

## Chapter 6 - ECOLOGICAL EFFECTS OF MERCURY

### A. Introduction

There is a huge and rapidly growing scientific literature on the distribution of mercury in ecosystems. Mercury has been measured in aquatic and terrestrial invertebrates, in a variety of plants, and in many higher organisms including humans. High concentrations of mercury have been associated with developmental and behavioral abnormalities, impaired reproduction and survival, and in some cases with direct mortality. There is evidence that mercury may act synergistically with organic pollutants such as PCBs (Gochfeld 1975). The Task Force was charged to address the question: "Does the current body of scientific knowledge indicate that mercury in the environment is at levels of potential wildlife and ecological impact?"

Mercury is among the most extensively studied of all the environmental pollutants, but the information on the distribution in various environmental or body compartments exceeds information on effects at the organism, population and ecosystem level. There remain substantial gaps in our understanding of the effects of mercury on different kinds of organisms, on different trophic levels, and on ecosystem function itself. Ecological effects can be measured by some impacts, presumably adverse, on microorganisms, plants, and animals that make up the decomposer, producer and consumer trophic levels of ecosystems. The endpoints in individuals exposed to mercury can include changes in behavior, physiology, reproduction, or longevity, as well as acute effects such as morbidity and mortality. Endpoints among species can include changes in survivorship and population structure, population declines or local extinction. Ecological endpoints include changes among the species interactions, usually reflected in food webs, as well as changes in the cycling of matter or the patterns of energy use and production.

As mercury is transferred from one trophic level to another, there can be direct contamination (uptake from the water column through gills or by ingestion) as well as uptake from food. These can be separated by raising organisms in both contaminated and "clean" water and providing them with food that has low and high levels of mercury. Simon and Boudou (2001a,b) have shown that crayfish and carp take up both  $Hg^{++}$  and MeHg by both routes. Carp have concentration factors (compared to water) from 1000 for  $Hg^{++}$  to 13,000 for MeHg. The trophic transfer rate for carp was estimated at 2% for  $Hg^{++}$  and 13% for MeHg (Simon and Boudou 2001a) and for crayfish at close to zero for  $Hg^{++}$ , but 20% for MeHg (Simon and Boudou 2001b).

Eisler (1987) summarized the ecotoxicology of mercury. Mercury and mercury compounds have no known beneficial biological function. Forms of mercury with relatively low toxicity can be transformed into forms with very high toxicity. Methylmercury can be accumulated through food chains resulting in higher exposure to upper trophic levels. Mercury is a mutagen and teratogen and the uses and releases of mercury should be reduced, "because the difference between tolerable background levels of mercury and harmful effects in the environment is exceptionally small." (Eisler 1987)

Although U.S. EPA (1997d,e) stated that the ecosystem effects of mercury are incompletely understood, and that no studies were found of the effects on intact ecosystems, they did identify the characteristics of ecosystems potentially at risk from mercury releases to air. These included systems located in areas that experience high levels of atmospheric deposition, those with surface waters already impacted by acid deposition, those possessing characteristics other than low pH that result in high levels of mercury bioaccumulation in

aquatic biota, and those with species which have been subject to point source discharges of mercury (e.g., industrial outfalls).

Many studies have shown that mercury becomes widespread in both abiotic and biotic components of ecosystems (Pillay et al. 1972; Peakall and Lovett, 1972). Much of the effort involving mercury investigations has focused on aquatic rather than terrestrial ecosystems due to the methylation and bioaccumulation of the highly toxic MeHg in these systems. In addition, most studies have examined the effects of mercury on individuals or species and have not examined ecosystem effects (e.g., biodiversity, food web structure, energy flows).

## B. Biomagnification

Biomagnification (sometimes called bioamplification) of mercury through a food chain is a primary cause for much of the concern with this metal. This term defines the process where at each level in a food chain, from bacteria to plankton to tiny crustacea, small fish, larger fish, and fish-eaters, organisms take in more mercury than they excrete thereby accumulating the excess in their organs. Thus the ultimate concentration in any organism is higher than the mercury concentration in its food. This results in elevated concentrations of mercury in higher trophic (feeding) levels of the food chain. These concentrations can be harmful to the organism itself, or to predators of those organisms. Figure 2.1 illustrates a hypothetical pattern of biomagnification, through which an infinitesimally low concentration of mercury (parts per trillion in water) can reach biologically dangerous concentrations (parts per million) in larger predators including humans. These theoretical values are reflected in Table 2.5. It should be noted that each step shown is for illustration purposes only and is not necessarily reflective of actual values measured in the environment.

**Table 2.5. Biomagnification of Methylmercury in a Hypothetical Aquatic Food Chain.**

Trophic Level	Concentration
Water	1 ng/L = 1 ppt
Bacteria and phytoplankton	10 ng/kg of water = 10 ppt
Protozoan/zooplankton	100 ng/kg = 100 ppt
Insect larvae	1 µg/kg = 1 ppb
Fish fry	10 µg/kg = 10 ppb
Minnows	100 µg/kg = 100 ppb
Medium-sized fish	1 mg/kg = 1 ppm
Large predators (fish, birds, humans)	10 mg/kg = 10 ppm = 10µg/g

Mercury bioaccumulation was demonstrated in a terrestrial forest ecosystem in Slovenia. Total mercury levels in soil exceeded 2000 ppm close to the Iridja smelter, and ranged from 14 to 886 ppm further afield of which less than 1% (0.01 to 0.6%) was MeHg. Plant samples ranged from 50 ppm down to less than 1 ppm (mostly non-accumulator species), of which up to 2% were MeHg. Air mercury was mostly in the 1-100 ng/m<sup>3</sup> range, with up to 1 µg/m<sup>3</sup> close to the smelter and 1.8 ng/m<sup>3</sup> at a distant reference site. Generally, sites closer to the

smelter had much higher total and lower percent of MeHg than sites further away. Large herbivores (Roe Deer and Chamois) had mercury levels in the 20 to 20,000 ppb ranges (kidney>liver>muscle), with a maximum kidney concentration of 56 ppm. Methylmercury comprised less than 1% of total mercury in kidney, 4-10% in liver, and 35-48% in muscle (Gnamus et al. 2000). They had only three carnivore samples (two Lynx and one Wolf) that did not show much higher levels than in the herbivores, but the MeHg comprised about 10-20% of kidney, 17-33% of liver and 50-83% of the muscle MeHg (Gnamus et al. 2000), which suggests a preferential retention of MeHg from the prey. Thus, whereas the concentration factor(lynx-deer) was less than one for total mercury it exceeded one for MeHg, and was proportionally higher in control animals with lower total mercury, suggesting a ceiling effect of high mercury on the concentration factor.

