the combustion of fossil fuels; mining and reprocessing of gold, copper, and lead; operation of chloralkali plants; and disposal of batteries and fluorescent lamps (NAS 1978; Das et al. 1982). The atmosphere plays an important role in the mobilization of Hg; 25% to 30% of the total atmospheric Hg burden is of anthropogenic origin (NAS 1978).

In the United States, mercury consumption rose from 1,305 metric tons in 1959 to 2,359 tons in 1969 (Table 1). The major use of mercury has been as a cathode in the electrolytic preparation of chlorine and caustic (Nriagu 1979). In 1968 this use accounted for about 33% of the total U.S. demand for Hg (EPA 1980). Of recent U.S. mercury consumption, electrical apparatus have accounted for about 27%; industrial and control instruments, such as switches, thermometers, and barometers, and general laboratory appliances, 14%; antifouling and mildew-proofing paints, 12%; Hg formulations to control fungal diseases of seeds, bulbs, and vegetables, 5%; and dental amalgams, pulp and paper manufacturers, pharmaceuticals, metallurgy and mining, and catalysts, 9% (EPA 1980). Mercury, however, is no longer registered for use in antifouling paints, or for the control of fungal diseases of bulbs (EPA 1980).

Mercury from natural sources enters the biosphere directly as a gas, in lava (from terrestrial and oceanic volcanic activity), in solution, or in particulate form; cinnabar (HgS), for example, is a common mineral in hot spring deposits and a major natural source of mercury (Das et al. 1982). The global cycle of Hg involves degassing of the element from the Earth's crust and evaporation from natural bodies of water, atmospheric transport (mainly in the form of Hg vapor), and deposition of Hg back onto land and water. Oceanic effluxes of Hg are tied to equatorial upwelling and phytoplankton activity and may significantly affect the global cycling of this metal. If volatilization of Hg is proportional to primary production in the world's oceans, oceanic phytoplankton activity represents about 36% of the yearly Hg flow to the atmosphere, or about 2,400 tons per year (Kim and Fitzgerald 1986). Mercury finds its way into sediments, particularly oceanic sediments, where the retention time can be lengthy (Table 2), and where it may continue to contaminate aquatic organisms (Lindsay and Dimmick 1983). Estimates of the quantities of Hg entering the atmosphere from degassing of the surface of the planet vary widely, but a commonly quoted figure is 30,000 tons annually (Clarkson et al. 1984). In aquatic ecosystems, removal of the source of anthropogenic Hg results in a slow decrease in the Hg content of sediments and biota (NAS 1978). The rate of loss depends, in part, on the initial degree of contamination, the chemical form of Hg, physical and chemical conditions of the system, and the hydraulic turnover time (NAS 1978).

Table 1. Industrial and other uses of mercury in the United States in 1959 and in 1969. All values are in metric tons (modified from Montague and Montague 1971).

	1959		1969	
Use	Tons	Percent	Tons	Percent
Chloralkali process	201	15.5	716	30.4
Electrical apparatus	308	23.6	644	27.3
Antifouling and mildew paints	121	9.3	336	14.2
Control devices	213	16.3	241	10.2
Dental preparations	63	4.8	105	4.5
Catalysts	33	2.5	102	4.3
Agriculture	110	8.4	93	3.9
Laboratory	38	2.9	71	3.0
Pharmaceuticals	59	4.5	25	1.1
Pulp and paper mill	150	11.5	19	0.8
Metal amalgamation	9	0.7	7	0.3
Total	1,305		2,359	

Table 2. Amount of mercury in some global reservoirs (NAS 1978), and residence time (Clarkson et al. 1984).

Reservoir	Hg content (metric tons)	Residence time
Atmosphere	850	6 to 90 days
Soils	21,000,000	1,000 years
Freshwater	200	-
Freshwater biota (living)	4	-
Ocean water	4,150,000,000	2,000 years
Oceanic biota (living)	3	-
Ocean sediments	330,000,000,000	>1 million years

CHEMICAL PROPERTIES

Mercury, a silver-white metal that is liquid at room temperature and is highly volatile, can exist in three oxidation states: elemental mercury (Hg^0), mercurous ion (Hg_2^{2+}), and mercuric ion (Hg^{2+}). It can be part of both inorganic and organic compounds (EPA 1980; Clarkson et al. 1984; Table 3). All mercury compounds interfere with thiol metabolism, causing inhibition or inactivation of proteins containing thiol ligands and ultimately leading to mitotic disturbances (Das et al. 1982; Elhassani 1983). The mercuric species is the most toxic inorganic chemical form, but all three forms of inorganic Hg may have a common molecular mechanism of damage in which Hg^{2+} is the toxic species (Clarkson and Marsh 1982; Figure 1).

Chemical speciation is probably the most important variable influencing ecotoxicology of Hg, but Hg speciation is difficult, especially in natural environments (Boudou and Ribeyre 1983). Mercury compounds in an aqueous solution are chemically complex. Depending on pH, alkalinity, redox, and other variables, a wide

variety of chemical species are liable to be formed, having different electrical charges and solubilities. For example, $HgCl_2$ in solution can speciate into $Hg(OH)_2$, Hg^{2+}, $HgCl^+$, $Hg(OH)^-$, $HgCl_3^-$, and $HgCl_4^{2-}$; anionic forms predominate in saline environments (Boudou and Ribeyre 1983). In the aquatic environment, under naturally occurring conditions of pH and temperature, Hg may also become methylated by biological or chemical processes, or both (Beijer and Jernelov 1979; EPA 1980; Ramamoorthy and Blumhagen 1984; Figure 1) -- although abiological methylation is limited (Callister and Winfrey 1986). Methylmercury is the most hazardous mercury species due to its high stability, its lipid solubility, and its possession of ionic properties that lead to a high ability to penetrate membranes in living organisms (Beijer and Jernelov 1979).

All mercury discharged into rivers, bays, or estuaries as elemental (metallic) mercury, inorganic divalent mercury, phenylmercury, or alkoxyalkyl mercury can be converted into methylmercury compounds by natural processes (Jernelov 1969). The mercury methylation in ecosystems depends on mercury loadings, microbial activity, nutrient content, pH and redox condition, suspended sediment load, sedimentation rates, and other variables (NAS 1978; Compeau and Bartha 1984; Berman and Bartha 1986; Callister and Winfrey 1986; Jackson 1986). The finding that certain microorganisms are able to convert inorganic and organic forms of Hg into the highly toxic methylmercury or dimethylmercury has made it clear that any form of Hg is highly hazardous to the environment (EPA 1980, 1985). The synthesis of methylmercury by bacteria from inorganic Hg compounds present in the water or in the sediments is the major source of this molecule in aquatic environments (Boudou and Ribeyre 1983). This process can occur under both aerobic and anaerobic conditions (Beijer and Jernelov 1979; Clarkson et al. 1984), but seems to favor anaerobic conditions (Olson and Cooper 1976; Callister and Winfrey 1986). Transformation of inorganic mercury to an organic form by bacteria alters its biochemical reactivity and hence its fate (Windom and Kendall 1979; Figure 1). Methylmercury is decomposed by bacteria in two phases. First, hydrolytic enzymes cleave the C-Hg bond, releasing the methyl group. Second, a reductase enzyme converts the ionic Hg to the elemental form, which is then free to diffuse from the aquatic environment into the vapor phase. These demethylating microbes appear to be widespread in the environment; they have been isolated from water, sediments, and soils and from the gastrointestinal tract of mammals--including humans (Clarkson et. al. 1984).

Table 3. Some properties of mercury and its compounds.[a]

Property	Elemental mercury	Mercurous chloride	Mercuric chloride	Methylmercury chloride
Empirical formula	Hg	Hg_2Cl_2	$HgCl_2$	CH_3HgCl
Molecular weight	200.59	472.09	271.52	251.09[c]
Chlorine, %	0	15.02	26.12	14.12[c]
Mercury, %	100	84.98	73.88	79.89[c]
Melting point, ° C	–38.87	sublimes at 400–500	277	170[c]
Density	13.534	7.15	5.4	4.063[c]
Solubility, mg/L (ppm)				
In water	0.056	2.0	74,070	~1,016[d]
In benzene	2.387[b]	insol.	5,000	~6,535[e]

[a]All data from Merck Index (1976), except where indicated.

[b]Spencer and Voigt (1968).

[c]Weast and Astle (1982).

[d]Eisler (unpubl.), 72 h equilibrium value.

[e]Eisler (unpubl.), 24 h equilibrium value.

Methylmercury is produced by methylation of inorganic mercury present in both freshwater and saltwater sediments, and accumulates in aquatic food chains in which the top-level predators usually contain the highest concentrations (Clarkson and Marsh 1982). Organomercury compounds other than methylmercury decompose rapidly in the environment, and behave much like inorganic Hg compounds (Beijer and Jernelov 1979). In organisms near the top of the food chain, such as carnivorous fishes, almost all Hg accumulated is in the methylated form, primarily as a result of the consumption of prey containing methylmercury; methylation also occurs at the organism level by way of mucous, intestinal bacteria, and enzymatic processes, but these pathways are not as important as diet (Huckabee et al. 1979; Boudou and Ribeyre 1983).

The biological cycle of Hg is delicately balanced, and small changes in input rates and the chemical form of Hg may result in increased methylation rates in sensitive systems (NAS 1978). For example, the acidification of natural bodies of freshwater is statistically associated with elevated concentrations of methylmercury in the edible tissues of predatory fishes (Clarkson et al. 1984). In chemically sensitive waterways, such as poorly buffered lakes, the combined effects of acid precipitation and increased emissions of Hg to the atmosphere (with subsequent deposition) pose a serious threat to the biota if optimal biomethylation conditions are met (NAS 1978).

Mercury binds strongly with sulfhydryl groups, and has many potential target sites during embryogenesis; phenylmercury and methylmercury compounds are among the strongest known inhibitors of cell division (Birge et al. 1979). Organomercury compounds, especially methylmercury, cross placental barriers and can enter mammals by way of the respiratory tract, gastrointestinal tract, skin, or mucous membranes (Elhassani 1983). When compared with inorganic mercury compounds, organomercurials are more completely absorbed, are more soluble in organic solvents and lipids, pass more readily through biological membranes, and are slower to be excreted (Clarkson and Marsh 1982; Elhassani 1983; Greener and Kochen 1983). Biological membranes, including those at the blood-brain interface and placenta, tend to discriminate against ionic and inorganic Hg, but allow relatively easy passage of methylmercury and dissolved Hg vapor (Greener and Kochen 1983). As judged by membrane model studies, it appears that electrically neutral mercurials are responsible for most of the diffusion transport of Hg, although this movement is modified significantly by pH and Hg speciation. It seems, however, that the liposolubility of methylmercury is not the entire reason for its toxicity and does not play a major role in its transport. This hypothesis needs to be examined further in studies with living membranes (Boudou et al. 1983).

Mercury-antagonistic drugs include 2,3-dimercaptopropanol, polythiol resins, selenium salts, vitamin E, and sulfhydral agents (in J.O. Nriagu (ed.). The biogeochemistry of mercury in the environment. Elsevier/North-Holland Biomedical Press, New York.Magos and Webb 1979; Elhassani 1983). Thiols (R-SH), which compete with Hg for protein binding sites, are the most important antagonists of inorganic mercury salts, and have been used extensively in attempts to counteract Hg poisoning in humans (Das et al. 1982). The protective action of selenium (Se) against adverse or lethal effects induced by inorganic or organic mercury salts has been reported for algae, aquatic invertebrates, fish, and mammals (Magos and Webb 1979; Heisinger 1979; Chang et al. 1981; Lawrence and Holoka 1981; Das et al. 1982; Gotsis 1982; Eisler 1985; Satoh et al. 1985). Selenite salts can release methylmercury from its linkage to proteins, and there is general agreement that a true antagonism exists between Se and Hg, although the exact mechanism is not fully established (Das et al. 1982). In marine mammals and humans, for example, Se and Hg concentrations are closely related, almost linearly in a 1:1 molar ratio, but this relation blurs in teleosts (in which Se is abundant) and fails in birds (Eisler 1985).

MERCURY IN MINAMATA, JAPAN

One of the earliest and most extensively documented cases of mercury poisoning occurred in the 1950's at Minamata Bay, in southwestern Kyushu, Japan--especially among fishermen and their families (Fujiki 1963, 1980; Irukayama 1967; Matida and Kumada 1969; Kojima and Fujita 1973; NAS 1978; Elhassani 1983; Nishimura and Kumagai 1983; Doi et al. 1984). The source of the mercury was in waste discharged from an acetaldehyde plant that used inorganic Hg as a catalyst; between 1932 and 1968, Minamata Bay received at least 260 tons of mercury, and perhaps as much as 600 tons. A severe neurological disorder was recognized in late 1953 and had reached epidemic proportions by 1956; 111 cases of poisoning were reported by the end of 1960 and 41 deaths by August 1965. By 1982, there were 1,800 verified human victims of mercury poisoning in a total regional population of 200,000; however, the total number of victims remains unconfirmed. Symptoms evidenced by human victims included sensory impairment, constriction of visual fields, hearing loss, ataxia, and

speech disturbances. Congenital cases were accompanied by disturbance of physical and mental development; about 6% of babies born in Minamata had cerebral palsy (vs. 0.5% elsewhere).

"Minamata disease" resulted from the discharge of methylmercury from chemical factories into Minamata Bay. Once diluted and diffused in the water, it was concentrated to a high level in fish and filter-feeding shellfish by several routes, including bioconcentration and food chain biomagnification. When these fish and shellfish were consumed by humans, methylmercury gradually accumulated to exceed a threshold value, causing intoxication. Spontaneously poisoned cats, dogs, rats, waterfowl, and pigs behaved erratically and died; flying crows and grebes suddenly fell into the sea and drowned; and large numbers of dead fish were seen floating on the sea surface (Doi et al. 1984). In laboratory studies, cats and rats fed shellfish from the Bay developed the same signs as those seen in animals affected spontaneously. Abnormal Hg content--i.e., more than 30 mg/kg fresh weight--was measured in fish, shellfish, and muds from the Bay, and in organs of necropsied humans and cats that had succumbed to the disease. Mercury contamination of fish and sediments was still evident in 1981, although discharges from the acetaldehyde plant ceased in 1971 (Doi et al. 1984).

There is a strong relation between the food of birds from Minamata and the Hg content in feathers; the content is highest in fish-eating seabirds and lowest in herbivorous waterfowl (Doi et al. 1984; Table 4). This same relation held in birds collected from China and Korea, although concentrations were significantly lower (Doi et al. 1984). There are close correlations between Hg contents of zooplankton and suspended particulate matter, and of sediments and fish muscle, suggesting a pathway from sediment to fish by way of suspended matter and zooplankton. The conversion from inorganic Hg to methylmercury is believed to have occurred primarily in zooplankton (Nishimura and Kumagai 1983).

