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# Guidelines for Interpretation of the Biological Effects of Selected Constituents in Biota, Water, and Sediment

## Mercury

*Participating Agencies:*

Bureau of Reclamation
U.S. Fish and Wildlife Service
U.S. Geological Survey
Bureau of Indian Affairs

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# Mercury

## Description

Mercury is the only metallic element that is liquid at normal environmental temperatures. It freezes at –39 °C and boils at 357 °C. Owing to its bright silvery color, the Romans called it *hydrargyrum* ("liquid silver"), which is why it is designated by the chemical symbol Hg. It has an atomic number of 80, an atomic weight of 200.6, and a specific gravity of 13.5. Ele-mental mercury has a low solubility in water, but some of its salts are highly soluble. Unlike many trace elements, mercury has no known biological function.

Mercury has three stable valence states—Hg(0) the native element, Hg(I) [$Hg_2^{2+}$], and Hg(II) [$Hg^{2+}$]—and it forms a variety of organic and inorganic compounds. The formation of methylmercury ($CH_3Hg^+$) is the most significant transformation because methyl-mercury is far more toxic and bioavailable than any other form of mercury. Methylation may be accomplished via bacteria in both sediments and water (Compeau and Bartha 1985). In some organisms and tissues, nearly all mercury is methylmercury.

Cinnabar (HgS), the most common ore of mercury, occurs either as long, slender, brilliant red crystals or as irregular red to gray or brownish masses. In powdered form, it is used as a pigment called "Chinese red." At some sites, minute globules of liquid ele-mental mercury are disseminated through the cinnabar.

### *Occurrence*

Primary sources of natural mercury emissions include volcanic eruptions and volatilization or solubilization of mercury from rocks, soils, and sediment. In rocks and soils, mercury most commonly occurs as mercuric sulfide (cinnabar). Mercury-enriched deposits are known in the Franciscan Formation of the coastal mountains of California, in the Green River Formation of the western Colorado Plateau, and in the vicinity of hot springs in many parts of the world. Mercury concen-trations in and around deposits of the Franciscan Formation may be in the 10 to 100 mg/kg range, whereas concentrations in the Green River Formation have been reported as high as 10 mg/kg (USGS 1970). The U.S. Geological Survey (USGS) reports mean background concentrations of mercury in surficial materials of the United States as 0.065 mg/kg (dw) (Schacklette and Boerngen 1984). Mercury occurs in coal at concentra-tions ranging from <0.01 to 8.0 µg/g depending on the geographic region and type of coal (Malani and Modetz 1981).

Atmospheric sources also contribute mercury to the environment (Haines 1991), although their concentrations are orders of magnitude lower than those of some geological sources. Atmospheric deposition is particularly important in environments where subsequent methylation is enhanced, as in the Everglades and other nutrient-enriched wetlands.

In the 20th century, mercury releases from artificial sources have been almost 10 times higher than calculated releases due to natural weathering (Moore and Ramamoorthy 1984). Mercury mining is a common source of mercury in the West; the mine wastes can be classified in five groups in order of increasing bioavailable mercury: (1) Waste rock, (2) low-grade unprocessed ore, (3) efflorescent salts, (4) processed ore tailings (calcines), and (5) soot or ash from the condenser system (J. Rytuba, USGS, pers. comm.). Other human activities that enhance mercury releases include the use of mercury as an amalgam in gold

mining; use of mercurials in seed dressings, fungicides, paints, and slimicides; fossil-fuel combustion; the industrial production of chlorine; and spills from field instruments (such as manometers) used to measure pressure at wellheads in gas fields.  One study estimated that, among the popula-tion of Sweden, the digestion and excretion of mercury from dental amalgams contribute about 100 kg of mercury to the environment each year (Skare 1995).

## Summary of Effects

Table 20 summarizes the predicted effects of environmental exposures to mercury, based on the information currently available.

## Field Case

Clear Lake, in Lake County, California, is a very large freshwater lake contaminated with more than 100 tons of mercury from the Sulphur Bank Mine, most of which is still present in the bed sediments.  The waste mercury is only a small percentage of more than 5000 tons of mercury that the mine had produced.  The lake and the mine are now included in an EPA Superfund cleanup site.  The ecological assessment of this site included an excellent survey of mercury bioaccumu-lation factors from sediment in a contaminated freshwater lake.  In sediment, total mercury concentrations range from 0.27 to 183 mg/kg (dw), and methylmercury concentrations range from 0.18 to 15.9 mg/kg (Suchanek et al. 1995).  Table 21 lists observed bioaccu-mulation factors for total mercury and methylmercury from sediment to oligo-chaetes, chironomids, and carp observed in this study.

The bioaccumulation factors in these results are consistently higher for methylmercury than for total mercury:  five orders of magnitude greater for fish and one to two orders of magnitude greater for benthic infauna.  Clearly, the parameters affecting net methylation are

controlling bioaccumulation of mercury.  For fish, trophic position was found to be the most influential factor in bioaccumulation at Clear Lake, a result supported by many other studies (Sorensen 1991).  BAFs are presented for sediment to carp because carp are directly grubbing about within the sediment.  However, carp had less mercury than other fish species.

Ecological effects on populations and communities were shown to be related to mercury concentrations in the sediment at Clear Lake.  Leech biomass was inversely correlated to both total mercury and methyl- mercury sediment concentrations (Suchanek and Richerson 1994).  Population numbers of *Chironomus* species exhibited an exponential decline as a function of sediment total mercury concentrations.  Benthic infaunal community diversity (as measured by both Shannon's index and Simpson's index) showed linear declines inverse to sediment mercury concentrations (Suchanek et al. 1995).

## Abiotic Factors Affecting Bioavailability

### Water

Analytical methods to detect mercury in water have dramatically improved in the last 5–10 years, resulting in lower detection limits and reduced interference from contamination. Because of the earlier analytical limitations, caution must be exercised when evaluating older mercury studies and reviews—especially studies that measured mercury concentrations in water. Background estimates of mercury in water conducted before the early 1980s likely report concentrations that are artificially high and bioconcentration factors that are too low.  Older studies that elucidated tissue residues and soil concentrations may still generally be relied upon. The role of global atmospheric transport, until recently, was underestimated, and the net flux of mercury from various aquatic and sediment compartments was inaccurately measured prior to the mid-1980s.