A discussion of the tissue concentrations and effects of mercury on various invertebrate and vertebrate groups is provided below as an example of the ecological impacts of mercury. Toxicity testing results have generally indicated that early developmental stages of organisms were the most sensitive, and organomercury compounds, especially methylmercury, were more toxic than inorganic forms (Eisler 1987).

### **C. Toxicity of Mercury to Algae and Micro- and Macroinvertebrate**

Sjoblom et al. (2000) found that direct uptake from water by fly larvae (*Chaoborus* sp.) was ten times higher for MeHg than for  $Hg^{++}$ . Organic matter in the form of humic acids decreased the uptake of both MeHg and  $Hg^{++}$  substantially (Sjoblom et al 2000). Similarly, Lawrence and Mason (2001) found that amphipods living in organic-rich sediment accumulated less mercury than those living in organic-poor sediments.

Planktonic unicellular algae, such as *Chorella vulgaris*, have a high capacity to bioaccumulate organic mercury, as do fresh water macrophytes such as *Elodea densa*. Experimental exposure of this plant to a 1 ppb concentration of methylmercury chloride for 10 days produced an average concentration in the leaves of 15 ppm, a 15,000-fold bioconcentration factor. Primary consumers, such as the crustacean *Daphnia*, readily accumulate mercury by eating contaminated algae. They too accumulate a higher concentration of MeHg than their algal food. Thus begins the biomagnification of mercury in the food chain when the *Daphnia* are consumed by other organisms such as insect larvae or small fish.

Toxicity tests with the rotifer, *Brachionus calyciflorus*, designed to incorporate natural stressors such as food shortage, Cyanobacteria blooms, and predation have been conducted to determine if ecological relationships affect the impact of anthropogenic toxicants on rotifer populations. Results indicated that natural stressors (i.e., food limitation and Cyanobacteria) exacerbate the stress due to contaminants, such as mercury (Ceccine 1997).

Planarians (*Dugesia dorotecephala*) are aquatic flatworms used as a test species for mercury toxicity because of their sensitivity to this metal. These animals (about 1 cm long) ordinarily have remarkable powers of regeneration and are able, for example, to grow a new head after decapitation. However, grossly visible abnormalities in head regeneration occurred when decapitated planarians were exposed to 0.1 ppm methylmercury. A much more sensitive indicator of toxic effects is the suppression of fissioning, a natural process of division in these animals. Concentrations as low as 0.03 ppb, a concentration approximating the lower mercury levels found in “unpolluted” US streams and ocean water, were sufficient to inhibit fissioning (Best et al 1981).

Mercury exposures of approximately 0.1 ppm in water have been shown to severely affect heart rate rhythms in the freshwater crab, *Potamon potamios* and in the crayfish, *Astacus astacus*, interfering with the normal circadian rhythm patterns in these animals. Such effects on the heart are then followed by substantial mortality in these animals under experimental conditions (Styrishave & Depledge 1996). Exposure of fiddler crabs to 0.5 mg/L of mercury resulted in the inhibition of limb regeneration and ecdysis (molting) (Callahan & Weis, 1983). These levels are far above background levels in surface water. Even in highly contaminated Berry's Creek, mercury levels range from 0.74 to 17.6 µg/L (total mercury) (Exponent 1998). These values appear to be well below the 100-500 µg/L effect levels in the crustacean studies.

#### **D. Toxicity of Mercury to Terrestrial Invertebrates**

Earthworms, as an example of terrestrial invertebrates, exhibited complete mortality at methylmercury concentrations of 25 mg/kg (ppm) in soil in a 12-week exposure (Beyer et al. 1985). Inorganic mercury concentrations of 0.79 mg/kg in soil were toxic to 50% of the earthworms in a 60-day study, and 100% mortality was observed at 5 mg/kg (Abbasi and Soni 1983). However, it is not possible to determine the relative toxicity of these two forms of mercury based on these two earthworm studies, which used different systems and two different families of earthworms (Efroymsen et al. 1997).

#### **E. Toxicity of Mercury to Fish**

There is a very large but non-systematic literature on mercury levels in fish, particularly with regard to tissue concentrations, which bear on whether they are safe to eat. Thus fish have been studied more as a vector of human exposure than as an ecological target of mercury. Young fish hatch with a burden of mercury acquired from the egg, but, as they grow in size, the diet-derived mercury and mercury taken up directly from water exceed the egg contribution. Comparing Walleye larvae from a contaminated lake with those from uncontaminated lakes indicates that the MeHg concentration of young fish involves both maternal contributions via the egg as well as ingestion of MeHg from water and food (Latif et al. 2001).

In many studies of mercury in fish there is an increasing concentration with the size and age of the fish (e.g., Latif et al. 2001, Redmayne et al. 2001). Where this is not found, it may be due in part to inadequate sample size, or inadequate range in mercury concentrations or fish size (Huckabee et al. 1979). Hatching success of Walleye eggs declined significantly with increasing waterborne MeHg, even in the range of 0.1-7.8 ng/L. Likewise embryonic heart rate declined, but larval growth was not affected (Latif et al. 2001).