Table 4. Mercury concentrations in selected biological and nonbiological materials collected from Minamata Bay, Japan and environs. Concentrations are in mg Hg/kg (ppm) fresh weight (FW), or dry weight (DW).

Sample, year of collection, and other variables	Concentration (mg/kg)	Reference[a]
Phytoplankton		
1974	Max. 0.32 DW	Nishimura and Kumagai 1983
Invertebrates		
1961		
Coelenterates	9.6 DW	Matida and
Tunicates	35–56 DW	Kumada 1969
Molluscs		
Pacific scallop, *Chlamys ferrei nipponensis*		
Soft parts	48 DW	
Pacific oyster, *Crassostrea gigas*		
Soft parts	10 DW; 5.6 FW	
Clam, *Hormomya mutabilis*		
Foot	18–48 DW	Fujiki 1963
Ganglion	181 DW	

Other tissues	20–73 DW	
Crustacean		
Crab, *Neptunus pelagicus*		
Muscle	39 DW	
Filter-feeding molluscs		
Soft parts		
1962	Max. 43 DW	Fujiki 1980
1963	Max. 40 DW	
1965	Max. 35 DW	
1967	Max. 60 DW	
1969	Max. 16 DW	
1971	Max. 16 DW	
1972	Max. 4 DW	
Zooplankton		
1974	Max. 1.1 DW	Nishimura and Kumagai 1983
Fish		
1961		
Largescale blackfish,		
Girella punctata		
Viscera	18–27 DW	Matida and
Muscle	12–20 DW	Kumada 1969
Scarbreast tuskfish,		
Choerodon azurio		
Muscle	309.1 DW	Fujiki 1963
Liver	85.0 DW	
Heart	36.4 DW	
Gill	13.3 DW	
Digestive gland	1.3 DW	
Black porgy, *Sparus macrocephalus*		
Muscle	16.5 DW	
Liver	32.2 DW	
Heart	18.3 DW	
Gill	9.1 DW	
Digestive gland	4.0 DW	
Muscle		
1961	23.0 DW	Fujiki 1980
1963	3.5 DW	
1965	11.5 DW	
1966–72	<0.6 DW	
1974	Max. 0.6 FW	Nishimura and Kumagai 1983
Birds		

1955–80		
Feather		
Fish-eating seabirds	7.1 DW	Doi
Omnivorous waterfowl	5.5 DW	et al. 1984
Predators	3.6 DW	
Omnivorous terrestrial birds	1.5 DW	
Herbivorous waterfowl	0.9 DW	
1965-66, found dead		
Feather	4.6–13.4 FW	Kojima and Fujita 1973
Mammals		
Cat, *Felis domesticus*, 1961		
Hair		
Naturally poisoned	40–52 DW	Jenkins 1980
Experimentally poisoned	22–70 DW	
Seawater		
1961		
Total	0.0016–0.0036 FW	EPA 1980
1974		
Filtered	0.0001 FW	Nishimura and
Suspended particulates	0.000075 FW	Kumagai 1983
Mud		
1963	28–713 DW	Fujiki 1980
1969	19–908 DW	
1970	8–253 DW	
1971	14–586 DW	
Sediments		
1973	>15–600 DW	Nishimura and Kumagai 1983

[a]Each reference applies to the values in the same row and in the rows that follow for which no other reference is indicated.

In aquatic environments where point sources of industrial contamination have been identified, the elimination of Hg discharges has usually improved environmental quality. Such improvement has been reported for Minamata Bay (Table 4); for sediments in Saguenay Fjord, Quebec, when chloralkali wastes were limited; for fish residues in Canada's Lake St. Clair after two chloralkali plants were closed; and in various sections of Europe and North America when industrial discharges were eliminated (Barber et al. 1984).

BACKGROUND CONCENTRATIONS

GENERAL

Mercury burdens in sediments and other nonbiological materials are estimated to have increased up to 5X prehuman levels, primarily as a result of man's activities (NAS 1978). The residence time of mercury is comparatively short (about 11 days) in the atmosphere, but is much longer (at least 1,000 years) in oceanic waters, soils, and sediments (Clarkson 1984).

An elevated concentration of mercury (i.e., >1.0 mg/kg fresh weight), usually as methylmercury, in any biological sample is often associated with proximity to human use of mercury. The elimination of Hg point-source discharges has usually been successful in improving environmental quality. However, elevated levels of mercury in biota may persist in contaminated areas long after the source of pollution has been discontinued (Rada et al. 1986). For example, Hg remains elevated today in resident biota of Lahontan Reservoir, Nevada, which received about 7,500 tons of mercury as a result of gold and silver mining operations during the period 1865 to 1895 (Cooper 1983). It is noteworthy that some groups of organisms with consistently elevated Hg residues may have acquired these concentrations as a result of natural processes, rather than from anthropogenic activities. These groups include older specimens of long-lived predatory fishes, marine mammals (especially pinnipeds), and organisms living near natural Hg-ore-cinnabar deposits.

NONBIOLOGICAL

Mercury burdens have increased 2X to 5X precultural levels in freshwater and estuarine sediments and freshwater lakes and rivers, but estimated increases in oceanic waters and terrestrial soils have been negligible (NAS 1978). Total mercury concentrations in uncontaminated natural waters (presumably unfiltered) now range from about 0.001 to 0.050 ug/l (Table 5). In sediments that were anthropogenically contaminated with Hg, concentrations were significantly elevated (usually >20.0 mg/kg) when compared with uncontaminated sediments (usually <1.0 mg/kg). The residence time of Hg in nonbiological materials is variable, and depends on a number of physicochemical conditions. Estimated half-time residence values for Hg are 11 days in the atmosphere, 1,000 years in terrestrial soils, 2,100 to 3,200 years in ocean waters, And >250 million years in oceanic sediments (NAS 1978, as quoted in Boudou and Ribeyre 1983); the estimate was 1 month to 5 years for water from the contaminated Saguenay River in Quebec (Smith and Loring 1981).

Table 5. Mercury concentrations in water and sediments.

Material (units)	Concentration	Reference[a]
Water (µg/L)		
Open ocean	0.0053 ± 0.0022	Nishimura et al. 1983
Open ocean	<0.01	Fitzgerald 1979
Coastal seawater	<0.02	
Estuarine seawater	<0.05	
Rivers and lakes	0.01 (Max. 0.05)	
Rainwater		
Open ocean	0.001	
Coastal ocean	0.01	
Continents	Often >0.05	
Sediment interstitial water	0.1	
Glacial waters	0.01	
Ground waters	0.05	
Sediments (mg/kg)		
Contaminated areas		
Near chloralkali plant		
Quebec, Canada	12.0	Smith and Loring 1981
Norway	250 (90.0–350.0)	Skei 1978
Thailand	8.4–58.0	Suckcharoen and Lodenius 1980

Near gold mining operations		
South Dakota	0.1–4.1	Martin and Hartman 1984
Australia	120.0	Bycroft et al. 1982
Near Hg-fungicide plant		
Denmark	22.0	Kiorboe et al. 1983
Near acetaldehyde plant		
Minamata Bay, Japan	28.0–713.0	Skei 1978
Near pulp and paper mill		
Finland	746.0	Paasivirta et al. 1983
Uncontaminated areas		
North Central U.S.	0.02–0.06 (Max. 0.11)	Martin and Hartman 1984
South Dakota	0.02–0.1	
Thailand	0.03	Suckcharoen and
Finland	0.02	Lodenius 1980
Various lakes	Usually <10.0, frequently <1.0	Skei 1978

[a]Each reference applies to the value in the same row and in the rows that follow for which no other reference is indicated.

Levels of mercury in sediments may be reflected by an increased mercury content in epibenthic fauna. For example, Hg concentrations in sediments near the Hyperion sewer outfall in Los Angeles, which ranged up to 820 ug/kg and decreased with increasing distance from the outfall, were reflected in the Hg content of crabs, scallops, and whelks. Concentrations of Hg were highest in organisms collected nearest the discharge, and lowest in those collected tens of kilometers away (Klein and Goldberg 1970).

BIOLOGICAL

Information on mercury residues in field collections of living organisms is especially abundant; accordingly, only a few selected data points are shown in Table 6. Additional, more extensive, information was given by Jenkins (1980), Eisler (1981), and Wren (1986).

In general, mercury concentrations in biota were usually <1.0 mg/kg fresh weight tissue in organisms collected from locations not directly affected by man's use of the element. However, they exceed 1.0 mg/kg--and are sometimes markedly higher--in animals and vegetation from the vicinity of chloralkali plants; agricultural users of mercury; smelteries; mining operations; pulp and paper mills; factories producing Hg-containing paints, fertilizers, and insecticides; sewer outfalls; sludge disposal areas; and other anthropogenic point sources of mercury. (Several notable exceptions to this generalization are discussed later.)

Certain species of macrophytes strongly influence mercury cycling. For example, *Spartina alterniflora*--a dominant salt marsh plant in Georgia estuaries--accounted for almost half the total mercury budget in that ecosystem (Windom et al. 1976). Mangrove vegetation plays a similarly important role in mercury cycling in the Florida Everglades (Tripp and Harriss 1976). These findings suggest that more research is needed on the role of higher plants in the mercury cycle.

Mercury was detectable in the tissues of almost all freshwater fishes examined, with the majority of the Hg (>80%) present as methylmercury (Huckabee et al. 1979). Methylmercury is absorbed more efficiently than inorganic Hg from water, and probably from food, and is retained longer regardless of the uptake pathway (Huckabee et al. 1979). Reservoir construction has often been inferred to be a cause of elevated mercury concentrations in fish. It is hypothesized that increases in mercury levels observed in fish were due to bacterial methylation of naturally occurring Hg in the flooded soils (Bodaly et al. 1984). Other factors that enhance accumulation of Hg in predatory teleosts from recently created reservoirs include low pH, high humus content, and low bioproduction (Lodenius 1983; Lodenius et al. 1983). In general, mercury levels are higher in fish from younger oligotrophic reservoirs, and lower in fish from older eutrophic reservoirs; in both situations, tissue Hg levels usually decline as the reservoirs age (Abernathy and Cumbie 1977). Concentrations >0.5 mg/kg (but <1.0 mg/kg) fresh weight have been reported in trout from several wilderness lakes in northern Maine (Akielaszak and Haines 1981) and from the Adirondacks region of New York (Sloan and Schofield 1983); these values are considerably higher than might be expected for fish inhabiting remote lakes. These elevated concentrations were usually associated with lakes of low pH, low calcium, low dissolved organic carbon concentrations, and low water hardness and alkalinity.

Table 6. Mercury concentrations in field collections of selected species of flora and fauna. Values shown are in mg Hg/kg fresh weight (FW), or dry weight (DW).

Taxonomic group, organism, tissue, and other variables	Concentration[a] (ppm)	Reference[b]
Fungi, Lichens, Mosses, Plants		
Mandarin orange, *Citrus tachibana*		
Japan		
Sprayed with Hg		
Fruit skin	0.03–0.24 FW	Jenkins 1980
Fruit pulp	0.01-0.4 FW	
Unsprayed		
Skin and pulp	0.01–0.05 FW	
Fungi, *Cortinarius* spp.		
Near smelter	9.5–35.0 DW	
Moss, *Dicranum scoparium*, whole		
Tennessee		
Exposed to fly ash	1.1 DW	
Remote areas	0.1 DW	
Great Smoky Mountains	0.07 DW	
Hawaii	0.16 DW	
Iceland	0.03 DW	
Water hyacinth, *Eichhornia crassipes*		
From sewage lagoon in		
Bay St. Louis, Mississippi		
Leaves	70.0 DW	Chigbo et al. 1982
Lichen, *Hypogymnia physodes*, whole		
Finland, 1982–83		
Distance, in km, from		
chloralkali plant		

0–1	18.0 FW	Lodenius and
1–5	2.0 FW	Tulisalo 1984
5–20	0.4 FW	
20–100	0.3 FW	
>100	0.3 FW	
Labrador tea, *Ledum* sp.		
Alaska, over cinnabar deposit		
Stem	1.0–3.5 DW	Jenkins 1980
Alfalfa, *Medicago sativa*		
From soil containing 0.4 ppm Hg		
Root	90.0 FW	
Leaf	0.13–0.4 FW	
From soil with <0.4 ppm Hg		
Leaf	0.16 FW	
Tobacco, *Nicotiana tabacum*		
Leaf		
Treated with Hg (Japan)	1.0–1.6 FW	
Untreated (USA)	<0.2 FW	
Rice, *Oryza sativa*, grain		
Sprayed with Hg	0.1–0.7 FW	
Unsprayed	0.02–0.1 FW	
Marine flowering plant, *Posidonia oceanica*		
Near sewer outfall, Marseilles, France		
Rhizomes	2.5 DW	Augier
Leaves	51.5 DW	et al. 1978
Roots	0.6 DW	
Cherry, *Prunus avium*		
Europe (Slovenia), bark		
Uncontaminated areas	0.06 FW	Jenkins 1980
High Hg in soil	6.0 FW	
Factory area	59.0 FW	
Mosses, *Sphagnum* spp., whole		
Finland, 1982–83		
Distance (km) from chloralkali plant		
0–1	3.8 (1.5–16.0) FW	Lodenius and
1–5	0.8 (0.2–2.6) FW	Tulisalo 1984
5–20	0.09 (0.04–0.2) FW	
20–100	0.05 (0.0–0.8) FW	
>100	0.02 FW	

Aquatic Invertebrates

Freshwater		
Annelids, 2 families		
From Hg-contaminated areas	0.3–0.6 FW	Huckabee
From uncontaminated areas	0.03–0.05 FW	et al. 1979
Arthropods		
Sow bug, *Asellus* sp.		
Sweden, whole		
20 km below paper mill	1.9 FW	Jenkins 1980
1–15 km above paper mill	0.06 FW	
Crustaceans, 2 families		
From Hg-contaminated areas	1.9–10.0 FW	Huckabee
From uncontaminated areas	0.06–0.56 FW	et al. 1979
Insects, 8 families		
From Hg-contaminated areas	0.5–5.0 FW	
From uncontaminated areas	0.05–0.21 FW	
Stonefly, *Isoperla* sp.		
Whole, Sweden		
17 km below paper mill	2.4 FW	Jenkins 1980
15 km above paper mill	0.07 FW	
Crayfish, *Orconectes virilis*		
Ontario, whole		
Central location	0.09–0.49 FW	
From chloralkali plant location	1.4–7.4 FW	
Crayfish, *Pacifiastacus* sp.		
Lahontan Reservoir, Nevada, 1981		
Abdomen	5.7 FW	Cooper 1983
Molluscs		
From Hg-contaminated areas	0.02–2.1 FW	Huckabee
From uncontaminated areas	0.05 FW	et al. 1979
Marine		
Annelids, whole, 3 spp.		
Georgia, USA, estuaries		
Hg-contaminated estuary		
Total Hg	0.7–4.5 DW	Windom and
Methyl Hg	Max. 0.8 DW	Kendall 1979
Control estuary		
Total Hg	0.1–0.6 DW	
Methyl Hg	Max. 0.13 DW	
Arthropods		
Crustaceans, whole, 2 spp.		
Georgia, USA, estuaries		

