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Ecological effects, transport, and fate of mercury: a general review

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Abstract

Mercury at low concentrations represents a major hazard to microorganisms. Inorganic mercury has been reported to produce harmful effects at 5 μg/l in a culture medium. Organomercury compounds can exert the same effect at concentrations 10 times lower than this. The organic forms of mercury are generally more toxic to aquatic organisms and birds than the inorganic forms. Aquatic plants are affected by mercury in water at concentrations of 1 mg/l for inorganic mercury and at much lower concentrations of organic mercury. Aquatic invertebrates widely vary in their susceptibility to mercury. In general, organisms in the larval stage are most sensitive. Methyl mercury in fish is caused by bacterial methylation of inorganic mercury, either in the environment or in bacteria associated with fish gills or gut. In aquatic matrices, mercury toxicity is affected by temperature, salinity, dissolved oxygen and water hardness. A wide variety of physiological, reproductive and biochemical abnormalities have been reported in fish exposed to sublethal concentrations of mercury. Birds fed inorganic mercury show a reduction in food intake and consequent poor growth. Other (more subtle) effects in avian receptors have been reported (i.e., increased enzyme production, decreased cardiovascular function, blood parameter changes, immune response, kidney function and structure, and behavioral changes). The form of retained mercury in birds is more variable and depends on species, target organ and geographical site. With few exceptions, terrestrial plants (woody plants in particular) are generally insensitive to the harmful effects of mercury compounds.

Keywords: Mercury; Invertebrates; Plants; Fish; Mammals; Birds; Fate and transport

1. Introduction

Mercury (Hg) is a liquid metal at ambient temperatures and pressures. It forms salts in two ionic states mercury (I) and mercury (II). Mercury (II), or mercuric salts, are much more common in the environment than mercury (I) or mercurous salts. These salts, if soluble in water, are bioavailable and considered toxic. Mercury also forms organometallic compounds, many of which have industrial and agricultural uses. Elemental mercury gives rise to a vapor that is only slightly soluble in water, but is problematic because of easy transport in the atmosphere. Lastly, the most common form of mercury is insoluble mercuric sulfide (naturally occurring cinnabar) which is non-toxic. Throughout the remainder of this document, 'mercury' will refer to the most bioavailable or toxic forms of mercury.

Two cycles are believed to be involved in the environmental transport and distribution of mercury. One is global in scope and involves the atmospheric circulation of elemental mercury vapor from sources on land to the oceans. The second cycle is local in scope and depends upon the methylation of inorganic mercury mainly from anthropogenic sources. The steps in this cycle remain poorly understood, but it likely involves the atmospheric

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circulation of dimethylmercury formed by bacterial action. Hence, environmental levels of methyl mercury depend upon the balance between bacterial methylation and demethylation.

Mercury is bound to the cell walls or membranes of microorganisms. Harmful effects appear to be related to both cell density and concentration of mercury in a substrate. The 96 h LC_{50} values for freshwater fish ranges between 33 and 400 μg/l, while the LC_{50} is generally higher for marine fish. There is little indication that fish themselves either methylate or demethylate mercury. As is the case with other groups of ecological receptors, organic mercury compounds are most toxic to birds. Sea birds and those feeding in estuaries are usually most contaminated (World Health Organization, 1989).

Contemporary measurements of atmospheric mercury together with historical records from lake sediments and peat indicate that the global reservoir of atmospheric mercury has increased by a factor of 2–5 since the beginning of the industrialized period. Because mercury vapor has a long atmospheric residence time, and because mercury contamination of lacustrine food webs appears geographically pervasive, mercury pollution is often viewed as a global problem that may defy state or national abatement efforts (Klaassen et al., 1986).

Natural mercury arises from the degassing of the earth's crust through volcanos, and probably, by evaporation from the oceans. Most indications are that atmospheric pollution from industrial production has decreased significantly in recent years, but contamination of water by mine tailings remains significant. Smelting of lead, copper and zinc ores incidentally emits mercury to the atmosphere in annual quantities estimated at 100 ton globally (4% of total) and about 9 ton in the US (3% of total) (Klaassen et al., 1986).

The burning of fossil fuels is also a source of mercury. The chloralkali, electrical equipment, paint, and wood pulping industries are the largest consumers of mercury, accounting for 55% of the total consumption. Some mercury compounds have been used in agriculture, principally as fungicides. Mercury has a wide variety of other uses in military applications, batteries, medicine and dentistry. Although the industrial use of mercury has been reduced in recent years due to stricter regulations, high concentrations are still present in sediments associated with industrial applications of mercury (Klaassen et al., 1986).

2. Mercury toxicity in aquatic receptors

2.1. Microorganisms

Wood (1984) discussed six protective mechanisms available to microorganisms (and certain higher organisms) that increase their resistance to mercury. These mechanisms are biochemical in nature, and generally, render the mercury ion ineffective in disturbing the normal biochemical processes of the cell. The mechanisms are:

1. Efflux pumps that remove the ion from the cell.
2. Enzymatic reduction of the metal to the less toxic elemental form.
3. Chelation by enzymatic polymers (i.e., metallothionein).
4. Binding mercury to cell surfaces.
5. Precipitation of insoluble inorganic complexes (usually sulfides and oxides), at the cell surface.
6. Biomethylation with subsequent transport through the cell membrane by diffusion. This mechanism renders the mercury more toxic to higher organisms.

2.2. Aquatic plants

The presence of sediment or humic material reduces the availability of mercury to aquatic plants via adsorption. Organomercury compounds, such as methyl- or butyl mercury chloride are more toxic to aquatic plants than inorganic forms (World Health Organization, 1989). In a study that exposed two-day-old sporelings of the red algae *Plumaria elegans* to mercuric chloride (Boney, 1971), 50% growth inhibition occurred after 6, 12 and 24 h at concentrations of 1.0, 0.5 and 0.25 mg/l, respectively. Various forms of organic mercury (methyl, butyl and propylmercuric chlorides) were also investigated, and found to be more toxic than inorganic mercury.

Although some aquatic plants have the capacity to absorb and accumulate heavy metals, the reported data on mercury uptake by wetland plants are sometimes contradictory, indicating that mercury accumulation may take place in roots rather than rhizomes or shoots (Boney, 1971). Many wetlands accumulate mercury largely as a result of direct atmospheric deposition, or through transport from the watershed. Mosses typify much of the vegetation in bogs and boggy wetlands, and are key species because they tend to accumulate and retain more mercury than other plants. Plant uptake has a potential to contribute substantial mercury to food webs, and may also recycle considerable mercury through decomposition products (Zillioux et al., 1993).

Stanley (1974) determined EC_{50} values in the presence of mercuric chloride, for various growth parameters of Eurasian milfoil (*Myriophyllum spicatum*) grown in soil with overlying water. The EC_{50} values (in mg/l) were 3.4 for root weight, 4.4 for shoot weight, 12.0 for root length, and 1.2 for shoot length. This study also reported that mercury had a strong adsorption affinity for the soil matrix.

De et al. (1985) exposed the floating water cabbage (*Pistia stratiotes*) for two days to mercuric chloride at

concentrations between 0.05 and 20.0 mg/l. The highest dose of mercury decreased chlorophyll content, protein, RNA, dry weight, catalase and protease activity, and increased production of free amino acids. In a study (Brown and Rattigan, 1979) using the Canadian pond weed (*Elodea canadensis*) and the free-floating duck weed (*Lemna minor*), both plants were exposed for 14 and 28 days to varying concentrations of mercuric chloride. Water concentrations of 7.4 and 1.0 mg/l produced 50% damage to the two plants, respectively. In a separate study, pond weed was exposed to mercury for 24 h in the dark and then oxygen evolution in the light was measured. Levels of 0.8 and 1.69 mg mercury/l reduced photosynthetic oxygen evolution by 50% (dark) and 90% (light), respectively.