Largemouth Bass upriver from a presumed contamination source in the Savannah River averaged 0.30 ppm total mercury in muscle, compared with an average of 0.68 ppm below it. Higher trophic level fish, bass and Bowfin, had higher levels of mercury than lower level fish such as sunfish (averages less than 0.25 ppm) (Burger et al. 2001b).

Toxicity values for freshwater and marine fish are given in Table 2.6. A wide range of acute toxicity values is evident with methylmercury being more toxic than inorganic mercury. Chronic toxicity values are much lower than acute values and highlight the adverse effects of relatively low concentrations of mercury and methylmercury in water (i.e., < 1 µg/L). Additional examples of the toxicity of mercury to fish can be found in Chapter 8, Section D.

**Table 2.6. Toxicity Values for Aquatic Species (EPA 1997d and 1997e).**

Organism	Hg <sup>++</sup> (µg/L) (as HgCl or HgNO <sub>3</sub> )	Methylmercury (µg/L)
<b>ACUTE (LC50) (1)</b>		
Freshwater invertebrates	2.2 (cladoceran)1 to 2,000 (insect larvae)	Not available
Freshwater fish	30 (guppy) to 1,000 (Tilapia)	Not available
Rainbow Trout	155 to 420	24 to 84
Saltwater invertebrates	3.5 (mysid shrimp)1 to 400 (soft clam)	Not available
Saltwater fish	36 (juvenile Spot) to 1,678 (flounder)	51.1 (Mummichog)
<b>CHRONIC (EC50) (1)</b>		
Fresh-water invertebrates (cladocerans or water fleas)	0.96 to 1,287	<0.04
Fresh-water fish	<0.23 (minnow) to <0.26 (minnow)	0.29 (Brook Trout) to 0.93 (Brook Trout)
Saltwater invertebrates	1.131 (mysid) (2)	Not available

(1) LC50 concentration that results in death in 50% of the organisms and EC50 = concentration at which 50% of the exposed animals show a particular effect.

(2) Common name for cladocerans is water flea; mysids are small shrimp (both are crustaceans).

Generally, acute mercury toxicity results in flaring of gill covers, increased respiratory movements, loss of equilibrium, and sluggishness in fish followed by death (Armstrong 1979). Chronic or sublethal exposures to mercury have been shown to adversely impact reproduction, growth, behavior, metabolism, blood chemistry, osmoregulation, and oxygen exchange in marine and freshwater organisms (Eisler 1987).

Studies that have compared fish tissue mercury concentrations to adverse effects show decreased weight, length, and gonad to body weight ratio in Walleye at whole body mercury concentrations of 1.7 to 3.1 mg/kg (ppm) (Friedmann et al. 1996). But no significant effect on body weight ratio was seen in Northern Pike at a muscle concentration of 0.6 mg/kg (Friedmann et al. 1996). Decreased weight and length were observed in Fathead Minnow at whole body concentrations as low as 1.3 mg/kg.

Fathead Minnows with whole body concentrations of 4.5 mg/kg failed to spawn (Snarski and Olson 1982). Rainbow Trout with whole body mercury concentrations of 4-27 mg/kg (muscle 9-52 mg/kg) exhibited decreased appetite and activity followed by death (Niimi and Kisson, 1994). Reduced growth was observed at muscle concentrations of 12-23 mg/kg (Wobeser 1975). Increased mortality, decreased growth, sluggishness, and deformities were observed in Brook Trout at whole body mercury concentrations of 5-7 mg/kg (muscle 10 mg/kg) (McKim et al. 1976).

Mercury is a potent teratogen. Exposure of Killifish eggs (blastula stage) to inorganic mercury at 0.03 to 0.1 mg/L resulted in a significant proportion of embryos exhibiting cyclopia (Weis & Weis 1977a). Exposure to methylmercury at 0.03 or 0.04 mg/L also resulted in cyclopia in many embryos and defects in the cardiovascular system of Killifish (Weis & Weis 1977b).

The impacts of mercury on aquatic life are complex. Mercury affects fish growth and behavior in the Mummichog (*Fundulus heteroclitus*) (Weis and Weis 1989). Fish from

polluted Piles Creek caught fewer shrimp and had higher brain mercury (mean 0.116  $\mu\text{g/g}$ ) levels than fish from a “clean” environment (Tuckerton, NJ) (mean 0.032  $\mu\text{g/g}$ ). They were also more susceptible to crab predation. Tuckerton fish raised in Pile Creek water for three weeks showed impairment of capture ability and increased brain levels (Smith and Weis 1997). Embryonic and larval exposure as low as 5  $\mu\text{g/L}$  altered the swimming behavior and increased susceptibility to predation of Mummichogs (Weis and Weis 1995a). The prey-capture ability of the larvae was also impaired initially but gradually improved to control levels after about one week (Weis and Weis 1995b).

## **F. Toxicity of Mercury to Birds**

### ***1. Introduction***

Studies of mercury in wild birds can be divided into three categories: eggs and reproduction, liver and other organs, and feathers. In the 1960's great concern over reproductive failures of many avian species prompted studies of various contaminant levels in eggs, particularly in eggs that failed to hatch. Many of these studies were “positive”, showing that unhatched eggs had higher contaminant levels (particularly chlorinated hydrocarbons), than hatched eggs and in many cases the contaminants levels were linked to eggshell thinning. Although mercury also causes eggshell thinning, most field studies did not analyze for mercury, and those that did often did not find significant increases in mercury.