Hg-contaminated estuary		
Total Hg	0.4–1.8 DW	
Methyl Hg	Max. 1.0 DW	
Control estuary		
Total Hg	0.1–0.4 DW	
Methyl Hg	Max 0.05 DW	
American lobster, *Homarus americanus*		
Muscle		
Chesapeake Bay	0.03–0.06 FW	Jenkins 1980
NW Atlantic	0.25–1.6 DW	
Nova Scotia	0.15–1.5 FW	
Spiny lobster, *Nephrops norvegicus*		
Tyrrhenian Sea, 1981		
Muscle	2.9 FW	Schreiber 1983
Shrimp		
Edible portions		
Total Hg	0.77 FW	Cappon
Methyl Hg	0.4 FW	and Smith 1982
Echinoderms		
Sea stars, 3 spp., 1981		
Venezuela, polluted area		
Gonads	3.8–8.7 DW; 0.9–1.6 FW	Iglesias and Panchaszadeh 1983
Molluscs		
From vicinity chloralkali plant, Israel, 1980–82, soft parts		
Gastropod, *Arcularia gibbosula*	18.2–38.7 DW	Hornung et al. 1984
Bivalve, *Donax venustus*	Max. 6.4 DW	
Bivalves, soft parts		
From Hg-polluted area, Denmark		
Deposit feeders	1.4–4.4 FW	Kiorboe
Suspension feeders	0.9–1.9 FW	et al. 1983
Edible portions		
Total Hg	0.04–0.22 FW	Cappon and
Methyl Hg	Max. 0.09 FW	Smith 1982
Soft parts, 2 spp.		
Georgia, USA, estuaries		

Hg-contaminated estuary	0.5–1.2 DW	Windom and
Control estuary	0.1–0.2 DW	Kendall 1979
Mussel, *Mytilus californianus*		
Soft parts		
Nationwide	<0.4 DW	Flegal
California	0.6–2.5 DW	et al. 1981
Mussel, *Mytilus edulis*		
Soft parts		
Belgium	1.0 DW	
Spain	1.5 DW	
New Brunswick	0.1 FW	
Netherlands	0.1–0.3 FW	
Great Britain	0.02–0.7 FW	
New Zealand	0.02–0.48 FW	
Norway	0.2–0.65 DW	
Softshell clam, *Mya arenaria*		
Soft parts		
Chesapeake Bay, MD	0.01–0.05 FW	
Nova Scotia	0.03–0.13 FW	
New Brunswick		
3 km below pulp mill	0.9 FW	
3 km below chloralkali plant	3.6 FW	
Terrestrial Invertebrates		
Aphid, *Macrosiphium gei*		
Whole, Illinois		
Fed on Hg-treated tomato plants	0.3–11.5 FW	
Control	0.08–0.81 FW	
Lacewing, *Chrysopa carnea*		
Whole, Illinois		
Fed on Hg-treated tomato plants	0.6–31.4 FW	
Control	0.0–1.1 FW	
Fish		
Rock bass, *Ambloplites rupestris*		
Muscle		
Ontario	0.6–4.6 FW	
Michigan	0.4 FW	
Western Ontario	1.1–10.9 FW	
Lake St. Clair	0.5–2.0 FW	
Blue hake, *Antimora rostrata*		
NW Atlantic, 2500 m depth		

Muscle		
1880	0.51 FW	Barber
1970	0.34 FW	et al. 1984
Freshwater drum, *Aplodinotus grunniens*		
Whole		
Age 0	0.05 FW	Busch 1983
Age I	0.13 FW	
Age II	0.18 FW	
Blacktail, *Diplodus sargus*, muscle		
Hg-polluted area	0.3–1.7 FW	Hornung
Unpolluted area	0.04–0.64 FW	et al. 1984
Northern pike, *Esox lucius*, muscle		
Sweden	0.2–9.8 FW	Jenkins 1980
Quebec	0.3–0.8 FW	
Norway	0.1 FW	
Saskatchewan	0.7–10.6 FW	
Canada (normal)	0.1 FW	
Canada (polluted)	0.5–0.7 FW	
Lake St. Clair	2.0–3.0 FW	
NW Ontario mining area	5.6 FW	
Wisconsin	0.9–1.4 FW	
Manitoba, manmade reservoir		
Preimpoundment (1971–73)	0.25–0.35 FW	Bodaly
Postimpoundment (1979–82)	0.67–0.95 FW	et al. 1985
Fish		
Muscle		
Freshwater		
Total Hg	0.27–1.7 FW	Cappon and
Methyl Hg	Max. 1.4 FW	Smith 1982
Marine		
Total Hg	0.11–5.7 FW	
Methyl Hg	Max. 4.5 FW	
Lahontan Reservoir, Nevada, 1981		
Muscle, 5 spp.	Max. 2.3–3.9 FW	Cooper 1983
Liver, 5 spp.	Max. 2.4–8.3 FW	
Heart, 4 spp.	Max. 1.1–2.1 FW	
Louisiana, Atchafalaya River, 1981		
Whole, 8 spp.	0.06–0.79 FW	Winger and Andreasen 1985
Nationwide, USA, whole		
1969–70	0.26 (0.05–1.7) FW	Henderson and
Pacific Coast and Alaska	0.25 (0.05–1.7) FW	Shanks 1973
Southwest	0.08 (<0.05–0.14) FW	

North Central	0.20 (<0.05–0.05) FW	
Northeast	0.23 (<0.05–0.08) FW	
Southeast	0.23 (<0.05–1.0) FW	
1972	0.15 FW	
1976–77	0.11 FW	Lowe et al.
1978–79	0.11 (0.01–1.1) FW	1985
1980–81	0.11 (0.01–0.77) FW	
Thailand		
Muscle		
Near chloralkali plant		
No waste water system, 1975–76	0.32–3.6 FW	Suckcharoen and Lodenius 1980
With waste water system, 1978	0.1–1.4 FW	
Control location	0.01–0.3 FW	
Various species		
Muscle		
From unpolluted areas	0.04–0.15 FW	NAS 1978
From moderately Hg-polluted areas	>1.0 FW	
From highly polluted areas	10.0–24.0 FW	
Blackfish, *Gadopsis marmoratus*		
Muscle		
From Hg-contaminated sediments	Max. 0.64 FW	Bycroft et al. 1982
From uncontaminated sediments	Max. 0.06 FW	
Channel catfish, *Ictalurus punctatus*		
Muscle		
Lake Erie	0.3–1.8 FW	Jenkins 1980
Lake St. Clair	0.5–2.0 FW	
Ohio	0.1–0.4 FW	
Illinois	0.03–0.2 FW	
Oregon	0.02–1.5 FW	
Georgia	0.1–1.9 FW	
Texas	0.2–2.5 FW	
Pumkinseed, *Lepomis gibbosus*		
16 lakes, Ontario, Canada, 1981		
Muscle	0.09–0.54 FW	Wren and MacCrimmon 1984
Black marlin, *Makaira indica*		
Muscle		
Pacific Ocean	0.6–4.3	Cappon and Smith 1982
NE Australia	0.5–16.5 FW	
Blue marlin, *Makaira nigricans*		

Muscle		
Hawaii	0.4–14.0 FW	
Total Hg	0.4–0.9 FW	
Methyl Hg	Max. 0.16 FW	
Largemouth bass, *Micropterus salmoides*		
Muscle		
Texas	0.1 FW	Jenkins 1980
Utah	0.3–7.3 FW	
California	0.1–0.6 FW	
Oregon	0.2–1.8 FW	
Washington	0.1–0.3 FW	
Georgia	0.1–5.4 FW	
Michigan	0.2–0.9 FW	
Illinois	0.03–1.2 FW	
Arizona	0.3 FW	
Striped bass, *Morone saxatilis*		
Lahontan Reservoir, Nevada, 1981		
Single specimen, 16 years old		
Muscle	9.5 FW	Cooper 1983
Heart	5.6 FW	
Liver	23.7 FW	
Muscle		
Body weight		
<3.2 kg	<0.5 FW	Alexander
3.2–5.7 kg	0.5 FW	et al. 1973
>5.7 kg	>0.5 FW	
Yellow perch, *Perca flavescens*		
Whole		
Age 0	0.07 FW	Busch 1983
Age I	0.13 FW	
Age II	0.22 FW	
Round whitefish, *Prosopium cylindraceum*		
Saginaw Bay, Michigan		
1977–78		
Fillets		
Methyl Hg	Max. 0.05 FW	Miller and
Total Hg	Max. 0.1 FW	Jude 1984
Trout, *Salmo* spp.		
Missouri		
Liver and muscle		

1946–50	3.0 FW	Lloyd
1973	0.1–0.3 FW	et al. 1977
Brook trout, *Salvelinus fontinalis*		
Adirondack lakes (15), New York		
Whole	<1.0 FW	Sloan and Schofield 1983
Lake trout, *Salvelinus namaycush*		
Muscle		
British Columbia	1.1–10.5 FW	Jenkins 1980
Ontario	0.3–1.3 FW	
Quebec	0.3–1.2 FW	
New York	0.3–0.6 FW	
Sharks, Australia, 1980		
Muscle, 7 spp.		
Carcharhinus spp.	Max. 4.3 FW	Lyle 1984
Sphyrna spp.	Max. 4.9 FW	
Walleye,		
Stizostedion vitreum vitreum		
Manitoba, manmade reservoir		
Muscle		
Preimpoundment (1971–77)	0.2–0.3 FW	Bodaly
Postimpoundment		et al. 1984
(1978–92)	0.6–0.8 FW	
Tunas, 1981, 5 spp.		
Muscle	1.0–6.3 FW	Schreiber 1983
Swordfish, *Xiphias gladius*		
Muscle		
NW Atlantic	2.0 FW; 8.1 DW	Jenkins 1980
Peru	1.1–1.8 FW	
Pacific	0.5–1.7 FW	
W. Atlantic	0.05–4.9 FW	
Gibralter Strait	1.0–2.0 FW	
Amphibians and Reptiles		
European toad, *Bufo bufo*		
Yugoslavia		
Control area		
Liver	1.5 FW	
Kidney	1.2 FW	
Lung	0.2 FW	
Muscle	0.2 FW	
Egg	0.06 FW	
Polluted mercury mining area		
Liver	21.8–25.5 FW	Jenkins 1980;
Kidney	22.8–24.0 FW	Terhivuo et al. 1985

Lung	1.7 FW	
Muscle	2.3–2.9 FW	
Egg	2.3 FW	
Loggerhead sea turtle, *Caretta caretta*		
Egg	0.01 FW	Hall 1980
Crocodile, *Crocodylus acutus*		
Egg	0.7 FW	
Bullfrog, *Rana catesbeiana*		
Lake St. Clair		
Carcass	0.1 FW	Jenkins 1980
Liver	0.3 FW	
Leopard frog, *Rana pipiens*		
Lake St. Clair		
Carcass	0.1–0.2 FW	
Liver	0.5–1.1 FW	
Florida		
Liver	0.1 FW	
Frog, *Rana temporaria*		
Yugoslavia, 1975		
From Hg-mining area		
Liver	21.0 FW	Terhivuo
Kidney	16.2 FW	et al. 1984
Muscle	3.4 FW	
Egg	1.3 FW	
From uncontaminated area		
All tissues	<0.08 FW	
Birds		
Goshawk, *Accipiter gentilis*		
Sweden		
Feather		
1860–1946	2.2 FW	Jenkins 1980
1947–65	29.0 FW	
1967–69	3.1–5.1 FW	
Finnish sparrowhawk, *Accipiter nisus*		
Feather		
Finland		
1899–1960	4.1 (2.1–7.7) DW	Solonen and
1961–70	11.1 (2.3–42.0) DW	Lodenius 1984
1971–82	7.4 (1.0–29.0) DW	
Germany		
1972–73	4.9 (0.4–20.3) DW	

Norway		
1976	2.0–20.0 DW	
Wood duck, *Aix sponsa*		
Tennessee, 1972–73		
Juveniles		
Liver	0.4 (0.1–1.1) FW	Lindsay and
Muscle	0.1 (0.05–0.4) FW	Dimmick 1983
Fat	0.1 (0.01–0.4)	
Adults		
Liver	0.2 (0.1–0.3) FW	
Muscle	0.08 (0.06–0.11) FW	
Fat	0.06 (0.01–0.11) FW	
Blue-winged teal, *Anas discors*		
Muscle		
Lake St. Clair	0.1–2.3 FW	Jenkins 1980
Ontario	3.8–10.4 FW	
Wisconsin	0.0–0.5 FW	
Illinois	0.05 FW	
Great blue heron, *Ardea herodius*		
Liver		
Lake St. Clair	97.0 (14.6–175.0) FW	
New Brunswick	4.5 FW	
Lake Erie	0.7–4.3 FW	
Wisconsin	0.5 (0.2–1.1) FW	
Birds		
Antarctic		
Liver		
1977–79		
4 spp.	0.5–1.3 FW	Norheim
3 spp.	2.7–2.9 FW	et al. 1982
1980		
5 spp.	0.5–2.1 FW	Norheim and Kjos-Hanssen 1984
Belgium, 1970–81		
Liver, 30 spp.		
Aquatic birds	0.11–35.0 FW	Delbeke
Terrestrial birds	ND-14.0 FW	et al. 1984
Hawaiian, 1980		
Egg, 3 spp.	0.12–0.36 FW	Ohlendorf and Harrison 1986
North America		
Feather		

From areas with mercury-treated seed dressing		
Seed-eating songbirds	1.6 DW	NAS 1978
Upland game birds	1.9 DW	
From untreated areas		
Seed-eating songbirds	0.03 DW	
Upland game birds	0.35 DW	
Northwestern Ontario, Canada		
From a heavily mercury-contaminated freshwater system		
Liver		
Scavengers	57.0 (13.8–121) FW	Fimreite 1979
Fish eaters	39.5 (1.7–91) FW	
Omnivores	26.6 (9.5–53) FW	
Invertebrate feeders	12.4 (3.2–28) FW	
Vegetarians	6.2 (1.9–28) FW	
Diving ducks	Max. 175.0	NAS 1978
Muscle		
Diving ducks	Max. 23.0 FW	
Mallard, *Anas platyrhynchos*	Max. 6.1 FW	
Eagle-owl, *Bubo bubo*		
Sweden, 1963–76		
Feather		
Inland populations	3.2 DW	Broo and
Coastal populations	6.5 DW	Odsjo 1981
1829–1933	0.3–3.6 FW	Jenkins 1980
1964–65	12.8–41.0 FW	
Peregrine, *Falco peregrinus*		
Feather		
1834–1849	2.5 DW	NAS 1978
1941–65	>40.0 DW	
Swedish gyrfalcon, *Falco rusticolus*		
Nestlings, feather		
Percent aquatic birds in diet		
None	0.035 FW	Lindberg 1984
4.8% biomass	0.66 FW	
10.6% biomass	1.22 FW	
American bald eagle, *Haliaeetus leucocephalus*		
Egg		
Maine (highest concentrations Nationwide)		
1974	0.35–0.58 FW	Wiemeyer