Czuba and Mortimer (1980, 1982) exposed plants of pond weed (*Elodea densa*), growing in flowing water, to concentrations of methyl mercuric chloride at 7.5×10^{-10} 7.5×10^{-9} or 7.5×10^{-8} mol/l, for 25 days. Toxicity was assessed by gross morphological examination of histological sections embedded in paraffin wax. Apical cells were most sensitive and developed aberrant nuclear and mitotic characteristics at lower concentrations than did roots. Root meristems showed total inhibition of mitotic activity at the middle concentration but no effect at the lowest concentration. Mitotic activity in bud meristems was absent in controls, but increased in the presence of methyl mercury; divisions were abnormal. Higher concentrations of methyl mercury chloride, up to 2.5×10^{-6} mol/l, stimulated the development of additional buds.

Glooschenko (1969) exposed the marine diatom (*Chaetoceros costatum*) to labeled mercury, and found no difference between uptake in the light or the dark in non-dividing cells. Dead cells took up twice as much mercury as living cells, presumably by surface adsorption. Hannerz (1968) demonstrated that there was no appreciable assimilation of mercury into the tissues of aquatic plants. Although concentrations were 10–20 times higher in submerged parts compared with emergent parts, this was also attributed to surface adsorption differences. De et al. (1985) grew *Pistia stratiotes* in nutrient solution containing mercuric chloride at concentrations ranging from 0.05 to 20 mg/l. Accumulation increased with increasing mercury concentrations, with maximum removal of mercury from water recorded at 6 mg/l or less, with only 20% mercury lost from plants receiving the highest concentration. Mercury accumulation into the roots was about four times higher than into the shoots at lower concentrations and about twice as high at 20 mg/l.

2.3. Aquatic invertebrates

Factors which impact the toxicity of mercury to aquatic invertebrates include the concentration and species of mercury, the developmental stage of the organism, temperature, salinity, hardness and in some cases, water flow rate. The larval stage is apparently the most sensitive of the invertebrate life cycle. Mercury toxicity increases with temperature and decreases with water hardness. Toxicity, appears to be higher in flow-through systems than in static systems. Levels of 1–10 μg/l normally cause acute toxicity for the most sensitive developmental stage of many different species of aquatic invertebrates.

Riisgard et al. (1985) transferred mussels (*Mytilus edulis*) from clean water to an area chronically polluted with mercury. Mussels were exposed for a duration of three months. They were then transferred back to the clean water and mercury depuration was measured. The biological half-life was 293 days; and 75% of the mercury detected in the mussels was inorganic. In another study, only 6% of the total mercury in a the molluscan *Macoma balthica*, a sediment-feeding bivalve, was methylated, a much lower percentage than in *Mytilus* from the same area. Gagnon and Fisher (1997) exposed mussels (*Mytilus edulis*) to both methylated and inorganic mercury from sediment and food. It was determined that the form of mercury (organic vs. inorganic) determined where the mercury would be accumulated. The methyl mercury was more likely to accumulate in soft (edible) tissues. The tissue distribution results were as follows: inorganic mercury = mantle>gland>shell> gills>foot; methyl mercury = mantle>gland>gills>foot> shell.

DeFreitas et al. (1981) found a net assimilation of 70–80% for methyl mercury and 38% for inorganic mercury when fed in the diet to shrimp *Hyalella azteca*. From water, inorganic mercury was assimilated 2–3 times more slowly than methyl mercury. Khayrallah (1985) found that the accumulation of ethyl mercuric chloride was almost twice as rapid as that of mercuric chloride in the amphipod *Bathyporeia pilosa*, although death occurred at similar concentrations. Ray and Tripp (1976) exposed grass shrimp (*Palaemonetes pugio*) to radiolabelled methyl mercury chloride and mercuric chloride for 24 and 72 h. After 24 h, the methylated form was mostly concentrated in the ventral nerve cord and to a lesser extent in the gills. The reverse was true for mercuric chloride. The concentration of mercury accumulated in the other tissues (exoskeleton > foregut > remainder) were similar for both compounds. After 72 h no order of tissue mercury concentrations was observed. There was an increase in the mercury levels in the exoskeleton, foregut and remainder tissues, while mercury levels in the gills remained unchanged and the ventral nerve cord concentration decreased.

Kraus et al. (1988) examined the tolerance to mercury in two populations of grass shrimp *Palaemonetes pugio* at different stages of the animal's life cycle. One population (PC) (collected from a mercury contaminated creek) was monitored in parallel with the other

population (BSC) (collected from an unpolluted creek). Larval grass shrimp showed no significant differences between groups in terms of mortality at 0.01 mg/l mercury chloride, although treated BSC metamorphosed significantly ($P < 0.05$) more slowly. While no BSC larvae survived the 0.0125 mg/l methyl mercury treatment, PC larvae survived as well as their respective controls under the same treatment, indicating an enhanced tolerance in this population. It was also determined that both populations were capable of producing a metallothionein like protein (MT) in response to mercury chloride treatment. The grass shrimp is capable of physiologically acclimating to mercury chloride through the production of MT-like binding proteins. This protein is not passed from generation to generation through the eggs, but is acquired thorough exposure to inorganic mercury. Observed tolerances to mercury may reflect a genetic adaptation.

Vernberg and O'Hara (1972) measured the uptake of labeled mercury into the gills and hepatopancreas of fiddler crabs (*Uca pugilator*) maintained in a solution containing 0.18 mg mercury/l (as mercuric chloride) for 72 h. Uptake was determined under various temperature (5°C and 30°C) and salinity (5 and 30 g/l) regimes. Total mercury levels in these organs were unaffected by the different regimes. However, the ratio of uptake into the two tissues was affected. At higher temperatures, the crabs were able to transport mercury from gill tissue to the hepatopancreas more efficiently than at lower temperatures. Crabs also possess the ability to produce methallothioneins in response to mercury contaminated environs (Vernberg and O'Hara, 1972).

In a recent study (Canli and Furness, 1995), Norway lobsters (*Nephrops norvegicus*) were fed a mercury-rich diet for up to 50 days or were exposed to sublethal concentrations of organic mercury or inorganic mercury in seawater for 30 days. Both organic and inorganic mercury were accumulated mainly in the gill, while highest concentrations were found in the hepatopancreas. Accumulation of organic mercury was higher than that of inorganic mercury. Distributions of organic and inorganic mercury also varied among tissues after uptake from seawater, with organic mercury being more evenly distributed among tissues than inorganic mercury, the latter being found predominately in the gill. Lastly, the dominant accumulation route of mercury in the hepatopancreas was from food. For the gill the main source of mercury was via uptake from water. For the tail muscle, accumulation from food was an important route, although accumulation from seawater was also pronounced, especially for organic mercury.

Midge (*Chironomus riparius*) larvae kept in a solution of 5 g/l for only 1 min accumulated 9.32 mg mercury/kg body weight. Adult midge accumulated 40% of the levels observed in larvae, suggesting that adults may be utilizing a means of mercury detoxification (World Health Organization, 1989). The acute toxicity of mercury to aquatic invertebrates is summarized in Tables 1 and 2.

2.4. Fish

Observations of various marine and freshwater fish species indicate that tissue concentrations of mercury increase with increasing age (as inferred from length) of the fish. In some species, males have been found to harbor higher mercury levels than females of equal age (World Health Organization, 1989).

The intestinal wall in fish is an effective barrier to mercury chloride but is permeable to methyl mercury, which can accumulate preferentially over time in muscle tissue to about 50% of the total dose (World Health Organization, 1989).