Studies of mercury in organs such as the liver were often made on moribund or dead birds to ascertain a cause of death. Widespread surveys of mercury in organs required the killing of large numbers of individuals. This was both inconvenient and undesirable. Hence many studies relied on measuring mercury in feathers. The affinity of mercury for the sulfhydryl groups on proteins, accounts for the relatively high concentrations of mercury deposited in growing feathers which are comprised mainly of the sulfhydryl rich protein, keratin. A substantial part of the body burden of MeHg is found in feathers (Braune and Gaskin 1987), which, like human hair, have repeatedly proven their value as a means for monitoring mercury concentrations in birds. Thompson et al. (1998) demonstrated the utility of feather mercury in documenting the temporal increase in mercury in the marine environment. Spalding et al. (2000a) found that the mercury levels in the feathers of growing chicks provided a good indication of the mercury dose and helped document the declining mercury contamination in the Everglades soon after curtailing emissions. Likewise, Hughes et al. (1997) showed that feathers were a better indicator of osprey exposure than concentrations in eggs. Burger (1993) provided a global review of mercury in feathers. At any one time up to 80% of the body burden of mercury is in the feathers (Braune and Gaskin 1987), and almost 100% of this mercury is MeHg (Thompson and Furness 1989). Lewis and Furness (1991) showed that about half of a single dose of MeHg was sequestered in feathers.

Feather sampling offers the advantage of being non-destructive (a sample can be removed from living birds with no impact on their subsequent health or survival), they require no special field preservation (i.e. freezing), and they allow comparison with museum specimens, which have been archived for more than a century. The ability to sample feathers without jeopardizing endangered species is particularly advantageous.

### ***2. Temporal Trends***

Although the hazards of environmental mercury were already well recognized by 1970, there was considerable speculation regarding the relative contribution of anthropogenic to natural sources for different compartments. Hammond (1971) questioned whether the magnitude of

human releases was sufficient to alter the concentration of mercury in the marine ecosystem. Thompson et al. (1998) addressed this question by showing that the mercury content of seabird feathers had increased between 65% and 394% (or 1-4% per year) in five species of North Atlantic seabirds for which pre-1931 and post-1979 feather samples were available.

Based on mercury levels in the feathers of museum specimens, mercury levels in carnivorous birds in Europe were low prior to the mid-20th century and then increased, reflecting the increased anthropogenic (both industrial and agricultural) contribution to the environment. Odsjö (1975) documented the dramatic jump in mercury concentration of Scandinavian Goshawk feathers from  $< 5 \mu\text{g/g}$  prior to 1940 to about  $20 \mu\text{g/g}$  after 1940. Peregrine Falcons averaged less than  $3 \mu\text{g/g}$  prior to 1940, jumping to almost  $38 \mu\text{g/g}$  (1964-1966) and declining to  $7-17 \mu\text{g/g}$  in the 1970s (Lindberg and Odsjö 1983) as agricultural uses and industrial pollution were curtailed. Smaller differences occur in non-predatory species, such as the Bar-tailed Godwit in the Netherlands where values increased from  $0.4 \mu\text{g/g}$  (1904-1963) up to  $2.0-4.9 \mu\text{g/g}$  (1979-1982).

### ***3. Impact on Birds***

The study of mercury levels in avian tissues has played an important role in understanding the dynamics of mercury in the environment. Early in the 1970's, several authors (Gochfeld 1971, Rappe 1973) pointed out that birds, like humans, are at the top of food chains and represent an important early warning system (Hays and Risebrough 1971) for mercury and other pollutants. Several authors in Scandinavia used museum collections to document changes in mercury concentrations over time (see Temporal Trends above).

Birds vary greatly in the amount of mercury in their bodies. In general, birds higher on the food chain, such as fish-eating (piscivorous) waterbirds and bird-eating raptors (hawks and eagles), have higher concentrations of mercury than seed-eating or fruit-eating birds. However, there are exceptions; birds eating grain that had been treated with mercurial fungicides have suffered mercury poisoning.

Most of the mercury in birds is in the form of methylmercury and comes from the diet. Although the consumption of fish is the main human exposure pathway for methylmercury, for the few people who eat piscivorous birds, such as fish-eating seabirds and ducks (e.g., mergansers), this is a potentially significant source of mercury exposure. Granivorous gamebirds such as doves, quail, and pheasants tend to have low mercury levels and pose little threat to human consumers. Even doves that have fed on hazardous waste sites, such as the contaminated lakebed of Par Pond, a Superfund site in South Carolina, have accumulated little mercury (Burger et al. 1997).

Eisler (1987) and Burger and Gochfeld (1997) reviewed data showing that mercury levels of 0.5-6 ppm in eggs are associated with decreased egg weight, malformations, lowered hatchability, and/or altered behavior in various species. Data relating feather levels to hatchability are more sparse, but in some species reduced hatching was observed in the 5-10 ppm range, while in others levels of 40-70 ppm in feathers were associated with lowered reproduction (Finley and Stendall 1978, Eisler 1987). Using 5 ppm in feathers as a criterion level, Burger and Gochfeld (1997) reported that Common Loons were at considerable risk with an average feather mercury level of 10 ppm.

### ***4. Experimental Mercury Poisoning in Birds***

Tejning (1967) reported an extensive study on domestic fowl fed an organic mercury compound (methylmercury dicyandiamide) used as a fungicide on grain. Many toxic endpoints were reported, including behavioral endpoints, and an increase in shell-less eggs, a phenomenon observed in NJ and New York Common Terns in the 1960's and early 1970's.

Fimreite and Karstad (1971) reported an experimental study feeding methylmercury-contaminated chicks to captive Red-tailed Hawks. Diet containing 10 µg/g of mercury resulted in death after one month from neurotoxicity. Liver of victims averaged 20 µg/g of mercury. Similarly Goshawks fed chicken averaging 13 µg/g of mercury died between 30 and 47 days (Borg et al. 1970). Toxicity emerged after the second week, included weakness, and loss of appetite and weight. Both studies found axonal changes and loss of myelin.

### ***5. Mercury in Raptors***

Raptorial birds (hawks, falcons, and eagles) are top predators in their respective ecosystems; therefore they are exposed to the relatively high levels of mercury and other bioaccumulative toxics (such as chlorinated pesticides and polychlorinated biphenyls) in their prey. Some species of hawks and eagles consume mainly mammals, others primarily birds, and a few, such as Osprey, eat mainly fish. Raptors have also been of interest as indicators of environmental pollution, because of the documented population crashes of many species in the 1940-1970 period, attributable primarily to organochlorine pesticides. The recovery of populations of hawks, falcons, eagles, and ospreys subsequent to the reduction in use of these persistent chemicals, attests to the benefits of regulating the release of persistent pollutants to the environment.