1975	0.22–0.63 FW	et al. 1984
1976	0.22–0.66 FW	
1977	0.28–0.90 FW	
1978	0.30 FW	
1979	0.84–1.2 FW	
Ringed herring gull,		
Larus argentatus		
Denmark, 1975–76		
Liver	0.65 (0.08–2.34) FW	Karlog and Clausen 1983
Ontario		
Egg	1.5–15.8 FW	Jenkins 1980
Albumin	16.1–22.7 FW	
Yolk	3.4–3.5 FW	
California seagull,		
Larus californicus		
Lahontan Reservoir, Nevada, 1981		
Muscle	0.4 FW	Cooper 1983
Liver	1.0 FW	
Egg	0.1–0.2 FW	
Osprey, *Pandion haliaetus*		
Sweden		
Feather		
1840–1940	3.5–5.0 FW	Jenkins 1980
1940–66	>17.0 FW	
Brown pelican,		
Pelecanus occidentalis		
Egg		
South Carolina	0.3–0.5 FW	
Florida	0.4 FW	
California	0.4 FW	
Liver	0.75 DW	Ohlendorf et al. 1985
Kidney	0.68 DW	
Feather	0.97 DW	
Ring-necked pheasant,		
Phasianus colchicus		
Muscle		
Denmark	0.01 FW	Jenkins 1980
Idaho	0.0–15.0 FW	
Indiana	0.06 FW	
Oregon	<0.5 FW	
Wyoming	0.2–0.6 FW	
Colorado	0.04–0.6 FW	

Utah	0.15 (0.01–2.1) FW	
California	1.6–4.7 FW	
Wisconsin	0.01–0.08 FW	
Illinois	0.02–0.03 FW	
Great crested grebe,		
Podiceps cristata		
Sweden		
Feather		
1865–1940	<10.0 FW	
1940–66	>14.0 FW	
Mourning dove, *Zenaida macroura*		
Liver		
Eastern United States	0.07–0.67 FW	
Mammals		
Australian fur seal,		
Arctocephalus pusillus		
Muscle	0.9 (0.1–1.9) FW	Bacher 1985
Liver	62.3 (1.0–170.0) FW	
Kidney	0.6 (0.1–1.7) FW	
Spleen	1.3 (0.0–3.8) FW	
Brain	0.7 (0.0–2.5) FW	
Hair	9.6 (1.1–19.8) DW	
Woodmouse, *Apodemus sylvaticus*		
Great Britain		
In field with Hg-treated		
wheat seed		
Liver	Max. 7.1 FW	Jenkins 1980
Kidney	Max. 11.7 FW	
In chloralkali area		
Liver	Max. 0.5 FW	
Kidney	Max. 1.3 FW	
Control area		
Liver	Max. 0.07 FW	
Kidney	Max. 0.3 FW	
Roe deer, *Capreolus capreolus*		
Males, Poland, 1977–78		
From Hg-contaminated habitat		
Muscle	0.047 FW	Krynski
Liver	0.036 FW	et al. 1982
Kidney	0.053 FW	
From uncontaminated area		
Muscle	0.013 FW	
Liver	0.015 FW	

Kidney	0.027 FW	
Beaver, *Castor canadensis*		
Wisconsin, 1972–75		
All tissues	<0.09 FW	Sheffy and St. Amant 1982
Cat, *Felis domesticus*		
Ate fish from below chloralkali plant, NW Ontario		
Brain	6.9–16.4 FW	Jenkins 1980
Pancreas	4.3–4.9 FW	
Kidney	0.8–13.4 FW	
Liver	14.2–67.1 FW	
Fur	121.0–392.0 FW	
River otter, *Lutra canadensis*		
Winnipeg River,		
Manitoba, Canada, 1979–81		
Males		
Liver	Max. 8.9 FW	Kucera 1983
Kidney	Max. 6.5 FW	
Brain	Max. 3.1 FW	
Females		
Liver	Max. 3.9 FW	
Kidney	Max. 1.8 FW	
Brain	Max. 0.6 FW	
Wisconsin, 1972–75		
Brain	0.7 FW	Sheffy and St. Amant 1982
Muscle	1.4 FW	
Liver	3.3 (Max. 23.6) FW	
Kidney	8.5 (Max. 20.9) FW	
Fur		
Industrial area	9.5 (Max. 63.2) FW	
Nonindustrial area	3.8 FW	
Bobcat, *Lynx rufus*		
Hair		
Georgia USA		
Upper coastal plain	13.1 DW	Jenkins 1980
Lower coastal plain	0.9 DW	
Mink, *Mustela vision*		
Wisconsin, 1972–75		
Brain	0.5 FW	Sheffy and St. Amant 1982
Muscle	1.3 FW	
Liver	2.1 (Max. 17.4) FW	
Kidney	2.3 (Max. 12.5) FW	

Fur		
Industrial area	10.5 (Max. 41.2) FW	
Nonindustrial area	3.0 FW	
Winnipeg River,		
Manitoba, Canada, 1979–81		
Males		
Liver	Max. 9.9 FW	Kucera 1983
Kidney	Max. 6.4 FW	
Brain	Max. 2.4 FW	
Females		
Liver	Max. 10.7 FW	
Kidney	Max. 8.1 FW	
Brain	Max. 2.1 FW	
Muskrat, *Ondatra zibethicus*		
Wisconsin, 1972–75		
All tissues	<0.06 FW	Sheffy and St. Amant 1982
Sheep, *Ovis aries*		
Grazing for 23 months on		
Hg-contaminated field		
Diet (grass)		
Winter	6.5 DW	Edwards and
Summer	1.9 DW	Pumphery 1982
Lung	Max. 4.0 FW	
Kidney	Max. 3.1 FW	
Liver	Max. 2.4 FW	
Brain	Max. 1.1 FW	
Flesh	<1.0 FW	
Harbor seal, *Phoca vitulina richardi*		
Liver		
California	269.0 (81.0–700.0) FW	Jenkins 1980
Oregon	0.3–68.0 FW	
Washington	1.3–60.0 FW	
Pribilof Islands	0.6–8.9 FW	
Raccoon, *Procyon lotor*		
Wisconsin, 1972–75		
Brain	<0.02 FW	Sheffy and
Muscle	0.08 FW	St. Amant 1982
Kidney	1.4 FW	
Liver	2.0 FW	
Fur	3.8 FW	
Gray squirrel, *Sciurus carolinensis*		

Hair, Florida, 1974		
Rural areas	0.43 FW	Jenkins 1980
Urban		
Age 0–1	1.0 (0.07–6.7) FW	
Age >2	2.7 (0.30–9.2) FW	
Striped dolphin, *Stenella coeruleoalba*		
Adults, Japan, 1977–80		
Muscle		
Total Hg	15.2 FW	Itano
Methyl Hg	5.3 FW	et al. 1984a
Liver		
Total Hg	205.0 FW	
Methyl Hg	7.0 FW	
Kidney		
Total Hg	14.7 FW	
Methyl Hg	3.2 FW	
Whole body		
Age 1 year		
Total Hg	0.8 FW	Itano
Methyl Hg	0.4 FW	et al. 1984b
Age 3 years		
Total Hg	1.8 FW	
Methyl Hg	1.0 FW	
Age 4 years		
Total Hg	3.0 FW	
Methyl Hg	1.5 FW	
Age 14 years		
Total Hg	4.5 FW	
Methyl Hg	2.6 FW	
Age 20 years		
Total Hg	10.7 FW	
Methyl Hg	3.5 FW	
Red fox, *Vulpes vulpes*		
Hair		
Georgia, USA		
Upper coastal plain	2.3 DW	Jenkins 1980
Lower coastal plain	0.5 DW	
Wisconsin, 1972–75		
Fur	0.6 FW	Sheffy and
Other tissues	<0.14 FW	St. Amant 1982
California sea lion, *Zalophus californianus*		

Liver		
Mother	73.0–1,026.0 DW	Jenkins 1980
Pup	0.9–16.0 DW	
Kidney		
Mother	4.1–43.2 DW	
Pup	0.6–6.7 DW	

[a]Concentrations are listed as mean, range, or maximum (Max.).

[b]Each reference applies to the value in the same row and in the rows that follow for which no other reference is indicated.

Nationwide monitoring of whole fish during the period 1969 to 1981 demonstrated that the highest Hg concentrations (0.33 to 1.7 mg/kg FW) were in northern squawfish (*Ptychocheilus oregonensis*) from the Columbia River basin in the Pacific Northwest (Henderson and Shanks 1973; Lowe et al. 1985). Elevated Hg concentrations in this piscivorous species were attributed primarily to the presence of major cinnabar deposits and with Hg use associated with mineral mining in the Columbia River basin. Northern squawfish may have a natural tendency to accumulate high concentrations of mercury in their flesh--as is well known for older specimens of long-lived predatory fishes such as tunas, billfishes, bluefish (*Pomatomus saltatrix*), striped bass (*Morone saxatilis*), northern pike (*Esox lucius*), and many species of sharks--but Hg uptake kinetics in squawfish requires further research (Lowe et al. 1985).

Of 159 species of finfish and sharks from the coastal waters of Alaska, Hawaii, and the conterminous United States, mercury concentrations in muscle were usually less than 0.3 mg/kg fresh weight (Hall et al. 1978). Mean Hg concentrations in excess of 0.5 mg/kg fresh weight muscle were recorded in 31 species, including 10 species of sharks and 4 species of billfishes; however, these 31 species accounted for less than 0.65% of the catch intended for human consumption (Hall et al. 1978).

In birds, it is generally acknowledged that mercury concentrations are highest in species that eat fish and other birds. Residues were highest in kidney and liver, but total Hg contents were significantly modified by food preference and availability, and by migratory patterns (NAS 1978; Delbeke et al. 1984). Also, there is an inverse relationship between total Hg and percent methylmercury in tissues of various avian species (Norheim et al. 1982; Karlog and Clausen 1983)--a pattern that seems to hold for all vertebrate organisms for which data are available. Diet and migration are the most important Hg modifiers in birds. For example, the higher levels of Hg in juvenile than in adult wood ducks (*Aix sponsa*) from Tennessee were related to dietary patterns: juveniles preferred insects, whereas adults preferred pondweed tubers; Hg residues were higher in the insects than in the pondweeds (Lindsay and Dimmick 1983). Concentrations of Hg in livers of Antarctic birds reflected Hg body burdens accumulated during migration, while the birds were overwintering near industrialized areas. Concentrations were highest in species that ate higher trophic levels of prey and were especially pronounced for skuas, *Catharacta* spp.; however, significant inherent interspecies differences were evident (Norheim et al. 1982; Norheim and Kjos-Hanssen 1984).

The significance of Hg residues in birds is not fully understood. For example, all eggs of the American bald eagle (*Haliaeetus leucocephalus*) collected Nationwide contained detectable levels of Hg, but the mean was 0.15 mg Hg/kg (fresh weight basis) in eggs from unsuccessful nests vs. 0.11 in eggs from successful nests (Wiemeyer et al. 1984). Many other contaminants--especially organochlorine compounds--were in eagle eggs, and several were present at levels that potentially interfere with eagle reproduction (Wiemeyer et al. 1984). It is not now possible to implicate Hg as a major cause of unsuccessful eagle reproduction.

Bird feathers have been used for some time as indicators of Hg loadings in terrestrial and marine environments. The keratin in bird feathers is not easily degradable, and Hg is probably associated firmly with the disulphide bonds of keratin. Consequently, it has been possible to compare Hg contents of feathers recently sampled with those from museum birds, thereby establishing a time series (Applequist et al. 1984). The most probable source of recent elevated Hg residues in feathers of the Finnish sparrowhawk (*Accipiter nisus*) was from consumption of avian granivores that had become contaminated as a result of eating seeds treated with

organomercury compounds; in 1981, 5.6 tons of methoxyethylmercury compounds were used in Finnish agriculture for protection of seeds against fungi (Solonen and Lodenius 1984). Captive Swedish eagle-owls (*Bubo bubo*), with low Hg content in feathers (<1.0 mg/kg DW), that were introduced into coastal areas quickly reflected the high (6.5 mg/kg) Hg levels in feathers of wild eagle-owls from that region. Captive birds released into inland territories, where Hg levels were near background, did not accumulate Hg in feathers (Broo and Odsjo 1981). Mercury levels in feathers of nestling Swedish gyrfalcons (*Falco rusticolus*) showed a better correlation with Hg levels in actual food items than with levels based on adult feathers. Mercury concentrations in feathers were higher in nestlings fed partly with aquatic bird species containing >0.07 mg Hg/kg in pectoral muscle than in nestlings fed willow grouse (*Lagopus lagopus*) and ptarmigan (*Lagopus mutus*), both of which contained <0.01 mg Hg/kg in pectoral muscle (Lindberg 1984). In some instances there was a substantial time lag, up to 10 years, between the introduction of a pesticide, such as alkylmercury, its subsequent banning, and measurable declines of Hg in feathers of several species of Swedish raptors; this was the case for various species of *Falco, Haliaeetus*, *Bubo, Buteo*, and *Accipiter* (Wallin 1984). Accordingly, a reduction in Hg content in feathers of free-living birds may be sufficient to establish an improved situation.

