Chapter 40

# Chlorinated Hydrocarbon Insecticides

**Synonyms**

*Organochlorines, OCs*

Chlorinated hydrocarbon insecticides (OCs) are diverse synthetic chemicals that belong to several groups, based on chemical structure. DDT is the best known of these insecticides. First synthesized in 1874, DDT remained obscure until its insecticidal properties became known in 1939, a discovery that earned a Nobel Prize in 1948. The means of synthesizing the cyclodiene group, the most toxic of the OCs, was discovered in 1928 and resulted in a Nobel Prize in 1950. The insecticidal properties of cyclodienes, which include aldrin, dieldrin, and endrin (Table 40.1), were discovered about 1945. OCs became widely used in the United States following World War II. Their primary uses included broad spectrum applications for agricultural crops and forestry and, to a lesser extent, human health protection by spraying to destroy mosquitoes and other potential disease carriers. These compounds also became widely used to combat insect carriers of domestic animal diseases.

## Cause

Chlorinated hydrocarbon insecticides are stored in body fat reserves or are lipophilic, and they remain in the environment for long periods of time after application. They bioaccumulate or are readily accumulated by animals through many exposure routes or repeated exposure and they tend to biomagnify or accumulate in higher concentrations in animals that are higher in the food chain. This combination of bioaccumulation and biomagnification can harm or kill wildlife, especially some species of birds. The highly toxic cyclodiene compounds cause direct mortality of birds as well as secondary poisoning, which results when birds prey on organisms dying from insecticide applications. Reproductive impairment is the primary effect of the less acutely toxic DDT and its metabolites, DDD and DDE. The cumulative storage of OC residues within body fat reserves presents an additional hazard for birds. Rapid use and depletion or mobilization of fat reserves during migration, food shortages, and other stressful conditions release OC residues into the blood. The residues are then carried to the brain, where they can reach toxic levels resulting in acute poisoning.

## Species Affected

Acute mortality from exposure to OCs has been documented in many bird species (Table 40.1). However, the toxicity for birds of different types of these insecticides varies greatly (Tables 40.2 and 40.3). In general, birds that are higher in the food chain are more likely to be affected by OCs present in the environment than birds that are lower in the food chain. This is especially true for fish-eating birds and raptors (Fig. 40.1). Environmental biomagnification of these contaminants can be seen in the mortality of robins and other birds from DDT. Leaves from trees that were sprayed with DDT to control Dutch elm disease had high residues of DDT (174–273 parts per million) shortly after spray applications. When the leaves dropped in the fall, they still contained 20–28 parts per million of DDT. This leaf litter, along with spray residue that reached the ground, produced high DDT residues in the top levels of soil. Earthworms that fed in those soils concentrated the residues to a level high enough to kill birds that fed on them. Another hazard is OC seed dressings, which are used to prevent insect damage to agricultural crops, that may be ingested by waterfowl and other seed or grain-eating birds.

## Distribution

Exposure to chlorinated hydrocarbon insecticides is global, and residues of these compounds are found in nearly every environment, even in Antarctica and the Arctic. Avian mortalities from OCs have been reported from Europe, Asia, North America, and South America. Poisoning may occur anywhere that birds are exposed to point sources of these chemicals or through bioaccumulation and biomagnification. Because of their environmental persistence and global movement, residues of chlorinated hydrocarbon insecticides impact bird health long after they become environmental contaminants and at locations far from the original application sites. For example, DDT compounds, polychlorinated biphenyls (PCBs), and dioxin-like compounds were recently found in black-footed albatross adults, chicks, and eggs on Midway Atoll in the Pacific.

## Seasonality

Exposure of birds to OCs is most likely during spring and summer in countries where these compounds are still used to control insect pests during the growing season, but exposure may occur any time that residues are present in food sources. For example, waterfowl and other birds that fed on

***Table 40.1*** *Examples of avian mortality events caused by chlorinated hydrocarbon insecticides.*

| Insecticide | Purpose of application | Means of bird exposure | Bird group affected | Principal species affected | Event location and time period |
|---|---|---|---|---|---|
| DDT | Spray application to control Dutch elm disease. | Biomagnification in terrestrial food chain. | Passerines | Robin and other small birds. | New England, Midwest; late 1940s to 1950s. |
| DDD | Spray application to control gnats. | Biomagnification in aquatic food chain. | Grebes | Western grebe | Clear Lake, California, 1950s. |
| Aldrin | Treatment of rice seed to combat agricultural pests. | Consumption of treated seeds, use and depletion of stored fat reserves during migration or periods of stress. | Waterfowl | Fulvous whistling duck, snow goose, blue-winged teal. | Texas, 1970s |
| Heptachlor | Treatment of wheat seed to control agricultural pests. | Consumption of treated seeds. | Waterfowl | Canada goose | Oregon, 1970s |
| Toxaphene | Spray application to control agricultural pests. | Direct contact with and consumption of contaminated food. | Waterfowl | Ducks and coot | California, 1960s |
| | Spray application for fisheries management. | Ingestion of contaminated food. | Waterfowl | Blue-winged teal, shoveler, mallard. | Nebraska, 1960s |
| Dieldrin | Spray application to control agricultural pests. | Biomagnification in food chain. | Raptors | Bald eagle, peregrine falcon | Nationwide, 1960s and 1970s. |
| Endrin | Spray application to control orchard rodents. | Direct contact with spray; consumption of contaminated food; biomagnification; use and depletion of fat reserves. | Gallina-ceous birds, raptors, geese | Quail, chukar partridge, goshawk, Cooper's hawk, barn owl, Canada goose. | Washington, 1960s 1970s, 1980s. |
| Chlordane | Dry formulations to control soil pests and termites. | Consumption of contaminated food; biomagnification. | Raptors, passerines | Great horned owl, American kestrel, Cooper's hawk, blue jay, robin, starling. | New York, Maryland, New Jersey, 1980s. |

***Table 40.2*** *Toxicity for the mallard duck of some chlorinated hydrocarbon insecticides.*
*[Modified from Heinz and others, 1979. $LC_{50}$ is the insecticide concentration, in parts per million, in feed that is required to kill 50 percent of birds during a given period of time. $LD_{50}$ is the insecticide amount, in milligrams per kilogram of body weight, in a single dose that is required to kill 50 percent of birds. ppm, parts per million; mg/kg, milligrams per kilogram; >, greater than; ≥, greater than or equal to. — no data available]*

| Insecticide | Subacute exposure $LC_{50}$ (ppm) | Acute exposure $LD_{50}$ (mg/kg) |
|---|---|---|
| Aldrin | 155 | 520 |
| Chlordane | 858 | 1,200 |
| DDT | 1,869 | >2,240 |
| Dieldrin | 169 | 381 |
| Endosulfan | 1,053 | 33 |
| Endrin | 22 | 5.6 |
| Heptachlor | 480 | ≥2,000 |
| Lindane | — | >2,000 |
| Mirex | >5,000 | >2,400 |
| Toxaphene | 538 | 71 |

***Table 40.3*** *Relative acute toxicity of chlorinated hydrocarbon insecticides for birds.*
*[Modified from Hudson and others, 1984. $LD_{50}$ is the insecticide amount, in milligrams per kilogram of body weight, in a single dose that is required to kill 50 percent of birds. mg/kg, milligrams per kilogram; >, greater than; <, less than.]*

| Species | $LD_{50}$ (mg/kg) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Aldrin | Chlordane | DDT | Dieldrin | Endosulfan | Endrin | Lindane | Mirex | Toxaphene |
| Canada goose | | | | <141 | | | | | |
| Mallard duck | 520 | 1,200 | >2,240 | 381 | 31–45 | 5.6 | 2,000 | 2,400 | 70.7 |
| Fulvous whistling duck | 29.2 | | | 100–200 | | | | | 99 |
| Sandhill crane | | | >1,200 | | | | | | 100–316 |
| Pheasant | 16.8 | 24–72 | 1,334 | 79 | 80 to >320 | 1.8 | | >2,000 | 40 |
| Sharp-tailed grouse | | | | | | 1.1 | | | 19.9 |
| Gray partridge | | | | 8.8 | | | | | 23.7 |
| Chukar partridge | | | | 25.3 | | | | | |
| Bobwhite quail | 6.6 | | | | | | | | 85.5 |
| California quail | | 14.1 | 595 | 8.8 | | 1.2 | | | 23.7 |
| House sparrow | | | | 47.6 | | | | | |
| Horned lark | | | | | | | | | 581 |

endrin-treated winter wheat seed have died in the autumn, and raptors have died yearround. Reproductive effects are manifested during the breeding season, but the exposure that causes these effects can occur at any time of year.

## Field Signs

Thin eggshells that often collapse under the weight of the nesting bird and eggs that break during incubation (Fig. 40.2) are classic signs of exposure to DDT and some other OCs. Clinically ill birds suffering from acute poisoning often exhibit signs of central nervous system disorders such as tremors, incoordination, and convulsions (Fig. 40.3). Other birds may be lethargic and exhibit additional behavioral changes (Table 40.4).

## Gross Lesions

Birds dying of chronic exposure to OCs are often emaciated (Fig. 40.4). Those that die acutely usually exhibit no lesions. The pathological effects attributed to exposure to these compounds (Table 40.4) are not unique and, therefore, they cannot be used as the only basis for diagnosis.

## Diagnosis

Residue analysis combined with necropsy findings, clinical signs, and an adequate field history are generally required for a diagnosis of chlorinated hydrocarbon insecticide poisoning. Brain is the tissue of choice for residue analysis because chemical concentrations that indicate poisoning in birds have been determined for several of these compounds. Take care not to contaminate tissues for residue analysis. Submit the entire carcass whenever possible, otherwise remove the head and send it intact to the laboratory. When it is necessary to remove the brain or other tissues for analysis, rinse the instruments with a solvent, such as acetone or hexane, to remove chlorinated hydrocarbon insecticide residues from them. Place the tissues in solvent-rinsed glass containers or wrap them in aluminum foil. The foil should not have been prepared by a manufacturer that uses oils made of animal fats. A "K" on the package label indicates that no animal fats were used in the manufacturing process.

Experimental studies have been done in an attempt to establish lethal brain levels for OCs in various species of birds (Fig. 40.5). DDE levels in the brains of bald eagles thought to have died from this contaminant have ranged from 212 to 385 parts per million (wet weight), and these levels are consistent with brain DDE levels of kestrels that died from experimental dosing studies (213–301 parts per million, wet weight). These findings are important for interpreting field data (Fig. 40.6). However, interpretation of residue values is complicated by the simultaneous occurrence of other contaminants that may combine with, interact with, or inhibit the toxic effects of any individual compound. Other factors, such as sex, age, and nutritional level also may affect toxicity.

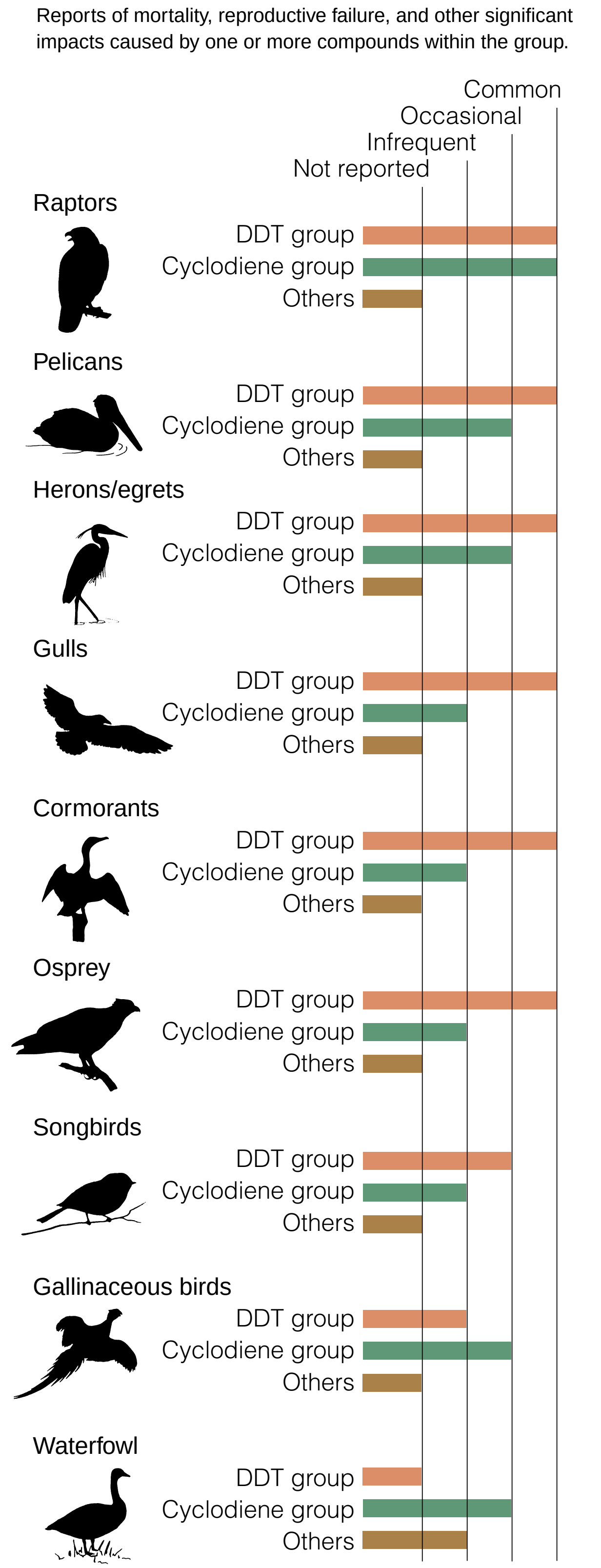


***Figure 40.1*** *Relative importance of health effects caused by chlorinated hydrocarbon insecticides in selected free-living birds.*

*Photo by Milton Friend*

***Figure 40.2*** *The flattened egg within this white-faced ibis clutch was caused by DDE.*

***A***

***B***

*Photos by Milton Friend*

***Figure 40.3*** *Damage to the central nervous system of birds by chlorinated hydrocarbon insecticides results in the type of aberrant posture seen in **(A)** this hen pheasant and **(B)** this mallard duck.*

***Table 40.4*** *Most commonly reported effects from chlorinated hydrocarbon insecticide exposures of birds.*

| General effect | Specific effects |
|---|---|
| Behavioral | Lethargy, slowness, depression |
| | Locomotive and muscle incoordination (ataxia) |
| | Tremors and convulsions |
| | Reduced nest attentiveness and nest abandonment |
| | Violent wing beating |
| | Aberrant wing and body carriage |
| | Muscle spasms causing the body to bend backwards and become rigid (opisthotonos) |
| Reproductive | Embryo mortality |
| | Decreased egg hatchability |
| | Decreased egg production |
| | Eggshell thinning |
| | Egg breakage during incubation |
| Pathological | Emaciation; muscle wasting and absence of fat |
| | Congestion of the lungs, kidneys, and especially the liver have been reported in pheasants dying from dieldrin poisoning |
| | Increased liver weight |
| | Small spleens have been reported |
| | In general, pathological changes are not readily evident at the gross level, and microscopic changes are not diagnostic |
| Immunological | Increased susceptibility to infectious disease |
| Other | Disruption of salt gland function by DDE |

Photo by James Runningen

***Figure 40.4*** *Chronic toxicity from chlorinated hydrocarbon insecticides can result in emaciation, demonstrated by the prominent keel and lack of subcutaneous fat in this black duck. In addition, emaciation caused by the rapid use and depletion of body fat stores due to stresses of migration, inadequate food supplies, and other causes can concentrate body residues of chlorinated hydrocarbons in the brain and cause acute toxicity.*