Growth rate and total mercury concentrations were determined in muscle tissue of pumpkinseed sunfish (*Lepomis gibbosus*) collected from waters in south–central Ontario (Wren and MacCrimmon, 1983). Mean water pH ranged from 5.6 to 8.4. Growth rate correlated positively with lake pH, whereas fish mercury levels were higher at lower pH. The general postulation that a reduced pH will enhance mercury uptake by fish has been largely promoted from mercury studies that show that the more toxic methyl mercury form found in fish, is much more prevalent at lower water pH. Differences in growth rate may be the result of both metal and acid stress in lower pH waters. In this study, water calcium (Ca) concentration was negatively correlated with mercury levels in pumpkinseed. A relationship between fish tissue concentrations of mercury and water Ca level was observed by McFarlane and Franzin (1980) for white suckers in five Manitoba lakes. Rodgers (1982) found that the uptake of methyl mercury by rainbow trout was less in hard water (385 mg $CaCO_3$/l) than in soft water (30 mg $CaCO_3$/l) and suggested that increased mercury uptake efficiency in soft water may partially explain elevated mercury levels observed in fish from lakes of low alkalinity and pH.

McMurtry et al. (1989) examined the concentrations of mercury in dorsal muscle tissue of lake trout from Ontario lakes. Mercury concentrations were positively correlated with dissolved organic carbon (DOC), which explained a significant amount of variation (37%) in mercury concentrations in lake trout. The relationship between DOC and mercury appeared to be strongest in lakes with DOC values of less than 4.0 mg/l. In contrast, mercury concentrations in smallmouth bass were correlated with variables affecting water hardness (Mg, Ca, conductivity) and acidity (pH, alkalinity). The relationship was inverse for the water hardness variables and positive for acidity. Regression analysis identified Ca, DOC, and latitude as significantly affecting variations in mercury accumulation in smallmouth bass.

Table 1
Toxicity of inorganic mercury (mercuric chloride) to marine invertebrates (World Health Organization, 1989)

Receptor	Life stage	Temperature (°C)	pH	Salinity (parts per thousand)	DO (mg/l)	Endpoint (LC_{50})	$HgCl_2$ (μg/l)
Starfish (*Asterias forbesi*)	Adult	20	7.8	20	>4	24 h	1800
	Adult	20	7.8	20	>4	96 h	60
	Adult	20	7.8	20	>4	168 h	20
Hard clam (*Mercenaria mercenaria*)	Embryo	25–27	7–8.5	25		48 h	4.8
Softshell clam (*Mya arenaria*)	Adult	20	7.8	20	>4	24 h	5200
	Adult	20	7.8	20	>4	96 h	400
	Adult	20	7.8	20	>4	168 h	4
American oyster (*Crassostrea gigas*)	Embryo	25–27	7–8.5	25		48 h	5.6
Pacific oyster (*Crassostrea gigas*)	Embryo	19–21	7.9–8.3	33.7–33.8	6.5–8.0	48 h	5.7
Oyster (*Ostrea edulis*)	Larvae	15				48 h	1.0–3.3
	Adult	15				48 h	4200
Cockle (*Cardium edule*)	Adult	15				48 h	9000
Mud Snail (*Nassarius obsoletus*)	Adult	20	7.8	20	>4	24 h	
	Adult	20	7.8	20	>4	96 h	
	Adult	20	7.8	20	>4	168 h	
American Lobster (*Homarus americanus*)	Stage I larvae	18–22		29.5–31.5	7.6–8.6	96 h	20
European Lobster (*Homarus gammarus*)	Larvae	15				48 h	33–100
Pink shrimp (*Pandalus montagui*)	Adult	15				48 h	75
White shrimp (*Penaeus setiferus*)	Post-larval	21–24		25		96 h	17
Brown shrimp (*Crangon crangon*)	Larvae	15				48 h	10
	Adult	15				48 h	3300–10,000
	Adult	15				96 h	100–330
Grass shrimp (*Palaemonetes vulgaris*)	Stage I larvae	26.5–27	6.3–6.9	32.7–33.3	5.6	48 h	10 (unfed)
	Stage I larvae	27	6.4–6.7			48 h	15.6 (fed)
Dungeness crab (*Cancer magister*)	1st stage zoeae	14–16	7.9–8.3	33.7–33.9	6.5–8.0	48 h	21.1
	1st stage zoeae	14–16	7.9–8.3	33.7–33.9	6.5–8.0	96 h	6.6
Shore crab (*Carcinus maenus*)	Larvae	15				48 h	14
	Adult	15				48 h	1200
Hermit crab (*Pagurus longicarpus*)	Adult	20	7.8	20	>4	24 h	2200
	Adult	20	7.8	20	>4	96 h	50
	Adult	20	7.8	20	>4	168 h	50

Table 1 (Continued)

Receptor	Life stage	Temperature (°C)	pH	Salinity (parts per thousand)	DO (mg/l)	Endpoint (LC_{50})	$HgCl_2$ (μg/l)
Crab	Adult	26.5–29.5	7–7.2			24 h	930
(*Scylla serrata*)	Adult	26.5–29.5	7–7.2			48 h	800
	Adult	26.5–29.5	7–7.2			72 h	680
	Adult	26.5–29.5	7–7.2			96 h	680
Polychaete	Juvenile		7.8			96 h	100
(*Neanthes*	Adult		7.8			96 h	22
arenaaceodentata)	Juvenile		7.8			28 d	90
	Adult		7.8			28 d	17
Polychaete	Larvae		7.8			96 h	14
(*Capitella*	Adult		7.8			96 h	>100
capitata)	Adult		7.8			28 d	100
Sandworm	Adult	20	7.8	20	>4	24 h	3100
(*Neris virens*)	Adult	20	7.8	20	>4	96 h	70
	Adult	20	7.8	20	>4	168 h	60

Wren et al. (1991) examined the relation between mercury levels in walleye (*Stizostedion vitreum vitreum*) and northern pike (*Esox lucius*) in Ontario lakes. The mean mercury concentrations in walleye and northern pike of standardized length from these lakes were 0.65 and 0.52 μg/g, respectively. Lacustrine characteristics positively correlated with mercury levels in both species including DOC and iron. Variables associated with acidity and hardness were negatively correlated with mercury concentrations in northern pike but not walleye.

The toxicity of inorganic and organic mercury to fish is summarized in Tables 3–5.

2.5. Marine mammals

Seals reportedly have a wide range of total mercury concentrations in liver (0.4 to over 300 mg/kg), with only a small fraction (2–17%) present in the methylated form. Gaskin et al. (1974) found liver total mercury levels ranging from 13 to 157 mg/kg in short-finned pilot whales and long-snouted dolphins from the Lesser Antilles. Selenium (Se) and mercury have been found in seal livers in a consistent 1:1 atomic ratio. Several studies have indicated that Se plays a protecting role against mercury toxicity. Koeman et al. (1975) determined mercury levels of 0.37–326 mg/kg in the livers of various marine mammals (seal, dolphins and porpoises) and reported a perfect 1:1 correlation between Se and mercury in each.

Honda et al. (1986) sampled striped dolphin (*Stenella coeruleoalba*) and found that accumulation of total mercury in bone correlated significantly with age (as documented in fish). Levels rose to 1.44 and 1.55 mg/kg for adult male and female, respectively, and similar trends were seen for methyl mercury. Falconer et al. (1983) found that in the common porpoise (*Phocoena phocoena*) the highest mercury levels were in the liver, where mean levels for females were 6.03 mg/kg and for males 3.42 mg/kg. In a similar study with grey seals from the British coast, the highest mercury levels were again found in the liver (4.9–326 mg/kg) (Falconer et al., 1983).

Concentrations of Hg and Se have been determined in liver and kidney of 92 harbor porpoises caught from the Norwegian coast. The hepatic and renal mercury concentrations ranged from 0.26 to 9.9 and 0.15 to 3.5 μg/g, respectively. The corresponding Se concentrations ranged from 0.74 to 14.2 and 0.60 to 8.6 μg/g, respectively. In all age classes, a significant, positive correlation between mercury and Se concentrations were found in both liver and kidney (Teigen et al., 1993).