Among mammals, marine pinnipeds usually contained the highest reported concentrations of Hg in tissues (Table 6). The relatively high concentrations appeared to be a result of natural processes rather than of anthropogenic activities, however, and probably did not represent a significant risk to pinniped health. In general, Hg concentrations increased significantly with increasing age of the organism, as shown (Figure 2) in the livers of harbor seal (*Phoca groenlandica*), grey seal (*Halichoerus grypus*), California sea lion (*Zalophus californianus*), and northern fur seal (*Callorhinus ursinus*). The mechanisms to account for this phenomenon in pinnipeds are similar to those reported by Itano et al. (1984 a,b, c) for the striped dolphin (*Stenella coeruleoalba*). They showed that tissue concentrations of mercury in striped dolphins increased with increasing age of the animal, reaching a plateau in 20 to 25 years; were highest in liver, although muscle accounted for about 90% of the total body Hg burden; were present in the methylated form in fetal and suckling stages, but the proportion of methylmercury decreased over time with no absolute increase after age 10 years; were excreted slowly by all developmental stages, and slowest in older dolphins (resulting in higher accumulations); and were correlated strongly with selenium concentrations in all age groups. It is probable that inorganic Hg and selenium were complexed in a 1:1 molar ratio, in a form biologically unavailable to marine mammals (and probably other mammals), thereby significantly decreasing the risk of mercury toxicosis to individuals with grossly elevated Hg body burdens (Eisler 1984, 1985). Large colonies of pinnipeds and, to a lesser extent, marine birds along the western coast of the United States may make Hg available to mussels (*Mytilus californianus*) through fecal elimination of large amounts of Hg, resulting in abnormally high Hg levels in mussels from several west coast sites (Flegal et al. 1981).

Among furbearers in the Wisconsin River drainage system, Hg burdens were higher in fish-eating than in herbivorous species--i.e., river otter (*Lutra canadensis*) > mink (*Mustela vison*) > raccoon (*Procyon lotor*) > red fox (*Vulpes fulva*) > muskrat (*Ondatra zibethicus*) > beaver (*Castor canadensis*) (Sheffy and St. Amant 1982). In general, fur contained the highest Hg levels, followed by liver, kidney, muscle, and brain, in that order (Table 6; Sheffy and St. Amant 1982). Mercury levels in piscivorous furbearers collected from the Wisconsin River basin paralleled Hg levels in fish, crayfish, and bottom sediments from that system; levels in all compartments were highest about 30 km downstream from an area that supported 16 pulp and paper mills and a chloralkali plant (Sheffy and St. Amant 1982). Mink and river otter accumulated about 10X more Hg than did predatory fishes from the same drainage areas--suggesting that these furbearers can serve as sensitive indicators of mercury, even at very low levels of Hg contamination (Kucera 1983).

Domestic sheep (*Ovis* sp.) allowed to graze for 23 months on grass contaminated with Hg (up to 6.5 mg/kg dry weight) caused by atmospheric emissions of a nearby chloralkali site, retained about 0.1% of the total Hg taken in by ingestion and inhalation, although residues in flesh were negligible (Edwards and Pumphery 1982). It was concluded that contamination of grass as a result of atmospheric discharges of inorganic He from

chloralkali sites causes no hazard, either directly to grazing animals or indirectly to humans who might ultimately consume their flesh.

ACUTE AND CHRONIC TOXICITY

GENERAL

For all organisms tested, early developmental stages were the most sensitive, and organomercury compounds--especially methylmercury--were more toxic than inorganic forms. Numerous biological and abiotic factors modify the toxicity of Hg compounds, sometimes by an order of magnitude or more, but the mechanisms of action are not clear.

Lethal concentrations of total Hg to sensitive, representative organisms varied from 0.1 to 2.0 ug/l of medium for aquatic fauna; from 2.2 to 31.0 mg/kg body weight (acute oral) and 4.0 to 40.0 mg/kg (dietary) for birds; and from 0.1 to 0.5 mg/kg body weight (daily dose) and 1.0 to 5.0 mg/kg (dietary) for mammals.

AQUATIC ORGANISMS

Toxic concentrations of mercury salts ranged from less than 0.1 ug/l to more than 200 ug/l for representative species of marine and freshwater organisms (Table 7). The lower concentrations of <2.0 ug/l recorded were usually associated with early developmental stages, long exposures, and flowthrough tests (Table 7). Among metals tested, mercury was the most toxic to aquatic organisms, and organomercury compounds showed the greatest biocidal potential (Eisler 1981). In general, toxicity was higher at elevated temperatures (Armstrong 1979), at reduced salinities in marine organisms (McKenney and Costlow 1981), and in the presence of other metals such as zinc and lead (Parker 1979).

Signs of acute mercury poisoning in fish included flaring of gill covers, increased frequency of respiratory movements, loss of equilibrium, and sluggishness (Armstrong 1979). Signs of chronic mercury poisoning included emaciation (due to appetite loss), brain lesions, cataracts, diminished response to change in light intensity, inability to capture food, abnormal motor coordination, and various erratic behaviors (Armstrong 1979; Hawryshyn et al. 1982). Mercury residues in severely poisoned fish that died soon thereafter ranged (in mg/kg fresh weight) from 26 to 68 in liver, 16 to 20 in brain, and 5 to 7 in whole body (Armstrong 1979).

Table 7. Toxicities of inorganic and organic mercury compounds to selected species of aquatic organisms.

Chemical species, ecosystem, taxonomic group, species, and other variables	Effect[a]	Concentration (µg Hg/L medium)	Reference
Inorganic Mercury			
Freshwater			
Crustaceans			
Crayfish, *Orconectes limosus*	LC-50 (30 d)	2.0	EPA 1980
Cladoceran, *Daphnia magna*	LC-50 (96 h)	5.0	EPA 1980
Cladoceran, *Daphnia magna*	LC-50 (LT)	1.3–1.8	EPA 1980
Scud, *Gammarus pseudolimnaeus*	LC-50 (96 h)	10.0	EPA 1980
Fish			
Rainbow trout, *Salmo gairdneri*			
Juveniles	LC-50 (96 h)	155.0–200.0	EPA 1980
Embryo-larva			
Static test	LC-50 (28 d)	4.7	Birge et al. 1979
Flowthrough test	LC-50 (28 d)	<0.1	Birge et al. 1979
Channel catfish, *Ictalurus punctatus*			
Embryo-larva			

Static test	LC-50 (10 d)	30.0	Birge et al. 1979
Flowthrough test	LC-50 (10 d)	0.3	Birge et al. 1979
Largemouth bass, *Micropterus salmoides*			
Embryo-larva			
Static test	LC-50 (8 d)	140.0	Birge et al. 1979
Flowthrough test	LC-50 (8 d)	5.3	Birge et al. 1979
Brook trout, *Salvelinus fontinalis*	LC-50 (LT)	0.3–0.9	EPA 1980
Fish, *Notopterus notopterus*	LC-50 (96 h)	440.0	Verma and Tonk 1983
Amphibians			
Narrow-mouthed toad, *Gastrophryne carolinensis*			
Embryo-larva	LC-50 (96 h)	1.3	Birge et al. 1979
Treefrogs, *Hyla* spp.			
Embryo-larva, 5 spp.	LC-50 (96 h)	2.4–2.8	Birge et al. 1979
Leopard frog, *Rana pipiens*			
Embryo-larva	LC-50 (96 h)	7.3	Birge et al. 1979
Cricket frog, *Acris* sp.			
Embryo-larva	LC-50 (96 h)	10.4	Birge et al. 1979
Anurans, 4 spp.			
Embryo-larva	LC-50 (96 h)	36.8–67.2	Birge et al. 1979
Marbled salamander, *Ambystoma opacum*			
Embryo-larva	LC-50 (96 h)	107.5	Birge et al. 1979
Marine			
Protozoans			
Ciliate, *Uronema marinum*	LC-50 (24 h)	6.0	Parker 1979
Molluscs			
Softshell clam, *Mya arenaria*	LC-50 (168 h)	4.0	Eisler and Hennekey 197
Hardshell clam, *Mercenaria mercenaria*			
Larva	LC-50 (48 h)	4.8	EPA 1980
Larva	LC-5 (9 d)	4.0	EPA 1980
American oyster, *Crassostrea virginica*			
Embryo	LC-5 (12 d)	3.3	EPA 1980
Larva	LC-50 (48 h)	5.6	EPA 1980
Adults	LC-50 (48 h)	5.5–10.2	EPA 1980
Blue mussel, *Mytilus edulis*	LC-50 (96 h)	5.8	EPA 1985
Slipper limpet, *Crepidula fornicata*			
Larva	LC-50 (96 h)	60.0	Thain 1984
Adults	LC-50 (96 h)	330.0	Thain 1984
Bay scallop, *Argopecten irradians*			
Juveniles	LC-50 (96 h)	89.0	Nelson et al. 1976
Crustaceans			

Fiddler crab, *Uca pugilator*			
Zoea	LC-50 (8 d)	1.8	EPA 1980
Mysid shrimp, *Mysidopsis bahia*			
Juveniles	LC-50 (96 h)	3.5	Gentile et al. 1983
Egg to egg	LC-50 (LT)	1.8	Gentile et al. 1983
Dungeness crab, *Cancer magister*			
Larva	LC-50 (96 h)	6.6	Glickstein 1978
Copepod, *Acartia tonsa*, adult	LC-50 (96 h)	10.0–15.0	EPA 1980
Prawn, *Penaeus indicus*			
Postlarva	LC-50 (48 h)	16.1	McClurg 1984
Postlarva	LC-50 (96 h)	15.3	McClurg 1984
Annelids			
Polychaete, *Capitella capitata*			
Larva	LC-50 (96 h)	14.0	EPA 1980
Fish			
Haddock, *Melanogrammus aeglifinus*			
Larvae	LC-50 (96 h)	98.0	EPA 1980
Organic Mercury			
Freshwater			
Planarians			
Flatworm, *Dugesia dorotocephala*			
Adult	LC-0 (10 d)	200.0	Best et al. 1981
Adult	LC-100 (5 d)	500.0	Best et al. 1981
Crustaceans			
Cladoceran, *Daphnia magna*	LC-50 (LT)	0.9–3.2	EPA 1980
Fish			
Rainbow trout			
Larva	LC-50 (96 h)	24.0	EPA 1980
Juvenile	LC-50 (96 h)	5.0–42.0	EPA 1980
Brook trout			
Yearling	LC-50 (96 h)	65.0	EPA 1980
Marine			
Crustaceans			
Amphipod, *Gammarus duebeni*	LC-50 (96 h)	150.0	EPA 1980

[a]Abbreviations: LT = lifetime exposure; h = hours; d = days.

BIRDS

Signs of mercury poisoning in birds included muscular incoordination, falling, slowness, fluffed feathers, calmness, withdrawal, hyporeactivity, hypoactivity, and eyelid drooping. In acute oral exposures, signs appeared as soon as 20 minutes postadministration in mallards and 2.5 hours in pheasants. Deaths occurred between 4 and 48 hours in mallards and 2 and 6 days in pheasants; remission took up to 7 days (Hudson et al. 1984). In studies with coturnix (*Coturnix coturnix coturnix*), (Hill 1981) found that methylmercury was always more toxic than inorganic mercury, and that young birds were usually more sensitive than older birds. Furthermore, some birds poisoned by inorganic mercury recovered after treatment was withdrawn, but chicks that were fed

methylmercury and later developed toxic signs usually died, even if treated feed was removed. Coturnix subjected to inorganic mercury, regardless of route of administration, showed a violent neurological dysfunction that ended in death 2 to 6 hours posttreatment. The withdrawal syndrome in coturnix poisoned by Hg 2^+ was usually preceded by intermittent, nearly undetectable tremors, coupled with aggressiveness towards cohorts; time from onset to remission was usually 3 to 5 days, but sometimes extended to 7 days. Coturnix poisoned by methylmercury appeared normal until 2 to 5 days posttreatment; then, ataxia and low body carriage with outstretched neck were often associated with walking. In advanced stages, coturnix lost locomotor coordination and did not recover; in mild to moderate clinical signs, recovery usually took at least 1 week (Hill 1981).

Mercury toxicity to birds varies with the form of the element, dose, route of administration, species, sex, age, and physiological condition (.Fimreite 1979). For example, in northern bobwhite chicks fed diets containing methylmercury chloride, mortality was significantly lower when the solvent was acetone than when it was another carrier such as propylene glycol or corn oil (Spann et al. 1986). In addition, organomercury compounds interact with elevated temperatures and pesticides, such as DDE and parathion, to produce additive or more-than-additive toxicity, and with selenium to produce less-than-additive toxicity (*Fimreite* 1979). Acute oral toxicities of various mercury formulations ranged between 2.2 and about 31.0 mg/kg body weight for most avian species tested (Table 8). Similar data for other routes of administration were 4.0 to 40.0 mg/kg for diet and 8.0 to 15.0 mg/kg body weight for intramuscular injection (Table 8).

Residues of mercury in experimentally poisoned passerine birds usually exceeded 20 mg/kg fresh weight, and were similar to concentrations reported in wild birds that died of mercury poisoning (Finley et al. 1979). Mercury levels in tissues of poisoned wild birds were highest (45 to 126 mg/kg fresh weight) in red-winged blackbirds (*Agelaius phoeniceus*), intermediate in starlings (*Sturnus vulgaris*) and cowbirds (*Molothrus ater*), and lowest (21 to 54) in grackles (*Quiscalus quiscula*); in general, Hg residues were highest in the brain, followed by the liver, kidney, muscle, and carcass. Some avian species are more sensitive than passerines (Solonen and Lodenius 1984): liver residues (in mg Hg/kg dry weight) in birds experimentally killed by methylmercury ranged from 17 in red-tailed hawks (*Buto jamaicensis*) to 70 in jackdaws (*Corvus monedula*); and values were intermediate in ring-necked pheasants, kestrels (*Falco tinnunculus*), and magpies (*Pica pica*). Experimentally poisoned grey herons (*Ardea cinerea*) seemed to be unusually resistant to Hg; lethal doses produced residues of 415 to 752 mg Hg/kg dry weight of liver (Van der Molen et al. 1982). However, levels of this magnitude were frequently encountered in livers from grey herons collected during a massive die-off in the Netherlands during a cold spell in 1976; the interaction effects of cold stress, mercury loading, and poor physical condition of the herons are unknown ((*Ardea cinerea*) in the Netherlands. Ardea 70:173-184Van der Molen et al. 1982).

Table 8. Toxicity to birds of mercury administered by oral, dietary, or other routes.