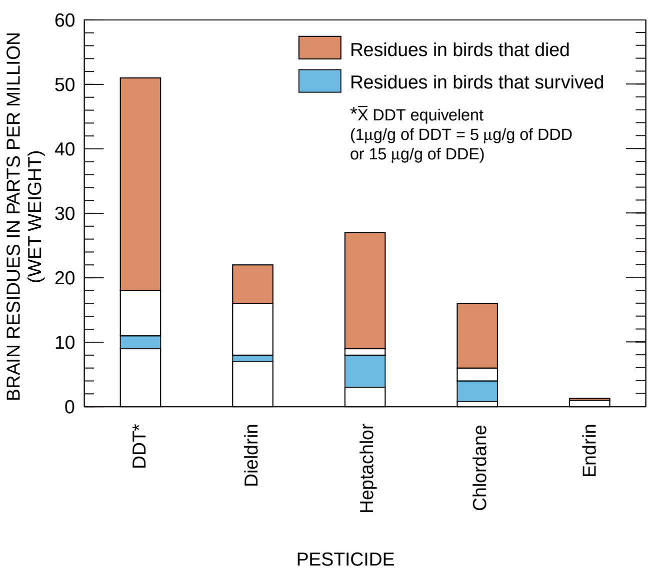


***Figure 40.5*** *Mean chlorinated hydrocarbon insecticide residues in brains of experimentally dosed passerines.*

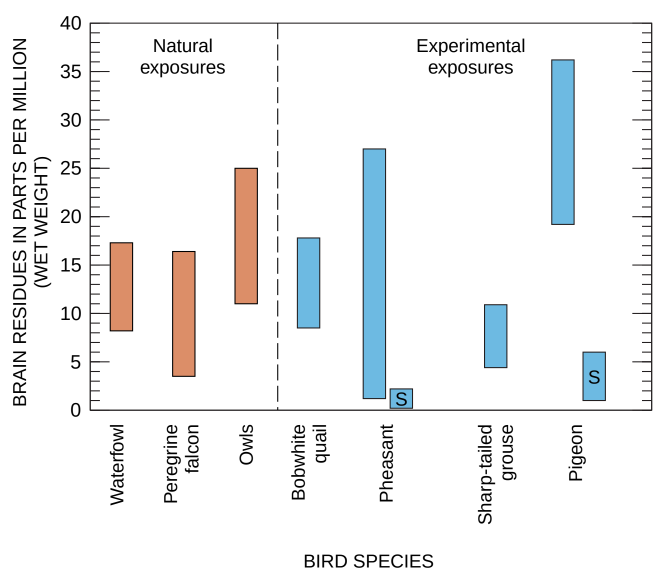


***Figure 40.6*** *Range of dieldrin residues in brains of selected avian species. [S is the range of concentration within which some of the birds survived.]*

## Control

Because uses of most OCs have been banned or greatly curtailed in the United States, controlling wildlife exposure to these compounds depends largely on properly disposing of existing stores, preventing leakage into the environment, and preventing malicious use. The spreading of these compounds to environments where they are no longer used will continue until suitable alternative pest controls are found. Also, migratory wildlife that are exposed to these compounds in areas where they are still used may not exhibit effects until they reach other areas on their migratory route.

## Human Health Considerations

As with many of the other toxins discussed in this section, residues of chlorinated hydrocarbons in birds are stored in tissues and are not transferred to humans through routine handling of carcasses. Exceptions include situations where a person could somehow come in contact with the pesticide, for example, in the stomach contents of a bird or on its skin or feathers. It is always wise to handle carcasses with disposable gloves, and to treat unknown mortalities as possible sources of infectious agents transferrable to humans.

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## Supplementary Reading


Blus, L.J., 1995, Organochlorine pesticides, *in* Hoffman, D.J., and others, eds., Handbook of ecotoxicology: Boca Raton, Fla., Lewis Publishers, p. 275–300.

Blus, L.J., Wiemeyer, S. N., and Henny, C.J., 1996, Organochlorine pesticides, *in* Fairbrother, A., and others, eds., Noninfectious diseases of wildlife (2nd ed.): Ames, Iowa, Iowa State University Press, p. 61–70.

Heinz, G.H., Hill, E.F., Stickel, W.H., and Stickel, L.F., 1979, Environmental Contaminant studies by the Patuxent Wildlife Research Center, *in* Kenaga, ed., Avian and mammalian wildlife toxicology: Philadelphia, Penn., American Society for Testing and Materials, Special Technical Publication 693, p. 9–35

Hudson, R.H., Tucker, R.K., and Haegele, M.A., 1984, Handbook of toxicity of pesticides to wildlife, (2d ed.): Washington, D.C., U.S. Department of the Interior, Fish and Wildlife Service, Resources Publication 153, 90 p.

Peakall, D.B., 1996, Dieldrin and other cyclodiene pesticides in wildlife, *in* Beyer, W.N., and others, eds., Environmental contaminants in wildlife: interpreting tissue concentrations: Boca Raton, Fla., Lewis Publishers, p. 73–97.

Chapter 41

# Polychlorinated Biphenyls

**Synonyms**

*PCBs, aroclors, chlorinated biphenyls*

Polychlorinated biphenyls (PCBs) are industrial compounds with multiple industrial and commercial uses (Table 41.1). PCBs are chemically inert and stable when heated. These properties contribute greatly to PCBs having become environmental contaminants. The chemical inertness and heat stability properties that make PCBs desirable for industry also protect them from destruction when the products in which they are used are discarded. These same properties also enable PCB residues to persist in the environment for long periods of time and to be transported worldwide when contaminated particulate matter travels through waters, precipitation, wind, and other physical forces.

PCBs have a physical structure similar to DDT, and, like DDT, they are classified as aromatic hydrocarbons which contain one or more benzene rings. The presence of chlorine results in DDT, PCBs, and other compounds with similar structures commonly being referred to as chlorinated hydrocarbons. The toxicity of these compounds is associated with the amount of chlorine they contain. The trade name of Aroclor® for PCBs that were produced by a manufacturer in the United States contains a numerical designation that specifies the amount of chlorine present in a particular formulation. For example, Aroclor® 1221 contains 21 percent chlorine while Aroclor® 1254 contains 54 percent chlorine. The first two digits designate the number of carbons in the formulation. The chemical structure of PCBs results in the possibility of many different forms or isomers, (more commonly called congeners) of these compounds. PCBs in other countries have different trade names than Aroclor® (Table 41.2).

## Cause

Like other chlorinated hydrocarbons, PCBs accumulate in the fat of animals or are lipophilic, and they tend to become concentrated at higher levels of the food chain. In general, persistence increases for PCBs that are made with higher amounts of chlorine. Birds are most susceptible to PCB compounds of the mid-chlorination range (42–54 percent).

## Species Affected

Mammals, especially mink, are more susceptible than birds and invertebrates to direct toxicity from PCBs. The highest tissue concentrations of these compounds are found among birds, especially marine species that are at the top of complex oceanic food webs and among fish-eating birds, such as cormorants, that use large inland water bodies. For example a 12.9-fold increase has been reported from plankton to fish in a Lake Michigan food web. Although direct toxicity for birds is generally low (Table 41.3), PCBs are powerful inducers of liver enzyme systems that increase the metabolism of hormones. PCBs may have caused thin eggshells in double-crested cormorants and white pelicans, and under experimental conditions, in ring-doves and (perhaps) in Coturnix quail and mallard ducks. Unfortunately, there is insufficient knowledge to clearly define the impacts of PCBs on bird reproduction, especially in field situations, because tissue residues are often highly correlated with other lipophilic compounds, such as organochlorines. Findings have generally been inconclusive, but the greatest effects have been seen in gallinaceous birds such as pheasants, chickens, and doves.

## Distribution

PCBs were first identified in the tissues of wildlife in Sweden, and they are now known to occur in a wide variety of wildlife and other species, including humans, throughout the world. PCBs are clearly global contaminants, and they are the most abundant of the chlorinated hydrocarbon pollutants in the global ecosystem with the possible exception of petroleum products. Industrial wastes released into aquatic systems, point sources of contamination from manufacturing facilities, landfills receiving waste from such facilities, and combustion and other disposal of products containing PCBs are generally recognized sources of contamination. Another less well-known source of PCB contamination of the environment was the use of PCBs during the 1950s and 1960s as additives to extend the residual life and effectiveness of expensive chlorinated insecticides such as chlordane, aldrin, dieldrin, and benzene hexachloride. It is estimated that more than 1.5 metric tons of PCBs have been produced worldwide. PCB manufacturing in the United States was discontinued in 1978.

The variable environmental distribution of PCBs results from their physical and chemical properties, which influence their rates of distribution, retention, and degradation in different environments. This results in great differences in the relative concentrations of the different forms of PCBs found in wildlife samples from different geographic areas and is also a reflection of the magnitude of local and regional con-

***Table 41.1*** *Uses of polychlorinated biphenyls (PCBs) in industry and products for society.*

| | |
|---|---|
| **Properties** | |
| | Heat stability |
| | Chemical stability |
| | Ability to be mixed with organic compounds |
| | Slow degradation |
| **Industrial uses** | |
| | Lubricants, hydraulic fluids, grinding fluids |
| | Heat transfer agents, insulators |
| | Plasticizers |
| | Dielectric sealants |
| | Dedusting agents |
| | Protective coatings |
| **Common products that have contained PCB additives** | |
| | Wire and cable coating |
| | Impregnants for braided cotton-asbestos insulation |
| | Printing inks and mimeograph inks |
| | Preparation of imitation gold leaf |
| | Pigment vehicle for decoration of glass and ceramics |
| | Essential components of coating for flameproofing cotton drill for outer garments and for rendering olive-drab canvas fire retardant, water-repellant, and rot-proof (tents, tarpaulins) |
| | Moistureproof coating for wood, paper, concrete, and brick |
| | Asphalt, roof coatings |
| | High quality precision casting wax; waxes used in making dental castings and costume jewelry |
| | Sealers for masonry, wood, fiberboard, and paper |
| | Window envelopes |
| | Polystyrene, polyethylene, neoprene, polybutene, silicone rubber, crepe rubber |
| | Plasticizers in paints |
| | Life extenders and sometimes toxicity synergists for pesticides containing DDT, dieldrin, lindane, chlordane, aldrin, and benzene |

tamination patterns, environmental transport processes, and the composition of PCB residues in the food chain.

## Seasonality

Exposure to PCBs is not seasonally dependent; except that in warm weather, PCB residues may vaporize or evaporate with liquid from contaminated areas, and thus, increase the risk of airborne exposure.

## Field Signs

Direct mortality of wild birds from exposure to PCBs rarely occurs. We are only aware of one such event having been documented. The number of different PCBs present in the environment further complicates evaluations because of different impacts and lethality associated with these different compounds. Nonspecific signs associated with acute exposure of birds to toxic levels of PCBs include lethargy, lack of locomotive and muscle coordination or ataxia, tremors, and other observations. Behavioral modifications and impaired reproductive performance may also occur and would be more readily detected at the population rather than individual level (Table 41.4).

## Gross Lesions

There are no diagnostic lesions associated with exposure to PCBs. Enlarged liver and kidneys, atrophy of the spleen and the bursa of Fabricius, emaciation, and excess fluids around the heart have been associated with chronic exposure.

Excess fluid or edema in tissues has been found in some cases of acute PCB exposure, and this suggests that PCBs may interfere with tissue permeability or cardiac function or both. PCBs have been shown to cause physical defects in embryos, or be teratogenic, in chickens, and they also cause

***Table 41.2*** *Trade names for polychlorinated biphenyls (PCBs).*

| Trade name | Country of manufacturer | Manufacturer |
|---|---|---|
| Aroclor® | United States of America | Monsanto |
| Clophens® | Germany | Bayer |
| Fenclors® | Italy | Caffaro |
| Phenoclors®; Pyralenes® | France | Prodelec |
| Kanechlors® | Japan | Kanegafuchi |
| Others have been produced in Czechoslovakia and the former USSR | | |

***Table 41.3*** *Relative toxicity of polychlorinated biphenyls (PCBs) for birds. [Adapted from Eisler, 1986. $LC_{50}$ is the contaminant concentration in the diet that is required to kill 50 percent of the test animals in a given period of time; by comparison, the $LC_{50}$ for mink to Aroclors® 1242 and 1254 is 8.6 and 6.7, respectively. mg/kg, milligrams per kilogram. >, greater than. —, no data available.]*

| | $LD_{50}$ (mg/kg of Aroclor®) | | | |
|---|---|---|---|---|
| Species | 1221 | 1242 | 1254 | 1260 |
| Bobwhite quail | >6,000 | 2,098 | 604 | 747 |
| Mallard duck | — | 3,182 | 2,699 | 1,975 |
| Ring-necked pheasant | >4,000 | 2,078 | 1,091 | 1,260 |
| Japanese quail | >6,000 | >6,000 | 2,898 | 2,186 |
| European starling, red-winged blackbird, brown-headed cowbird | — | — | 1,500 | — |

***Table 41.4*** *Reported effects of polychlorinated biphenyls (PCBs) in birds.*

| Type of impact | Examples |
|---|---|
| Behavioral | Lethargy |
| | Locomotive and muscle incoordination or ataxia |
| | Tremors and convulsions |
| | Reduced nest attentiveness and protection of eggs |
| Reproductive | Embryo mortality resulting in decreased hatchability of eggs |
| | Decreased egg production |
| | Egg shell thinning |
| Pathological | Accumulation of fluid within the pericardial sac or hydropericardium |
| | Excess fluid or edema in body tissues and organs |
| | Atrophy of bursa of Fabricius, spleen, and other lymphoid tissues |
| | Enlarged livers that are firm and light colored |
| | Bill and foot deformities (from embryonic exposure) |
| Immunological | Increased susceptibility to infectious disease |
| Other | Weight loss |
| | Debilitation |

a condition analogous to chick edema disease. This condition results in the leakage of body fluids into various organs and tissues. However, the presence of dioxins as contaminants within the PCB formulations may be the actual cause of these lesions.

## Diagnosis

Diagnosis of acute poisoning is based on PCB residues in tissues, and as for most other chlorinated hydrocarbons, mortality is best diagnosed from residues found in brain tissue. However, the concentrations of PCBs that indicate poisoning vary greatly with the specific formulation of PCBs, the species of bird, and, often, the presence of other environmental contaminants. Detection of subacute effects, such as poor reproductive performance and immunosuppression, is also confounded by these same factors. Comparison of residues in the tissues of birds suspected of being poisoned with residues in tissues of normal birds of the same species in nearby or regional sites can be diagnostically useful along with knowledge of PCB deposition and discharges in the area. Comparisons are sometimes difficult because of the varying effects of different PCB mixtures and the interactions that occur between PCBs, other pollutants, and other disease agents. Many toxic and biochemical responses from PCB exposure occur in multiple species and body organ systems.

Residue levels alone will generally not be sufficient data for making a diagnosis. Necropsy findings combined with laboratory analyses, including residue evaluations, knowledge of environmental conditions and events at the field site, and response of different species to PCB exposure are all needed for sound judgements to be reached.

## Control

Prevention of the entry of PCBs into the environment and containment or removal of PCB contamination that is already present are necessary to reduce exposure of wildlife. PCB sales in the United States were stopped in the 1970s, but large amounts are still present in the environment due to environmental persistence and to global transport by winds and other means from locations where PCBs are still used. Improper disposal of products that contain PCBs through landfills and incineration at temperatures that are too low (below 1,600 °C) to destroy PCBs can cause further environmental contamination. However, more stringent air-quality standards in the United States and other nations have diminished the potential that PCBs in incinerated materials will be added to the environment through combustion.