## Table 20.—Summary table for predicted mercury effect levels

[All matrix values expressed as total mercury (includes organic and inorganic forms). All criteria relate mercury risk to populations, not individuals.]

| Matrix | No effect[1] | Level of concern[2] | Toxicity threshold[3] | Explanation |
|---|---|---|---|---|
| Water (µg/L) | - - - | - - - | >30 | Sublethal effects to fish (Eisler 1987) |
| Sediment (mg/kg dw) | <0.065 | >0.15 | 0.2 | 0.065, surficial materials background (Shacklette and Boerngen 1984); 0.15, ERL of Long et al. (1990); 0.2, threshold to protect clapper rail (Schwarzbach et al. 1993) |
| | | | 0.24 | Toxic to guppies (Gillespie and Scott 1961) |
| Fish, whole body (mg/kg ww): | | | | |
|    Warm-water sp. | 0.11 | - - - | - - - | Background in bluegill (table 23). |
|    Cold-water sp. | - - - | - - - | 1.0 | FDA action level |
| Birds, diet (mg/kg bw/day) | - - - | - - - | 0.064 | Effects in mallards (Heinz 1979) |
| Birds, diet (mg/kg ww) | - - - | - - - | 0.3 | Loon reproductive and behavioral effects (Barr 1986) |
| | | | 0.1 | Mallard reproductive and behavioral effects (Heinz 1979) |
| Bird eggs (mg/kg fww) | 0.1 | 0.2–1.0 | 0.5–1.5 | 0.1, no effects in osprey; 0.5–1.5, low hatchability for pheasant (table 24) |
| | | | 0.86 | Mallard reproductive and behavioral effects (Heinz 1979) |
| | | | 5.0 | Mallard brain lesions (Heinz 1975) |
| Bird brain (mg/kg ww) | 0.13 | 0.13–1 | | 0.13 = mean in controlled, nonexposed population (Finley and Stendell 1978) |
| | | | 1 | Obvious signs of intoxication (Scheuhammer 1988) |
| | | | 4 | Lethal in embryos (Finley and Stendell 1978) |
| | | | 15 | Lethal in adults (Scheuhammer 1988) |
| Bird feathers (mg/kg dw) | 5 | 5–40 | 40 | Effects highly variable; sample other tissues. 5, upper end of background range; 20, reflects >1 mg/kg in diet (Scheuhammer 1991). Reproduction impaired over range of 5–40 (Eisler 1987) |
| Bird kidney (mg/kg ww) | <2 | - - - | 20 | Varies depending on species, sex, form of Hg, and Hg:Se ratio. Toxicity likely whenever kidney conc. > liver conc. See Littrel (1991), Heinz (1996). |
| Bird liver (mg/kg ww) | <1 | 1–2 | 3 | 1–2, behavioral effects (Zillioux et al. 1993); 3, reproductive harm (Barr 1986) |
| | | | 5 | Threshold for adult waterbirds (Zililoux et al. 1993) |
| | | | 25 | Kidney disease, gout in herons (Spalding et al. 1994) |

[1] Concentrations below this level are close to background and are not known to cause adverse effects.

[2] Concentrations at this level are above background but rarely appear to cause any adverse effects.

[3] Concentrations exceeding this level seem to cause some adverse effects, including reproductive impairment and sublethal impacts.

**Table 21.—Mercury bioaccumulation factors from sediment to oligochaetes,
chironomids, and carp in Clear Lake, California**
[Compiled from data in Suchanek et al. 1993.  ND, not determined]

| Site | Hg in sediment (mg/kg dw) | | Hg bioaccumulation factors | | | | | |
|------|-------|--------|-------|--------|-------|--------|-------|--------|
| | | | Oligochaetes | | Chironomids | | Carp | |
| | Total | Methyl | Total | Methyl | Total | Methyl | Total | Methyl |
| WB–1 | 77.80 | 10.36 | 0.14 | 1.15 | 0.07 | 2.0 | 0.01 | 124 |
| WB–2 | 27.16 | ND | .17 | ND | .1 | ND | ND | ND |
| WB–3 | 16.68 | ND | .09 | ND | .11 | ND | ND | ND |
| WB–4 | 4.13 | 7.39 | .2 | 1.0 | .2 | 1.4 | ND | ND |
| WB–5 | 2.61 | 4.16 | .26 | 1.6 | .23 | 2.5 | .16 | 185 |
| WB–6 | 8.85 | 7.1 | .16 | 2.8 | .05 | 1.1 | .0797 | ND |
| WB–7 | 1.40 | 2.43 | .24 | 2.6 | .16 | 23.6 | .39 | ND |

Water concentrations are typically used to assess mercury hazards to fish and aquatic life.  Gill and Bruland (1990) have shown that total dissolved mercury concentrations are not as useful in predicting concentrations in fish as are the dissolved concentrations of organic mercury compounds.  Concentrations are typically measured in picomolar (p$M$) quantities (5 p$M$ Hg is roughly equivalent to 1 ng of Hg).  Estimates of background total Hg concentrations in freshwater, prior to 1980, were incorrectly measured in the range of 50 to 250 p$M$ (10 to 50 ng/L).  Freshwater background concentrations are now thought to be less than 50 p$M$ (10 ng/L).  Some exceptionally pristine areas have concentrations less than 1 ng/L (Gill and Bruland, 1990).  The concept of "background" concentration is somewhat complicated by the global distribution of mercury through atmospheric water and may not be a very useful concept.  Concentrations greater than "background" are routinely found in continental rainwater, and levels of 120 ng/L or more have been documented (Dvonch et al. 1995).

Much of the research on mercury in freshwater systems has been conducted in poorly buffered systems, in mesic environments dominated by atmospheric mercury sources rather than geologic sources.  The low pH of these systems (Canada, Sweden), due to acid rain, appears to promote mercury bioaccumulation.  In the Western United States, lakes in areas of mercuriferous rocks and soils tend to be more alkaline; the climate in these areas tends to be arid to semiarid.  Clear Lake, California, and the Carson River in Nevada are two such areas in the west, and intense research is currently underway in both areas.  Results of these studies should produce new insights into mercury cycling in aquatic ecosystems typical of the Western United States.

Table 22 shows an extraordinary range of variability of estimated and measured effects of mercury in water.  Differences in mercury toxicity between taxa are greater than differences between the organic and inorganic forms of mercury.  Fish toxicity concentrations (96-h LC50), vary by two orders of magnitude, from 11 to 1800 µg/L (Sorensen 1991).  At 10 °C, methylmercury is about seven times more toxic than $Hg^{+2}$ to fingerling rainbow trout (Macleod and Pessah 1973).  Eisler (1987) concluded that total mercury concentrations in water of 100 to 2000 µg/L were fatal to sensitive aquatic species, and concentrations between 30 and 100 µg/L caused significant sublethal effects in fish.