Wagemann (1989) studied Hg content in ringed seals (*Phoca hispida*) from the Canadian Arctic. Strong positive correlations were found between seal age and mercury content in liver. There was also a positive correlation between levels of Se and mercury in the seals, compared to a reference group.

3. Mercury toxicity in amphibia

Mercury exerts toxicity in amphibian tadpoles similar to that for fish. There is considerable species variability in susceptibility to the metal. There is no information on effects on adult amphibians. The toxicity of mercury to amphibians is summarized in Tables 6 and 7.

Table 2
Toxicity of inorganic mercury (mercuric chloride) to freshwater invertebrates (World Health Organization, 1989)

Organism	Temperature (°C)	Alkalinity (mg $CaCO_3$/l)	Hardness (mg $CaCO_3$/l	pH	DO (mg/l)	Endpoint (LC_{50})	$HgCl_2$ (μg/l)
Mussel	28–32	9.5–9.9	32–38	7.0–7.3	5.4–6.2	24 h	7390
(*Lamellidens*	28–32	9.5–9.9	32–38	7.0–7.3	5.4–6.2	48 h	5910
maginalis)	28–32	9.5–9.9	32–38	7.0–7.3	5.4–6.2	72 h	3690
Snail (egg)	17		50	7.6	6.2	24 h	6300
(*Amnicola* sp.)	17		50	7.6	6.2	96 h	2100
Snail (adult)	17		50	7.6	6.2	24 h	1100
(*Amnicola* sp.)	17		50	7.6	6.2	96 h	80
Snail (*Pila globosa*)	28–32	9.5–9.9	32–38	7.0–7.3	5.4–6.2	24 h	1108
	28–32	9.5–9.9	32–38	7.0–7.3	5.4–6.2	48 h	369
	28–32	9.5–9.9	32–38	7.0–7.3	5.4–6.2	72 h	296
Pulmonate snail	25.5–29.5	240–278	290–335	7.4–8.1	6.0–8.1	24 h	330
(*Lymnaea luteola*)	25.5–29.5	240–278	290–335	7.4–8.1	6.0–8.1	48 h	188
	25.5–29.5	240–278	290–335	7.4–8.1	6.0–8.1	96 h	135
Crab (*Oziotelphusa*	28–32	9.5–9.9	32–38	7.0–7.3	5.4–6.2	24 h	739
senex senex)	28–32	9.5–9.9	32–38	7.0–7.3	5.4–6.2	48 h	591
	28–32	9.5–9.9	32–38	7.0–7.3	5.4–6.2	72 h	443
Crayfish	15–17			7.0		96 h	20
(*Austropotamobius*	15–17			7.0		30 day	2
pallipes pallipes)	15–17			7.0		30 day	<2
Crayfish	15–17			7.0		96 h	50
(*Orconectes*	15–17			7.0		30 day	2
limosus)	15–17			7.0		30 day	<2
Copepod (*Cyclops* *abyssorum*)	10	0.5 meq/l		7.2		48 h	2200
Copepod (*Eudiaptomus* *padanus*)	10	0.5 meq/l		7.2		48 h	850
Water flea (*Daphia* *hyalina*)	10	0.5 meq/l		7.2		48 h	5.5
Water flea	17–19	41–50	44–53	7.4–8.2		48 h	5.0
(*Daphnia magna*)	17–19	41–50	44–53	7.4–8.2		21 day	13
	11.5–14.5	390–415	235–206	7.4–7.8	5.2–6.5	24 h	4890
	11.5–14.5	390–415	235–260	7.4–7.8	5.2–6.5	48 h	3610
Bristle worm	17		50	7.6	6.2	24 h	1900
(*Nais* sp.)	17		50	7.6	6.2	96 h	1000
Stonefly (*Acroneuria* *lycorias*)	16–20	40	44	7.25	9.2	96 h	2000
Mayfly (*Ephemerella* *subvaria*)	16–20	40	44	7.25	9.2	96 h	2000
Caddisfly (*Hydropsyche* *betteni*)	16–20	40	44	7.25	9.2	96 h	2000
Caddisfly	17		50	7.6	6.2	24 h	5600
(*unidentified* sp.)	17		50	7.6	6.2	96 h	1200

Table 2 (Continued)

Organism	Temperature (°C)	Alkalinity (mg $CaCO_3$/l)	Hardness (mg $CaCO_3$/l	pH	DO (mg/l)	Endpoint (LC_{50})	$HgCl_2$ (μg/l)
Damselfly	17		50	7.6	6.2	24 h	3200
(*unidentified* sp.)	17		50	7.6	6.2	96 h	1200
Midge	17		50	7.6	6.2	24 h	60
(*Chironomus* sp.)	17		50	7.6	6.2	96 h	20
Midge (*Chironomus*	20		50	6.8		24 h	1074
riparius,	20		50	6.8		48 h	316
intermittent flow conditions)	20		50	6.8		96 h	100
Midge (*Chironomus*	20		50	6.8		24 h	1028
riparius, static	20		50	6.8		48 h	750
conditions)	20		50	6.8		96 h	547

4. Toxicity in terrestrial receptors

4.1. Birds

In exposed birds, the highest mercury levels are generally found in liver and kidneys. Methyl mercury is more readily absorbed than inorganic mercury and it exhibits a longer biological half-life. Depending on speciation, mercury occurs in different compartments of the birds' eggs; methyl mercury tends to concentrate in the white and inorganic mercury in the yolk (World Health Organization, 1989). In aquatic systems, piscivorus birds tend to have higher mercury levels than non-fishing birds. Of these, double-crested cormorants appear to be most efficient at accumulating high levels of mercury.

In terrestrial systems, seed-eating birds, small mammals, and their predators can retain mercury at high levels in areas where methyl mercury fungicides are used. Levels of mercury in non-piscivorus birds are lower, but in many cases still significantly higher than area background. In general, greater accumulations occurred in the livers of fish-eating birds (0.89–30.9) than in the kidneys of non-fish eating birds (0.27–0.60) (Rand, 1995).

In one study, mercury in the liver of adult birds from a contaminated freshwater system showed increasing average concentrations with increasing trophic position: vegetarians, 6.64 ppm (w/w); invertebrate feeders, 12.4; omnivores 26.6; fisheaters; 40.2; scavengers, 54.4 (Zillioux et al., 1993). Braune (1987) surveyed nine species of marine birds from the New Brunswick region of Canada, including cormorants, eiders, guillemots, phalaropes, gulls, terns and kittiwakes. He reported a progressive decrease in mercury concentration from the innermost (secondary) to the outermost (primary) feathers in Bonaparte's gulls, herring gulls, black-legged kittiwakes and Arctic terns. Cormorants, guillemots and eiders, which feed predominantly on fish, had the higher tissue mercury levels, whereas birds such as kittiwakes and phalaropes, which consume mainly pelagic invertebrates, had the lowest mercury levels.

In a 1984 paper profiling mercury exposures in mallards, pheasants and bald eagles (Wiemeyer et al., 1984), mallards that were fed a diet containing 0.5 ppm mercury laid fewer eggs and produced fewer young than controls; average residues in eggs from three generations ranged from 0.79 to 0.86 ppm. Pheasants given mercury in their diet produced significantly fewer eggs and had higher embryo mortality compared to controls; mercury residues in their eggs ranged from 0.9 to 3.1 ppm. Mercury residues in eggs of pheasants with significantly reduced hatchability ranged between 0.5 and 1.5 ppm in one study, and 1.3 and 2.0 ppm in another study (Wiemeyer et al., 1984). Only 10 bald eagle eggs in the study contained more than 0.50 ppm mercury, a level at which adverse effects on reproduction might be expected.