Route of administration (units), organism, and mercury formulation	Concentration	Exposure interval	Effect	Reference[a]
Acute oral (mg Hg/kg body weight)				
Chukar, *Alectoris chukar*				
Ethyl	26.9	Within 14 d posttreatment	LD-50	Hudson et al. 1984
Mallard, *Anas platyrhynchos*				
Methyl	2.2–23.5	"	LD-50	
Ethyl	75.7	"	LD-50	
Phenyl	524.7	"	LD-50	
Northern bobwhite, *Colinus virginianus*				

Methyl	23.8	"	LD-50	
Coturnix, *Coturnix coturnix coturnix*				
Methyl	11.0–27.0	"	LD-50	Hill 1981
Inorganic	26.0–54.0	"	LD-50	
Japanese quail, *Coturnix japonica*				
Methyl	14.4–33.7	"	LD-50	Hill and Soares 1984; Hudson et al. 1984
Ethyl	21.4	"	LD-50	Hudson et al. 1984
Inorganic	31.1	"	LD-50	Hill and Soares 1984
Rock dove, *Columba livia*				
Ethyl	22.8	"	LD-50	Hudson et al. 1984
Fulvous whistling duck, *Dendrocygna bicolor*				
Methyl	37.8	"	LD-50	
Domestic chicken, *Gallus domesticus*				
Phenyl	60.0	"	LD-50	Mullins et al. 1977
House sparrow, *Passer domesticus*				
Methyl	12.6–37.8	Within 14 d posttreatment	LD-50	Hudson et al. 1984
Gray partridge, *Perdix perdix*				
Ethyl	17.6	"	LD-50	
Ring-necked pheasant, *Phasianus colchicus*				
Ethyl	11.5	"	LD-50	
Methyl	11.5–26.8	"	LD-50	
Phenyl	65.0–101.0	"	LD-50	Mullins et al. 1977; Hudson et al. 1984
Prairie chicken, *Tympanuchus cupido*				
Ethyl	11.5	"	LD-50	Hudson et al. 1984

Dietary (mg Hg/kg diet)				
Coturnix				
Inorganic	32.0	Hatch-9 weeks	LD-0	Hill 1981
Inorganic	2,956–5,086	5 d + 7 d pt	LD-50	
Methyl	4.0	Hatch-9 weeks	LD-0	
Methyl	8.0	5 d	Some deaths	Hill and Soares 1984
Methyl	31.0–47.0	5 d + 7 d pt	LD-50	Hill 1981
Ring-necked pheasant				
Ethyl	4.2	70 d	LD-0[b]	Spann
Ethyl	12.5	70 d	LD-50	et al. 1972
Ethyl	37.4	28 d	LD-50	
Ethyl	112.0	15 d	LD-50	
Japanese quail				
Inorganic				
In dry salt	500	28 d	LD-86	El-Begearmi
In ethanol, methanol, or water	500	28 d	LD-55	et al. 1980
In casein premix	500	28 d	LD-33	
Birds, 4 spp.				
Methyl	40.0	6 to 11 d	LD-33	Finley et al. 1979
Intramuscular injection (mg Hg/kg body weight)				
Coturnix				
Methyl	8.0–33.0	Single dose	LD-50	Hill 1981
Inorganic	15.0–50.0	Single dose	LD-50	
Rock dove				
Inorganic	10.0	Daily, 17 d	Some deaths	Leander et al. 1977
Yolk sac injection (ug Hg/egg)				
Chicken				
Methyl	15.0	Single dose	Some deaths	Greener and Kochen 1983
Methyl	40.0-50.0	Single dose	LD-50	
Applied to Egg Surface ug Hg/egg				
Mallard				
Methyl	3.0	Single dose	LD-0	Hoffman and
Methyl	9.0	Single dose	Some deaths	Moore 1979

In drinking water (mg Hg/L)				
Chicken				
Inorganic	500.0	3 d	Some deaths	Grissom and Thaxton 1985

[a]Reference cited applies to data in the same row and in the rows that immediately follow for which no reference is indicated.

[b]Reduction in egg production, 55% to 80%; embryonic survival sharply reduced.

Table 9. Toxicity of organomercury compounds to selected mammalian species.

Organism	Dose, route of administration, and other variables	Effects	Reference
Dog, *Canis familiaris*	0.1 to 0.25 mg/kg body weight during entire pregnancy; oral route	High incidence of stillbirths	Khera 1979
Cat, *Felis domesticus*	0.25 mg/kg body weight daily for 90 days (total 80–90 mg Hg); dietary route	Mean survival time 78 days. Convulsions starting at day 68; all with signs by day 90. Liver residues of survivors were 40.2 and 18.1 mg/kg fresh weight for total mercury and inorganic mercury, respectively	Eaton et al. 1980
Pig, *Sus* spp.	0.5 mg/kg body weight during pregnancy; oral route	High incidence of stillbirths	Khera 1979
Rhesus monkey, *Macaca mulatta*	0.5 mg/kg body weight during days 20–30 of pregnancy	Maternally toxic, and abortient	Khera 1979
Mink, *Mustela vison*	1.0 mg/kg in diet	Fatal to 100% in about 2 months 2 months	Sheffy and St. Amant 1982
River otter,	>2.0 mg/kg in diet	Fatal	Kucera 1983

Lutra canadensis			
Mink	5.0 mg/kg in diet	All dead in 30 to 37 days. Elevated residues in kidney (37.7 mg/kg fresh weight) and liver (55.6) prior to death	Sheffy and St. Amant 1982
Humans	Various	Lethal residues in tissues, in mg/kg fresh weight, were >6.0 in brain, >10.0 in liver, and >17.0 in whole body	Khera 1979
Mule deer, *Odocoileus hemionus hemionus*	17.88 mg/kg body weight; single oral dose	LD-50	Hudson et al. 1984
Harp seal, *Pagophilus groenlandicus*	25.0 mg/kg body weight daily; oral route	Dead in 20 to 26 days. Blood Hg levels just before death were 26.8 to 30.3 mg/L	Ronald et al. 1977

MAMMALS

Methylmercury affects the central nervous system in man--especially the sensory, visual, and auditory areas concerned with coordination; the most severe effects lead to widespread brain damage, resulting in mental derangement, coma, and death (Clarkson and Marsh 1982). In mule deer (*Odocoileus hemionus hemionus*), after acute oral Hg poisoning was induced experimentally, additional signs included belching, bloody diarrhea, piloerection (hair more erect than usual), and loss of appetite (Hudson et al. 1984). The kidney is the probable critical organ in adult mammals due to the rapid degradation of phenylmercurials and methoxyethylmercurials to inorganic Hg compounds and subsequent translocation to the kidney (Suzuki 1979), whereas in the fetus the brain is the principal target (Khera 1979). Most human poisonings were associated with organomercury compounds used in agriculture as fungicides to protect cereal seed grain (Elhassani 1983); judging from anecdotal evidence, many wildlife species may have been similarly afflicted. Organomercury compounds, especially methylmercury, were the most toxic mercury species tested. Among sensitive species of mammals, death occurred at daily organomercury concentrations of 0.1 to 0.5 mg/kg body weight, or 1.0 to 5.0 mg/kg in the diet (Table 9). Larger animals such as mule deer and harp seals appear to be more resistant to Hg than smaller mammals such as mink, cats, dogs, pigs, monkeys, and river otters; the reasons for this difference are unknown, but may be related to differences in metabolism and detoxication rates. Tissue residues in fatally poisoned mammals (in mg Hg/kg fresh weight) were 6.0 in brain, 10.0 to 55.6 in liver, 17.0 in whole body, about 30.0 in blood, and 37.7 in kidney (Table 9).

OTHER GROUPS

Methylmercury compounds at concentrations of 25.0 mg Hg/kg in soil were fatal to all tiger worms (*Eisenia foetida*) in 12 weeks; at 5.0 mg/kg, however, only 21% died in a similar period (Beyer et al. 1985). Inorganic mercury compounds were also toxic to earthworms (*Octochaetus pattoni*); in 60 days, 50% died at soil Hg levels of 0.79 mg/kg, and all died at 5.0 mg/kg (Abbasi and Soni 1983).

SUBLETHAL EFFECTS

GENERAL

Mercury is a known mutagen, teratogen, and carcinogen. At comparatively low concentrations in birds and mammals, it adversely affects reproduction, growth and development, behavior, blood and serum chemistry, motor coordination, vision, hearing, histology, and metabolism. It has a high potential for bioaccumulation and biomagnification, and is slow to depurate. Organomercury compounds were more effective in producing adverse effects than were inorganic Hg compounds; however, effects were significantly enhanced or ameliorated by numerous biotic and nonbiological modifiers. For sensitive aquatic species, adverse effects were observed at water concentrations of 0.03 to 0.1 ug Hg/l. For sensitive species of birds, harmful levels were 640 ug Hg/kg body weight daily, or 50 to 500 ug Hg/kg in the diet; for sensitive mammals, these levels were 250 ug Hg/kg body weight daily, or 1,100 ug Hg/kg diet.

AQUATIC ORGANISMS

Mercury at comparatively low concentrations adversely affects the reproduction, growth, behavior, metabolism, blood chemistry, osmoregulation, and oxygen exchange of marine and freshwater organisms. In general, the accumulation of mercury by aquatic biota is rapid, and depuration is slow. It is emphasized that organomercury compounds, especially methylmercury, were significantly more effective than inorganic mercury compounds in producing adverse effects and accumulations.

Reproduction was inhibited among sensitive species of aquatic organisms at water concentrations of 0.03 to 1.6 ug Hg/l. In the planarian (Best et al. 1981); in the slipper limpet (*Crepidula fornicata*), spawning was delayed and fecundity was decreased at 0.25 ug Hg^{2+}/l (Thain 1984); in the zebrafish (*Brachydanio rerio*), hatching success was reduced at 0.1 ug Hg^{2+}/l and egg deposition was reduced at 0.8 ug/l (Armstrong 1979); fathead minnows (*Pimephales promelas*) exposed to 0.12 ug methylmercury/l for 3 months failed to reproduce (Birge et al. 1979); the leopard frog (*Rana pipiens*) did not metamorphose during exposure to 1.0 ug methylmercury/l for 4 months (EPA 1980); and in the mysid shrimp (*Mysidopsis bahia*), the abortion rate increased and population size decreased after lifetime (i.e., 28 days) exposure to 1.6 ug/l of mercury as mercuric chloride (Gentile et al. 1983). For sensitive marine invertebrates such as hydroids, protozoans, and mysid shrimp, reproduction was inhibited at concentrations between 1.1 and 2.5 ug Hg^{2+}/l; this range was 5 to 71 ug/l for more resistant species of marine invertebrates (Gentile et al. 1983).

Reduced growth of sensitive species of aquatic organisms has been recorded at water concentrations of 0.04 to 1.0 ug Hg/l. The rainbow trout (*Salmo gairdneri*) was the most sensitive species tested; growth reduction was observed after 64 days in 0.04 ug Hg/l as methylmercury, or 0.11 ug Hg/l as phenylmercury (EPA 1980). In adults of the marine mollusc *Crepidula fornicata*, growth was reduced after 16 weeks in 0.25 ug Hg^{2+}/1 (Thain 1984). Growth inhibition was recorded in freshwater algae after exposure of 24 hours to 10 days to 0.3 to 0.6 ug organomercury/l, in brook trout (*Salvelinus fontinalis*) alevins after exposure for 21 days to 0.79 ug organomercury/l (EPA 1980), and in the marine alga *Scripsiella faeroense* exposed to 1.0 ug Hg^{2+}/l for 24 hours (Kayser 1976).

Adverse effects of mercury to aquatic organisms, in addition to those listed on reproduction and growth, have been documented at water concentrations of 0.88 to 5.0 ug/l: enzyme disruption in brook trout (*Salvelinus fontinalis*) embryos immersed for 17 days in solutions containing 0.88 ug/l, as methylmercury (EPA 1980); an increased incidence of frustule abnormalities and burst thecae in two species of marine algae during exposure to 1.0 ug Hg^{2+}/l for 24 hours (Kayser 1976; Saboski 1977); arrested development of sea urchin larvae at 3.0 ug Hg^{2+}/l for 40 hours (EPA 1980); decreased rate of intestinal transport of glucose, fructose, glycine, and

tryptophan in the murrel (*Channa punctatus*) at 3.0 ug Hg^{2+}/l for 30 days (Sastry et al. 1982); altered blood chemistry in striped bass (*Morone saxatilis*) at 5.0 ug Hg^{2+}/l in 60 days (Dawson 1982); and decreased respiration in striped bass 30 days postexposure after immersion for 30 to 120 days in 5.0 ug Hg^{2+}/l (Armstrong 1979; EPA 1980). In marine molluscs exposed to water concentrations of 6 to 10 ug Hg^{2+}/l for 96 hours, the feeding of adults ceased and the swimming rate of larval stages declined (Thain 1984). At 44 ug Hg^{2+}/l for 30 days, the freshwater fish *Notopterus notopterus* showed generalized metabolic derangement (Verma and Tonk 1983). In freshwater planarians exposed to 80 to 100 ug/l as methylmercury, behavior was modified and regeneration retarded (Best et al. 1981). And at high sublethal concentrations of methylmercury, rainbow trout were listless and darkly pigmented; appetite was reduced, and digestion was poor (Rodgers and Beamish 1982).

At lower trophic levels, the efficiency of mercury transfer was low through natural aquatic food chains; however, in animals of higher trophic levels, such as predatory teleosts and fish-eating birds and mammals, the transfer was markedly amplified (Eisler 1978, 1981).

High uptake and accumulation of mercury from the medium by representative species of marine and freshwater teleosts and invertebrates have been documented (Kopfler 1974; Eisler 1978, 1981; Birge et al. 1979; Huckabee et al. 1979; EPA 1980, EPA. 1985; Stokes et al. 1981; Rodgers and Beamish 1982; Hirota et al. 1983; Clarkson et al. 1984; McClurg 1984; Niimi and Lowe-Jinde 1984; Ramamoorthy and Blumhagen 1984; Ribeyre and Boudou 1984; Thain 1984). Accumulation patterns were enhanced or significantly modified by numerous biological and abiotic factors (NAS 1978; Eisler 1978, 1981, 1984, 1985; EPA 1980, 1985; Stokes et al. 1981;Rodgers and Beamish 1982; Clarkson et al. 1984; Ramamoorthy and Blumhagen 1984; Ribeyre and Boudou 1984). In general, the accumulation of mercury was markedly enhanced at elevated water temperatures, reduced water salinity or hardness, reduced water pH, increased age of the organism, and reduced organic matter content of the medium; in the presence of zinc, cadmium, or selenium in solution; after increased duration. of exposure; and in the presence of increased nominal concentrations of protein-bound mercury. Uptake patterns were significantly modified by sex, sexual condition, prior history of exposure to Hg salts, the presence of complexing and chelating agents in solution, dietary composition, feeding niche, tissue specificity, and metabolism; however, trends were not consistent between species and it is difficult to generalize. In one example, Ribeyre and Boudou (1984) immersed rainbow trout in solutions containing 0.1 ug Hg/l, as methylmercury: after 30 days, bioconcentration factors (BCF) ranged from 28,300 for brain to 238,000 for spleen; values were intermediate for muscle (30,000), whole fish (36,000), blood (102,000), liver (110,000), kidney (137,000), and gill (163,000). The values may have been higher if exposure had extended beyond 30 days; Rodgers and Beamish (1982) showed that whole body Hg residues in rainbow trout subjected to mercury insult continued to increase for the first 66 days before stabilizing. When mercury was presented as inorganic mercuric ion at 0.1 ug/l for 30 days, BCF values were usually lower than in trout exposed to methylmercury: 2,300 for muscle; 6,800 for brain; 7,000 for whole trout; 14,300 for blood; 25,000 for liver; 53,000 for kidney; 68,600 for gill; and 521,000 for spleen (Ribeyre and Boudou 1984). The high BCF values recorded for rainbow trout were probably due to efficient uptake from water, coupled with slow depuration (Rodgers and Beamish 1982). Whole body levels up to 100 mg Hg/kg were reportedly not lethal to rainbow trout, although 20 to 30 mg/kg were associated with reduced appetite, loss of equilibrium, and hyperplasia of gill epithelium (Niimi and Lowe-Jinde 1984). However, brook trout showed toxic signs and death at whole body residues of only 5 to 7 mg/kg (Armstrong 1979). In another example, the marine copepod *Acartia clausi*, subjected to 0.05 ug/l of mercury and higher, reached equilibrium with the medium in only 24 hours. In that study (Hirota et al. 1983), BCF values for whole *Acartia* after 24-hour exposures were 14,360 for inorganic mercuric ion (0.05 ug/l) and, for methylmercury, 179,200 (0.05 ug/l) and 181,000 (0.1 ug/l).