**Table 22.—Mercury concentrations in water and associated effects on wildlife**

| Concentration (µg/L)[1] | Species | Comments/Effects | Reference |
|---|---|---|---|
| 0.00092–0.0036 (4.6–18 p*M*) | — | Background in Great Lakes water (50 times lower than pre-1980 measures) | Gill and Bruland 1990 |
| 0.0025 | — | Background concentration in freshwater lakes with atmospheric source of Hg only | Sorensen et al. 1990 |
| 0.012 | Aquatic life | EPA freshwater chronic criterion for aquatic life protection (current) | EPA 1986 |
| 0.18 | Birds | Great Lakes Initiative proposed criterion | EPA 1993 |
| 0.77 | Aquatic life | Proposed EPA freshwater chronic criterion for aquatic life protection | EPA 1997a |
| 1.4 | Aquatic life | Proposed EPA freshwater acute criterion for aquatic life protection | EPA 1997a |
| 1.6 | Mammals | Great Lakes Initiative proposed criterion | EPA 1993 |
| 2.4 | Aquatic life | EPA freshwater acute criterion for aquatic life protection (current) | EPA 1986 |
| 240 | Fathead minnow | No effect (MeHg, 48 mo) | Olson et al. 1975 |
| 290–930 | Brook trout | MATC, maximum acceptable toxicant concentration ($\approx$0.4 to 1.3% of 96-h LC50) | McKim et al. 1976 |
| 900 | Brook trout | LOEC, lowest observed effect concentration, behavioral effect | McKim et al. 1976 |
| 1000 | *Rana pipiens* | Arrested metamorphosis (MeHg, 4 mo) | EPA 1980 |
| 2930 | Brook trout | Early embryo death (3-generation exposure) | McKim et al. 1976 |

[1] Total Hg, unless otherwise noted.

The mercury concentrations proposed for the Great Lakes Initiative (table 22) are below the concentrations found in continental rain and, presumably, below background levels for freshwater lakes. The proposed mammalian value is an order of magnitude higher than the avian value, principally because an arbitrary species sensitivity factor of 0.1 was applied due to differences between observed test animals and the target birds. These proposed wildlife numbers probably represent safe concentrations but may be ultraconservative and unachievable in many environments. Controlling the processes and factors affecting bioaccumulation and methylation of mercury may ultimately be more important than maintaining low total mercury concentrations. Controlling bioaccumulation and methylation will require a good deal more understanding than we now have.

## Bottom Sediment

Sediment may be both a sink for mercury and a source of it, with changing physical and biological conditions. The effects of mercury in sediment were extensively investigated during the assessment of the contamination at Clear Lake, California, as described above. Equally high natural concentrations are sometimes noted in geothermal areas: measurements at Yellowstone National Park show contents as

high as 500 mg/kg (dw) in sediments from springs and pools and
150 mg/kg in fine-grained muds from mudpots and mud volcanoes.

Transport of mercury from watersheds depends strongly on the content of organic matter, which is usually greatest downstream from wetlands (Zillioux et al. 1993). Dis-turbance of wetland sediments may facilitate mercury transport by changing oxidation states, lowering pH, and resuspending sediment-bound mercury complexes in the water column. Limited measurements of methylmercury (2–14 percent of total Hg) show that disturbed wetlands produce more of it than undisturbed wetlands. Freshwater systems show strong correlations between dissolved organic carbon and filtered total mercury (Zillioux et al. 1993).

Sediment is definitely a source of methyl-mercury to biota and the water column. Even relatively low concentrations may result in bioaccumulation. Guppies (*Poecilia reticulata*) exposed to "control level" sediment (0.24 mg Hg/kg dw) at 21–23 °C achieved whole-body mercury concentrations of 1 mg/kg ww in only 60 days (Gillespie and Scott 1971). Schwarzbach (1993) proposed a sediment toxicity threshold of 0.2 mg/kg (dw) total mercury in sediment to protect the clapper rail (*Rallus longirostrus obsoletus*), a benthic forager, in San Francisco Bay. This sediment toxicity threshold, proposed for guiding sediment criteria in new wetlands created with dredge spoils, was based upon the ratio of the LOAEL for bird eggs (500 mg Hg/kg fww) to the observed bioaccumulation factors for mercury in sediment to mercury in rail eggs in four independent marshes within the bay (Schwarzbach 1993).

Criteria have not been established by the EPA for either total or methylmercury in sediment. Long and Morgan (1991) evaluated a wide variety of *marine* sediment toxicity studies in lab and field for the effects of sediment concentrations on benthic organisms. They

established Effects Range-Low (ERL) and Effects Range-Median (ERM) concentrations for each constituent evaluated. The ERL is the lower 10 percentile toxicity value in the database, and the ERM is the median toxicity value. For total mercury the ERL is 0.15 mg/kg (dw) and the ERM is 1.3 mg/kg. Fresh-water sediment criteria for mercury have been proposed by Canada (Smith et al. 1996). These show a threshold effect level of 0.174 mg/kg (dw) and a probable effect level of 0.486 mg/kg. These draft values were calculated based on information compiled in BEDS (biological effects database for sediments) as of January 1994. This is the same data base described by Long et al. (1995). These values are based on toxicity to benthic organisms and not on biological transfer coefficients to benthic predators.

## Biotic Effects

The biokinetics and toxicology of organo-mercurials, particularly methylmercury, have been more extensively studied than those of the inorganic form. This is because the methylated form has both greater toxicity and greater bioaccumulation than the inorganic forms. Intestinal absorption of inorganic mercury is limited to a few percent, whereas absorption of methylmercury is nearly
100 percent (Scheuhammer 1987). The ability to demethylate mercury almost certainly confers some relative resistance to mercury toxicity and, together with excretion mechanisms, may account for the high variability in sensitivity to mercury observed between taxa. The half-life of mercury in seabirds has been estimated to be about
60 days (Monteiro and Furness 1995). Inorganic mercury appears to have the greatest effect upon the kidneys, whereas methylmercury is highly toxic to embryos and the nervous system. MeHg readily penetrates the blood-brain barrier, produces brain lesions, spinal cord degeneration, and central nervous system dysfunctions.