Bird feathers are useful for biological monitoring for methyl mercury exposure. Analysis of feathers can allow recapitulation of past exposure. In general, liver and kidney have much higher levels of mercury than other bird tissues (World Health Organization, 1989). However, feathers are of particular interest because non-destructive sampling would allow acquisition of more data points; molting and feather-replacement patterns can be used to relate mercury concentration with migration history and source area; as a mechanism of waste elimination. Up to 70% of the body burden of mercury can be found in feathers; and a high degree of correlation has been shown between mercury accumulation in feathers and other tissues (Braune, 1987).

As in other receptors, organomercury compounds are more toxic in birds than inorganic forms, and cause reproductive impairment (World Health Organization,

Table 3
Toxicity of inorganic mercury (mercuric chloride) to fish (World Health Organization, 1989)

Organism	Temperature (°C)	Alkalinity (mg $CaCO_3$/l)	Hardness (mg $CaCO_3$/l)	pH	DO (mg/l)	Endpoint (LC_{50})	Water Conc. (μg/l)
Tilapia (*Tilapia mossambica*)	28–30				>4.8	48 h	1000
	28–32	7.7-11.7	32–38	7.0–7.3	5.4–6.2	24 h	1256
	28–32	7.7–11.7	32–38	7.0–7.3	5.4–6.2	48 h	1108
	28–32	7.7–11.7	32–38	7.0–7.3	5.4–6.2	72 h	739
Catfish (*Heteropneustes fossilis*)	28					96 h	350
Catfish (*Sarotherodon mossambicus*)	28–32	7.7–11.7	32–38	7.0–7.3	5.4–6.2	24 h	1700
	28–32	7.7–11.7	32–38	7.0–7.3	5.4–6.2	48 h	1500
	28–32	7.7–11.7	32–38	7.0–7.3	5.4–6.2	72 h	1000
	28					96 h	75
Catfish (*Channa marulius*)	24–27.5	165–190	245–285	7.1–7.7	5.5–8.2	24 h	860
	24–27.5	165–190	245–285	7.1–7.7	5.5–8.2	96 h	314
	24–27.5	165–190	245–285	7.1–7.7	5.5–8.2	240 h	131
Rainbow trout (*Oncorhynchus mykiss*) length (mm) (0.6–3.0)	9.3–10.7	70	101	8.55	>8.0	24 h	903
Rainbow trout [9.1–15.5]	5		90	7.5–7.8		48 h	650
	5		90	7.5–7.8		96 h	400
Rainbow trout [13.2–21.3]	10		90	7.5–7.8		48 h	450
	10		90	7.5–7.8		96 h	280
Rainbow trout [18.5–27.8]	20		90	7.5–7.8		48 h	300
	20		90	7.5–7.8		96 h	220
Rainbow trout [51–76] (Study performed with mercurous nitrate)		82–132		6.4–8.3	4.8–9.0	96 h	33
Flounder (adult) (*Platichthys flesus*)	15					48 h	3300
Banded killifish (*Fundulus diaphanus*)	28		55	8.0	6.9	24 h	270
	28		55	8.0	6.9	48 h	160
	28		55	8.0	6.9	96 h	110
Striped bass (*Roccus saxatilis*)	28		55	8.0	6.9	24 h	220
	28		55	8.0	6.9	48 h	140
	28		55	8.0	6.9	96 h	90
Pumpkinseed (*Lepomis gibbosus*)	28		55	8.0	6.9	24 h	410
	28		55	8.0	6.9	48 h	390
	28		55	8.0	6.9	96 h	300
White perch (*Roccus americanus*)	28		55	8.0	6.9	24 h	420
	28		55	8.0	6.9	48 h	340
	28		55	8.0	6.9	96 h	220
Carp (*Cyprinus carpio*)	28		55	8.0	6.9	24 h	330
	28		55	8.0	6.9	48 h	210
	28		55	8.0	6.9	96 h	180
American eel (*Anguilla rostrata*)	28		55	8.0	6.9	24 h	250
	28		55	8.0	6.9	48 h	190
	28		55	8.0	6.9	96 h	140
Mummichog (*Fundulus heteroclitus*)	20			8.0		96 h	2000
	20			7.8	<4	24 h	23 000
	20			7.8	<4	96 h	800
	20			7.8	<4	168 h	800

Table 4
Toxicity of organic mercury (methyl mercuric chloride) to fish (World Health Organization, 1989)

Organism weight (g)	Temperature (°C)	Alkalinity (mg $CaCO_3$/l)	Hardness mg $CaCO_3$/l)	pH	DO (mg/l)	Endpoint (LC_{50})	H_3CHgCl (μg/l)
Blue gourami	26–28			7.4	10	24 h	123
(*Trichogaster trichopterus*)	26–28			7.4	10	48 h	94.2
(1.5–2.0)	26–28			7.4	10	96 h	89.5
Rainbow trout (fry)	9.3–10.7	70	101	8.55	>8.0	24 h	84
(*Oncorhynchus mykiss*)	9.3–10.7	70	101	8.55	>8.0	48 h	45
	9.3–10.7	70	101	8.55	>8.0	96 h	24
Rainbow trout (fingerling)	9.3–10.7	70	101	8.55	>8.0	24 h	125
[0.6–3.0]	9.3–10.7	70	101	8.55	>8.0	48 h	66
	9.3–10.7	70	101	8.55	>8.0	96 h	42
Rainbow trout (Juvenile)	10		90	7.5–7.8		24 h	25
[22.9] (Study conducted	18		250			24 h	5
using phenyl mercuric	18		250			48 h	4
acetate)							
Brook trout (juvenile)	11–13	41–44	45–46	6.9–7.6	7.7	96 h	75
(*Salvelinus fontinalis*)							
Lamprey (larvae)	12	150	146	8–8.5		24 h	>166
(*Petromyzon marinus*)	12	150	146	8–8.5		48 h	88
(0.3–3.0)	12	150	146	8–8.5		96 h	48

Table 5
Toxicity of inorganic mercury to the embryo-larval stages of fish (World Health Organization, 1989)

Organism	Static/Flow-through	LC_{50} (μg/l)	95% confidence limits
Rainbow trout	Static[a]	4.7	4.2–5.3
	Flow[b]	<0.1	
Channel catfish	Static	30.0	26.9–33.2
	Flow	0.3	0.2–0.4
Bluegill sunfish	Static	88.7	73.5–106.3
Goldfish	Static	121.9	112.3–132.1
	Flow	0.7	0.6–0.8
Redear sunfish	Static	137.2	115.0–162.8
Largemouth bass	Static	140.0	128.7–151.9
	flow	5.3	5.0–5.6

Exposure was initiated 30 min. to 2 h after spawning and continued through to four days post hatching. Hatching times were 24 days for rainbow trout, 6 days for channel catfish, and 3–4 days for the other fish. Therefore, total exposure was as follows: rainbow trout 28 days; channel catfish 10 days; and all other fish 7–8 days.

[a] *Static*: static conditions with water renewed every 12 h.

[b] *Flow*: flow-through conditions (mercury concentration in water continuously maintained).

1989). Interpretation of the results of laboratory experiments on birds should take into account that practically all studies have been carried out using predatory and gallinaceous (seed eating) birds, which are unrepresentative of bird species as a whole.

The toxicity of mercury to avian receptors is summarized in Table 8.

