# Chapter 45 Mercury

#### Synonyms

#### Minamata disease

Mercury has been used by humans for over 2,000 years and was associated with premature deaths of cinnabar (mercuric sulfide) miners as early as 700 B.C. More recent human poisonings have been related to agricultural and industrial uses of mercury. One of the best documented of these cases occurred in the 1950s in Minamata Bay, Japan, when mercury was discharged into the environment and accumulated in fish and shellfish used as human food. In addition to human poisonings, mercury poisoning or toxicosis has been identified in many other species.

Mercury is sometimes used to recover gold from stream sediments, and it may pose hazards to wildlife if it is released to the environment during ore recovery. Fungicidal treatment of seeds with mercury was common in the 1950s and 1960s, but this agricultural practice has been largely halted in the Northern Hemisphere.

#### Cause

Mercury is a heavy metal that is nonessential and toxic to vertebrates, and it occurs in both organic and inorganic forms. The organic forms, such as methylmercury, are generally the most toxic. However, inorganic mercury can be transformed into organic forms through a variety of biological processes. Mercury occurs naturally in soils and sediments, but it is also introduced into the environment by human activities (Fig. 45.1).

## **Species Affected**

Birds affected by mercury include species that are exposed to high levels of the metal because of their feeding behavior (Fig. 45.2). Exposure may occur through accumulation of mercury in the aquatic food chain, agricultural uses of mercury as a fungicidal seed treatment, and from point-source industrial and mining discharge to the environment.



Figure 45.1 Sources of mercury contamination.

Species group and representative species



Loons Common loon, red-throated loon



Wading birds Common egret, great blue heron, black-crowned night heron



**Pelicans** Brown pelican, white pelican, gannets



**Cormorants** Double-crested cormorant



Mergansers Common merganser, red-breasted merganser



Gulls and terns Herring gull, common tern



**Pelagic seabirds** Fulmars, shearwaters



Raptors Bald eagle, osprey, golden eagle, owls



Gallinaceous birds Ring-necked pheasant, chukar partridge, grouse, quail



Sources of mercury exposure for birds that live, nest, or feed in or near aquatic systems include industrial discharge, acid precipitation, and high mercury levels in fish and sediments. Gallinaceous birds, such as turkey and pheasant, may be exposed when they consume mercury-treated grain. Raptors, such as golden eagle and owls, may be secondarily exposed when they consume birds or small mammals that died from eating treated grain.

Major bird die-offs from mercury poisoning are rarely reported. Mortality from mercury poisoning is more of an insidious problem involving scattered mortalities. Some instances where mercury has been associated with mortality or sublethal effects are listed in Table 45.1.

## Distribution

Mercury is present in fossil fuels and in some soils and sediments. The release of mercury into the atmosphere from burning of fossil fuels, the conversion of inorganic mercury to organic methylmercury and its cycling in aquatic systems, and accumulation through the food chain can expose wildlife to mercury and potential toxicity. Problems with mercury poisoning in birds traditionally have occurred in northern latitudes in areas affected by acid precipitation, at pointsources of industrial discharge, and in agricultural areas where mercury-based seed treatments have been used.

## Seasonality

Seasonality is dependent only on the movement and foraging of birds that may put them at risk of mercury exposure while they feed in contaminated habitats.

## **Field Signs**

Clinical signs of mercury poisoning in birds have been documented primarily from experimental feeding studies, and they include incoordination, tremors, weakness, ruffled feathers, and drooping eyelids. Experimental exposure of birds to high levels of mercury has caused acute death in less than 1 hour with few signs of intoxication. In free-ranging birds, most cases of mercury poisoning are probably more insidious, resulting in an emaciation syndrome and a variety of sublethal effects that may act together to cause eventual death (Table 45.2).

## **Gross Lesions**

Birds suspected of having died of mercury poisoning often are emaciated, but no other gross lesions are noted.

## Diagnosis

A diagnosis of mercury poisoning as cause of death can seldom be made on the basis of mercury concentrations in tissues alone. A complete necropsy examination with appropriate laboratory evaluations should be done by a qualified diagnostic laboratory. A diagnosis is generally based on total mercury concentrations of 20 parts per million wet weight or more in the liver or the kidneys and by the presence of microscopic lesions in tissues consistent with mercury poisoning. A definitive diagnosis is difficult, however, because the amounts of residues that would indicate mercury poisoning have not been determined for most bird species. Also, seabirds may naturally accumulate and tolerate higher levels of mercury than nonmarine birds. Another confounding factor is that selenium, which is an element that is essential to health, has been found to reduce the toxicity of mercury, and residues of both of these elements are often found in birds. A thorough history of field observations and background information about potential agricultural and industrial uses of mercury is an invaluable supplement to the specimens submitted.

**Table 45.1** Reports of mercury exposure associated with mortality and sublethal effects in free-ranging birds.

Location	Species	Effect
Sweden	Pheasants, partridge, pigeon, magpie, passerines	Mortality
Sweden	Goshawk, Eurasian sparrow- hawk, white-tailed eagle, peregrine falcon	Mortality
The Netherlands	Various raptors	Morbidity and mortality
Canada	Loons, turkey vulture	Mortality
Canada	Common tern	Poor reproduction
Scotland	Golden eagle	Poor reproduction
United States	Bald eagle	Poor reproduction
Canada	Loons	Poor reproduction

Table 45.2 Sublethal effects of mercury exposure from experimental studies.

Species	Effect(s)	
Pheasants	Decreased egg weight, fertility, and hatchability	
Starling	Microscopic kidney lesions	
Mallard duck	Microscopic brain lesions, skeletal deformities; reduced clutch size, hatchability, embryonic growth; behavioral changes	
Black duck	Reduced clutch size and hatchability	
Red-tailed hawk	Neurologic signs of weakness and incoordination	

## Control

Prevention of exposure is required to control the lethal and sublethal effects of mercury poisoning in avian populations. Elimination of mercury discharge in industrial, mining, and sewage wastes, reduction of fossil fuel (especially coal) combustion, reduced inputs to (and thus releases from) municipal incinerators, and elimination of agricultural uses will reduce the amount of mercury entering the environment as a result of human activities. One factor to consider in the development of new wetlands is that the accumulation of mercury in aquatic biota is enhanced when terrestrial habitats are flooded. Little control is possible over low-level exposure to naturally occurring sources of mercury from soils and sediment.

#### **Human Health Considerations**

Mercury is a well-documented human health hazard. Avoid exposure to elemental mercury, which is volatile and can be inhaled in significant amounts in enclosed areas, mercury-based seed treatments, and mercury-contaminated food. One should wear gloves when handling carcasses, but birds thought to have died of mercury poisoning present no special hazard because the mercury is biologically bound to tissues within the carcass.

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## **Supplementary Reading**

- Eisler, R., 1987, Mercury hazards to fish, wildlife, and invertebrates: a synoptic review: U.S. Fish and Wildlife Service Biological Report 85(1.10), 90 p.
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- Thompson, D.R., 1996, Mercury in birds and terrestrial mammals, in Beyer, W.N., and others, eds., Environmental contaminants in wildlife: interpreting tissue concentrations: Boca Raton, Fla., Lewis Publishers, p. 341–356.
- Wren, C.D., Harris, S., and Harttrup, N., 1995, Ecotoxicology of mercury and cadmium, *in* Hoffman, D.J., and others, eds., Handbook of ecotoxicology: Boca Raton, Fla., Lewis Publishers, p. 392–423.