## Fish

Bioconcentration of mercury in fish (table 23) is influenced by many water quality variables, including temperature, pH, hardness, and mercury speciation. Mercury concentrations in water are reflected, in a dose-dependent manner, in residue levels in fish. The primary focus for most monitoring has been to evaluate the hazard of mercury in fish flesh to human consumption. This focus has utilized either fillets or, less often, whole-body concentrations. The old U.S. EPA aquatic life criterion for mercury (EPA 1980) was not based upon the hazard to fish but rather the hazard to human consumption. The criterion regulated that concentration in water which can be expected to result in a concentration of 1 mg/kg ww in fish—the FDA action level for mercury in the United States. As of 1992, fish mercury levels high enough to warrant consumption advisories had been observed in portions of 26 States (Clean Water Fund 1992). (In recent years, though, most such advisories have been directed at "sensitive subpopulations," such as children and pregnant women, rather than at the general population.) It should also be noted that many State and national governments (Canada, Germany, Florida, etc.) have adopted a more restrictive human health advisory standard of 0.5 mg/kg, consistent with the National Academy of Sciences recommendations (NAS 1978). In a survey of 370 surface water bodies, the Environmental Protection Agency found that fish from 15 percent of the water bodies had mercury concentrations above the 0.5 mg/kg level (EPA 1997a).

### Table 23.—Effects of mercury residues in fish and herptile tissues

| Species | Hg concentration (mg/kg ww) | Tissue | Effect or interpretation | Reference |
|---|---|---|---|---|
| **Fish** | | | | |
| Redbreast sunfish (*Lepomis auritus*) | 0.08 | Skinless fillet | Background mean | Southworth et al. 1994 |
| Bluegill (*Lepomis machrochirus*) | 0.11 | Skinless fillet | Background mean | Southworth et al. 1994 |
| Brook trout | 2.7 | Whole body | Mercury intoxication | McKim et al. 1976 |
| Rainbow trout (*Oncorhyncus mykiss*) | 1–5 | Whole body | Chronic effects estimate | Niimi and Kissoon 1994 |
| | 10–20 | Whole body | Lethal estimate | |
| | 13 | Based on 25 mg/kg in diet for 189 d | Minimata disease[1] | Southworth et al. 1994 |
| **Amphibians/Reptiles** | | | | |
| Amphibian | 0.04–0.49 | Muscle | "Uncontaminated" | Byrne et al. 1975 |
| American alligator (*Alligator mississippiensis*) | 0.08 | Brain | Background | Heaton-Jones et al. 1994 |
| | 1.37 | | Irreversible visual impairment suspected | |

[1] Symptoms include a rolling swim, inability to stop in front of obstacles, and visual disturbances (Matida et al. 1972).

In addition to human health, another focus of mercury monitoring has been to examine the status of mercury contamination or the trophic transfer of mercury in various aquatic sys-tems. Unfortunately, only a small number of studies have examined mercury in fish as a hazard to the fish themselves. Whole-body concentrations appear most useful for evaluating both the bioaccumulation of mercury and its biological or toxicological hazard to fish. Niimi and Kissoon (1994) strongly advocate the use of whole-body mercury concentrations for evaluating mercury risk to fish, rather than water or any specific tissue concentrations. Whole-body fish residues were specifically recommended over individual organs or fillet tissue con-centrations because of the large degree of uncertainty in identifying the tissues critical
to fish health.

Most mercury in fish is methylmercury (Sorensen 1991). The measurement of total mercury alone in fish is therefore entirely sufficient for evaluating mercury risk. The half-life of methylmercury in fish muscle is estimated at 2–3 years (Sorensen 1991).

There is both field and laboratory evidence that diet is the most important route of fish exposure to mercury; it contributes more than 90 percent of the methylmercury accumulated. The assimilation efficiency for uptake of dietary methylmercury in fish is probably 65 to 80 percent or greater. To a lesser extent, fish may obtain mercury from water passed over the gills, and fish may also methylate inorganic mercury in the gut (Wiener and Spry 1996). However, trout have been shown to be about seven times more efficient at extracting methylmercury from dietary sources than from water via the gills (Phillips and Buhler 1978), and Hall et al. (1994) experimentally confirmed the dietary route of exposure as the most important one for fish.

As is the case for top avian, reptilian, and mammalian predators in aquatic systems, piscivorous fish, particularly long-lived

species, may be at risk from mercury bioconcentration and biomagnification. As noted above, measurements of mercury in water prior to the mid-1980s were unreal-istically high. Tissue measurements, however, were reasonably accurate, and hence the bioconcentration factors (BCFs) estimated at that time were unrealistically low (the result of dividing fairly accurate tissue concentrations by overstated water concentrations). Zillioux et al. (1993) point out that BCFs of 23,000 and 81,700 for methylmercuric chloride exposure of brook trout and fathead minnows were used to calculate freshwater final residue values, whereas recent BCF estimates derived from sampling and analyses using clean techniques generally exceed 1 million. The Mercury Study Report to Congress (EPA 1997b) listed median BCFs (called "bio-accumulation factors" in that report) for methylmercury in fish at two different trophic levels. For those at trophic level 3 (fish that feed on plants and plankton), this factor is 1,600,000; for those at trophic level 4 (fish that feed on other fish), it's 6,800,000.

The Mercury Study Report (EPA 1997b) also expressed these BCFs in terms of total dissolved mercury (concentration of all mercury species remaining in the water after filtering), based on the assumptions that
(1) essentially all of the mercury measured in fish tissue is methylmercury, and (2) methyl-mercury averages 7.8 percent of the total dissolved mercury in water. Therefore, the BCFs for total mercury are 124,800 for trophic level 3 and 530,400 for trophic level 4.

These BCFs may be applied to tissue concen-trations associated with harmful effects in order to derive values for possibly harmful total mercury concentrations in water. For instance, McKim et al. (1976) reported reproductive impairment in brook trout that had whole-body mercury concentrations of
2.7 mg/kg (=2,700,000 ng/kg). Applying the total-mercury BCF for trophic level 4 fish gives:

$$\frac{2,700,000 \text{ ng/kg}}{530,400} = 5.0 \text{ ng/kg} \quad \text{OR} \quad 5.0 \text{ ng/L}$$

as the total dissolved mercury concentration at which salmonid reproduction is impaired.

Although the Mercury Study Report to Congress (EPA 1997b) generated data on a range of national BCFs, that report emphasized the value of applying site-specific and field-derived BCFs when developing criteria for specific regions. Factors which affect these site-specific BCFs are many and varied. These include the number of trophic levels present and food web structure, the abundance of sulfur-reducing bacteria, and the concentration of sulfates, dissolved oxygen, temperature, organic carbon availability, pH, the nature of the mercury source, and a number of other parameters (Porcella et al, 1995).

Developing embryos are the most vulnerable life stage to mercury exposure. In all vertebrates, including fish, the transfer of methylmercury to the embryo represents the greatest hazard. According to Wiener (1995), "methylmercury derived from the adult female probably poses greater risk than waterborne mercury for embryos in natural waters." Sublethal and lethal effects on fish embryos are associated with mercury residues in eggs that are perhaps 1 to 10 percent of the residues associated with toxicity in adult fish. Mercury-intoxicated rainbow trout have between 4 and 30 mg/kg in whole bodies, while intoxicated embryos contain 0.07 to 0.1 mg/kg (Weiner 1995).