4.2. Plants

The accumulation of mercury in terrestrial plants increases with increasing soil mercury concentration. Soil type has considerable influence on this process (i.e., a high organic matter content will decrease uptake). Generally, the highest concentrations of mercury are

Table 6
Toxicity of mercuric chloride to amphibians (World Health Organization, 1989)

Organism	Temperature (°C)	Alkalinity ($mgCaCO_3/l$)	Hardness ($mgCaCO_3/l$)	pH	Endpoint (LC_{50})	$HgCl_2$ (μg/l)
Frog (tadpole stage) (*Rana hexadactyla*)	13–16	24–40	13–80	6.2–6.7	24 h	762
	13–16	24–40	13–80	6.2–6.7	48 h	121
	13–16	24–40	13–80	6.2–6.7	72 h	68
	13–16	24–40	13–80	6.2–6.7	96 h	51
Clawed toad (3–4 week larva) (*Xenopus laevis*)	19–21				48 h	100
Toad (tadpole stage) (*Bufo melanostictus*)	29–34	120–160	165–215	7.1–7.6	12 h	69.8
	29–34	120–160	165–215	7.1–7.6	24 h	52.8
	29–34	120–160	165–215	7.1–7.6	48 h	45.6
	29–34	120–160	165–215	7.1–7.6	96 h	43.6

Table 7
Toxicity of inorganic mercury to the embryo-larval stage of amphibians (World Health Organization, 1989)

Organism	LC_{50} (μg/l)	95% Confidence limits
Narrow-mouthed toad (*Gastrophryne carolinensis*)	1.3	0.9–1.9
Southern grey tree frog (*Hyla chrysoscelis*)	2.4	1.5–3.4
Squirrel tree frog (*Hyla squirrella*)	2.4	1.5–3.8
Barking tree frog (*Hyla gratiosa*)	2.5	1.7–3.4
Grey tree frog (*Hyla versicolor*)	2.6	1.2–4.2
Spring peeper (*Hyla crucifer*)	2.8	1.9–3.9
Leopard frog (*Rana pipiens*)	7.3	4.8–10.0
Cricket frog (*Acris crepitans blanchardi*)	10.4	8.5–12.6
Red-spotted toad (*Bufo punctatus*)	36.8	18.3–51.1
Green toad (*Bufo debilis debilis*)	40.0	25.6–52.2
River frog (*Rana heckscheri*)	59.9	53.8–65.9
Fowlers toad (*Bufo fowleri*)	65.9	44.0–84.0
Pig frog (*Rana grylis*)	67.2	54.3–79.5
Marbled salamander (*Ambystoma opacum*)	107.5	72.5–153.5

Exposure was under static conditions (water renewed every 12 h), and was initiated 30 min to 2 h after spawning and continued to four days post-hatching. Hatching times varied from 2.6 to 3.4 days, therefore total exposure was between 6.6 and 7.4 days.

found at the roots, but translocation to other organs occurs. In contrast to higher plants, mosses are known to take up mercury via atmospheric deposition.

Huckabee et al. (1983) monitored levels of mercury in vegetation in the vicinity of a mercury mine in Spain. Mean concentrations of total mercury in vegetation ranged from >100 mg/kg within 0.5 km of the mine to 0.20 mg/kg 20 km from the mine. Moreover, there was a significantly higher mercury content in vegetation 25 km upwind from the mine (approximately 10 times the background level). Mosses were found to contain greater concentrations of mercury (7.58 mg/kg) than herbaceous plants (2.25 mg/kg). Vegetation samples were collected and analyzed in the spring. There was a correlation between distance from the mine and plant mercury content for woody plants and mosses but not for herbaceous plants. No quantifiable levels of methyl mercury were found in any of the plants, although traces were seen in several samples, indicating a methyl mercury content of less than 10 picograms (pg) per sample.

Suszcynsky and Shann (1995) conducted an experiment on the absorption, phytotoxicity, and internal distribution of mercury in tobacco plants (*Nicotiana miersii*). The plants were exposed to elemental mercury (Hg^0) through the shoot or ionic mercury (Hg^{2+}) through the root. Tobacco plants were grown for a 10 day period at varying concentrations in hydroponic chambers. Plants exposed to elemental mercury accumulated mercury in the shoots with no movement to roots. Visible signs of elemental mercury stress were apparent at 1.0 mg/m^3 exposure levels and greater. Root-exposed plants showed accumulation of mercury in the roots with movement to the shoots by day 10. Inhibition of root and shoot growth occurred at treatment levels of 1.0 μg/ml and greater with very limited tissue damage at higher treatment levels.

Table 8
Toxicity of mercury to birds (World Health Organization, 1989)

Species	Age	Compound[a]	Endpoint[b]	Concentration (mg/kg)
Japanese quail (*Coturnix conturnix japonica*)	14 days	Methyl mercuric chloride	Acute LD_{50}[c]	11
	14 days	Mercuric chloride	Acute LD^{c}_{50}	42
	2 months	Ceresan M	Acute LD^{c}_{50}	668
	4 months	Ceresan L	Acute LD^{c}_{50}	1498
	14 days	Methyl mercuric chloride	5 day LC_{50}	47
	14 days	Mercuric chloride	5 day LC_{50}	5086
	14 days	Methoxyethylmercury chloride	5 day LC_{50}	1750
	14 days	Phenyl mercuric acetate	5 day LC_{50}	614
	14 days	Morsodren	5 day LC_{50}	45
	14 days	Ceresan M	5 day LC_{50}	1
Pheasant (*Phasianus colchicus*)	12 months	Ceresan M	Acute LD^{c}_{50}	360
	3–4 months	Ceresan L	Acute LD^{c}_{50}	1190
	3–4 months	Phenyl mercuric acetate	Acute LD^{c}_{50}	169
	10 days	Mercuric chloride	5 day LC_{50}	3790
	10 days	Methoxyethylmercury chloride	5 day LC_{50}	1102
	10 days	Phenyl mercuric acetate	5 day LC_{50}	2350
	10 days	Morsodren	5 day LC_{50}	64
	10 days	Ceresan M	5 day LC_{50}	146
Mallard duck (*Ana platyrhynchos*)	6–8 days	Ceresan M	Acute LD^{c}_{50}	>2262
	3 months	Ceresan M	Acute LD^{c}_{50}	>2262
	3 months	Ceresan L	Acute LD^{c}_{50}	>2000
	3–4 months	Phenyl mercuric acetate	acute LD^{c}_{50}	878
	10 days	Mercuric chloride	5 day LC_{50}	>5000
	10 days	Methoxyethylmercury chloride	5 day LC_{50}	280
	10 days	Phenyl mercuric acetate	5 day LC_{50}	1175
	5 days	Morsodren	5 day LC_{50}	51
	10 days	Morsodren	5 day LC_{50}	60
	5 days	Ceresan M	5 day LC_{50}	54
	10 days	Ceresan M	5 day LC_{50}	50
Bobwhite quail (*Colinus virginianus*)	2–3 months	Ceresan L	acute LD^{c}_{50}	1060
	14 days	Ceresan M	5 day LC_{50}	70
Prairie chicken (*Tympanuchus cupido*)	unknown	Ceresan M	Acute LD^{c}_{50}	360
Chukar partridge (*Alectoris chukar*)	4 months	Ceresan M	Acute LD^{c}_{50}	841
Grey partridge (*Perdix perdix*)	9–20 months	Ceresan M	Acute LD^{c}_{50}	550
Rock dove (*Columba livia*)	unknown	Ceresan M	Acute LD^{c}_{50}	714
Fulvuous whistling duck (*Dendrocygna bicolor*)	3–6 months	Ceresan L	Acute LD^{c}_{50}	1680

[a] Morsodren = cyano methyl mercury guanidine (1.51% mercury); ceresan M = N(ethylmercury)-p-toluenesulfonalide (3.2% mercury); ceresan L = methyl mercury 2,3-di-hydroxyl propyl mercaptide+ methyl mercury acetate (2.25% mercury).
[b] Concentrations expressed as mg/kg food, unless stated otherwise.
[c] Concentrations as mg compound per kg body weight in a single oral dose (i.e., birds were fed with a dosed diet for five days followed by a 'clean' diet for three days).