Bird use of heavily contaminated sites should be prevented to the extent feasible by habitat manipulation, physical barriers, scaring devices, and other appropriate means. Knowledge of PCB levels in specific environments should be gained prior to developing those areas for wildlife, including the use of dredge material to create artificial islands for bird nesting habitat. PCB and heavy metal loads in sediments should also be considered in decisions regarding dumping dredge materials.

## Human Health Considerations

PCBs are known to accumulate in humans, and health advisories are often issued about consuming wildlife from heavily contaminated environments. Residues in wildlife can only be transferred to humans by consuming contaminated tissues. As with most chlorinated hydrocarbons, the greatest concentrations of residues are in fat tissue, and removing fatty parts of the carcass prior to cooking can significantly reduce potential human exposure. Although PCB residues cannot be transferred to humans from wildlife by means other than consumption, the cause of death is seldom known when dead wildlife are encountered and the risk of exposure to disease agents that can be transmitted by contact should not be taken. Always wear gloves or use other physical barriers to prevent personal contact with the carcass.

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## Supplementary Reading


Eisler, R., 1986, Polychlorinated biphenyl hazards to fish, wildlife, and invertebrates: a synoptic review: Fish and Wildlife Service Biological Report 85(1.7), 72 p.

Hoffman, D.J., Rice, C.P., and Kubiak, T.J., 1996, PCBs and dioxins in birds, *in* Beyer, W.N., and others, eds., Environmental contaminants in wildlife: interpreting tissue concentrations: Boca Raton, Fla., Lewis Publishers, p. 165–207.

O'Hara, T.M., and Rice, C.D., 1996, Polychlorinated biphenyls, *in* Fairbrother, A., and others, eds., Noninfectious diseases of wildlife (2nd ed.): Ames, Iowa, Iowa State University Press, p. 71–86.

Rice, C.P., and O'Keefe, P., 1995, Sources, pathways, and effects of PCBs, dioxins, and dibenzofurans, *in* Hoffman, D.J., and others, eds., Handbook of ecotoxicology: Boca Raton, Fla., Lewis Publishers, p. 424–468.

# Chapter 42
# Oil

### Synonyms

*Petroleum*

Each year, an average of 14 million gallons of oil from more than 10,000 accidental spills flow into fresh and saltwater environments in and around the United States. Most accidental oil spills occur when oil is transported by tankers or barges, but oil is also spilled during highway, rail, and pipeline transport, and by nontransportation-related facilities, such as refinery, bulk storage, and marine and land facilities (Fig. 42.1). Accidental releases, however, account for only a small percentage of all oil entering the environment; in heavily used urban estuaries, the total petroleum hydrocarbon contributions due to transportation activities may be 10 percent or less. Most oil is introduced to the environment by intentional discharges from normal transport and refining operations, industrial and municipal discharges, used lubricant and other waste oil disposal, urban runoff, river runoff, atmospheric deposition, and natural seeps. Oil-laden wastewater is often released into settling ponds and wetlands (Fig. 42.2). Discharges of oil field brines are a major source of the petroleum crude oil that enters estuaries in Texas.


***Figure 42.2*** *Wastewater laden with petroleum being discharged into a settling pond.*

## Cause

Birds that are exposed to spilled or waste petroleum can be affected both externally and internally. Oil contamination of feathers (Fig. 42.3) disrupts their normal structure and function, and it results in the loss of insulation for warmth and waterproofing. Oiled birds lose the ability to fly, and they frequently die from hypothermia, starvation, exhaustion, or drowning. Birds that are exposed to oil during their reproductive season can also transfer lethal doses of the contaminant to their eggs during incubation. Even small quantities of oil (5–20 microliters) externally applied to eggs can kill embryos. Birds can also ingest, inhale, or absorb oil when exposed to a spill or while preening contaminated plumage. The toxic effects of ingested oil vary, depending on the type of oil and on the species of birds affected. These effects include gastrointestinal irritation and hemorrhaging, anemia, reproductive impairment, depressed growth, and osmoregulatory dysfunction (Table 42.1). Polycyclic aromatic hydrocarbons (PAH) contribute to the toxicity of crude petroleum and refined petroleum products, but the amounts of PAH in petroleum products vary greatly.

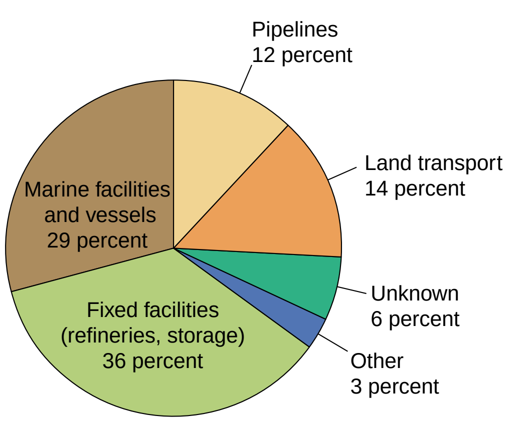



***Figure 42.1*** *Origin of petroleum spills, 1987–94.*

Unfortunately, the effects of petroleum pollution can persist long after the visible spill is cleaned or dispersed. Petroleum persistence in the water column is usually less than 6 months, but it can be much longer (more than 10 years) in other components of the environment. Chronic losses may result when birds ingest oil in contaminated food items. For example, oil from the 1989 Exxon Valdez spill is still sequestered in bivalve communities within the areas of contamination and, thus, is still available to birds and other wildlife that feed on bivalves. Subtle effects on reproduction, such as decreased egg production, reduced fertility and hatchability, and decreased sperm production, as well as reduced immunologic function and impaired disease resistance, may occur as a result of ingesting oil-contaminated food (Table 42.1).

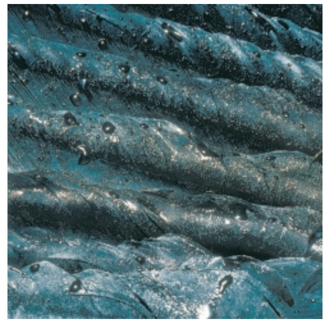



***Figure 42.3*** *Oiling disrupts normal feather structure and function.*

***Table 42.1*** *Commonly reported effects of oil toxicosis in birds.*

| Impact | Consequences |
|---|---|
| **Mechanical** | |
| Loss of waterproofing and insulation value of feathers | Wetting, chilling, and hypothermia leading to death |
| | Exhaustion due to depletion of body stores, inability to feed, and greater expenditure of energy to maintain body heat and stay afloat |
| | Altered behavior |
| | Drowning |
| **Toxicological** | |
| Pathological changes in tissue | Inflammation of gastrointestinal lining |
| | Malformations |
| | Embryotoxicity |
| Physiologic disruptions | Altered endocrine function |
| | Liver and kidney disorders |
| | Altered blood chemistry |
| | Blood disorders including anemia |
| | Impaired salt (nasal) gland function resulting in disruption of osmoregulation |
| **Reproductive** | |
| | Embryotoxicity |
| | Impaired reproduction |
| **Other** | |
| | Reduced growth and development |
| | Reduced immunologic function |
| | Impaired disease resistance |

## Species Affected

A wide variety of birds and other wildlife have been affected by oil. The bird species affected depend on the location of the oil and the behavior of the birds. Species that suffer the greatest losses are gregarious, spend most of their time on the water, often near shipping lanes, and dive into the water to find food or to avoid disturbance. Seabirds, such as auks, guillemots, murres, puffins, sea ducks, and penguins, are particularly susceptible to contamination from oil spills (Fig. 42.4). In addition, annual losses of marine birds occur due to natural oil seeps along the Santa Barbara Channel of the California coast.

## Seasonality

Species with high reproductive rates may quickly recover from a spill, but for species with low reproductive rates, such as brown pelicans, oil pollution can cause catastrophic losses and it may take decades for populations to return to prespill numbers. Even oiled brown pelicans that have been successfully rehabilitated have reduced reproductive success.

Winter storms increase the likelihood of transport spills, making January, February, and March the peak spill season. This is also the time of year when seabirds and waterfowl congregate in wintering areas, resulting in an increased potential for significant bird losses.

Sea and bay ducks (scoters, scaups, oldsquaws, canvasback) that tend to concentrate on wintering grounds and diving birds (grebes, loons, and mergansers) that overwinter in marine environments or on large water bodies with commercial shipping are quite vulnerable to oil pollution, especially during winter months. Eiders are vulnerable most of the year.

## Distribution and Extent of Mortality

The oiling of migratory birds is not limited to specific geographic areas. Accidental oil spills have occurred in all 50 States including inland waters, such as rivers and non-navigable waters, and in open coastal waters, ports and harbors. Although it is not possible to accurately estimate the number of birds lost to oil pollution, in many cases the mortality has been substantial (Table 42.2). Bird losses of 5,000 or more are common for larger oil spills. Reports are usually of the numbers of oiled birds found dead or moribund on the shore, but these estimates may be inaccurate because of search biases, accessibility of the shore, losses of birds that have sunk to the bottom, and other factors. An important source of error in estimating losses in marine environments is the unknown proportion of oiled birds that die at sea but that do not reach the coast.

In addition to accidental spills, other opportunities for animal exposure to oil occur in association with oil production, petroleum refining, and highly industrialized locations throughout the United States. Persistent oil pollution is a chronic problem around marinas and ports due to discharges from shipping and boating activities and storage tank clean-

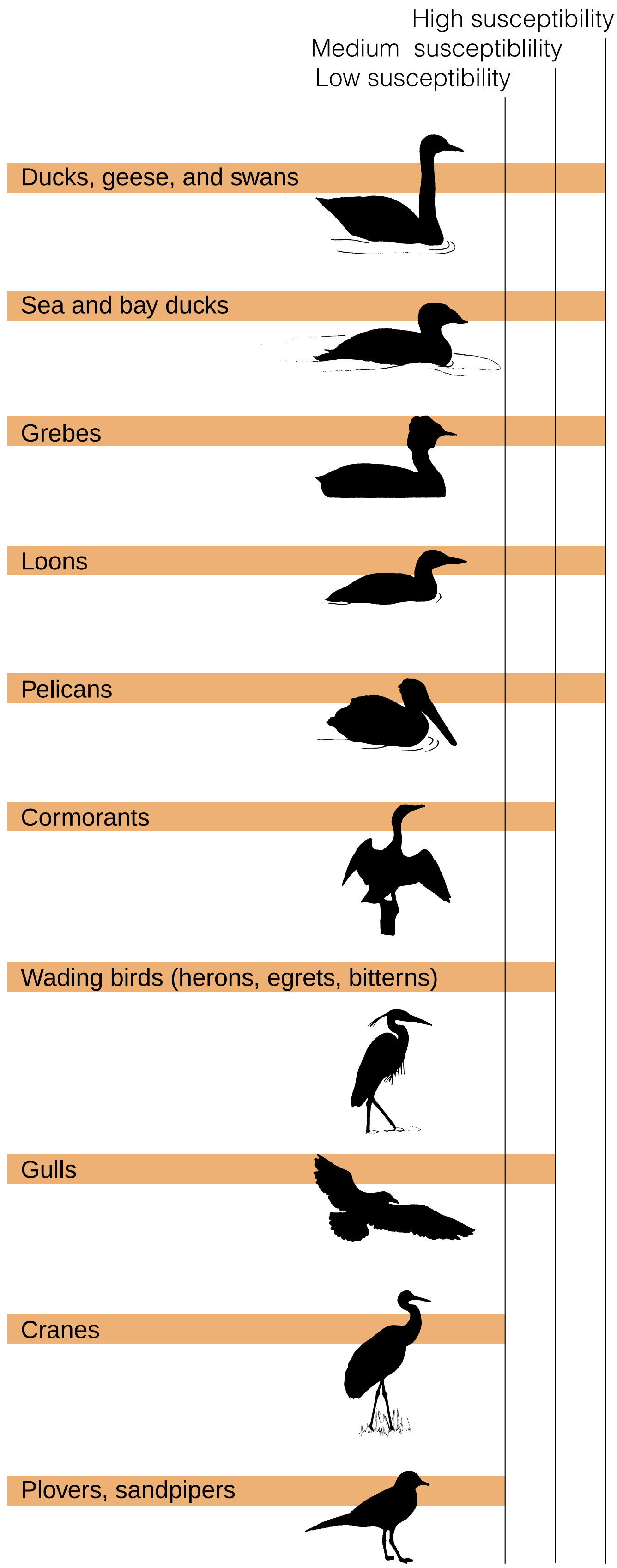



***Figure 42.4*** *Relative susceptibility of birds to oiling.*

*Table 42.2* *Examples of bird mortality from oil spills.*

| Vessel or source | Year | Site | Estimated bird mortalities |
|---|---|---|---|
| Exxon Valdez | 1989 | Prince William Sound, Alaska | 350,000–390,000 |
| Nestucca | 1988 | Grays Harbor, Washington | 50,000 |
| Amoco Cadiz | 1978 | Brittany, France | 20,000 |
| Barge STC-101 | 1976 | Chesapeake Bay, Virginia | 20,000–50,000 |
| Torrey Canyon | 1967 | English Channel | 30,000 |
| Gerda Maersk | 1955 | Elbe River, Germany | 500,000 |

ing, but, unfortunately, the numbers of birds affected by oil pollution in these areas are unknown. In the Playa Lakes regions of eastern New Mexico, northwestern Texas, and western Oklahoma, open pits and tanks containing oil and oil-field wastes have been reported to claim the lives of approximately 100,000 birds each year (Fig. 42.5).

Photo by James Runningen

***Figure 42.5*** *These oiled birds were recovered from oil-field wastewater pits in the southwestern United States.*

## Field Signs

Major oil spills are frequently accompanied by intensive media coverage, and they may be well publicized before slicks or affected birds appear. However, small spills, especially those of unknown origin, often go unnoticed except for the appearance of a few contaminated birds. Oiled birds are frequently wet and chilled because the oil damages feather waterproofing and insulating properties (Fig. 42.6); birds may ride lower in the water than normal because they have lost feather buoyancy. Oiling is suggested when water birds leave the water for islands, rocks, pilings, and other surfaces because they are chilled (Fig. 42.7). Birds that survive for 48 hours or more after oiling are often thin, and even close to starvation, because they have stopped feeding and are using first body fat and then muscle tissue to produce heat in response to chilling.

Matting of the feathers occurs from external oiling. Oil can usually be seen or smelled on the feathers, but some light, transparent oils may be difficult to detect. One useful technique for detecting oiling is to place a few feathers from the bird in a pan of water and watch for an oil sheen to appear (Fig. 42.8). An enzyme-linked immunosorbent assay (ELISA), which detects PAH in oil, can provide quick confirmation of the presence of petroleum products on fur or feathers.