Both size and species of fish are important variables in mercury sensitivity. Smaller fish tend to accumulate mercury at greater rates than larger fish due to higher metabolic rates (Reinert et al. 1974).

In an extensive evaluation of mercury bio-accumulation in aquatic and benthic life in the contaminated waters of Clear Lake, California, Suchanek et al. (1995) showed that mercury

concentrations in fish increased with increasing body size, and they noted the following species-specific differences: carp < silversides < channel catfish < largemouth bass (Suchanek et al. 1993). Bioconcentration factors for mercury from water to silversides in Clear Lake were in the range of $10^4$ to $10^5$ for total mercury and from $10^6$ to $10^7$ for methylmercury. Concentrations in large-mouth bass were further increased by as much as 26 times over silversides.

In fish as in other groups, taxonomic differences can also influence mercury susceptibility. Bluegills are capable of demethylation of mercury in the liver (Burrows and Krenkel 1973), but it is doubtful whether rainbow trout have the same ability (Olson et al. 1978). Methylation can occur in the livers of some fish (e.g., albacore and yellowfin tuna) but not rainbow trout or mackerel (Sorensen 1991).

Mercury causes histopathological effects in nearly every fish tissue evaluated (gill, kidney, exocrine pancreas, bile ducts, liver, and erythrocytes). It appears that inorganic mercury is trapped in mucus of the gills, whereas methylmercury traverses this boundary readily.

### Amphibians/Reptiles

In a review of metal accumulations in amphibians, Hall and Mulhern (1984) reported that adult amphibians from areas uncontaminated with mercury had mercury concentrations in muscle ranging from 0.04 to 0.49 mg/kg ww; muscle of amphibians from contaminated areas ranged from 1.39 to 2.85 mg/kg ww (Byrne et al. 1975). Hall and Mulhern concluded that amphibians do not seem to accumulate mercury as much as some other species (i.e., predatory birds and some fish). Zoll et al. (1988) examined genotoxicity and bioaccumulation in laboratory studies of the newt *Pleurodeles waltl*. They observed cells with broken chromosomes and others with improper numbers of

chromosomes in blood smears from larvae exposed to both mercuric chloride ($HgCl_2$) and methyl mercuric chloride ($CH_3HgCl$). Bioaccumulation ratios after 12 days were 600 for mercuric chloride and 1,200 for methyl mercuric chloride.

### Birds

Symptoms of acute methylmercury poisoning in birds include reduced food intake leading to weight loss; progressive weakness in wings and legs; difficulty flying, walking, and standing; and an inability to coordinate muscle movements (Scheuhammer 1987). For acute mercury poisoning, brain residues are most diagnostic. Kidney disease and gout also seem to be strongly associated with elevated mercury in the liver (>25 mg/kg ww) (Spalding et al. 1994). If birds have been found dead, and mercury poisoning is suspected, the birds' brain, liver, and kidney concentrations of mercury should be determined in order to confirm the cause.

Birds may show significant adverse effects even at relatively low tissue concentrations if these concentrations result from chronic mercury exposures (table 24). In great white herons, liver mercury contamination >6 mg/kg ww correlated with mortality from chronic diseases (Sundlof et al. 1994). Reproduction is one of the most sensitive physiological processes and may show toxic effects even at very low dietary concentra-tions. Concentrations in the egg are typically most predictive of mercury risk to avian reproduction, but concentrations in the liver have also been evaluated for predicting reproductive risk. The documented effects of mercury on reproduction range from embryo lethality to sublethal behavioral changes in juveniles at low dietary levels. Effects of mercury include reduced hatchability due to increases in early mortality of embryos; some amount of eggshell thinning; reduced clutch size; increased numbers of eggs laid outside

the nest; aberrant behavior of juveniles; and potentially may include impaired hearing of juveniles.

The dietary concentrations of methylmercury required to produce significant reproductive impairment are about one-fifth those required to produce overt toxicity in adult birds of the same species (Scheuhammer 1991). In some cases, overall reproductive success in birds has decreased as much as 35–50 percent due to dietary methylmercury exposure insufficient to cause obvious signs of intoxication in adults. Heinz (1979) fed methylmercury to three generations of mallards at the level of 0.5 mg/kg dw (0.1 mg/kg ww). Females laid fewer eggs and produced fewer ducklings. Moreover, the ducklings that survived were less responsive to taped maternal warning calls and were hypersensitive to fright stimulus. Barr (1986) made similar observa-tions in a field study of the common loon in northwestern Ontario. Egg laying and territorial fidelity were both reduced where the mean mercury concentra-tion in loon prey was 0.3–0.4 mg/kg fresh weight; loons established few territories, and none laid any more than a single egg. The eggs contained mercury residues as high as 1.39 mg/kg ww. Around waters where the mean mercury concentrations of prey exceeded 0.4 mg/kg fresh weight, the loons raised no progeny.

The kidney is the major reservoir of inorganic mercury in birds as well as mammals. In renal tissue, mercury binds to metallothionein. Not surprisingly, the major toxic effect of inorganic mercury is kidney damage—specifically, necrosis of the proximal tubular cells (Ware et al. 1975). Spalding et al. (1994) found kidney disease and gout were present in great white herons that had >25 mg/kg ww liver mercury. In the same field study of great white herons, liver mercury contamination >6 mg/kg correlated with mortality from chronic diseases. However, the authors urged caution in interpreting these results because they examined only birds that had been found

**Table 24.—Observed effects of mercury residues in bird eggs and tissues**

[dw, dry weight; ww, wet weight]