The effect of leaf litter from trees may complicate interpretation of mercury deposition. In addition to root uptake and translocation to the leaves, tree leaves apparently trap atmospheric mercury (Godbold, 1991). After leaf fall, the amount of deposition will be greater than in areas that do not contain trees and shrubs.

Moss samples and peat cores that contain leaf litter will likely have higher mercury concentrations, leading to higher estimates of mercury deposition (Godbold, 1991).

In higher plants, exposure to mercury reduced photosynthesis and transpiration, water uptake, and chlorophyll synthesis (Godbold and Huttermann, 1986). In spruce (*Picea abies*) seedlings exposed to mercury and methyl mercury, changes in photosynthesis and transpiration were attributed to mercury-induced root damage rather than the direct action of metals in the needles. The authors of this study suggest that in seedlings exposed to mercury, the primary damage to roots affects nutrient and water supply to the needles. In the young root tips the effect of mercury on mineral levels were more pronounced than in older root parts. Exposure to both organic and inorganic mercury resulted in a loss of potassium (K^+), magnesium and manganese, and accumulation of iron. A dramatic decrease in the level of K^+ in the root tip nuclei of the mercury seedlings supports the idea that both inorganic and organic mercury may cause changes in root tip cell membrane integrity. However, there are differences in how these two forms of mercury carry out cell injury. While inorganic mercury ($HgCl_2$ in particular) affects the plasma membrane, methyl mercury may primarily affect organelle metabolism in the cytoplasm which subsequently affects membrane integrity. Mercury induced root damage may have serious consequences for nutrient and water supply to above ground plant parts, and should be taken into account when assessing the effect of mercury on the physiology of leaves or needles (Godbold and Huttermann, 1986). In another study on mercury toxicity in spruce seedlings (Godbold, 1991), methyl mercury was found to significantly decrease root elongation, while mercury chloride had no impact on root length. The authors concluded that the greater toxicity of methyl mercury is due to a higher toxicity to root metabolism, rather than a greater root uptake. It was also determined that several factors probably contributed to the seedling receiving a greater exposure to methyl mercury via soil acidification, decreases in soil pH, an increase in humus layer depth, and microbial activity.

Some researchers have implemented mercury biomonitoring using grass cultures (Temmerman et al., 1986). Grass cultures can provide a reliable indication of the average concentrations found in leafy vegetables. However, for washed leafy vegetables, the concentrations are usually lower than those found in grass. Mercury accumulation in herbs, especially in perennials, is far higher than that in grasses and leafy vegetables. A clear relationship exists between the mercury concentrations found in herbs, and those in grass cultures. However, no such relationship was found in roots, tuber, bulbs (10 times less accumulation than in grass cultures), fruits or leguminous vegetables.

4.3. Soil invertebrates

The experimental information available on the effects of mercury on terrestrial invertebrates is insufficient to make any proper appraisal. The following describes some of the best information available.

Marigomez et al. (1986) fed the terrestrial slug (*Arion ater*) on a diet containing mercuric chloride at 0, 10, 25, 50, 100, 300 or 1000 mg/kg for 27 days. Slug mortality was low in all treatments (a maximum of three deaths out of 24 animals per treatment) and unrelated to the dose. These results indicate that slug exposure to mercury at levels likely to be found in the environment will not kill them. However, a significant reduction in food consumption was noted at exposures of >10 mg/kg, with this effect increasing with higher dosage. Additionally, a significant dose-related reduction in growth rate also occurred.

Lindqvist et al. (1995) studied excretion and distribution of inorganic mercury and methyl mercury in the predatory beetle (*Pterostichus niger*). Beetles were fed mercury-spiked insect larvae for a 30-day period. After two weeks, inorganic mercury had decreased to approximately 1% of the ingested amount, while higher levels of methyl mercury were retained. At day 30, 60% of the ingested methyl mercury was retained in the beetle. Inorganic mercury was predominately harbored in the gut, while methyl mercury was evenly distributed in most body tissues. Results of other studies have shown similar results. Methyl mercury was found to be accumulated from food in nymphs of a mayfly about 60-fold more than the inorganic form (Saouter et al., 1993). Methyl mercury was also transferred to a higher degree than was inorganic mercury from fish carcass to fly larvae and further concentrated in predatory beetle preying on the fly larvae (Nuorteva and Nuorteva, 1982).

Abassi and Soni (1983) kept adult earthworms (*Octochaetus pattoni*) in cement tanks at a density of 120 animals/m^3, the average density of this species in the wild. Mercuric chloride was added to a mixture of soil and animal dung in the tanks at dose levels of 0, 0.5, 1.0, 2.0 or 5.0 mg mercury/kg. The experiment duration was 60 days and estimates of mortality were used to give LC_{50} values. The LC_{50} was 2.39 mg/kg at 10 days and had decreased to 0.79 mg/kg over the 60-day exposure period. As mortality progressed throughout the experimental period, the surviving earthworms reproduced significantly more than controls. The reason for this effect is unclear. Beyer et al. (1985) exposed the earthworm (*Eisenia foetida*) to soil containing methyl mercuric chloride at 0. 1, 5, 25 or 125 mg/kg. All worms dosed at 25 or 125 mg/kg died within 12 weeks. Survival

rates within the other treatment group were 97%, 92% and 79%, for doses of 0, 1 and 5 mg/kg, respectively. Regeneration of amputated segments was normal after treatment with methyl mercuric chloride at 1 mg/kg soil, but reduced or eliminated in the 5 mg/kg treatment group.

4.4. Mammals

Few studies have been published on truly wild, non-laboratory mammals. To date, almost all of the work in this field has focused on mink and prairie vole. The available evidence indicates that toxic effects, including reproductive changes, can be produced through exposure to some mercury species. Methyl mercury has been found to be more toxic than inorganic mercury in these terrestrial mammals. It has been shown to be a potent neurotoxin to most vertebrates, because they lack external barriers or internal detoxification systems (Klaassen et al., 1986).

Aulerich et al. (1974) dosed the diet of mink with either 5 mg/kg methyl mercury (as contained in Ceresan L, which contains 2.25% mercury) or 10 mg mercuric chloride/kg. No adverse effects in either group was observed for three weeks. However, after 25 days, the mink dosed with organic mercury showed ataxia, loss of balance, anorexia and loss of weight. By day 29, paralysis, tremors and death were observed. Attempts to arrest the above symptoms were made by reverting back to a control diet, along with injections of either EDTA (a synthetic metal chelator) or methionine (a natural biochemical chelator). Such attempts had no effect on the mink symptoms or time to death. In the mercuric chloride treatment group, no adverse effects were observed. Mercuric chloride did not affect reproductive performance and teratological effects were absent. Although there was a significant reduction in the weight of the kits from treated parents at birth, this had recovered by four weeks of age.

Wren et al. (1987a,b) fed adult mink a daily diet containing methyl mercury at 1 mg/kg for three months. Later, due to a high rate of mortality, the diet was administered every other day for another three month period. The initial, daily-dosed diet resulted in the death of 8 out of 12 females, and 1 out of 4 males. There were no observed effects of these treatments on the thyroid, pituitary or adrenal glands, or on serum triiodothyronine (T3) or thyroxine (T4) during the experimental period. In addition, mortality could not be exclusively attributed to mercury since the animals were kept outside during the winter and therefore could have suffered from cold stress. The fertility of male mink and number of kits born per female were not affected in either mercury treatment group.

Hartke et al. (1976) calculated an acute LD_{50} of 10 mg/kg body weight for phenyl mercuric acetate (PMA) in female prairie voles (*Microtus ochrogaster*), after intra peritoneal injection. Female voles were also injected on days 8, 9 and 10 of gestation with 0.06–5.0 mg PMA/kg body weight. Some normal fetuses and some resorption sites (where implantation had occurred but the fetal material had been reabsorbed) were found in voles injected with 0.5 mg/kg or less on days 8 and 9 of gestation. Animals treated with >1.0 mg/kg had no live fetuses, but all maintained resorption sites in the uterus. Similar results were found for voles treated on day 10.