Raptors have been studied extensively because of the documented impact of contaminants on survival and reproduction. Although much of this work has been done on European birds, there are a substantial number of North American studies. Borg et al. (1970) attributed the decline of the White-tailed Eagle in Sweden to mercury.

In a study mainly of organochlorines, Snyder et al. (1973) reported on mercury levels in 24 eggs from Cooper's Hawk nests in Arizona and New Mexico. Their level of detection was 0.007 µg/ml of contents, and 23 eggs exceeded that level with a mean of 0.023 µg/ml (approximately 23 ppb).

Elliott et al. (1996) sampled Bald Eagle eggs from six locations along the British Columbia Coast. Geometric mean values ranged from 0.08 µg/g (wet) to 0.29 µg/g, with a maximum of 0.40 µg/g. Bowerman et al. (1994) sampled feathers of adult and young Bald Eagles from six Great Lakes locations and found that feather levels of adults averaged > 20 µg/g at most sites. Although Bowerman et al. (1994) suggested that mercury might contribute to reproductive failure, the major contributor was organochlorines (Grier 1974). In one of the earliest such studies in North America, Fimreite et al. (1970) found relatively low levels of mercury (0.75 µg/g) in livers of the insectivorous Kestrel, compared with moderate levels in Prairie Falcon (1.26 µg/g), and high levels in the carnivorous Short-eared Owl (6.8 µg/g). Table 2.7 summarizes data on concentrations of mercury in raptor species that occur in the United States (even if data were obtained elsewhere).



**Table 2.7. Tissue Concentrations of Mercury in Raptors that Occur in the United States (N=1 unless otherwise indicated).**

Species	Location	Tissue	Years	Concentration (µg/g)	Reference
Turkey Vulture (C)	California	Feathers	1980s	0.12	Wiemeyer et al. 1986
American Kestrel (I)	NY: Greenport, LI	Liver	1980s	0.22	NYSDEC 1981
American Kestrel (I)	Alberta	Adult liver	1960s	0.75	Fimreite et al. 1970
Peregrine Falcon (B)	Sweden	Feathers	1980s	1.38	Lindberg & Odsjö 1983
Peregrine Falcon (B)	Sweden	Feathers	1980s	0.05-1.7	Lindberg 1984
<b>Peregrine Falcon (B)</b>	<b>NJ: Barnegat Bay</b>	<b>Carcasses, n=3</b>	<b>1989-90</b>	<b>1.47 wet</b>	<b>Day et al. 1991</b>
<b>Peregrine Falcon (B)</b>	<b>NJ: Atlantic Coast</b>	<b>Eggs, n=12</b>	<b>1990-91</b>	<b>0.38 wet</b>	<b>Frakes et al. 1997</b>
<b>Peregrine Falcon (B)</b>	<b>NJ: Delaware Bay</b>	<b>Eggs, n=3</b>	<b>1991</b>	<b>0.01 wet</b>	<b>Frakes et al. 1997</b>
Goshawk (B)	Scandinavia	Feathers, n=16	pre-1940	<5	Odsjö 1975
Goshawk (B)	Scandinavia	Feathers, n=35	1940's	20	Odsjö 1975
Goshawk (B)	Netherlands	Feathers	1966-67	34.7	Spronk & Hartog 1970
Goshawk (B)	Netherlands	Feathers	1966-67	43.3	Spronk & Hartog 1970
Goshawk (B)	S. Finland	Feathers	1980-82	2.7	Solonin & Lodenius 1984
Cooper's Hawk (B)	SW US	Eggs, n=23		0.023	Snyder et al. 1973
Red-tailed Hawk (M)	Alberta	Adult liver		0.48	Fimreite et al. 1970
Northern Harrier (M)	Saskatchewan	Adult liver		0.07	Fimreite et al. 1970
Osprey (F)	S. Finland	Feathers	1980-82	11.6	Solonin & Lodenius 1984
Osprey (F)	NY: Suffolk Co.	Liver		2.4 (wet or dry?)	NYSDEC 1981
<b>Osprey (F)</b>	<b>NJ: Delaware Bay</b>	<b>Eggs, n=11</b>	<b>1985-1988</b>	<b>Gm=0.09 wet</b>	<b>Steidl et al. 1991</b>
<b>Osprey (F)</b>	<b>NJ: Atlantic Coast</b>	<b>Eggs, n=12</b>	<b>1985-1988</b>	<b>Gm=0.17 wet</b>	<b>Steidl et al. 1991</b>
<b>Osprey (F)</b>	<b>NJ: Maurice River</b>	<b>Eggs, n=2</b>	<b>1985-1988</b>	<b>Gm=0.10 wet</b>	<b>Steidl et al. 1991</b>
<b>Osprey (F)</b>	<b>NJ: Coastal Atlantic</b>	<b>Eggs, n=1</b>	<b>1999</b>	<b>0.15 wet</b>	<b>Clark &amp; Niles 1999</b>
<b>Osprey (F)</b>	<b>NJ: Maurice River</b>	<b>Eggs, n=1</b>	<b>1999</b>	<b>0.14 wet</b>	<b>Clark &amp; Niles 1999</b>
<b>Osprey (F)</b>	<b>NJ: Delaware Bay</b>	<b>Eggs, n=1</b>	<b>1999</b>	<b>0.08 wet</b>	<b>Clark &amp; Niles 1999</b>
Bald Eagle (FB)	W. Canada	Eggs	1990-1992	Gms=0.08-0.29	Elliott et al. 1996.
Bald Eagle (FB)	NW Ontario	Eggs	1971-1972	2.49 dry	Grier 1974
Bald Eagle (FB)	MI: Lower peninsula	Adult feathers		Gm=21	Bowerman et al. 1994
Bald Eagle (FB)	MI: Upper peninsula	Adult feathers		Gm=21	Bowerman et al. 1994
Bald Eagle (FB)	Lake Superior	Adult feathers		Gm=22	Bowerman et al. 1994
Bald Eagle (FB)	Lake Michigan	Adult feathers		Gm=20	Bowerman et al. 1994