| Species | Hg concentration (mg/kg) | Tissue | Effects | Reference |
|---|---|---|---|---|
| Black duck | 4–6 dw | Brain | Eggs failed to hatch | Finley and Stendell 1978 |
| Black-footed albatross | 37.4 dw (tot. Hg) 6.2 dw (MeHg) | Kidney | No adverse effect observed | Kim et al. 1996 |
| | 306 dw (tot. Hg) 20.4 dw (MeHg) | Liver | | |
| Common loon | >2 ww | Brain | Reduced egg laying; reduced nest-site and territory fidelity | Barr 1986 |
| | 29.73 ww | Liver | Reduced nesting success | Barr 1986 |
| | 51.9 ww | | Reduced hatching success | Fimreite 1974 |
| Common tern | 1.0 ww | Egg | Successful reproduction | Fimreite 1974 |
| | 3.65 ww | | 27% hatching success; 10–12% fledging rate | |
| | 1.06 ww | Liver | No effect | Gochfeld 1980 |
| | 2.22 ww | | Abnormal feather loss—juveniles | |
| | 9.08 ww | | Successful nesting | Fimreite 1974 |
| | 20.7 ww | | 27% hatching success; 10–12% fledging rate | |
| | 27.5 ww | | 10–12% fledge rate | |
| Gannet | 97.7 dw | Liver | Death | Parslow et al. 1973 |
| Grackle | 40.4 ww | Kidney | LD33 | Finley et al. 1979 |
| | 54.5 ww | Liver | | |
| Great white heron | >6 ww | Liver | Correlated mortality from chronic disease | Spalding et al. 1994 |
| | 7.2 ww | | Increased disease and emaciation | Spalding and Forrester 1991 |
| | >25 ww | | Kidney disease, articular gout | Spalding et al. 1994 |
| Grebe | 23.3 ww | Liver | Death | Littrel 1991 |
| Grey heron | 58.4 dw (11.7 ww) | Kidney | Death | Van der Molen et al. 1982 |
| | 95.5 dw | Liver | | |
| Herring gull | 2–16 ww | Egg | No decrease in hatchability | Fimreite 1974 |
| Mallard | 0.86 ww | Egg | Aberrant nesting behavior | Heinz 1979 |
| Merlin | 1–5 dw (0.2–1.0 ww) | Egg | Reduced productivity in ½ of populations | Newton and Haas 1988 |

**Table 24.—Observed effects of mercury residues in bird eggs and tissues—Continued**

[dw, dry weight; ww, wet weight]

| Species | Hg concentration (mg/kg) | Tissue | Effects | Reference |
|---|---|---|---|---|
| Osprey | 0.05–0.11 ww | Egg | No adverse reproductive effects | Audet et al. 1992 |
| | 1.5–3.0 dw (0.3–0.6 ww) | Egg | Decrease in number of young fledged | Odsjö 1982 |
| | 35  ww | Liver | Death | Wiemeyer et al. 1987 |
| Pheasant | 0.5–1.5 ww | Egg | Decrease in hatchability | Fimreite 1971 |
| Red-winged blackbird | 74.3 ww | Kidney | LD33 | Finley et al. 1979 |
| | 126.5 ww | Liver | | |
| Starling | 86.4 ww | Kidney | LD33 | Finley et al. 1979 |
| | 103.6 ww | Liver | | |
| Various species | 30. ww | Liver | Neurologic effects | Scheuhammer 1991 |
| Water birds, generally | 1.0–3.6 ww | Egg | Residue threshold for significant toxic effects | Zillioux et al. 1993 |
| | 5. ww | Liver | Conservative residue threshold for major toxic effects | |
| White tailed eagle | 33. ww | Liver | Death | Falandysz et al. 1988 |
| | 56. ww | Kidney | | |
| Zebra finch | 20. ww | Brain | 25% mortality | Scheuhammer 1988 |

dead.  Zillioux et al. (1993) found in their review of the literature that concentrations in the liver between 1 and 2 mg Hg/kg ww may result in behavioral effects, whereas liver-mercury concentrations of about 11 mg/kg ww and above lead to high embryo and duckling mortality and to brain lesions.  Spalding and Forrester (1991) suggested neurological effects may be associated with liver-mercury levels in birds as low as 5 mg/ kg ww.  Zillioux et al. (1993) concluded that a conservative residue threshold for major toxic effects in waterbirds would be 5 mg/kg ww in the liver.

However, apparently normal seabirds have been found with mercury concentrations many times this level in the liver, but analysis has shown these concentrations to be pri-marily inorganic mercury (Kim et al. 1996).  In some species, especially Procellariformes, it appears that demethylation of mercury is an important

detoxification strategy.  Therefore, characterizing the different forms of mercury in tissues is increasingly recognized as important to meaningful interpretation of residue data.

In the majority of wild birds sampled, liver concentrations of mercury are higher than kidney concentrations.  However, in some cases of mercury poisoning, kidney con-centrations are found to be nearly the same as the liver concentration (Lewis and Furness 1991).  Kidney concentrations of 20 mg/kg ww have been noted in birds found dead in mercury-contaminated environments (Littrel 1991).

Brain mercury as low as 3–7 mg/kg ww can be lethal to ducklings.  Concentrations four times this high are required to cause direct mortality in adults.  The lowest concentration of mercury in the brain to produce obvious signs of

intoxication in adults is 5 mg/kg dw or 1 to 1.6 mg/kg ww (Scheuhammer 1991). Heinz and Locke (1975) found that the brains of mallard ducklings found dead with brain lesions contained an average of 6.17 and 5.19 mg Hg/kg ww in two successive years.

The toxic effects of mercury in bird eggs have been documented by many investigators in both laboratory and field studies (Barr 1986; Birge et al. 1976; Fimreite 1971, 1974; Heinz 1974, 1979; Heinz and Locke 1975; Hoffman and Moore 1979; Finley and Stendell 1978; Tejning 1967; etc.). Mercury is an extremely potent embryo toxicant, and dietary mercury is transferred to avian eggs in a dose-dependent manner. Reproductive impair-ment is one of the most sensitive endpoints of mercury toxicity. Mercury accumulates particularly in the egg-white proteins, which derive from serum proteins. Egg concentra-tions, therefore, more closely reflect mercury from recent dietary uptake than from accumu-lated tissue stores. There is also evidence that the ovalbumin fraction of egg white has a specific affinity for dietary mercury, while the ovoglobulin fraction tends to accumulate low levels of "nondietary" mercury. Because of the strong dietary connection, Walsh (1990) suggested that eggs provide a particularly good indicator of mercury exposure in the vicinity of the nesting site in the immediate pre-laying season. One can expect methylmercury to predominate in eggs, particularly within the albumen fraction. Because mercury is predominantly deposited in albumen, more intra-clutch variation in mercury content is also to be expected than in contaminants preferentially distributed to yolk. Becker (1992) reports that, among the Charadriiformes, the last egg of a clutch commonly has lower mercury content than the first egg. The first egg laid contained as much as 39 percent more mercury than the second or third egg. Becker et al. (1994) predict that the toxic effects of mercury would be more pronounced in *a*-chicks (the chick from the first laid egg). In elevated mercury environments, this will result in

abnormally high losses of *a*-chicks, a reversal of the normal situation. Barr (1986) documented adverse effects on loons associated with egg concentrations of 1.39 mg/kg ww.