Photo by U.S. Fish and Wildlife Service Region 7

***Figure 42.6*** *Oiled birds become wet and chilled because oil damages feather waterproofing and insulating properties.*

## Gross Lesions

Necropsy findings of birds that die from oil exposure are highly variable. Birds are often emaciated, and oil may be present in their trachea, lungs (Fig. 42.9), digestive tract, and around the vent. The lining of the intestine may be reddened, or the intestine may contain blood. The salt glands, which


***Figure 42.7*** *Common murre out of water due to oiling.*

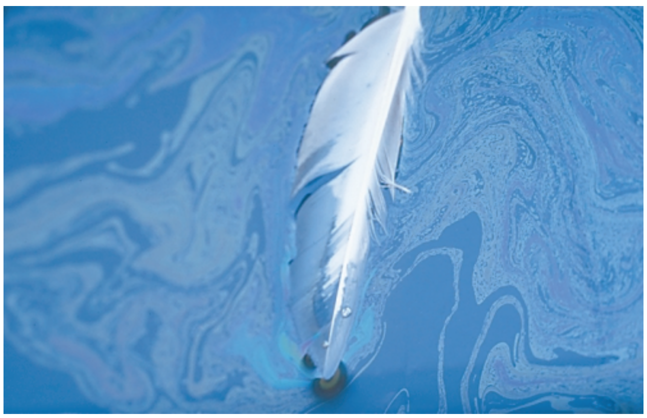



***Figure 42.8*** *If external oiling is suspected, place feathers on water and watch for oil sheen.*

are located over the eyes, may appear swollen (Fig. 42.10), and the adrenal glands may be enlarged. A variety of other changes in the normal appearance of tissues and organs may also be present, but no specific or consistent lesion is typical in animals that are exposed to oil.

## Diagnosis

Diagnosis of oiling is seldom a problem; visible oil on the bird or in the environment usually suffices (Fig. 42.11). However, proving that oil has caused mortality is more complex. For damage assessments and cause-of-death determinations, it must be determined that oiling did not occur after the death of the animals in question.

Chemical analyses of tissues or eggs are difficult to use for diagnosis because the chemical composition of petroleum products is complex. Therefore, good background information and field observations are an integral part of specimen submission to diagnostic laboratories (see Chapter 1, Recording and Submitting Specimen History Data). Submit whole carcasses whenever possible.

## Control

Treatment of oil spills within the States, territorial possessions, and territorial waters of the United States is legislatively mandated by the Oil Pollution Act of 1990. The Act mandates the inclusion of a fish and wildlife response plan within the National Contingency Plan and the creation of Area Contingency Plans. These plans provide for an integrated response to a spill with assigned agency responsibilities for protecting fish and wildlife and environmental cleanup.

**In the event of a spill, contact the National Response Center at the 24-hour, toll free number 1-(800)-424-8802.** The National Response Center will advise the responsible agencies (Coast Guard, Environmental Protection Agency,


***Figure 42.9*** *In severe cases, oil may be inhaled and may discolor the lungs, such as in this Canada goose.*

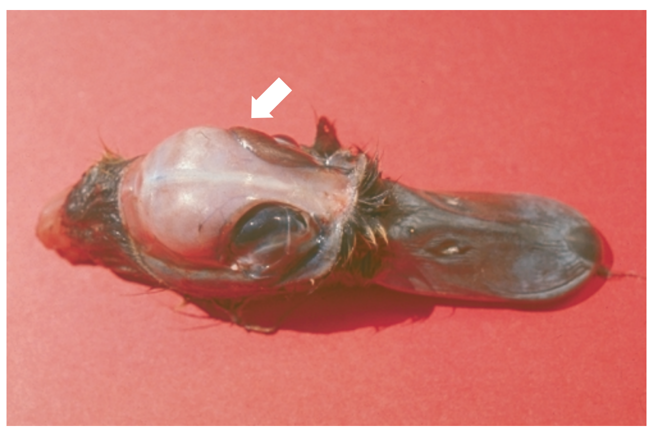
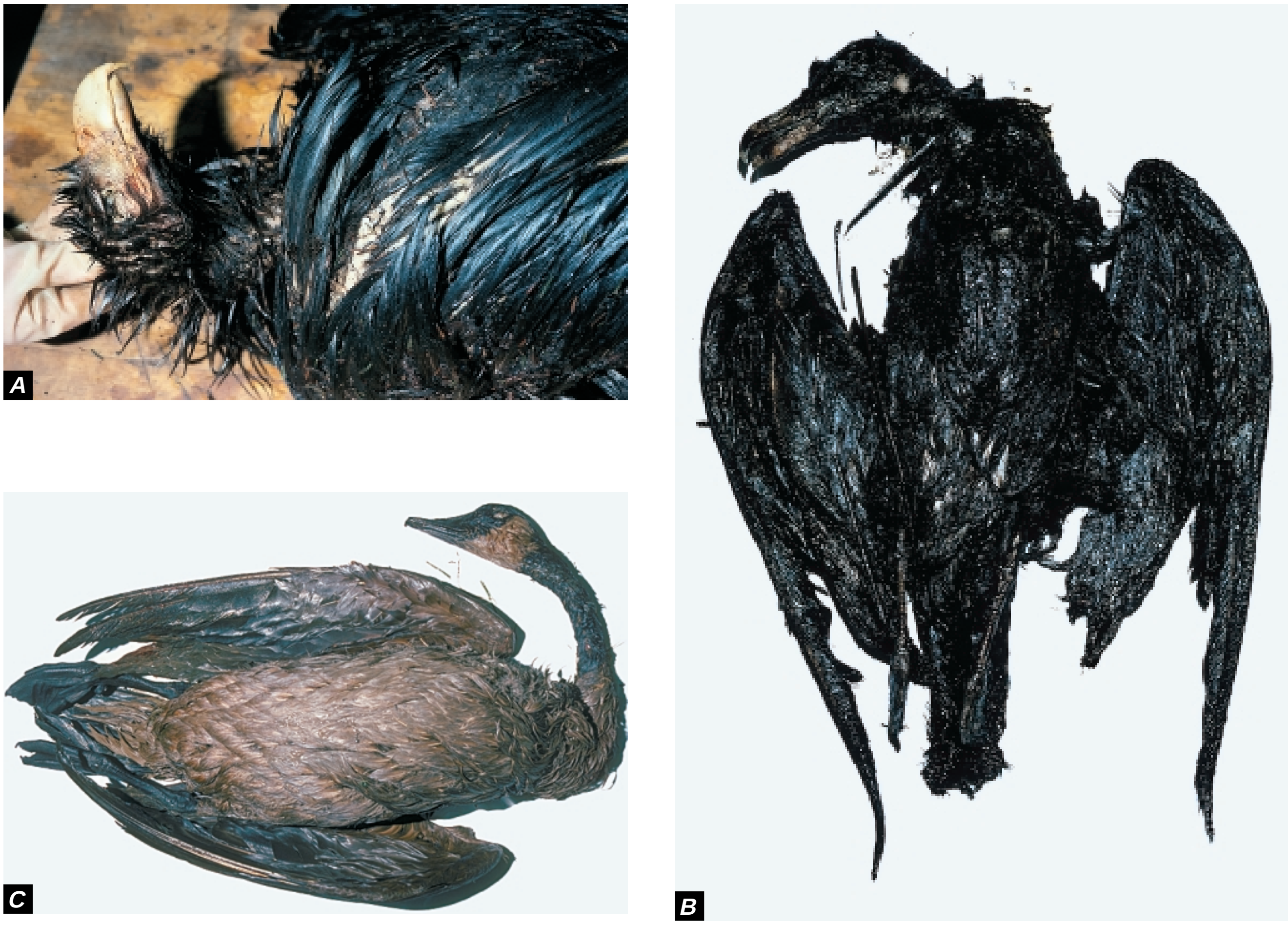



***Figure 42.10*** *Swollen salt glands.*




Photos by James Runningen

***Figure 42.11*** *Diagnosis of oiling is facilitated when oil is plainly visible on the carcass, such as on this bald eagle* ***(A)****, herring gull* ***(B)****, and Canada goose* ***(C)****.*

and natural resource trustees) who will then respond to the event. In some States, notably California, State agencies may have lead responsibility for oil spills.

Cleaning oiled birds may not be justified on a "population" basis, but it is desired by the public, required by both State and Federal laws, and warranted when rare, threatened, or endangered species are involved. Contingency plans that were developed under the Oil Pollution Act address wildlife rehabilitation. Do not attempt to rehabilitate oiled animals without knowledge of cleaning techniques. For situations that do not require a response mandated by the Act, obtain advice from State wildlife resource agencies and the private sector (Table 42.3).

Scaring devices and other forms of disturbance can be used to discourage bird use of oil-polluted areas. If a polluted area is being used or is likely to be used by endangered species, it may be helpful to initiate actions that will attract the birds to other locations while the spill is contained and cleaned. All actions taken, including wildlife rehabilitation, should be in concert with those mandated to address oil spills.

## Human Health Considerations

Direct contact with petroleum, handling oiled wildlife, and activities associated with the cleanup are all potentially hazardous to humans. Health impacts due to the toxic effects of petroleum include contact dermatitis, increased skin cancer risk, eye irritation, and problems associated with inhaling volatile components of petroleum products. These products may be contaminated with other chemicals including polychlorinated biphenyls (PCBs) and organophosphates. Wear protective clothing to prevent direct exposure of oil to skin surfaces. Preventing injuries during spill containment and cleanup requires a cool head, advice from experts, and close supervision of workers — especially volunteers. Two major concerns are drowning and hypothermia.

Workers should not enter the water, climb slippery cliffs, or put themselves in hazardous situations to rescue birds. Also, the birds themselves present a hazard. Many sea birds have sharp, "spearing" beaks and often aim for the eyes of their predators — and their caretakers. Always wear goggles when handling these birds.

*Table 42.3* ***Table 42.3*** *Sources of information for rehabilitation of oiled birds.*

Many individuals and groups have expertise in the rehabilitation of oiled birds and other wildlife. The following are major programs that conduct this type of activity.

| Program and address | Telephone |
|---|---|
| Tri-State Bird Rescue and Research, Inc.<br>110 Possum Hollow Rd., Newark, DE 19711 | 302-737-9543 |
| California Department of Fish and Game<br>Office of Oil Spill Prevention and Response<br>Oiled Wildlife Care Network<br>Wildlife Health Center<br>University of California, Davis, CA 95616 | 530-752-4167 |
| International Bird Rescue Research Center<br>699 Potter St., Berkeley, CA 94710 | 510-841-9086 |

*Tonie E. Rocke*

## Supplementary Reading


Albers, P.H., 1995, Oil, biological communities and contingency planning, *in* Fink, L., and others, eds., Wildlife and oil spills: response, research, and contingency planning: Hanover, Pa., The Sheridan Press, p. 1–10.

Albers, P.H., 1995, Petroleum and individual polycyclic aromatic hydrocarbons, *in* Hoffman, D.J., and other, eds., Handbook of Ecotoxicology: Boca Raton, Fla., Lewis Publishers, p. 330–355.

Bourne, W.R.P., 1976, Seabirds and pollution *in* Johnson, R., ed., Marine Pollution: London, Academic Press, p. 403–502.

Burger, A.E., 1993, Estimating the mortality of seabirds following oil spills: effects of spill volume. Marine Pollution Bulletin, v. 26, p. 140–143.

Flickinger, E.L., 1981, Wildlife mortality at petroleum pits in Texas: Journal of Wildlife Management, v. 45, p. 560–564.

Hoffman, D.J., 1990, Embryotoxicity and teratogenicity of environmental contaminants to bird eggs: Reviews of Environmental Contamination and Toxicology, v. 115, p. 39–89.

Chapter 43

# Lead

**Synonym**

*Plumbism*

Lead poisoning of waterfowl is neither a new disease nor a subject without controversy. The use of lead shot for waterfowl hunting within the United States has been prohibited and efforts are underway to ban the use of lead fishing sinkers and prohibit the use of lead shot for nonwaterfowl hunting. The first documented reports within the United States of lead-poisoned waterfowl were from Texas in 1874. Numerous other reports and studies added to those findings during the years and decades that followed. However, strong opposition to nontoxic shot requirements prevented full implementation of them until 1991. A full transition to nontoxic shot shells for all hunting and to nontoxic fishing sinkers and jig heads for fishing within the United States will not happen easily. The continued use of lead shot and lead fishing weights and the large amounts of these materials previously deposited in environments where birds feed assure that lead poisoning will remain a common bird disease for some time.

## Cause

Lead poisoning is an intoxication resulting from absorption of hazardous levels of lead into body tissues. Lead pellets from shot shells, when ingested, are the most common source of lead poisoning in birds. Other far less common sources include lead fishing sinkers, mine wastes, paint pigments, bullets, and other lead objects that are swallowed.

## Species Affected

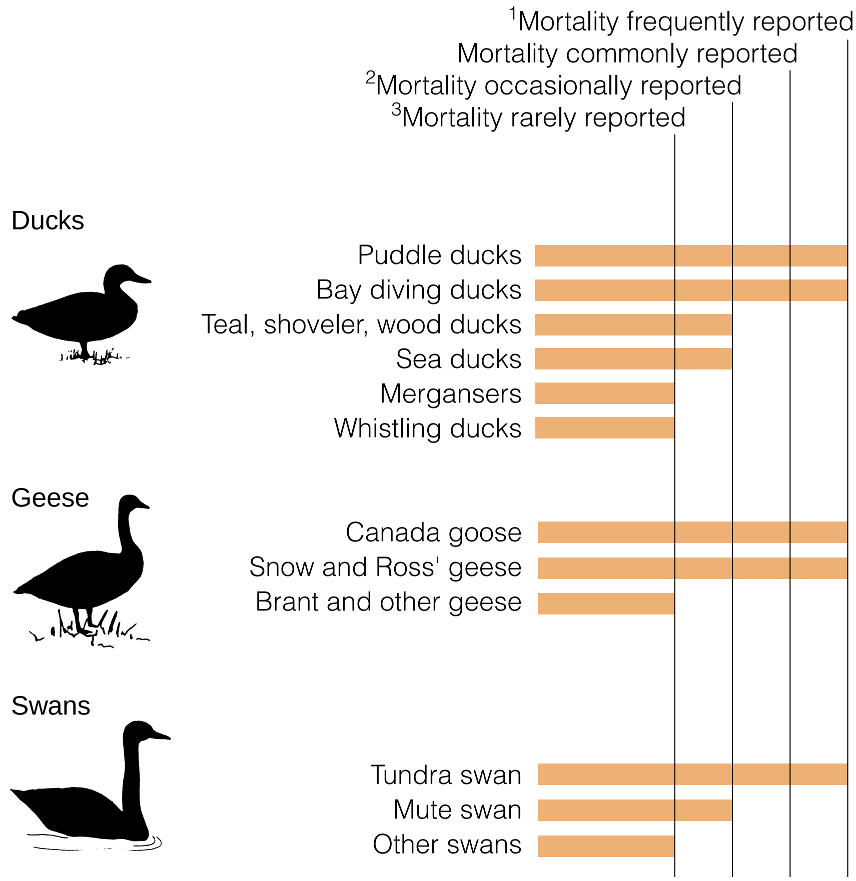
Lead poisoning has affected every major species of waterfowl in North America and has also been reported in a wide variety of other birds. The annual magnitude of lead poisoning losses for individual species cannot be precisely determined. However, reasonable estimates of lead-poisoning losses in different waterfowl species can be made on the basis of mortality reports and gizzard analyses. Within the United States, annual losses from lead poisoning prior to the 1991 ban on the use of lead shot for waterfowl hunting were estimated at between 1.6 and 2.4 million waterfowl, based on a fall flight of 100 million birds. Followup studies have not been conducted since the ban on lead shot to determine current losses from lead poisoning. This disease still affects waterfowl and other species due to decades of residual lead shot in marsh sediments, continued deposition from allowable use of lead shot during harvest of other species, noncompliance with nontoxic shot regulations, target shooting over areas where birds may feed, and from other sources of lead.