Bald Eagle (FB)	Lake Erie	Adult feathers		Gm=13	Bowerman et al. 1994
Bald Eagle (FB)	Great Lakes	Juvenile feathers		Gm=3.7-20	Bowerman et al. 1994
<b>Bald Eagle (FB)</b>	<b>NJ: Gloucester</b>	<b>Eggs, n=1</b>	<b>1993</b>	<b>0.67 (wet)</b>	<b>Roberts &amp; Clark 1995</b>
Bald Eagle (FB)	DE: Bombay Hook	Eggs, n=1	1982	0.04 wet	Wiemeyer et al. 1993
Bald Eagle (FB)	DE: Cool Springs	Eggs, n=1	1983	0.05 wet	Wiemeyer et al. 1993
Bald Eagle (FB)	DE: Bombay Hook	Eggs, n=1	1977	0.03 wet	Wiemeyer et al. 1984
Bald Eagle (FB)	DE: Bombay Hook	Eggs, n=1	1978	0.19 wet	Wiemeyer et al. 1984
Bald Eagle (FB)	DE: Bombay Hook	Eggs, n=1	1978	Replicate, 0.19 Wet	Wiemeyer et al. 1984
<b>Bald Eagle (FB)</b>	<b>Southern NJ</b>	<b>Chick red cells, N=8</b>	<b>1992-93</b>	<b>0.23</b>	<b>Roberts and Clark 1995</b>
<b>Bald Eagle (FB)</b>	<b>Southern NJ</b>	<b>Chick feather, N=12</b>	<b>1992-93</b>	<b>2.92</b>	<b>Roberts and Clark 1995</b>
Golden Eagle (M)	NY: Westchester	Muscle, n=1		1.7 (wet or dry?)	NYDEC 1981
Golden Eagle (M)	NY: Westchester	Liver, n=1		2.7 (wet or dry?)	NYDEC 1981

*Data obtained on NJ birds is shown in boldface. The letters in parentheses designate the main prey as follows: M=Mammals, B=Birds, F=Fish, I=Insects, C=Carrion. The location of the study is indicated, as is the tissue analyzed. The published concentrations have been converted to micrograms per gram (parts per million). All results for feathers as well as results for most other tissues are reported on a dry weight basis (which is about three times higher than the corresponding wet weight basis). In some cases, the basis cannot be determined from the paper. "Gm" indicated that a geometric mean is reported. This is usually about 20% lower than the corresponding arithmetic mean.*

## 6. Mercury in Coastal Waterbirds

In addition to raptors, birds such as pelicans, cormorants, herons, and egrets are also top trophic level predators on fish. Pelicans were among the most prominent victims of the chlorinated hydrocarbons.

Black-crowned Night Herons from Lake Erie were found to average 3.1 ppm mercury in liver and 11.5 ppm in feathers (Hoffman and Curnow 1973). In a California population of Great Egrets showing poor reproduction, liver mercury averaged 6.08 ppm in six birds. There was inadequate data at the time for the authors to draw a conclusion regarding the role of mercury vs. organochlorines in the impaired reproduction (Faber et al. 1972).

Probably no area with mercury pollution has been as extensively studied as the Everglades in south Florida. The MeHg concentrations are elevated in many species of wetland wildlife and have been extensively studied in Great Egrets (*Egretta alba*), one of the top trophic level piscivorous species. Dietary MeHg reduced the appetite and growth rates of baby birds. In the baby birds fed MeHg (Spalding et al. 2000a) at 0.5 mg/kg body weight (comparable to doses encountered in the wild) anemia, feather abnormalities, neurological changes, and immunology damage occurred. At higher doses birds showed gait disturbances. Birds in the wild died at lower doses than laboratory birds, presumably due to multiple stressors (Spalding et al. 2000b). Recent reports (Lange et al. 2000), indicate that the mercury levels in Everglades Egrets has declined in 2000, a rapid response to the reduction in atmospheric inputs to the Everglades.

Herring Gulls from Captree, Long Island, averaged 4.5 ppm of mercury in feathers of males and 3.8 ppm in females. Herring Gulls found dead did not have higher levels in feathers than feathers sampled from live gulls (Burger 1995). However, Herring Gulls from the Mediterranean averaged 8.7 ppm (Lambertini 1982). Additional studies are required to clarify whether the differences are temporal (1970s vs. 1990s) or geographic.

### **7. Mercury in Seabirds**

Marine birds such as albatrosses, shearwaters, petrels, and auks breed on islands remote from local sources of industrial pollution. Their propensity to bioaccumulate mercury and other pollutants serves as important indicators of global mercury transport and marine pollution. The increase in mercury levels in such species during the 20th century has demonstrated that much of the mercury load in the oceans is due to anthropogenic rather than natural sources. (Thompson et al. 1992).

Marine birds appear tolerant of relatively high mercury concentrations and have a lower proportion of their mercury in organic form (Burger and Gochfeld 2001b). Likewise, marine mammals appear to have low susceptibility to mercury (Wang et al. 2001).

### **8. Mercury in Waterfowl**

Considering the popularity of waterfowl hunting and the number of people who eat wild ducks and geese, it is surprising that there have not been more published studies on mercury levels in waterfowl. Vermeer et al. (1973) sampled waterfowl from a mercury-contaminated ecosystem in Ontario, and found a dramatic trophic level effect with mercury concentration in muscle ranging from 0.5 ppm up to 12.3 ppm in fish-eating mergansers. However, some primarily vegetarian species, such as mallards, averaged 6.1 ppm.