Hoffman and Moore (1979) treated mallard eggs with externally applied methylmercury chloride. Effects were dose related and included decreased embryo weights, developmental abnormalities, and embryonic death. The lowest dose applied which affected survival was 27 micrograms. Given an average mallard egg weight of 55 grams, this dose corresponds to about 0.5 mg/kg. With increasing concentrations, abnormalities progressed in severity from mostly minor skeletal deformities to gross external ones such as micromelia, gastroschisis, and eye defects as well as internal deformities such as brain defects and a reduction in liver size. Such laboratory work is useful because it may efficiently elucidate the types of effects that can be produced, but these results should not be literally extrapolated to the field. External mercury exposures by Hoffman and Moore had more pronounced effects at lower doses than organic mercury incorporated into the egg through the hen's metabolism (Heinz 1974) presumably because the applied mercury was not completely bound to the ovalbumin and ovoglobulin.

Reproductive effects may extend beyond the embryo and may reduce the rate of juvenile survival. Mercury in the eggs of mallards has caused brain lesions in hatched ducklings. Heinz and Locke (1975) reported on mallards that were fed 3.0 mg/kg methylmercury dicyandiamide (equivalent to 0.6 mg Hg/kg in a natural succulent duck diet) over two successive years. Mercury accumulated in the eggs to an average of 7.18 and 5.46 mg/kg ww in the two years. Lesions included demyelina-tion, neuron shrinkage, necrosis, and hemor-rhage in the meninges overlying the cerebellum.

In a field study of total mercury in eggs of common terns, Fimreite (1974) estimated the threshold level for toxic effects to be between 1.0 and 3.6 mg/kg ww. Heinz (1979) was able

to relate egg concentrations to subtle behavioral effects in mallard ducklings. As described earlier, he fed ducks a diet including 0.5 mg Hg/kg dw over three generations and found decreased reproductive success and altered behavior of ducklings. The mean mercury concentration in eggs in this study was 0.86 mg/kg (fww). In a study of ring-necked pheasants, Fimreite (1971) found a significant reduction in hatchability associated with dietary mercury levels between 0.5 and 1.5 mg/kg ww. The low end of this effect range continues to be the lowest observed adverse effect level (LOAEL) for mercury in bird eggs.

Establishing effect levels based on mercury concentrations in feathers must be considered with caution. Feathers represent a route of excretion and not a target organ. Mercury is deposited in feathers at the time of molt, when there is active feather growth and a corresponding blood supply to the growing feather (Goede and deBruin 1984; Furness et al. 1986; Braune 1987). Once mercury is in feathers, it is bound to the sulfide bonds of feather keratin and is not physiologically available for redistribution to target organs. Mercury content of feathers will vary with time to last molt, feather type, and age and species of the bird (Monteiro and Furness 1995). Feathers have the advantage of being a nondestructive exposure-assessment matrix which may be resampled in the same individual and which may also be compared with museum specimens (Applequist et al. 1984). The concentration of mercury in tissues may actually decrease during molting as mercury is mobilized from tissues into feathers (Furness et al. 1986). In sequential feather-loss patterns, the first primary feather to be grown back has the greatest mercury concentration, and the concentrations decrease in subsequent feathers (Lewis and Furness 1991; Braune 1987; Braune and Gaskin 1987). Becker et al. (1994) found results in three species of larids which implied that mercury in the first down of chicks was a consequence of mercury levels in the egg, whereas levels in feathers of chicks were largely due to mercury ingested in

food. Lewis and Furness (1991) found that in laboratory-reared black-headed gulls, 49 per-cent of the administered mercury was accumulated in the plumage independent of the dose administered. The percentage of the mercury body burden found in plumage varies from species to species.

Almost all feather mercury is the organic form (Thompson and Furness 1989). Species that are effective in demethylating mercury, such as the Procellariiformes, will tend to have a lower percentage of their total mercury body burden partitioned into the feathers than other species do (Kim et al. 1996). This character-istic has been interpreted as an adaptation to the slow molt of feathers in Procellariiformes; inasmuch as they do not shed feathers as quickly as other species, the feathers are less useful as a medium for the sequestration and ultimate excretion of methylmercury (Kim et al. 1996).

The molt pattern of any given species will also have a large influence on the variation of mercury content between different feathers within an individual bird (Applequist et al. 1984). Greater variation in mercury with feather type should also be expected in more contaminated environments (Becker et al. 1994). The timing of feather growth may also influence mercury accumulation in other tissues if the levels of mercury exposure differ greatly between the birds' wintering and breeding grounds. For meaningful quantitative monitoring of mercury using feathers, the feather/mercury pattern for a species should be established and similarly sampled among those individuals or populations which are to be compared. For historic comparisons using older museum specimens, especially if preservation methods are vaguely recorded, it may be prudent to determine both total mercury and methyl-mercury in feathers to evaluate the relative contribution of mercurial used in preservation of the avian skins.

In a review of effects related to mercury concentrations in feathers, Eisler (1987)

reported that concentrations between 5 and 40 mg/kg in feathers are linked to impaired reproduction. Sterility was observed in the Finnish sparrow hawk (*Accipiter nisus*) at feather mercury concentrations of 40 mg/kg. Bowerman et al. (1994) found that feathers of bald eagles in the Great Lakes region had mercury concentrations of 13 to 21 mg/kg, but they could make no association between mercury concentrations and bald eagle repro-duction. Scheuhammer (1991) suggests that feather mercury concentrations >20 mg/kg can result from diets containing >1 mg Hg/kg and that these concentrations probably indicate a wetland that poses a mercury risk to birds. He estimates the normal background of mercury in raptor feathers to be 1–5 mg/kg.

### Mammals

Though far fewer studies have been conducted assessing mercury toxicity in mammals than in birds, many of the general mechanisms are similar. Like birds, mammals accumulate mercury from various environ-mental matrices, but those living in or near water tend to accumulate the most. Kucera (1983) reports that mink and river otter, in drainage areas supporting 16 pulp and paper mills and a chlor-alkali plant, accumulated 10 times more mercury than predatory fish from the same drainage areas. Generally, carnivorous or piscivorous animals tend to have the highest body burdens, omnivores have intermediate body burdens, and herbivores tend to have the lowest body burdens. There is also an age-related effect: older animals tend to have higher body burdens than younger ones. This is probably due to a combination of factors including the length of time the older animals have had to bioaccumulate mercury, the younger animals' higher metabolic rate, and the older animals' slower rate of mercury excretion.

In mammals, the highest mercury concentra-tions are generally found in hair or in liver or kidney tissue, depending upon the species (table 25). Muscle and brain tissue also tend to accumulate mercury. The primary route of excretion is through the hair. Animals that grow heavy winter coats and shed them in summer tend to excrete more mercury than those that don't. Mercury excreted through the hair is bound to proteins, just as in bird feathers.