Lead poisoning is common in mallard, northern pintail, redhead, and scaup ducks; Canada and snow geese; and tundra swan. The frequency of this disease decreases with increasing specialization of food habits and higher percentages of fish in the diet. Therefore, goldeneye and merganser ducks are seldom affected (Fig. 43.1). A surprising recent finding has been lead poisoning in spectacled and common eiders on their Alaskan breeding range, where the intensity of hunting is far less than in the contiguous 48 States. These findings demonstrate that lead poisoning can afflict birds even without heavy hunting pressure. Among land birds, eagles are most frequently reported dying from lead poisoning (Fig. 43.2). Lead poisoning in eagles and other raptors generally is a result of swallowing lead shot embedded in the flesh of their prey. With the exception of waterfowl and raptors, lead poisoning from ingesting lead shot is generally a minor finding for other species (Table 43.1). However, lead poisoning has been reported in partridge, grouse, and pheasants subjected to intensive shooting in uplands of Europe. Lead poisoning in pheasants in Great Britain was reported as early as 1875.

Lead poisoning due to ingesting lead fishing weights has been reported in numerous species. The greatest number of reports are from swans as a group, common loon, brown pelican, Canada goose, and mallard duck (Fig. 43.3). Laysan albatross chicks on Midway Atoll suffer high lead exposures and mortality from ingesting lead-laden paint chips flecking off of vacant military buildings (Fig. 43.4).

## Distribution

Losses occur coast-to-coast and border-to-border within the United States. Documented lead poisoning in birds varies widely between States and does not necessarily reflect true geographic differences in the frequency of occurrence of this condition. For example, although the geographic distribution of lead poisoning in bald eagles is closely associated with their wintering areas, the number of lead poisoning cases from Wisconsin and Minnesota is disproportionately high. Because submission of bald and golden eagles for examination from different areas is highly variable, no direct comparison can be made between States regarding the number of lead-poisoned eagles (Fig. 43.5A). The reported distribution of lead poisoning in eagles and waterfowl depends




[1]Including individual die-offs of hundreds to thousands of birds
[2]Lead shot ingestion studies generally indicate low levels of exposure to lead shot
[3]Lead shot ingestion studies genreally indicate little or no lead shot ingestion

***Figure 43.1*** *Relative occurrences of lead shot poisoning in North American waterfowl.*

on the numbers of birds submitted for complete disease diagnostic evaluations. In areas where few birds are examined, the frequency of lead poisoning and other diseases will be underestimated. Even where many bird carcasses are adequately evaluated, the number of diagnoses made reflects minimum numbers of lead-poisoning cases. The general distribution of this disease in waterfowl on the basis of lead shot-ingestion surveys and documented mortality prior to nontoxic shot requirements is shown in Fig. 43.5B.

Lead poisoning has also been reported as a cause of migratory bird mortality in other countries (Fig. 43.6). Several of these countries have implemented nontoxic shot requirements and several others are beginning to address this issue.

## Seasonality

Birds can can die from lead poisoning throughout the year, although birds are most often poisoned by lead after the waterfowl hunting season has been completed in northern areas and during the later part of the season in southern areas of the United States. January and February are peak months for cases in tundra swans, Canada geese, and puddle ducks. Spring losses are more commonly reported for diving ducks. Tundra swans are also frequently lead poisoned during spring migration.

## Field Signs

Lead-poisoned waterfowl are often mistaken for hunting season cripples. Special attention should be given to waterfowl that do not take flight when the flock is disturbed and to small groups of waterfowl that remain after most other birds of that species have migrated from the area. Lead-poisoned birds become reluctant to fly when approached and those that can still fly are often noticeably weak flyers — unable to sustain flight for any distance or flying erratically

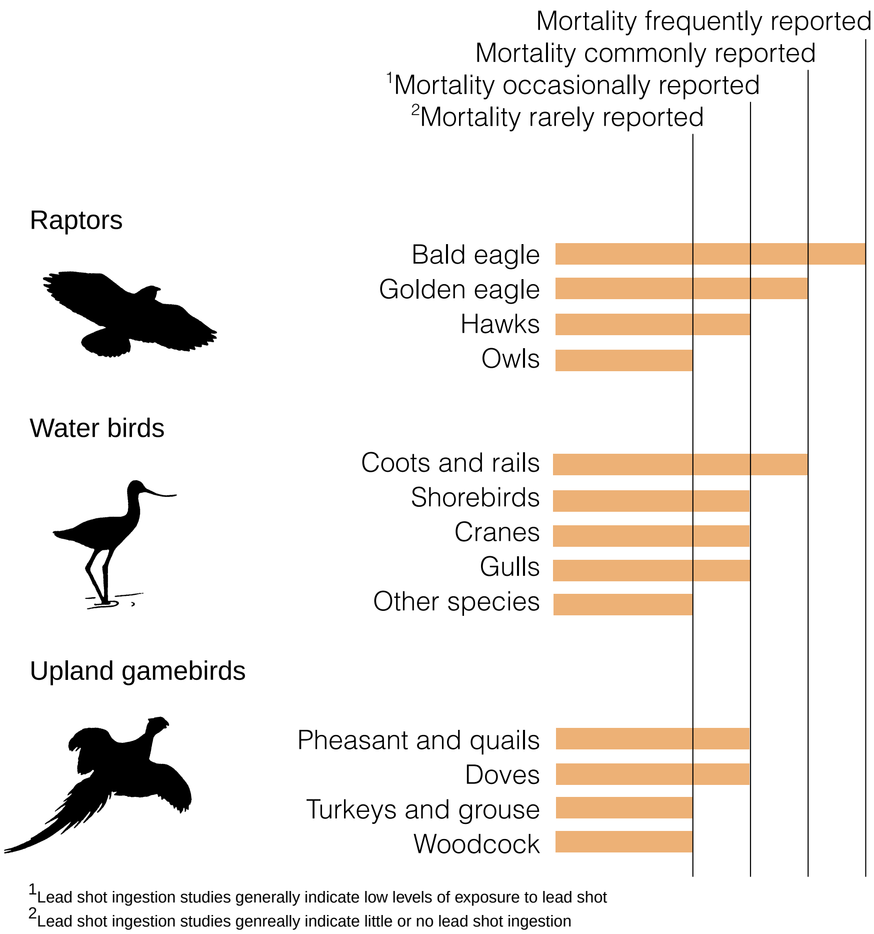



***Figure 43.2*** *Relative occurrence of lead shot poisoning in groups of birds other than waterfowl.*

and landing poorly. Birds that attempt to escape pursuit by running may exhibit an unsteady gait. In lead-poisoned Canada geese, the head and neck position may appear "crooked" or bent during flight; a marked change in the tone of call is also sometimes evident in this species. As the disease progresses and waterfowl become flightless, the wings are held in a characteristic "roof shaped" position (Fig. 43.7), which is followed by wing droop as the birds become increasingly moribund (Fig. 43.8). Fluid may discharge from the bill and often a bird may not attempt to escape in the presence of humans.

Lead-poisoned waterfowl are easily captured during advanced stages of intoxication (Fig. 43.9). Because severely affected birds generally seek isolation and protective cover, well-trained retrieving dogs can help greatly to locate and collect these birds. An abundance of bile-stained feces on an area used by waterfowl (Fig. 43.10) is suggestive of lead poisoning and warrants ground searches even if other field signs have not been observed. Green-colored feces can also result from feeding on green wheat and other plants, but the coloration is somewhat different.

## Gross Lesions

Lead-poisoned waterfowl are often emaciated because of the prolonged course of the illness and its impact on essential body processes. Therefore, many affected birds appear to be starving; they are light in weight, have a "hatchet-breast" appearance (Fig. 43.11), and the undersurface of their skin is devoid of fat (Fig. 43.12). The vent area of these birds is often stained with a bright green diarrhea (Fig. 43.13). The heads of Canada geese may appear puffy or swollen because serum-like fluids accumulate in the tissues of the face (Fig. 43.14).

Lesions observed at necropsy of lead-poisoned birds that

***Table 43.1*** *Documented North American cases of lead poisoning in free-ranging nonwaterfowl species.*

| | | | |
|---|---|---|---|
| **Nonendangered species** | | | |
| **Upland gamebirds** | | | |
| Ring-necked pheasant | Hungarian partridge | Bobwhite quail | Scaled quail |
| Wild turkey | Mourning dove | | |
| **Raptors** | | | |
| Golden eagle | Northern harrier | Rough-legged hawk | |
| Red-tailed hawk | Prairie falcon | Turkey vulture | |
| **Wetland birds** | | | |
| Common loon | Double-crested cormorant | Greater sandhill crane | Lesser sandhill crane |
| White pelican | American coot | Royal tern | Flamingo |
| Great blue heron | White ibis | Great egret | Snowy egret |
| Sora rail | American avocet | Black-necked stilt | Marbled godwit |
| Pectoral sandpiper | Western sandpiper | Long-billed dowitcher | Laughing gull |
| Herring gull | Glaucous-winged gull | California gull | Laysan albatross[1] |
| **Endangered species** | | | |
| California condor | Brown pelican | Whooping crane[2] | |
| Bald eagle | Mississippi sandhill crane | Peregrine falcon | |

[1]The cause of poisoning was ingestion of paint chips rather than lead shot, bullets, or fishing tackle.
[2]The cause of poisoning was particulate lead of unknown origin but not lead shot or fishing tackle.

have died after a prolonged illness generally consist of the following:

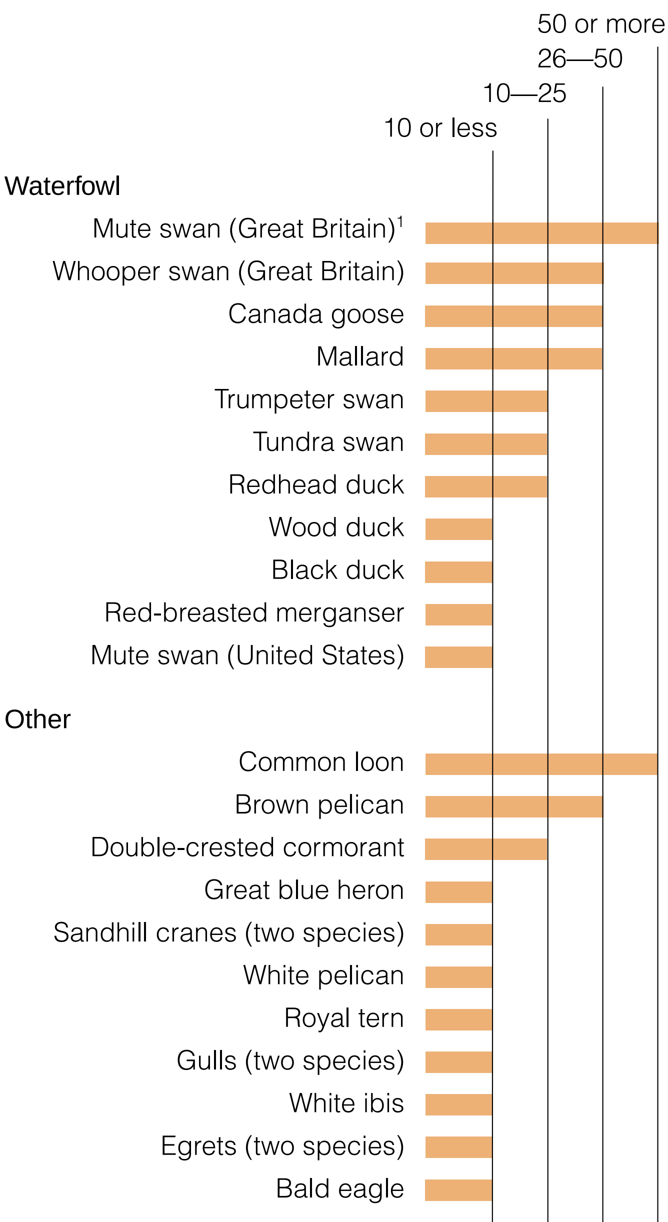
1. Severe wasting of the breast muscles (Fig. 43.11).
2. Absent or reduced amounts of visceral fat (Fig. 43.12).
3. Impactions of the esophagus or proventriculus in approximately 20–30 percent of affected waterfowl. These impactions may contain food items, or combinations of food, sand, and mud. The extent of impaction may be restricted to the gizzard and proventriculus, extend to the mouth, or lie somewhere in between (Fig. 43.l5).
4. A prominent gallbladder that is distended, filled with bile, and dark or bright green (Fig. 43.l6).
5. The normally yellow gizzard lining is discolored a dark or bright green (Fig. 43.l7). Gizzard contents are also often bile-stained.
6. Lead pellets or small particles of lead are often present among gizzard and proventricular contents. Pellets that have been present for a long time are well worn, reduced in size, and disk-like rather than spherical (Fig. 43.18). Careful washing of contents is required to find smaller lead fragments. X-ray examination is often used to detect radiopaque objects in gizzards, but recovery of the objects is necessary to separate lead from other metals. Flushing contents through a series of progressively smaller sieves is one method of pellet recovery.

Less obvious pathological changes include wasting of internal organs such as the liver, kidneys, and spleen; areas of paleness in the heart muscle; a flabby-looking heart; and paler-than-normal-looking internal organs and muscle tissue.

The above field signs and gross lesions provide a basis for a presumptive diagnosis of lead poisoning. However, none of these signs or lesions is diagnostic by itself and all can result from other causes. Also, many of the above signs and lesions are absent in birds that die acutely following an overwhelming lead exposure.

## Diagnosis

A definitive diagnosis of lead poisoning as a cause of death is based on pathological and toxicological findings supplemented by clinical signs and field observations. The presence or absence of lead shot or lead particles in the gizzard contents is useful information and should be recorded, but it is not diagnostic. The liver or kidneys are the tissues of choice for toxicology analysis, with liver tissue being more commonly used. If you suspect lead poisoning and cannot submit whole birds to the diagnostic laboratory, remove the liver or kidney tissue, wrap the specimens separately in aluminum foil, and freeze them until they are submitted for analysis. Collect the entire liver or one entire kidney. However,




[1] Great Britain banned the use of lead sinkers in 1987.