Elimination of accumulated mercury, both organic and inorganic, from aquatic organisms is a complex multicompartmental process, but appears to be largely dependent on its rate of biological assimilation. This rate, in turn, varies widely (20% to 90%) between species, for reasons as yet unexplained (NAS 1978). For example, mercury associated with dietary components that are not assimilated is eliminated rapidly with feces. The rest is absorbed across the gut and incorporated into tissues. This assimilated fraction of mercury is depurated much more slowly, at a rate positively correlated with the organism's metabolism (NAS 1978; Rodgers and Beamish 1982). Time to eliminate 50% of biologically assimilated mercury and its compounds (Tb 1/2) is variable. Among various species of freshwater teleosts, Tb 1/2 values (in days) were 20 for guppies

Poecilia reticulatus, 23 for goldfish *Carassius auratus*, 100 for northern pike, and 1,000 each for mosquitofish *Gambusia affinis*, brook trout, and rainbow trout (Huckabee et al. 1979). similar range in. Tb 1/2 values was recorded for invertebrates and marine fishes: 297 days for the crayfish *Astacus fluviatilis*, 435 days for mussel, 481 days for the clam *Tapes decussatus* 1,030 days for the eel *Anguilla vulgaris*, and 1,200 days for the flounder *Pleuronectes flesus* (NAS 1978).

Mercury-tolerant strains of bacteria (Colwell et al. 1976), protozoa (Berk et al. 1978), crustaceans (Green et al. 1976; Weis 1976), and fish (Weis 1984) have been reported. It has been suggested that the mercury-resistant strains of bacteria that have been cultured or discovered may have application in mobilization or fixation of mercury from contaminated aquatic environments to the extent that polluted areas may become innocuous (Colwell et al. 1976). The marine protozoan *Uronemia nigricans*, after feeding on Hg-laden bacteria, acquired mercury tolerance within a single generation (Berk et al. 1978). The white shrimp (*Penaeus setiferus*), preexposed for 57 days to 1 ug Hg/l, did not differ from controls during either exposure or subsequent Hg stress experiments (Green et al. 1976); this observation suggested that nonsensitization or adaptation mechanisms are involved. The fiddler crab (*Uca pugilator*) seemed unusually resistant and showed negligible uptake or effects during exposure to 100 ug Hg/l for 2 weeks (Weis 1976). Reasons to account for Hg adaptation of the estuarine cyprinodontiform teleost *Fundulus heteroclitus* to both methylmercury and inorganic mercury are under investigation (Weis 1984).

BIRDS

Sublethal effects of mercury on birds, administered by a variety of routes, included adverse effects on growth, development, reproduction, blood and tissue chemistry, metabolism, and behavior; histopathology and bioaccumulation were also noted.

The dietary route of administration is the most extensively studied pathway of avian Hg intake. Domestic chickens fed diets containing as little as 50 ug/kg of mercury, as methylmercury, contained elevated total Hg (2.0 mg/kg fresh weight) residues in liver and kidney after 28 weeks; at 150 ug/kg, residues ranged from 1.3 to 3.7 mg/kg in heart, muscle, brain, kidney, and liver, in that general order; at 450 ug/kg in diets, residues in edible chicken tissues (3.3 to 8.2 mg/kg) were considered hazardous to human consumers, although no overt signs of mercury toxicosis were observed in the chickens (March et al. 1983). High inorganic mercury levels (500 mg/l) in drinking water of chickens decreased growth rate and food and water consumption, and elevated hemoglobin, hematocrit, and erythrocyte content within 3 days (Grissom and Thaxton 1985). The dietary concentration of 0.5 mg Hg/kg dry weight (equivalent to about 0.1 mg/kg fresh weight) in the form of methylmercury was fed to three generations of mallards (Heinz 1979). Females laid a greater percentage of their eggs outside nest boxes than did controls, and also laid fewer eggs and produced fewer ducklings. Ducklings from parents fed methylmercury were less responsive than controls to tape-recorded maternal calls, but were hyperresponsive to a fright stimulus in avoidance tests. The tissues and eggs of ducks and other species of birds collected in the wild have sometimes contained levels of mercury equal to, or far exceeding, those associated with reproductive and behavioral deficiencies in domestic mallards (e.g., 9 to 11 mg/kg in feathers; >2.0 mg/kg in other tissues); therefore, it is possible that reproduction and behavior of wild birds have been modified by methylmercury contamination (Heinz 1979). Tissue mercury residues of wild-strain mallards and game-farm mallards were not significantly different after the birds were fed diets containing 0.5 mg Hg/kg as methylmercury for extended periods--indicating that game-farm mallards are suitable substitutes for wild mallards in toxicological evaluations (Heinz 1980). Dietary concentrations of 1.1 mg total Hg/kg have been associated with kidney lesions in juvenile starlings (*Sturnus vulgaris*) and with elevated residues in the liver (6.5 mg/kg dry weight and kidney (36.3 mg/kg), after exposure for 8 >weeks (Nicholson and Osborn 1984). In American black ducks (*Anas rubripes*) fed diets containing 3.0 mg Hg/kg as methylmercury for 28 weeks, reproduction was significantly inhibited; tissue residues were elevated in kidney (16.0 mg/kg fresh weight) and liver (23.0 mg/kg); and brain lesions characteristic of mercury poisoning were present (Finley and Stendell 1978). Japanese quail (*Coturnix japonica*) fed diets containing 8 mg Hg/kg of inorganic mercury for 3 weeks had depressed gonad weights; those fed 3 mg/kg inorganic mercury or 1 mg/kg methylmercury for 9 weeks showed alterations in brain and plasma enzyme activities (Hill and Soares 1984). Grossly elevated tissue residues of 400 mg/kg in feathers and 17 to 130 mg/kg in other tissues were measured in gray partridge (*Perdix perdix*) after dietary exposure of 20 to 25 mg total Hg/kg for 4 weeks (McEwen et al. 1973).

Mercury exposure by immersion and oral administration have caused reproductive and behavorial modifications. Brief immersion of mallard eggs in solutions of methylmercury resulted in a significant incidence of skeletal embryonic aberrations at dosages of 1.0 ug Hg/egg, and higher; no increases in embryonic malformations were noted at 0.3 ug Hg/egg (Hoffman and Moore 1979). Reduced reproductive ability was noted in grey pheasants ingesting 640 ug Hg (as organomercury)/kg body weight daily for 30 days (McEwen et al. 1973); similar results were observed in ring-necked pheasants (Spann et al. 1972; Mullins et al. 1977). Behavioral alterations were noted in pigeons (*Columba livia*) given 3,000 ug inorganic Hg/kg body daily for 17 days (Leander et al. 1977) or 1,000 ug/kg body weight of methylmercury for 5 weeks (Evans et al. 1982). Observed behavioral changes in posture and motor coordination of pigeons were permanent after the brain accumulated >12,000 ug Hg/kg fresh weight, and were similar to the "spastic paralysis" observed in wild crows during the Minamata, Japan, outbreak of the 1950's, although both species survived for years with these signs (Evans et al. 1982).

Mercury residues of 790 to 2,000 ug/kg in egg, and 5,000 to 40,000 ug/kg in feathers, are linked to impaired reproduction in various bird species (Spann et al. 1972; NAS 1978; Heinz 1979; Fimreite 1979; Solonen and Lodenius 1984). Residues in eggs of 1,300 to 2,000 ug Hg/kg fresh weight were associated with reduced hatching success in white-tailed sea-eagles (*Haliaeetus albicilla*), the common loon (*Gavia immer*), and in several seed-eating species (Fimreite 1979); this range was 900 to 3,100 ug/kg for ring-necked pheasant (Spann et al. 1972), and 790 to 860 ug/kg for mallards (Heinz 1979). Residues of 5,000 to 11,000 ug Hg/kg in feathers of various species of birds have been associated with reduced hatch of eggs and with sterility (NAS 1978). Sterility was observed in the Finnish sparrow hawk (*Accipiter nisus*) at mercury concentrations of 40,000 ug/kg in feathers (Solonen and Lodenius 1984). Chicks of the common tern (*Sterna hirundo*) from a colony in Long Island, New York, with abnormal feather loss, had significantly elevated mercury levels in blood and liver (Gochfeld 1980); however, the linkage of feather loss to mercury contamination requires further examination.

Interaction effects of mercury with other contaminants, such as herbicides and pesticides, could intensify hazards to avian populations (Mullins et al. 1977). For example, a striking parallel exists between levels of Hg and of DDT and its metabolites in birds of prey, suggesting the existence of common ecotoxicological mechanisms (Delbeke et al. 1984; Wiemeyer et al. 1984); additional research is clearly needed.

MAMMALS

Mercury has no known physiological function (EPA 1985). In humans and other mammals, it causes teratogenic, mutagenic, and carcinogenic effects; the fetus is the most sensitive life stage (NAS 1978; Chang 1979; Khera 1979; EPA 1980, 1985; Elhassani 1983; Greener and Kochen 1983; Clarkson et al. 1984). Methylmercury irreversibly destroys the neurons of the central nervous system. Frequently, a substantial latent period intervenes between the cessation of exposure to Hg and the onset of signs and symptoms; this interval is usually measured in weeks or months, but sometimes in years (Clarkson et al. 1984). At high sublethal doses in man, mercury causes cerebral palsy, gross motor and mental impairment, speech disturbances, blindness, deafness, microcephaly, intestinal disturbances, tremors, and tissue pathology (Chang 1979; EPA 1980, 1985; Elhassani 1983; Clarkson et al. 1984). Pathological and other effects of Hg may vary from organ to organ, depending on factors such as the effective toxic dose in the organ, the compound involved and its metabolism within the organ, the duration of exposure, and the other contaminants to which the animal is concurrently exposed (Chang 1979). Many compounds--especially salts of selenium--protect humans and other animals against mercury toxicity, although their mode of action is not clear (NAS 1978; Chang 1979; EPA 1980, 1985; Eisler 1985).

Adverse effects of organomercury compounds to selected species of mammals have been recorded at administered doses of 0.25 mg Hg/kg body weight daily, dietary levels of 1.1 mg/kg, and blood Hg levels of 1.2 mg/l (Table 10).

Mercury transfer and biomagnification through mammalian food chains is well documented (Galster 1976; NAS 1978; Eaton et al. 1980; Eisler 1981; Huckabee et al. 1981; Sheffy and St. Amant 1982; Kucera 1983; Clarkson et al. 1984; Wren 1986), but considerable variation exists. Among terrestrial mammals, for example, herbivores such as mule deer, moose (*Alces alces*), caribou (*Rangifer tarandus*), and various species of rabbits usually contained less than 1.0 mg Hg/kg fresh weight in liver and kidney, but carnivores such as the marten (*Martes martes*), polecat (*Mustela putorius*), and red fox (*Vulpes vulpes*) frequently contained more than 30 mg/kg NAS 1978). The usually higher mercury concentrations in fish-eating furbearers than in herbivorous

species seemed to reflect the amounts of fish and other aquatic organisms in the diet. In river otter and mink from the Wisconsin River drainage system, Hg levels paralleled those recorded in fish, crayfish, and bottom sediments at that location. Highest Hg levels in all samples were recorded about 30 km downstream from an area that supported 16 pulp and paper mills and a chloralkali plant; residues were highest in the fur, followed by the liver, kidney, muscle, and brain (Sheffy and St. Amant 1982).

In marine mammals, more than 90 % of the mercury content is inorganic; however, enough methylmercury occurs in selected tissues to result in the accumulation of high tissue concentrations of methylmercury in humans and wildlife consuming such meat (Clarkson et al. 1984). The liver of the ringed seal (*Phoca hispida*) normally contains 27,000 to 187,000 ug Hg/kg fresh weight, and is a traditional and common food of the coastal Inuit people (Eaton et al. 1980). Although levels of Hg in hair (109,000 ug/kg) and blood (37 ug/l) of Inuits were grossly elevated, no symptoms of Hg poisoning were evident in the coastal Inuits. Similar high concentrations have been reported for Alaskan Eskimo mothers who, during pregnancy, ate seal oil twice a day, and seal- meat or fish from the Yukon-Kuskokwim Coast every day (Galster 1976). Despite the extremely high total Hg content of seal liver, only the small organomercury component was absorbed and appeared in the tissues. Cats fed a diet of seal liver (26,000 ug Hg/kg fresh weight) for 90 days showed no neurologic or histopathologic signs (Eaton et al. 1980). It seems that the toxic potential of seal liver in terms of accumulated tissue levels in cats (up to 862 ug total Hg/l blood, and 7,600 ug total Hg/kg hair) is better indicated by the organomercury fraction in seal liver than by the concentration of total Hg (Eaton et al. 1980).

Table 10. Sublethal effects of organomercury compounds administered to selected species of mammals.