### **9. Mercury in Reptiles**

There are few studies of mercury concentrations or effects in reptiles or amphibians. Over much of the southeastern United States, the American Alligator (*Alligator mississippiensis*) is a top level predator in wetlands, feeding on large fish, birds and other organisms. Alligators accumulate mercury in their tissues. In the Everglades, the concentration factors (organ:water) for alligators was about 100 million (Khan and Tansel 2000). Adults had about 50-70% higher mercury levels than juveniles (less than four year old alligators). All piscivorous wildlife in the Everglades are at elevated risk of mercury poisoning, with 100% of alligators exceeding a chronic risk threshold (Duvall and Barron 2000). Alligators had 36 ppm (dry weight basis) mercury in their kidneys (equivalent to about 10 ppm wet weight) (Yanochko et al. 1997). In central Florida, where alligators have been significantly impacted by organochlorine pollutants, mercury levels were low and were considered to have had a negligible impact on reproductive impairment (Burger et al. 2000).

## **G. Toxicity to Non-Human Mammals**

Piscivorous mammalian wildlife are exposed to mercury primarily via the food chain (i.e., consumption of contaminated fish) and bioaccumulate mercury in concentrations higher than those observed in their prey (i.e., biomagnification). Concentrations of mercury in wildlife have been observed at concentrations causing adverse effects in laboratory studies using the same species (U.S. EPA 1997d). Toxic effects from the consumption of contaminated fish have been observed in mammalian wildlife in areas with point sources of mercury emission (U.S. EPA 1997d).

U.S. EPA (1997d) investigated population impacts on piscivorous wildlife and estimated an adverse effect level for methylmercury in trophic level 3 fish (between 0.077 and 0.3 µg/g). This indicates that it is possible that individuals of some highly exposed wildlife subpopulations are experiencing adverse toxic effects due to mercury in the food chain.

U.S. EPA (1997d) examined several wildlife populations including mink, otter, and Florida Panther for the impacts of airborne mercury emissions. They concluded that field data were insufficient to determine whether the mink, otter or other piscivorous mammals suffered adverse effects. However, field data indicated that levels in panthers were high enough to cause toxic effects and contribute to the decline of this endangered animal. In the Arctic, Polar Bears had higher mercury levels in liver and kidney than the Ringed Seals on which they fed (Dietz et al. 2000) and adult bears had higher mercury levels than young ones.

Concentrations of mercury in marine mammals have also been studied. More than 90% of the mercury in marine mammals is in the inorganic form, suggesting that they can readily demethylate MeHg. However, the concentration of methylmercury in the tissues results in accumulation of high concentrations of methylmercury in humans and wildlife consuming these mammals (Clarkson et al. 1984). In another study, average mercury concentrations in the liver of Ringed Seal (*Phoca hispida*) ranged from 1.0 to 230.0 µg/g (wet weight; Wagemann & Muir, 1984). In Gray Whales (*Eschrichtius robustus*) stranded on the Pacific coast, liver mercury concentrations ranged from 9 to 120 ng/g. The concentrations of potentially toxic contaminants in these filter feeding whales was considered low relative to the concentrations in tissues of marine mammals feeding on higher trophic level species (e.g., fish; Varanasi et al. 1993).

An oral dose of 25 mg/kg body weight/day of methylmercury was fatal to Harp Seals in 20 to 26 days (Ronald et al. 1977). Sublethal doses (250 µg/kg body weight daily) adversely affected the cells of the inner ear of Harp Seals, and resulted in liver, kidney, and muscle methylmercury residues of 47.2 –82.5 mg/kg (wet weight; Ronald et al. 1977; Ramprasad & Ronald, 1977). Selenium has a mildly protective effect against the toxic effects of MeHg. Quail fed MeHg in corn-soy vs. a tuna diet, survived well with the latter, which also provided selenium (Ganther et al. 1972), and sodium selenite also protected quail against MeHg (Stoewsand et al. 1974). Marine fish accumulate selenium from sea water (Ganther 1980), which may reduce their vulnerability to the MeHg. Selenium reduces the amount of MeHg that reaches target organs (Komsta-Szumaska 1983). Whether the protective mechanism occurs, the level of uptake, tissue distribution, or end organ or cellular toxicity remains to be determined. Recently, Southworth et al. (2000) demonstrated that Largemouth Bass in a Tennessee lake showed a great increase in mercury concentrations after the cessation of selenium-rich effluent into the river. The elimination of the effluent resulted in a three-fold decline in selenium concentrations of fish tissue. In a study of 11 Savannah River fish species, mercury and selenium were strongly positive correlated in only two species (Yellow Perch and Red-breasted Sunfish; Burger et al. 2001a).

## **H. Wildlife Criteria and Reference Dose**

U.S. EPA (1997d) calculated a wildlife criterion for the protection of piscivorous avian wildlife of 61 pg/L (0.061 ppt) for methylmercury. Wildlife criterion is defined as the concentration of mercury in water that protects wildlife (e.g., avian and mammalian) from adverse effects resulting from ingestion of surface waters and from ingestion of aquatic life taken from these surface waters (U.S. EPA 1997d). For protection of piscivorous mammalian wildlife, EPA calculated a surface water wildlife criterion of 50 pg/L (0.05 ppt) for

methylmercury. Burger and Gochfeld (1997) identified 0.5 ppm (wet weight) in eggs and 5 ppm in feathers as levels that have been associated with adverse reproductive outcomes.

Wildlife criteria are difficult to determine, and show a range of values, dependent on the form of mercury chosen. Nichols et al. (1999) describe in detail the procedures and uncertainties associated with the estimated Wildlife Criterion Value of 1.3 ng/L (based on total mercury in unfiltered water) published in its Great Lakes Initiative report (1995) and of 0.05 ng/L for methylmercury published in EPA's Report to Congress in 1997.