***Figure 43.3*** *Number of reported lead poisoning occurrences following ingestion of lead sinkers and jigs through 1994.*

Photo by Louis Sileo

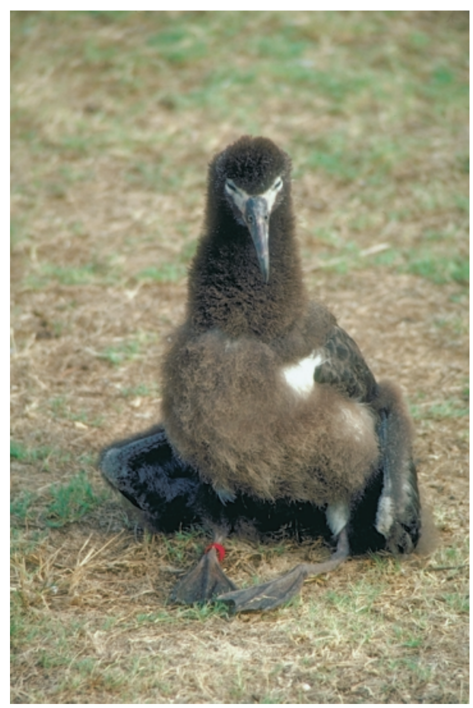


***Figure 43.4*** *The droopy wings and unthrifty appearance of this Laysan albatross chick are the result of lead poisoning caused by ingestion of lead-laden chips that flecked off abandoned buildings. The paint had high concentrations of lead.*

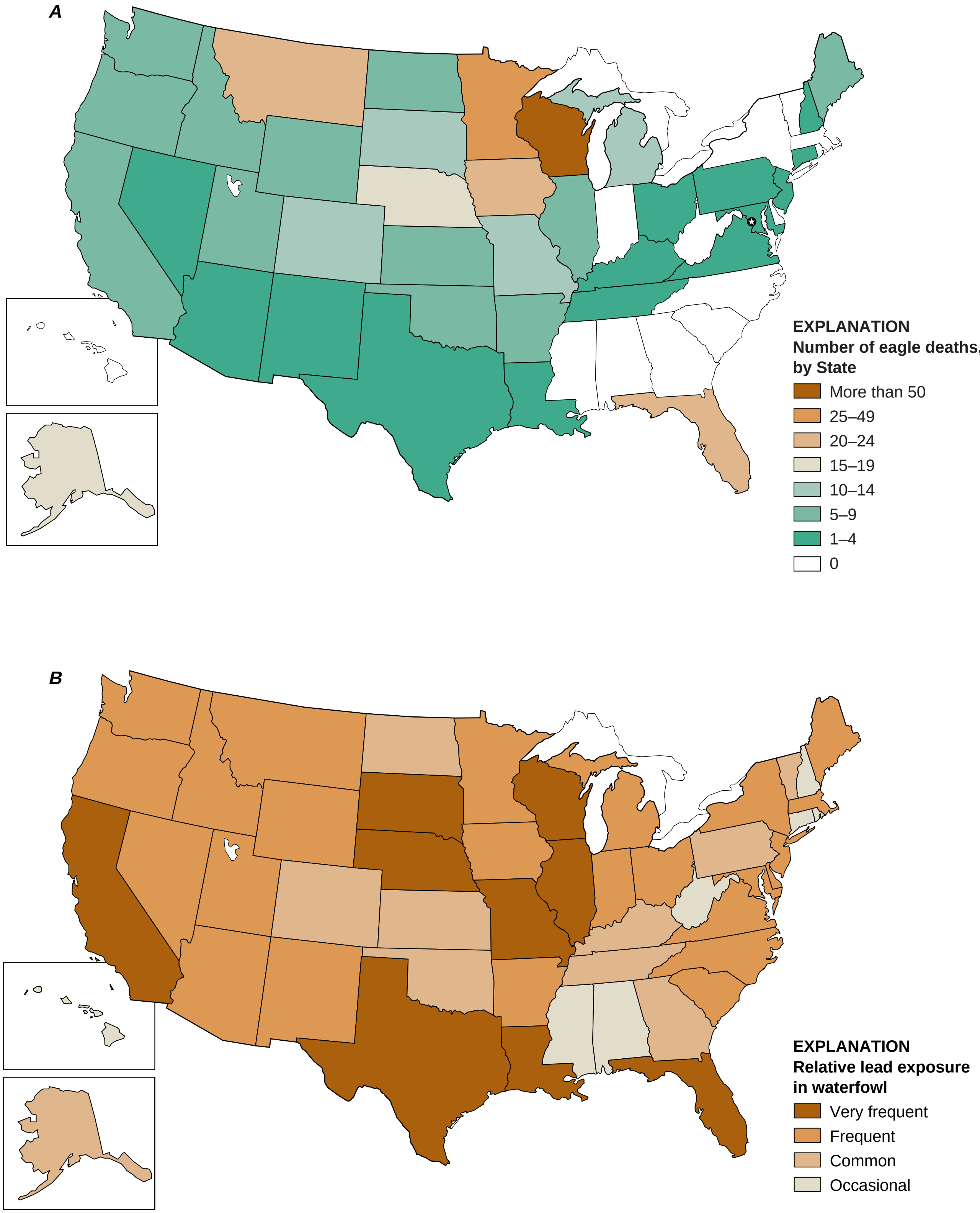



*Figure 43.5* ***(A)*** *Diagnosed cases of lead poisoning in bald eagles though mid-April, 1996.* ***(B)*** *Relative occurrence of lead exposure in waterfowl prior to the 1991 ban on use of lead shot for waterfowl hunting. Evaluation is based on gizzard analysis and reported mortality.*

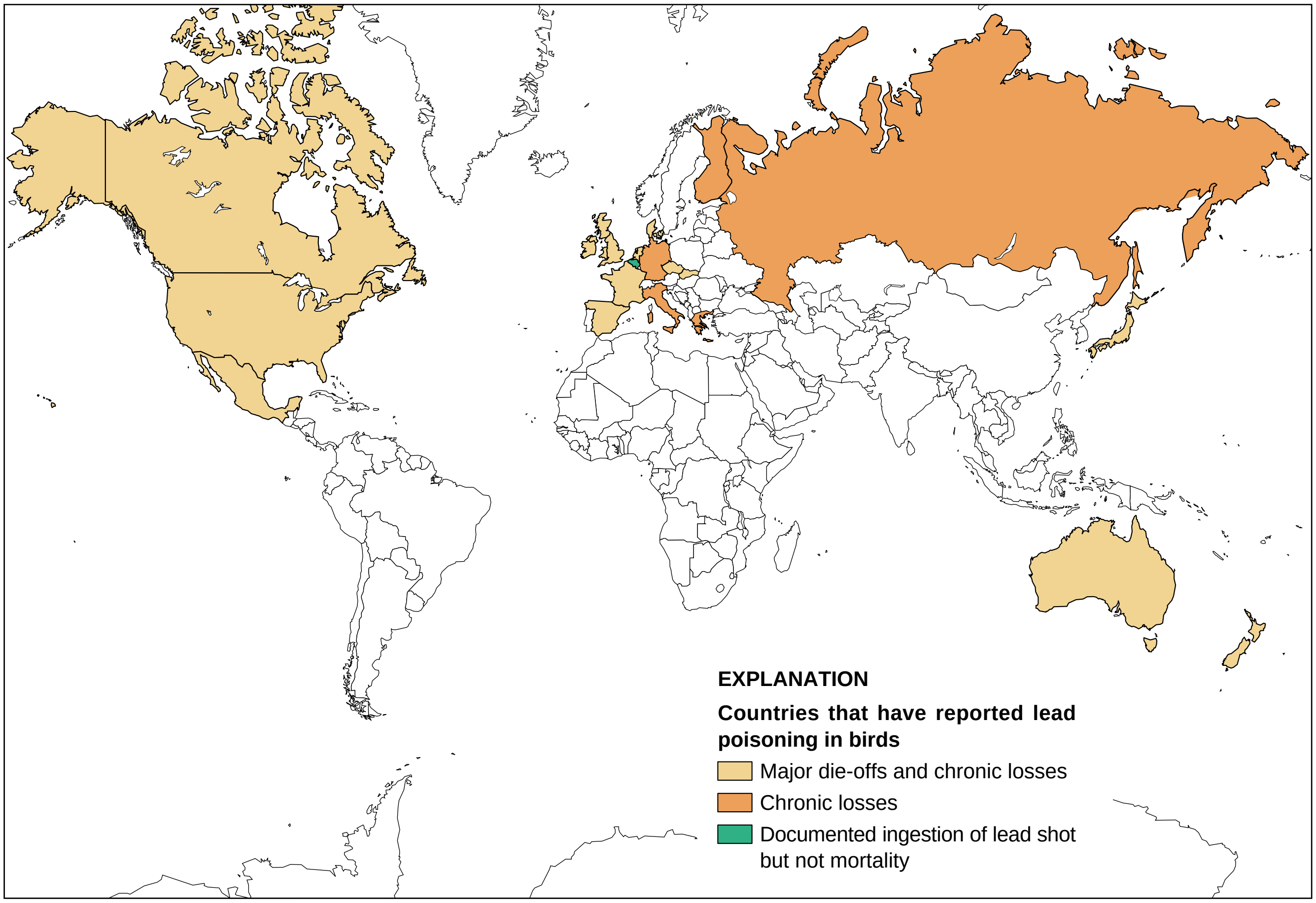



***Figure 43.6*** *Countries that have reported lead poisoning in birds.*

because toxicology is but one aspect of reaching a lead-poisoning diagnosis, make every effort to submit whole birds for analysis. Liver lead values of 6–8 parts per million or higher on a wet-weight basis or 20–30 parts per million on a dry-weight basis are suggestive of lead poisoning when other signs of lead poisoning are present.

Lead levels in populations of live birds can be evaluated by using whole blood. Collect a minimum of 2–5 milliliters of blood in lead-free tubes containing an anticoagulant such as sodium citrate or EDTA. Evidence of lead exposure can also be obtained through indirect measurements involving blood enzymes. Measurement of protoporphyrin IX in red blood cells is the most popular assay because only a few drops of blood are needed and testing is inexpensive once appropriate instrumentation is obtained. Elevated blood protoporphyrin levels are correlated with lead exposure and serve as a sensitive screening assay, but they do not provide direct measurement of the amount of lead in blood. This technique has its greatest value in identifying populations from which more direct measurements should be taken and for screening blood samples to determine which should be tested for blood lead concentrations. Confirm correct procedures for collecting blood samples for lead analysis with the diagnostic laboratory before collecting the samples. Keep blood samples chilled until submitting them for analysis, regardless of the assay that will be used. Write the date and time of collection on the tube along with the specimen number and other information identifying the sample and its origin.

The diagnosis of lead poisoning as a disease or poisoning syndrome, but not as a cause of death, can be made from tissue residues alone when there are sufficient residue data for the species in question or closely related species. The amount of tissue residue variability that exists between species can be considerable and it is also influenced by the route of lead exposure such as ingestion vs. inhalation (Fig. 43.19). For example, rock doves (pigeon) are highly resistant to high concentrations of lead when they are compared with other birds, but most lead exposure in rock doves is from automobile emissions in cities. Rock doves that have ingested lead shot have greatly increased tissue lead levels, can exhibit behavioral changes consistent with lead toxicity in other species, and can die from the toxic effects of lead.

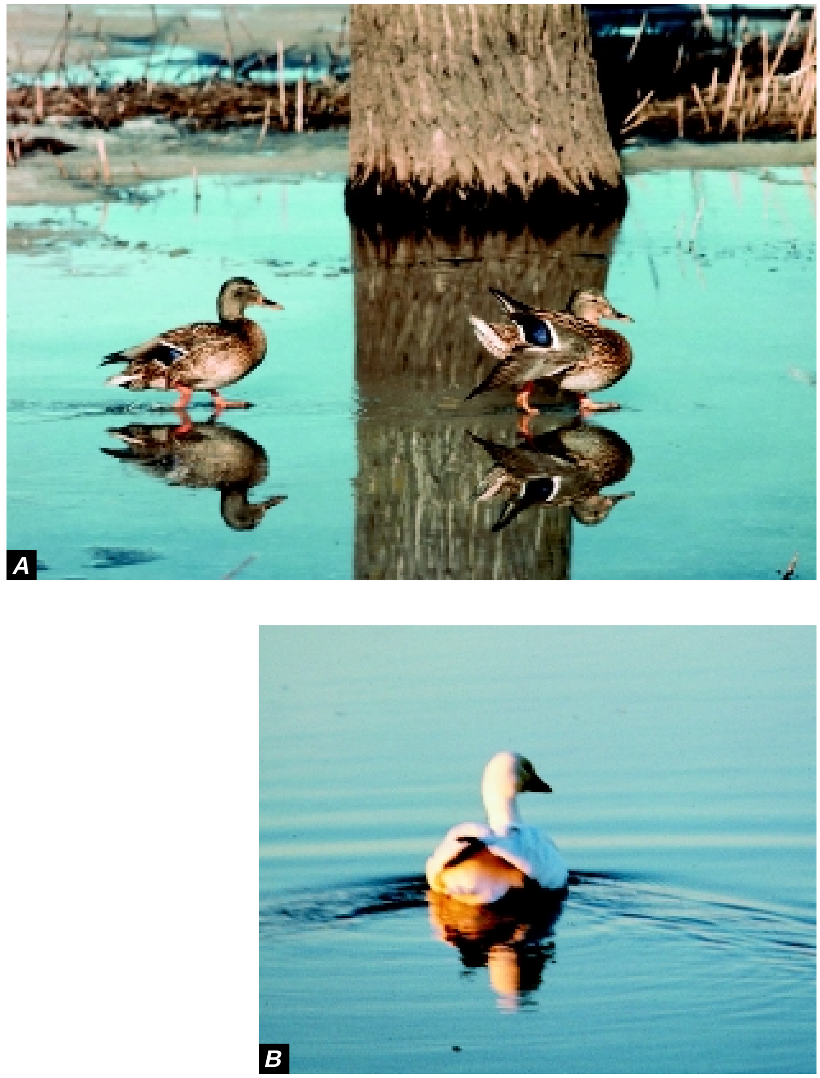



Photo by James Runningen

Photo by Milton Friend

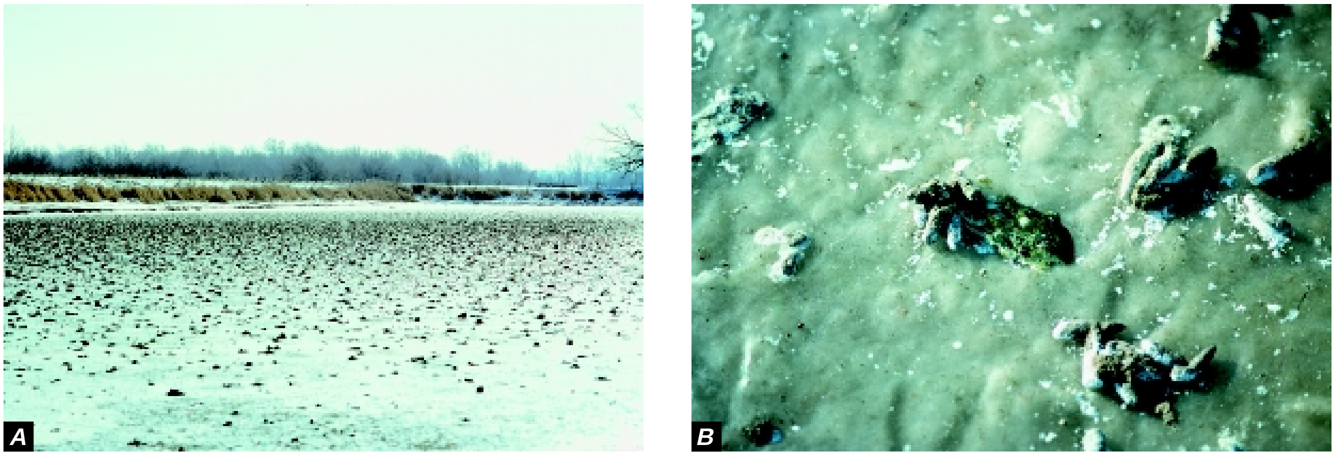
*__Figure 43.7__ Characteristic "roof-shaped" position of the wings in __(A)__ a lead-poisoned mallard (leading bird) and __(B)__ a snow goose.*

Photo by Milton Friend

*__Figure 43.8__ Wing droop in a tundra swan in advanced stages of lead intoxication.*

Photo by Milton Friend

*__Figure 43.9__ Inability of these lead-poisoned Canada geese to escape capture by humans illustrates their great vulnerability to predation.*



Photos by Milton Friend

**Figure 43.10** *Waterfowl feces provide presumptive evidence of lead poisoning. Examination of **(A)** feces where waterfowl are concentrating and **(B)** observations of an abundance of bright green-colored feces should be reason to search for sick birds and carcasses.*