Organism	Dose, and other variables	Effect	Reference
Rhesus monkey, *Macaca mulatta*	16 µg/kg body weight daily on days 20 to 30 of pregnancy	No measurable eff ect on reproduction	Khera 1979
Human, adult	50 µg/day	Risk of paresthesia, 0.3% (burning-prickling sensation of skin)	Clarkson et al. 1984
Human, adult	200 µg/day	Risk of paresthesia, 8%	Clarkson et al. 1984
Cat, *Felis domesticus*	250 µg/kg body weight daily on days 10 to 58 of gestation; oral route	Increased incidence of anomalous fetuses	Khera 1979
Harp seal, *Pagophilus groenlandicus*	250 µg/kg body weight daily for 90 days; dietary route	Residues of 47,200 to 82,500 µg/kg fresh weight in liver, kidney, and muscle; histopathology	Ronald et al. 1977; Ramprashad and Ronald 1977

		of middle ear	
Rat, *Rattus* sp.	500 µg/kg body weight daily; oral route	Reduced fertility	Khera 1979
Human, adult	1,000 µg/day	Risk of paresthesia, 50%	Clarkson et al. 1984
Mink, *Mustela vison*	1,100 µg/kg in diet	Residues of 7,100 to 9,300 µg/kg in brain; signs of poisoning	Kucera 1983
Rat	2,000 µg/kg in diet (as Pacific blue marlin); gestation through post-natal day 16	Adverse behavioral changes in offspring	Suzuki 1979
Rat	13,300 to 50,000 µg/kg body weight daily for 5 days; subcutaneous injection	Impaired cutaneous sensitivity and hearing up to one year post-treatment	Wu et al. 1985
Monkeys, *Macaca* spp.	Various	Visual disturbances at blood Hg levels of 1,200 to 4,000 µg/L or brain Hg levels of 6,000 to 9,000 µg/kg; tremors at blood Hg levels of 2,000 to 10,000 µg/L; kidney pathology at brain Hg levels of 1,500 µg/kg	Suzuki 1979
Human, adult	Various	Symptoms of poisoning evident at residues of 1,200 µg Hg/L blood, 2,000 to 3,000 µg/kg whole body, or 3,400 µg/kg hair	Suzuki 1979

Mice, *Mus* spp.	Various	Residues of 2,000 to 5,000 µg/kg hair or >10,000 µg/kg brain associated with motor incoordination and decreased swimming ability; no observable effect at <2,000 µg/kg hair	Suzuki 1979
Human, infant	Various	Severe effects at blood Hg levels of 3,000 µg/L	Elhassani 1983

Retention of mercury by mammalian tissues is longer for organomercury compounds (especially methylmercury) than for inorganic mercury compounds (NAS 1978; Clarkson and Marsh 1982; Elhassani 1983; Clarkson et al. 1984). Excretion of all Hg species follows a complex, variable, multicompartmental pattern; the longer-lived chemical Hg species have a biological half-life that ranges from about 1.7 days in human lung to 1.36 years in whole body of various pinnipeds.

OTHER GROUPS

Methylmercury compounds have induced abnormal sex chromosomes in the fruit fly (*Drosophila melanogaster*) (NAS 1978; Khera 1979). Earthworms (*Eisenia foetida*) exposed to soil containing methylmercury concentrations of 5.0 mg Hg/kg--typical of soil Hg levels near chloralkali plants--showed a significant reduction in the number of segments regenerated after 12 weeks, and contained 85 mg Hg/kg on a whole body fresh weight basis. Regeneration was normal at soil Hg levels of 1.0 mg/kg, although body burdens up to 27 mg/kg were recorded. It was concluded that soil contaminated with methylmercury posed a greater hazard to the predators of earthworms than to the earthworms (Beyer et al. 1985). Studies with a different species of earthworm (*Octochaetus pattoni*) and mercuric chloride, demonstrated a progressive initial increase in reproduction as soil mercury levels increased from 0.0 to the 60-day lethal level of 5.0 mg/kg (Abbasi and Soni 1983).

CURRENT RECOMMENDATIONS

Proposed mercury criteria for the protection of sensitive aquatic organisms, birds, and mammals, as well as human health, are shown in Table 11. In almost every instance, these criteria are listed as concentrations of total Hg, with most, if not all, the Hg present as an organomercury species.

In 1980, the U.S. Environmental Protection Agency's proposed mercury criteria for freshwater aquatic life protection were 0.00057 ug/l (24-hour average), not to exceed 0.0017 ug/l at any time; these criteria seemed to afford a high degree of protection to freshwater biota, as judged by survival, bioconcentration, and biomagnification. Literature documented in this paper showed that mercury concentrations in water of 0.1 to 2.0 ug/l were fatal to sensitive aquatic species and that concentrations of 0.03 to 0.1 ug/l were associated with significant sublethal effects. The 1980 proposed freshwater criteria provided safety factors for acute toxicities of 175X to 3,508X based on the 24-hour average, and 58X to 1,176X based on the maximum permissible concentration (Table 11). For protection against sublethal effects, these values were 53X to 175X based on the 24-hour mean, and 18X to 59X based on the maximum permissible concentration (Table 11). However, the most current freshwater criteria of 0.012 ug/l, not to exceed 2.4 ug/l (Table 11; EPA 1985), dramatically reduces the level of protection afforded aquatic biota: safety factors for acute toxicities are now 8X to 167X based on the 96-hour average, and only 0.04X to 0.8X based on the maximum permissible concentration. For protection against sublethal effects, these values were 2X to 8X based on the 4-day average, and only 0.01X to 0.04X based on the maximum permissible concentration, or essentially no significant protection.

The proposed saltwater criteria of EPA (1980) for mercury and marine life were unsatisfactory. Proposed saltwater values of 0.025 ug/l (24-hour average), not to exceed 3.7 ug/l at any time (Table 11), provided safety factors of 4X to 80X against acute toxicity (based on 24-hour average), but less than 0.5X based on the maximum permissible level. For protection against sublethal damage effects, the safety factors computed were 1.2X to 4X (based on 24-hour average) and less than 0.03X (based on maximum allowable concentration). The most recent saltwater criteria of 0.025 ug/l, not to exceed 2.1 ug/l (Table 11; EPA 1985), does not appear to offer a substantive increase in protection to marine life, when compared to criteria proposed 5 years earlier (EPA 1980). It seems that some downward modification is needed in the proposed Hg saltwater criteria if marine and estuarine biota are to be provided even minimal protection.

Table 11. Proposed mercury criteria for protection of various resources and human health.

Resource and criterion (units in parentheses)	Mercury concentration	Reference
Aquatic life		
Freshwater (µg/L)	Total recoverable Hg <0.00057 (24 h average), not to exceed 0.0017 at any time	EPA 1980
	<0.012, 4-day average (not to be exceeded more than once every 3 years; <2.4, one-hour average (not to be exceeded more than once every 3 years)[a]	EPA 1985
Saltwater (µg/L)	Total recoverable Hg <0.025 (24 h average), not to exceed 3.7 at any time	EPA 1980
	<0.025, 4-day average (not to be exceeded more than once every 3 years); <2.1, one-hour average (not to be exceeded more than once every 3 years)[a]	EPA 1985
Freshwater (µg/L)		
Inland surface waters, India	<10.0 from point source discharge	Abbasi and Soni 1983
Fish (µg/kg fresh weight)		
Brook trout, *Salvelinus fontinalis*		

Whole body	<5,000	EPA 1980, 1985
Wildlife		
Birds		
Tissue residues (µg/kg fresh weight)		
Feather	<5,000	NAS 1978
Egg		
Mallard, *Anas platyrhynchos*	<900	Heinz 1979
Ring-necked pheasant, *Phasianus colchicus*	<900	Spann et al. 1972
Various species	1,300–2,000	Fimreite 1979
Diet (µg/kg fresh weight)	50 to <100	Heinz 1979; March et al. 1983
Daily dose (µg/kg body weight)		<640 Spann et al. 1972; McEwen et al. 1973; Mullins et al. 1977
Mammals		
Tissue residues (µg/kg fresh weight)		
Kidney	<1,100	EPA 1980
Blood	<1,200	Suzuki 1979
Brain	<1,500	Suzuki 1979
Hair		<2,000 Suzuki 1979
Diet (µg/kg fresh weight)	<1,100	Kucera 1979
Daily dosage (µg/kg body weight)	<250	Ramprashad and Ronald 1977; Ronald et al. 1977; Khera 1979
Human Health		
Tissue residues (µg/kg fresh weight)		
Blood	<200	Galster 1976
Hair		<6,000 Lodenius et al. 1983
Water (µg/L)		
Potable	<2.0	NAS 1978
Protection from toxic properties of Hg through consumption of contaminated aquatic organisms	<0.146	EPA 1980

Protection from toxic properties of Hg from ingestion of water plus consumption of resident aquatic organisms	<0.144	EPA 1980
Fish and seafood		
Acceptable intake (µg)		
Daily, 60 kg adult	25	Khera 1979
Weekly, 70 kg adult	200	Khera 1979
Weekly, adult	500	Lodenius et al. 1983
Pregnant women (µg/kg fresh weight)	<250	Khera 1979
Various locations (µg/kg fresh weight)		
Japan	<400	NAS 1978
Canada, West Germany, U.S.A.	<500	NAS 1978
Australia	<500 (mean), not to exceed 1,500 in any sample	Lyle 1984
Finland	<1,000	Lodenius et al. 1983
Scandinavia	<1,000	Suckcharoen and Lodenius 1983
Sweden	<1,000	NAS 1978
U.S.A.	<1,000	EPA 1980, 1985; Barber et al. 1984; Miller and Jude 1984
Foods of animal origin (µg/kg fresh weight)		
Livestock tissues	<500	Best et al. 1981
Wildlife tissues	<50	Krynski et al. 1982
Breast muscle		
Domestic poultry	<500	NAS 1978
Ducks (wildlife)	<1,000	Lindsay and Dimmick 1983
All foods		
Adult weekly intake (µg)		
As total Hg	<150	NAS 1978
As methylmercury	<100	NAS 1978
Various locations (µg/kg fresh weight)		
Australia	10 to 100	NAS 1978

Benelux countries	<30	NAS 1978
Brazil	<50	NAS 1978
Canada	<500	Bodaly et al. 1984
U.S.A.	<1,000	Bodaly et al. 1984

[a]All mercury that passes through a 0.45-mm membrane filter after the sample is acidified to pH 1.5 to 2.0 with nitric acid.

The significance of elevated Hg residues in tissues of aquatic organisms is not fully understood. Concentrations >1,000 ug Hg/kg fresh weight can occur in various tissues of selected species of fish and aquatic mammals eaten by humans. But it would be incorrect to assume that aquatic food chains--especially marine food chains--incorporate Hg exclusively from anthropogenic activities (Barber et al. 1984). Some organisms, however, do contain Hg tissue residues associated with known adverse effects to the organism and its predators. Thus, whole body residues of 5,000 to 7,000 ug Hg/kg fresh weight in brook trout eventually proved fatal to that species (EPA 1980). To protect sensitive species of mammals and birds that regularly consume fish and other aquatic organisms, total mercury concentrations in these food items should probably not exceed 100 ug/kg for avian protection, or 1,100 ug/kg for small mammals (Table 11). By comparison, proposed Hg levels in fish and seafood, in ug/kg fresh weight, for human health protection should not exceed 250 for expectant mothers, and 400 to 1,000 for adults worldwide (Table 11).

Since long-lived, slow-growing, high-trophic-positon aquatic organisms usually contain the highest tissue mercury residues, some fisheries managers have proposed a legal maximum limit based on fish length or body weight, or alternatively, constraining the mean Hg concentration of the entire catch to a nominated level. In the Australian shark fishery, for example, implementation of a maximum length restriction (to a nominated level of 500 ug Hg/kg), would result in retention of less than half of the present catch of seven species (Lyle 1984).

Among sensitive avian species, adverse effects--predominantly on reproduction--have been reported at mercury concentrations (in ug/kg fresh weight) of 5,000 in feather, 900 in egg, 50 to 100 in diet, and daily administered doses of 640 on a body weight basis (Table 11). Although low Hg concentrations--e.g., 50 ug/kg in the diets of domestic chickens--sometimes produced no adverse effects on chickens, the tissue residues of Hg were sufficiently elevated to pose a hazard to human consumers (March et al. 1983). In contrast, in eggs of the American bald eagle (with 150 ug Hg/kg and low hatch), other contaminants present--especially organochlorine compounds--probably had a greater effect on hatchability than did Hg (Wiemeyer et al. 1984).

Mammals, such as the domestic cat and the harp seal, showed birth defects, histopathology, and elevated tissue residues at doses of 250 ug Hg/kg body weight daily (Table 11). The mink, at dietary levels of 1,100 ug Hg/kg, had signs of mercury poisoning; Hg residues in mink brain at this dietary level ranged from 7,100 to 9,300 ug/kg (Kucera 1983). Tissue residues in kidney, blood, brain, and hair in excess of 1,100 ug Hg/kg in other nonhuman mammals are usually considered presumptive evidence of significant Hg contamination (Table 11).

At this point, it seems that four courses of action are warranted. First, toxic mercurials in agriculture and industry should be replaced by less toxic substitutes. Second, controls should be applied at the point of origin to prevent the discharge of potentially harmful Hg wastes. Third, continued periodic monitoring of fishery and wildlife resources is important, especially in areas with potential for reservoir development, in light of the hypothesis that increased flooding increases the availability of Hg to biota. And finally, additional research is needed on mercury accumulation and detoxication in comparatively pristine ecosystems.

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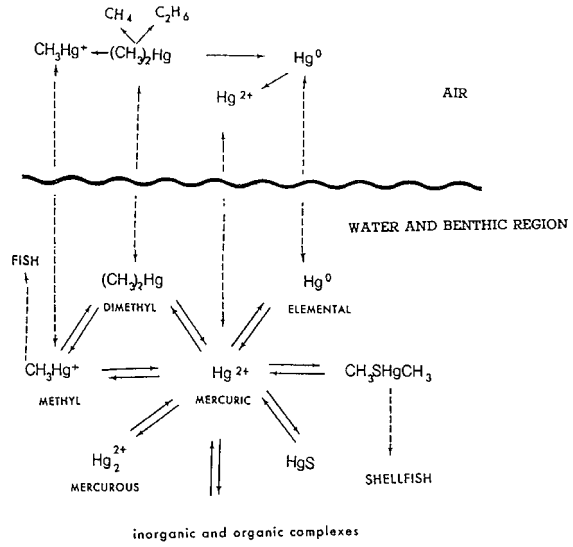

Figure 1. Major transformations of mercury in the environment (modified from Beijer and Jernelov 1979).

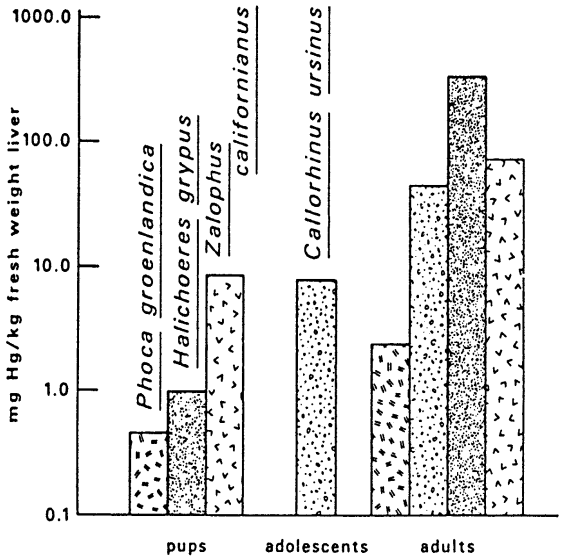

Figure 2. Mercury concentrations in livers of four species of pinniped mammals (from Eisler 1984).