Manifestations of mercury poisoning in mammals include loss of muscle coordination, loss of appetite, and sensory impairment. Sensory impairment has been described in humans and monkeys as constriction of the visual field and loss of hearing. Organic mercury is more toxic to mammals than inorganic mercury; methylmercury irreversi-bly destroys the neurons of the central nervous system. Animals exposed to sub-lethal mercury concentrations, or suffering from chronic low-level exposure, may appear normal. When stressed, though, they may not be able to perform adequately to survive the rigors of living in the wild.

Mercury crosses the placental barrier and reaches the developing fetus. A great deal of research has been done studying the impacts of occupational and accidental mercury exposures to human fetuses. From this research, the fetus is known to be much more sensitive than the adult to the ill-effects of mercury (Girard and Dumont 1995). For all organisms tested, early developmental stages have been shown to be the most sensitive to mercury poisoning (Eisler 1987). Methyl-mercury is furthermore a known teratogen and mutagen.

## Bioaccumulation

Mercury strongly bioaccumulates and even biomagnifies through trophic levels in aquatic systems. Biomagnification of mercury has been documented in birds (Fimreite 1974), fish, and even zooplankton communities (Watras and Bloom 1992). Within aquatic

### Table 25.—Mammalian mercury exposures and associated effects

| Species | Hg concentration (mg/kg dw) | Tissue | Effects | Reference |
|---|---|---|---|---|
| Cat (*Felis domesticus*) | 121–392 (fw) | Hair | Death | Jenkins 1980 |
| Mink (*Mustela vison*) | 30–40 | Liver | Death; also suffering from cold stress | Wren et al. 1987 |
| | 4–18 | Brain | | |
| | 7.8 | Muscle | Nervous system pathology | Burton et al. 1977 |
| | 25.4 | Liver | Nervous system pathology | Hallbrook et al. 1994 |
| | 58.2 | Liver | Death | Wobeser et al. 1976 |
| | 34.9 | Fur | | |
| | 31.9 | Kidney | | |
| | 15.2 | Muscle | | |
| | 13.4 | Brain | | |
| Monkeys | 1.2–4 | Blood | Visual disturbance | Suzuki 1979 |
| | 6–9 | Brain | Visual disturbances | Burton et al. 1977 |
| Mountain lion | 110 | Liver | Death | Roelke 1990 |
| River otter (*Lutra canadensis*) | 13.5 | Muscle | Death | Hallbrook et al. 1994 |
| | 30 | Liver | Death | |
| White-footed mouse (*Peromyscus maniculatus*) | 0.31 | Hair | No effect | Burton et al. 1977 |
| | 10.8 | | Normal appearance; poor performance under stress | |

systems, the net rate of methylation/ demethylation processes in water and sediments ultimately governs the bio-availability of mercury. Both processes are biological, and demethylation is better understood than methylation (Zillioux et al. 1993). Several factors have been shown to influence the net rate of methylmercury production. Those that increase production include high dissolved organic carbon content, low pH, the presence of sulfides, and high temperatures.

Within a watershed, wetlands enhance the rate of methylmercury production (Lee and Hultberg 1990), and so do reservoirs, particularly new reservoirs. The "new reservoir effect," whereby a pulse of methylmercury is produced in the first few years of reservoir filling, is usually credited to the initial nutrient input from decaying terrestrial vegetation killed by the rising waters. It should be noted that this effect is not limited to those regions that have local geological or industrial sources; atmospheric input alone may be sufficient to produce a detectable pulse of methylmercury. Although mercury bioconcentration factors (BCFs) from water to fish of over a million are not uncommon (Watras et al. 1994; Bloom et al. 1991; Porcella 1994; Suchanek et al. 1993), Porcella has cautioned that these BCFs may not be applicable over a wide range of water qualities because so many factors influence mercury bioavailability.

## Interactions

Mercury is known to interact toxicologically with other elements in additive, synergistic, and antagonistic ways. Its interactions with selenium are of particular interest. Both mercury and selenium bioaccumulate, both bind to organo-thiol groups, and both have their greatest toxic effect through dietary exposure to the organic forms. Interactions between selenium and mercury have been extensively documented (Cuvin-Aralar and Furness 1991; Sorensen 1991), although many conflicting results have been reported. These interactions vary greatly in character and in strength, depending on whether the forms of the two elements are organic or inorganic. The exact chemical speciation, and other factors, can cause the toxicity of selenium to be increased, reduced, or unaffected by the presence of mercury. Selenite has been shown

to protect against kidney poisoning caused by inorganic mercury salts. El-Begearmi et al. (1977) demonstrated that sodium selenite reduced the toxicity of methylmercury and increased the survival of Japanese quail. However, Heinz and Hoffman (1998) found conflicting results when they studied the interactions of selenomethionine and methylmercury in the diets of captive mallards: the selenium and mercury together were less likely to poison adult birds than either element separately but more likely to impair reproduction. The presence of methylmercury in the diet also greatly enhanced the storage of selenium in both eggs and livers, and, similarly, the presence of selenium enhanced the storage of mercury. Teratogenesis was most severe in the eggs of mallards that had been fed both methyl-mercury and selenomethionine.

## Regulatory Standards

| U.S. Environmental Protection Agency Standards and Criteria [See Appendix II for explanation of terms. Source: EPA 1986, 1995, 1997a, 1997b] | |
|---|---|
| Status | EPA priority pollutant |
| Drinking water MCL | 2 µg/L |
| Drinking-water health advisories for 70-kg adult | Reference dose: 0.3 µg/kg/day<br>Long-term HA: 2 µg/L<br>Lifetime HA: 2 µg/L<br>DWEL: 10 µg/L |
| Freshwater criteria (for dissolved Hg) | 2.4 µg/L for acute exposure<br>0.012 µg/L for chronic exposure |
| 1/1,000,000 cancer risk | 0.144 µg/L (water and organisms)<br>0.146 µg/L (organisms only) |
| Human health criterion | 0.05 µg/L total Hg (water; based on bioconcentration in fish) |
| Wildlife criterion for protection of bald eagles | 0.000082 µg/L methylmercury (based on biomagnification through multiple trophic levels) |
| Wildlife criterion for protection of piscivorous species | 0.00005 µg/L methylmercury ($\approx$0.00064 µg/L total mercury) |

The U.S. Food and Drug Administration action level for methylmercury in the edible portions of fish, shellfish, crustaceans, and other aquatic animals is 1.0 mg/kg ww (FDA 1992).

For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water-quality officials in the 17 Western States.

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