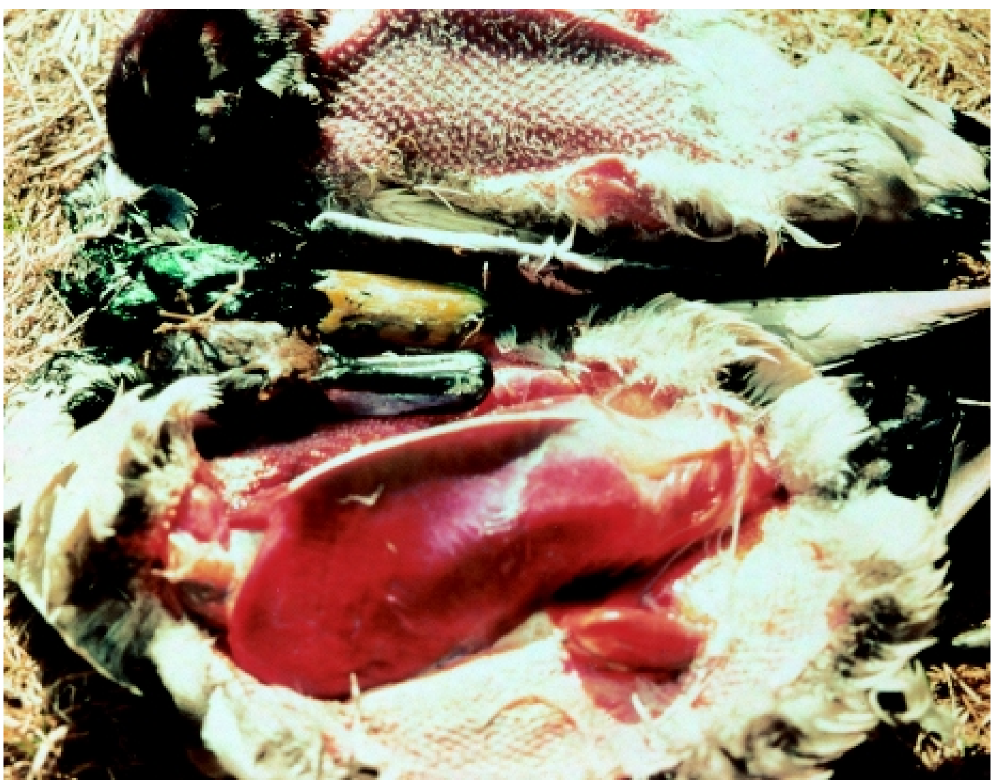


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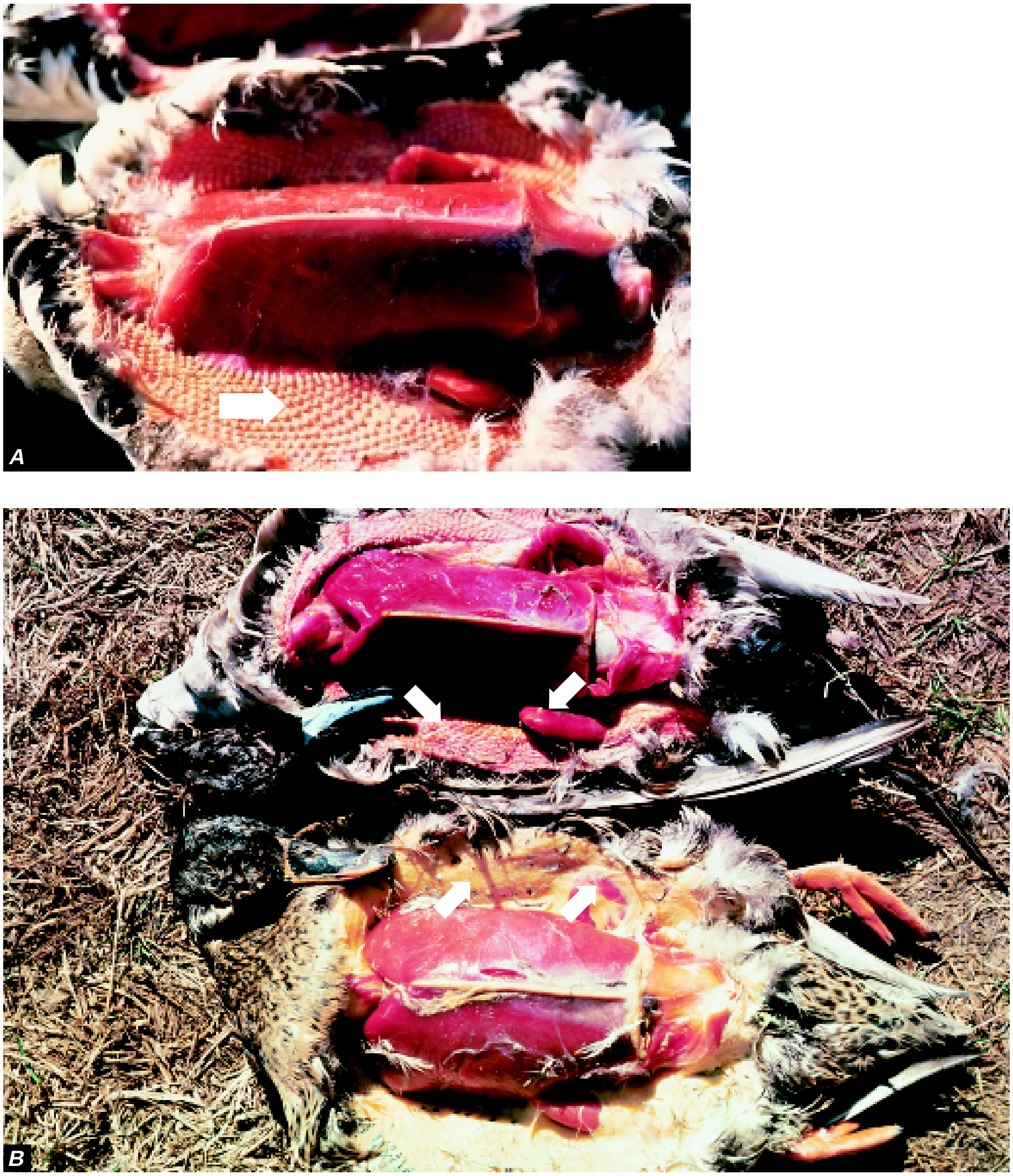
**Figure 43.11** *"Hatchet-breast" appearance of a lead-poisoned mallard (top bird) and northern pintail. The skin has been removed from the breast of the pintail to further illustrate the severe loss of muscle tissue.*




*Photos by Milton Friend*

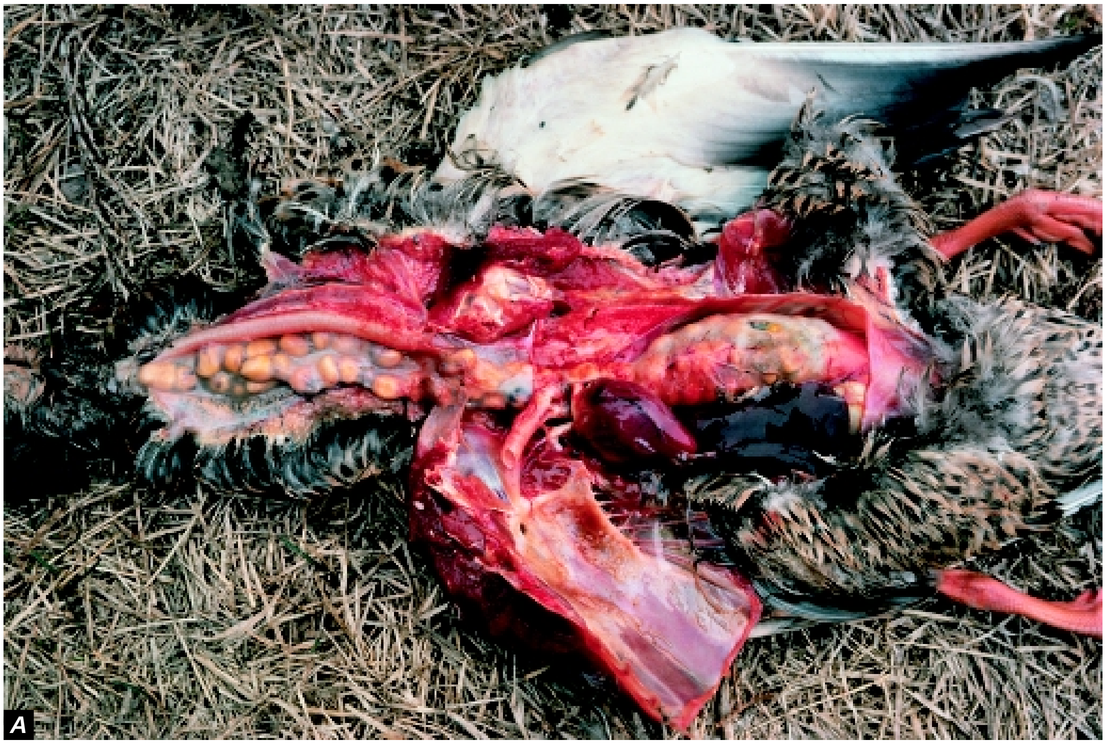
***Figure 43.12*** *Loss of subcutaneous fat is often extreme in lead-poisoned birds.* ***(A)*** *The undersurface of the skin of this pintail is totally devoid of fat, in contrast with* ***(B)*** *the abundance of yellow fat present in the mallard (bottom bird) that had died of avian cholera. Note also the absence of fat in the visceral area and along the knees of the northern pintail (top bird) in comparison with the mallard.*

Photo by Milton Friend

***Figure 43.13*** *Bright green staining of the vent area is often indicative of lead poisoning.*

Photo by Milton Friend

***Figure 43.14*** *The heads of lead-poisoned Canada geese often appear puffy or swollen.*



Photos by Milton Friend

***Figure 43.15*** *Examples of impactions in lead-poisoned birds.* ***(A)*** *Impaction of corn in digestive tract of a hen mallard, extending from the gizzard to the mouth;* ***(B)*** *snow goose with an impaction of grasses.* ***(C)*** *Tundra swan with impaction of grasses and some seeds, extending from the mouth to the gizzard; and* ***(D)*** *a more limited impaction in a drake mallard.*


C


D


Photo by Milton Friend

***Figure 43.16*** *The gallbladder (top arrow) of lead-poisoned birds is often distended and filled with bright green bile. Note also the lead shot present in the gizzard (bottom arrow) of this bird.*


Photo from U.S. Fish and Wildlife Service files

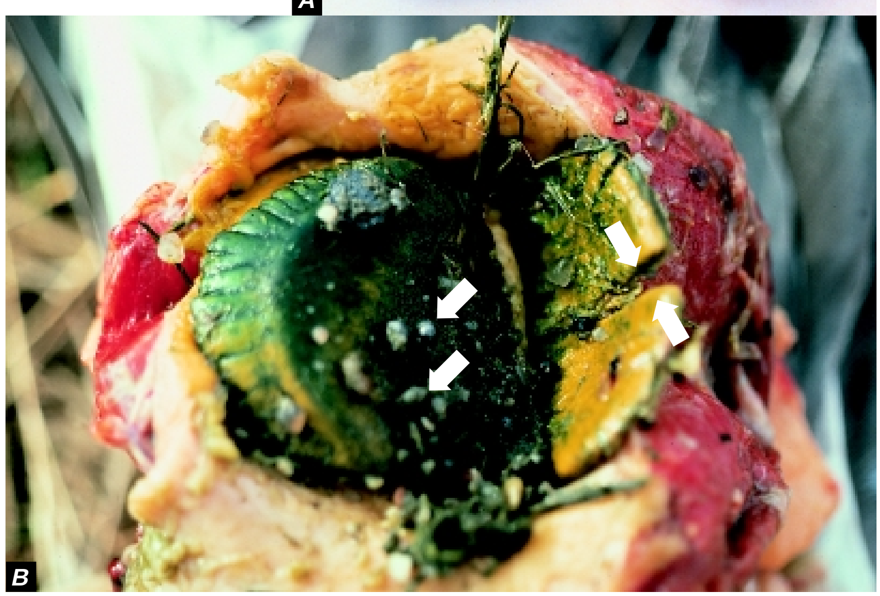


Photo by Milton Friend

***Figure 43.17*** ***(A)*** *Comparison between the appearance of the gizzard lining of a lead-poisoned mallard (left) and a normal mallard (right).* ***(B)*** *Pathological changes in the gizzard of a lead-poisoned bird. Note green-stained coloration and hard appearance of tissue. The gizzard lining has split (arrow) because the tissue has become so brittle. Note also the presence of lead shot among the grit in the center of the pad.*

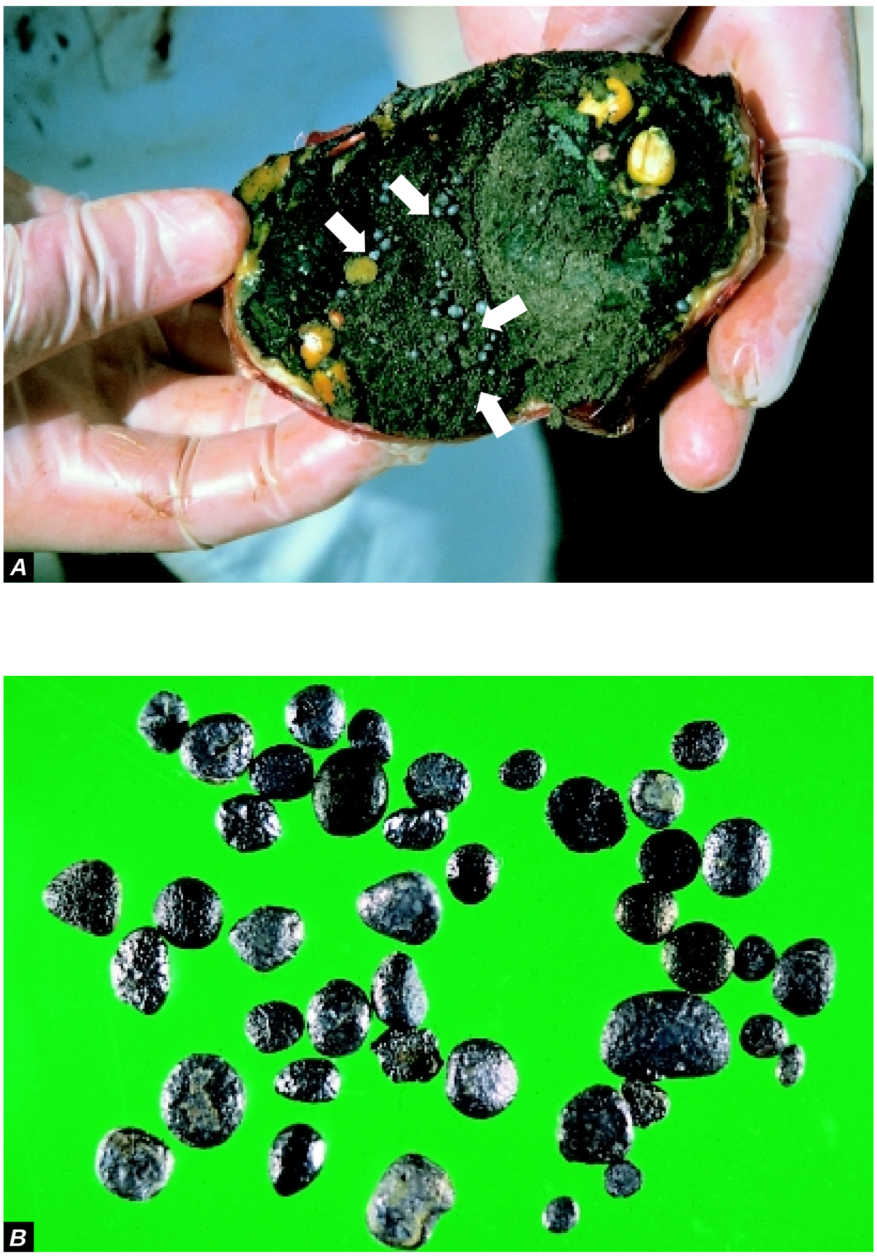



Photo by Milton Friend

Photo by James Runningen

***Figure 43.18*** ***(A)*** *Lead shot in Canada goose gizzard. Note the presence of corn. Corn and other cereal grains intensify the toxicity of lead.* ***(B)*** *Lead shot, originally spherical, that have been worn down in the waterfowl gizzard. Note the flattened, disk-like shape of many of these pellets.*

## Control

Two actions can often be taken to reduce the magnitude of mortality from lead poisoning when die-offs occur: denying birds use of problem areas, and rigorous pickup and proper disposal of dead and moribund birds.