5. Transport and fate of mercury

5.1. Environmental matrices

Atmospheric transport of elemental mercury (Hg^0) in the vapor phase represents the major pathway of global deposition. Calculations based on mercury content of the Greenland ice cap show an increase from as early as the year 1900 to recent times, and suggest that the incremental increase in background levels in rain water is related to man-made release. As much as one-third of atmospheric mercury may come from industrial sources. Regardless of source, both organic and inorganic forms of mercury may undergo environmental transformation. Metallic mercury (Hg^0) may be oxidized to inorganic divalent mercury (Hg^{2+}), particularly in the presence of organic material. Divalent inorganic mercury may, in turn, be reduced to metallic mercury when conditions are appropriate for reducing reactions to occur. This is important in terms of the global mercury cycle since this may be a significant source of mercury vapor released into the atmosphere. A second potential conversion of divalent mercury is methylation to di methyl mercury by anaerobic bacteria, the by-product of which diffuses into the atmosphere and eventually returns to the earths crust or bodies of water via rainfall (Klaassen et al., 1986).

Methyl mercury does not follow ideal liquid–liquid partitioning (K_{ow}) behaviors. Although it accumulates in aquatic organisms to a high degree (on the order of 10,000–100,000 times water concentrations), the small K_{ow} does not reflect this potential. Transfer from water to organic phases occurs mainly in the form of neutral ion pairs (CH_3 HgCl and CH_3 HgOH) (Rand, 1995).

Wetlands are known to be effective at trapping and releasing mercury (Zillioux et al., 1993). Colloidal manganese oxides may sorb inorganic mercury and thereby affect cycling in lake waters. In intact peat, mercury remains sequestered with the organic matter and does not appreciably leach during seasonal aerobic and anaerobic cycles. However, drying of the peat can substantially increase leaching, probably because phys-

ical properties of the peat are changed. Wetland drainage waters in Sweden were measured for methyl mercury. It was determined that disturbed wetlands produced significantly more methyl mercury than in undisturbed wetlands. In Wisconsin, a study of 41 surface waters showed a strong correlation between DOC and filtered total mercury. In fact, the highest mercury concentration occurred in the drainage from a brown water wetland having concentrations of 30–40 mg/l of DOC near Lake Superior, remote from any local mercury sources (Zillioux et al., 1993).

Increased mobility of organic-matter associated mercury occurs when newly formed reservoirs are flooded. This suggests that mineral mercury and atmospherically deposited mercury that has accumulated over time might be mobilized as a result of disturbance of wetland systems. Natural and anthropogenic wetland disturbance could result from peat mining; hydrologic disturbance; drying of soils rich in organic matter; fires; and successional changes in vegetation (Zillioux et al., 1993).

5.2. Biological matrices

The liver is the main site of methyl mercury biotransformation in animals. The liver transforms harmful compounds into metabolites which are excreted directly into bile for continued detoxification. Once a compound is excreted into bile and enters the small intestine, it is either reabsorbed in the gut or eliminated in feces. However, methyl mercury is reabsorbed in a process called enterohepatic recirculation (Gordon and Skett, 1986). When methyl mercury undergoes this enterohepatic cycle the overall result is that mercury is retained by the organism and has a substantially increased half-life. If the organomercury concentration is high enough, this metabolism-based cycle can result in prolonging the pharmacological activity of mercury (Gordon and Skett, 1986).

In a study on mercury distribution in the Blue Tilapia (*Oreochromis aureus*), exposure to Hg^{2+} at 0.5 and 0.1 mg/l for varying periods resulted in excretion of mercury in the bile, reaching 3.92 μg mercury/l bile after exposure to 0.1 mg/l mercury for one week. This also demonstrates an efficient methylation response to inorganic mercury exposure. Therefore, bile seems to play an important role in the inter-organ distribution of mercury in fishes (Allen, 1994).

Methyl mercury passes the blood–brain barrier and nuclear membranes to react directly with both cellular and nuclear components. Accumulation of mercury in the brain, compared to blood and muscle, is much less in fish than in mammals. The blood-to-brain mercury ratio has been shown to be 100 times higher in some terrestrial mammals than in fish (Gordon and Skett, 1986).

6. Conclusions

When evaluating the environmental hazards of mercury it is necessary to extrapolate from laboratory experiments to ecosystems. This must be done with extreme caution for the following reasons:

- Speciation of mercury, and its adsorption to matrices such as soil, sediment, organic matter, and biota limit its availability to other organisms.
- Environmental variables such as temperature, pH and chemical composition of water, soil type, and geology have all been shown to affect uptake and effects of mercury.
- There are few data measuring mercury availability to organisms. Most data represent nominal or total metal concentration, rather than the more bioavailable, and therefore most toxic, fraction of total mercury impacting an organism.
- Organisms are most commonly exposed to mixtures of metals in the environment, however data is limited on the behavior of mixtures of metals from controlled experimental work.
- Some studies have shown that selenium may be capable of inhibiting the negative effects of mercury.
- It is probable that subtle disturbances to a community occur at much lower concentrations (chronic exposures) of mercury than those suggested in this paper (largely based on acute effects).

Aquatic organisms at all levels accumulate mercury into tissues. This mercury is retained for long periods if it is in an organic (methylated) form. A number of factors affect the susceptibility of aquatic organisms to mercury. These include the life-cycle stage (the larval stage is particularly sensitive), the development of tolerance, water temperature and hardness. Some basic limitations to keep in mind are that toxic effects have been produced experimentally at concentrations much higher than those found in non-polluted aquatic environs, and most studies have focused on acute lethality, using only inorganic mercury compounds.

Mercury compounds are acutely toxic to freshwater microorganisms. Using photosynthesis and/or growth as parameters, the no-observed-effect-level (NOEL) for inorganic mercury lies between 1 and 50 μg/l, depending on the organism, density of cells in culture, and experimental conditions. For organomercury compounds, the NOEL is 10–100 times lower. Aquatic plants sustain damage after exposure to inorganic mercury at concentrations of 800–1200 μg/l. Many aquatic invertebrates are sensitive to mercury, particularly as larvae. For the most sensitive species, i.e., *Daphnia magna*, the NOEL for reproductive impairment is 3 μg/l for inorganic mercury and <0.04 μg/l for methyl mercury. In both aquatic plants and invertebrates, organic mercury compounds are toxic at concentrations 10–100 times less than inorganic mercury.

Freshwater fish show lethal responses to mercury in acute nominal concentrations starting from approximately 30 μg/l. Larvae under the same (static) conditions are 10 times more sensitive. In flow-through tests, fish are up to 100 times more sensitive than in static microcosms. In both static and flow-through tests, organomercury compounds are approximately 10 times more toxic than inorganic forms. In some fish receptors, the NOEL may be well below 0.01 μg/l. Aquatic developmental stages of amphibia demonstrate sensitivity to mercuric compounds similar to that of fish.

Mercury has been shown in laboratory studies, to be toxic to terrestrial organisms over a broad range of concentrations. However, most of these studies have used unrealistically high exposure levels (particularly in birds) or unrealistic exposure routes (hydroponic culture of plants).

Acute effects may not be seen in terrestrial plants growing in natural soils, nor in terrestrial birds or mammals, other than by exposure to mercurials used in fungicides. Other effects seen in birds are derived from their exposure in marine environments. Birds, particularly coastal species or those eating prey that feed in estuaries, are most impacted by mercury contamination. Mercury has adversely affected breeding and may have influenced avian population stability.

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