U.S. EPA (1997d) calculated a reference dose (RfD) for methylmercury for avian species based on the chronic no observed adverse effect level (NOAEL) from studies on mallards. The RfD is defined as the daily intake (in  $\mu\text{g}$  mercury/kg body weight per day) that may occur without appreciable risk of any adverse effect on the organism. The value calculated was 26  $\mu\text{g}/\text{kg}$  bw/d. However, the comparable value for humans is on the order of 0.1  $\mu\text{g}/\text{kg}$  bw/d, suggesting that either the endpoint or procedure for developing wildlife RfD should be reconsidered. However, such a comparison may not be valid since the human RfD is based on studies that involved detailed testing of subtle markers of neurological performance involving interactive communication between tester and subject. Such testing is more difficult with birds. U.S. EPA (1997d) calculated a RfD for mammals of 18  $\mu\text{g}/\text{kg}$  bw/d for methylmercury.

U.S. EPA (1997d) concluded that piscivorous avian and mammalian species are the receptors receiving the greatest exposure to mercury. Several avian wildlife populations, including Common Loon, Wood Stork and Great Egret, were examined by U.S. EPA (1997d) for the impacts of mercury emissions. Field data were deemed insufficient to make a determination on whether adverse effects have occurred to these and other piscivorous wading birds due to airborne mercury emissions.

## **I. Interactions of Mercury with Other Pollutants**

Although mercury is one of the most ubiquitous and toxic of the environmental pollutants, it does not occur alone, nor does it necessarily act alone. In tissues of higher trophic level fish, mammals, and birds, methylmercury often co-occurs with other bioaccumulative pollutants, particularly chlorinated hydrocarbons such as PCBs. Polychlorinated biphenyls (PCBs) are also widely distributed in nature and occur as contaminants in fish and other organisms (Rice 1995). Studies on developmental defects in seabirds associated with elevated levels of both mercury and PCBs indicated that an interaction between the two might be causal (Gochfeld 1975). Subsequent research has identified a synergistic mechanism validating this prediction, at least for the central nervous system (Bemis and Seegal 1999). PCBs from fish also cause neurodevelopmental abnormalities in humans (Jacobson and Jacobson 1996; Schantz, 1996). The co-occurrence of PCBs in the Faroe Islands has been suggested as a possible explanation for the neurological effects seen there, although subsequent statistical analysis demonstrated that MeHg was associated with impairment independently of PCBs. The extent to which there may be synergistic or other interactions with PCBs and the endpoints that may be involved should be studied as such information has important public health ramifications. Bemis and Seegal (1999) suggest that the potential for interactions should be considered in the development of fish-consumption guidelines.

On the other hand, selenium has been identified as conferring a possible protective effect against the actions of mercury, at least in certain circumstances. Although selenium is an essential element at low concentrations, it can be highly toxic to aquatic wildlife at high concentrations (Schuler et al. 1990).

## **J. Summary and Conclusions on Ecological Effects of Mercury**

Mercury compounds have been widely distributed in the environment. Due to the discharge and transport of mercury, organism exposure in aquatic and terrestrial ecosystems has resulted in the bioaccumulation of mercury. Mercury, primarily methylmercury, is quickly accumulated by aquatic biota, and methylmercury is the principal form of mercury that causes adverse effects. Biomagnification of mercury up the food chain has been shown, especially in aquatic systems; those predators at the top of the food chain (i.e., piscivorous species) accumulate the highest concentrations of mercury. Mercury accumulation by organisms has resulted in adverse effects ranging from sublethal effects to deaths. Mercury is a teratogen, mutagen, and carcinogen, and causes embryocidal, cytochemical, and histopathological effects (Eisler 1987). Ecosystem-level effects are not well characterized and additional study and data are needed to ascertain the impacts of mercury at this scale. Nonetheless, it is clear that piscivorous species, including birds, fish, and mammals, are especially at risk from the effects of mercury.

EPA developed a Water Quality Criterion (WQC) for mercury for the Protection of Wildlife (1.3 ng/L or ppt) for surface waters of the Great Lakes. In addition, EPA has calculated a surface water wildlife criterion of 0.05 ng/L for methylmercury for protection of piscivorous mammals. These values are well below current Water Quality Criteria for the protection of aquatic life, and indicate that current surface water criteria may not adequately protect wildlife. Therefore, issuance of similar criteria should be considered for protection of wildlife nationally.

## **K. Recommendations**

**Address critical information gaps concerning the quantities and chemical species of mercury emissions and releases, the fate and transport of mercury in the environment, and the exposure pathways. To accomplish this, NJ should:**

- **Encourage federal agencies to expand existing national research on the ecological effects of mercury, particularly on piscivorous (fish-eating) fish, birds and mammals (particularly marine mammals).**

**(From recommendation “M.3.” in Volume 1).**

**Reduce mercury levels in fish and other biota. Mercury concentrations in freshwater and estuarine fish in New Jersey should, at a minimum, be in compliance with the EPA's recent Surface Water Criterion of 0.3 µg/g methylmercury in tissue. This guidance value, aimed at protecting human health, may not be adequate to protect the health of the fish. Therefore mercury levels in surface water and fish tissue should achieve levels protective of aquatic life and of wildlife (the criterion for which is currently under development). Assessing this criterion requires the use of improved analytic methodologies that lower detection levels by at least an order of magnitude.**  
**(From recommendation “Q” in Volume 1).**

The EPA is encouraged to issue National Water Quality Criteria for the Protection of Wildlife for mercury/methylmercury. NJ should have a comprehensive monitoring program for mercury and other bioaccumulative pollutants in representative trophic and ecosystems to document spatial and temporal trends in the state.

Federal agencies should continue existing national research and monitoring and initiate additional study of the impacts of mercury on piscivorous fish, birds, and mammals

(including marine mammals). This should include the examination of ecosystem-level effects and the monitoring of tissue levels in target species to track long-term trends in mercury bioaccumulation. Tissue levels should be compared to national and international mercury reduction efforts and other media measurement (e.g., air, water and sediment) as an environmental indicator of progress.