Denying birds use of problem areas requires knowing where the birds are picking up the lead. This is complicated by the fact that signs of intoxication may not appear until 1 week after lead ingestion, and birds may not start dying until 2–3 weeks after lead ingestion. Habitat modification of contaminated areas is also useful in some instances, but differences in feeding habits must be considered. For example, placing additional water on an area may protect puddle ducks from reaching lead shot on the bottom of wetlands, but this may create an attractive feeding area for diving ducks. Similarly, draining an area may prevent waterfowl from using an area and ingesting shot, but it may create an attractive feeding area for shorebirds or pheasants. Therefore, control plans must consider the broad spectrum of wildlife likely to use the area at the time action will be taken. Rigorous pickup and proper disposal of lead-contaminated waterfowl carcasses is required to prevent raptors and other scavenger species from ingesting them. The high percentage of waterfowl with embedded body shot provides a continual opportunity for lead exposure in raptors that far exceeds the opportunity for ingestion of shot present in waterfowl gizzards.

Other management practices that have been used to reduce losses from lead poisoning on site-specific areas include tillage programs to turn lead shot below the surface of soil so that shot is not readily available to birds, planting food crops other than corn and other grains that aggravate the effects of lead ingestion, and requiring the use of nontoxic shot in hunting areas. The potential contributions of the first two practices toward reducing lead-poisoning losses among birds are, at best, limited and temporary. Supplemental grit has also been placed in wetlands in the belief that

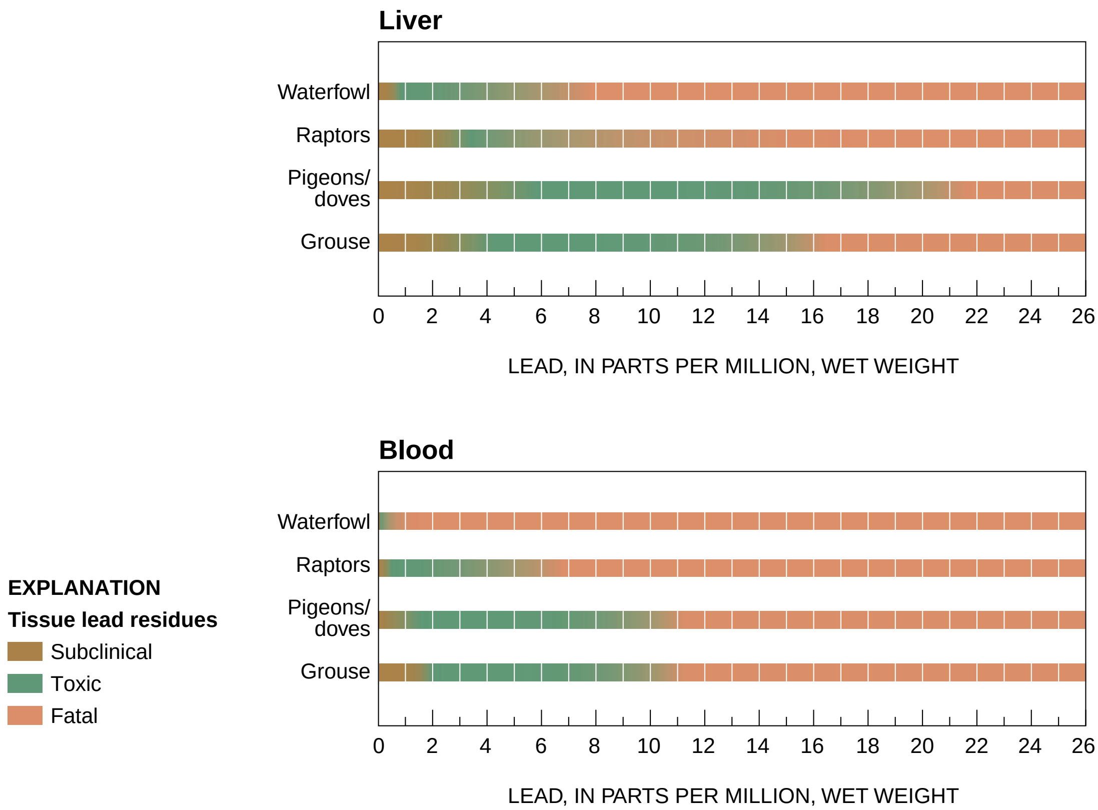



***Figure 43.19** Lead residues in the liver and blood associated with subclinical, toxic, and mortality findings in several groups of birds. Variability within groups occurs because of differences in species and a variety of factors affecting toxicity within species.*

birds intentionally ingest shot because grit is not available; as with tillage and food crops, any benefits are limited and temporary. The use of nontoxic shot is the only long-term solution for significantly reducing migratory bird losses from lead poisoning.

The strong correlation between exposure of waterfowl to lead and the use of lead shot for hunting waterfowl was vividly demonstrated by National Wildlife Health Center sponsored studies that compared tissue lead levels and gizzard analyses in a subpopulation of Canada geese as they migrated from their breeding grounds to their wintering grounds. Nontoxic shot requirements were in place at some sampling sites but not at others. Lead exposure was significantly less where nontoxic shot requirements existed.

Since lead shot has been banned for hunting waterfowl in the United States, attention has turned to regulating the use of lead fishing sinkers and lead jig heads. The Environmental Protection Agency has been petitioned to address the problem of bird mortality from these sources (Fig. 43.20). Prohibitions against using lead fishing weights below certain sizes have already been initiated on some Federal lands and other areas. The number of cases of lead poisoning in swans in the Thames Valley of England was reduced by 70 percent in 2 years following enactment of the 1987 ban on use of split lead shot and other fishing sinkers up to 1 ounce in size. Sizes larger than those that can be ingested by birds have not yet become a focus for concern.

The use of lead shot for target shooting and hunting on uplands is also receiving increased attention. In general, ingestion rates for lead shot in upland species are far less than those for waterfowl, even for doves (Table 43.2). The harvest of doves is somewhat analogous to waterfowl hunting in that large numbers of shells are often fired over the same location year after year (Fig. 43.21.). However, the duration of intense shooting on specific sites tends to be much less for doves than for waterfowl and the hunting area is generally tilled annually for agricultural purposes.

Photo by James Runningen

*Figure 43.20* *Fishing weights found in the stomachs and gizzards of birds that died from lead poisoning.*

Veterinary treatment of lead-poisoned birds is generally not a reasonable approach. However, endangered species or other birds of high individual value that are lead poisoned may warrant treatment. In those instances, treatment should be done only by qualified persons familiar with and skilled in the proper use of lead-chelating chemicals. Under the best of circumstances, the results of treatment are unpredictable and the success rate low.

## Human Health Considerations

People do inadvertently consume lead-poisoned birds. Although this is not desirable, no appreciable risks to human health exist. Most lead present in the body of a lead-poisoned bird is in organs such as the liver and kidneys rather than in the flesh. The dose relation (milligrams of lead per kilogram of body weight) and lead excretion processes are such that a great number of lead-poisoned birds would need to be consumed in a relatively short time before toxic levels of lead could build up in the human body. Persons who eat

***Table 43.2*** *Percentage of upland gamebirds reported with ingested lead shot, by State.*

| Species | State | Percentage with ingested lead shot |
|---|---|---|
| Mourning doves | Alabama | 1.0 |
| | Eastern seaboard (Maryland to South Carolina) | 2.4 |
| | Indiana | 2.3 |
| | Maryland | 1.0–6.5 |
| Scaled quail | New Mexico | 0.4 |
| Bobwhite quail | New Mexico | 1.8 |

Photo by Milton Friend

***Figure 43.21*** *High bag limits and the large number of shells generally expended to reach a bag limit on swift-flying mourning doves results in large amounts of lead shot being deposited in uplands. Because most of the doves are harvested over agricultural fields, tillage helps to reduce the potential for that shot being ingested.*

the liver, kidneys, and other soft tissues from lead-poisoned birds would consume more lead than those who eat only the muscle tissue of these birds. Persons who consume waterfowl bones would be additionally exposed to lead, because lead is stored long-term in bone.

There are a few documented cases of humans developing lead poisoning after having accidentally ingested lead shot embedded in the meat they ate. This type of lead poisoning is rare, perhaps due to caution exercised when eating hunter-killed wildlife so as to avoid potential damage to teeth from biting into shot. Lead shot that is ingested can also become lodged in the appendix, resulting in appendicitis. This does not happen often, and it happens most in people who hunt waterfowl for subsistence. It is also possible that humans may ingest tiny fragments of lead that may be present in tissues of wildlife killed with lead shot.

*Milton Friend*

## Supplementary Reading

Franson, J.C., 1996, Interpretation of tissue lead residues in birds other than waterfowl, *in* Beyer, W. N., and others, eds., Environmental contaminants in wildlife, interpreting tissue concentrations: Boca Raton, Fla., Lewis Publishers, p. 265–279.

Franson, J.C., Petersen, M.R., Meteyer, C.U., and Smith, M.R., 1995, Lead poisoning of spectacled eiders (*Somateria fischeri*) and of a common eider (*Somateria mollissima*) in Alaska: Journal of Wildlife Diseases, v. 31, no. 2, p. 268–271.

Sanderson, G. C., and Bellrose, F. C., 1986, A review of the problem of lead poisoning in waterfowl: Illinois Natural History Survey, 172, Special Publication 4, 34 p.

Scheuhammer, A. M., and Norris, S. L., 1996, The ecotoxicology of lead shot and lead fishing weights: Ecotoxicology, v. 5, p. 279–295.

Scheuhammer, A. M., Perrault, J.A., Routhier, E., Braune, B.M., and Campbell, G.D., 1998. Elevated lead concentrations in edible portions of game birds harvested with lead shot. Environmental Pollution, v. 102, p. 251–257.

# Chapter 44

# Selenium

**Synonyms**

*Selenosis*

## Cause

Selenium is a naturally occurring element that is present in some soils. Unlike mercury and lead, which also are natural environmental components, selenium is an essential nutrient in living systems. The amount of dietary selenium required by animals depends upon many factors, including the availability of certain other metals such as zinc and copper, as well as vitamin E and other nutrients. Muscle damage results if dietary selenium is deficient, but dietary excess can be toxic.

## Species Affected

Selenium poisoning or toxicosis has been documented in many avian species as well as in mammals and humans. The vulnerability of animals to selenium poisoning is primarily associated with the use of heavily contaminated habitats. Plants and invertebrates in contaminated aquatic systems may accumulate selenium in concentrations that are toxic to birds that consume them. In an experimental study with mallard ducklings, it was demonstrated that exposure to selenium in contaminated food items enhanced the birds' susceptibility to infectious diseases.

## Distribution

The potential for selenium poisoning exists wherever bird habitat is created over sites with high soil concentrations of selenium and where point-source releases of selenium, for example from smelter emissions and sewage sludge, contaminate the environment.

Kesterson Reservoir in California is a classic example of bioaccumulation of selenium in wetlands created in an area with selenium-rich soils. The reservoir became a sump for wastewater return flows from irrigated soils that were rich in selenium. The continual addition of selenium-laden return wastewater leads to toxic concentrations of selenium in food items of birds. The result is reproductive failure caused by embryonic deformities and death, as well as mortality of adult birds.

## Seasonality

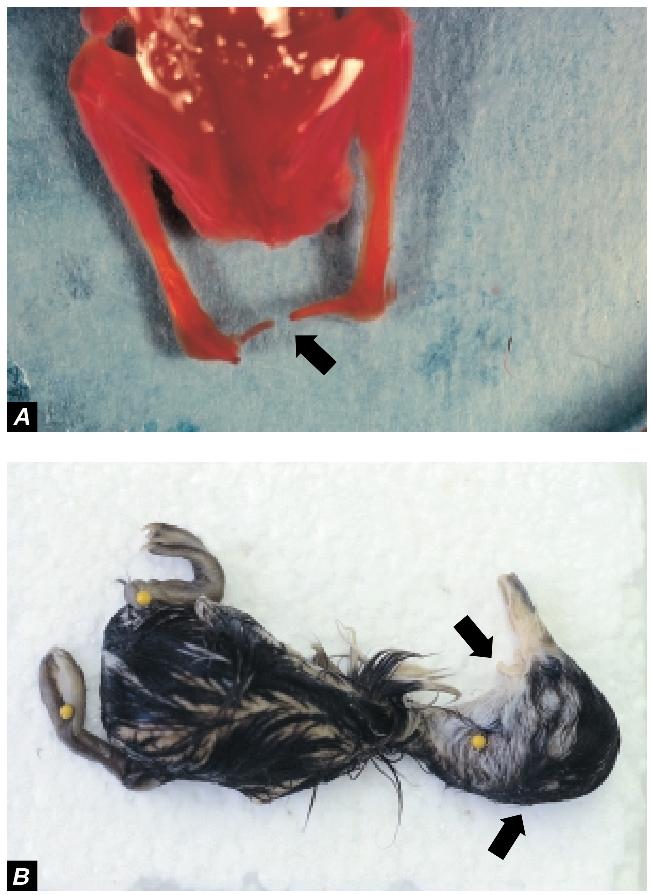
The seasonality of selenium poisoning depends on when birds use habitats that have high selenium concentrations.

## Field Signs

There are no unique clinical signs of selenium poisoning. The primary field indications that selenium poisoning may be occurring in an area are poor avian reproductive performance, embryonic deaths and deformities, and occasional mortality of adults.

## Gross Lesions

Deformities caused by selenium poisoning may include missing or abnormal body parts, especially wings, legs, eyes, and beaks, as well fluid accumulation in the skull (Fig. 44.1). Affected adults often are emaciated, but other gross lesions generally are absent.




*Photos by David Hoffman, U.S. Geological Survey, Patuxent Wildlife Research Center*

***Figure 44.1*** *Embryonic deformities may be seen in the offspring of birds exposed to high levels of selenium.* ***(A)*** *A cleared and stained preparation of a coot embryo with fusion of the digits of both feet (arrow).* ***(B)*** *This mallard embryo has fluid accumulation over the back of the skull, and the mandible is only a remnant of normal (arrows).*

## Diagnosis

Diagnosis of selenium poisoning is complicated by its biological interactions with other elements, particularly mercury. These two elements often lessen or prevent the toxicity of each other when both are present. The diagnosis of selenium poisoning depends upon a history of potential exposure; gross developmental defects; microscopic lesions, primarily evidence of chronic liver damage, that are consistent with selenium toxicosis; and selenium levels in tissues and environmental samples such as food items, water, and sediment. In birds found dead at Kesterson Reservoir, mean selenium concentrations in livers and kidneys were about 95 parts per million dry weight, which is about 10 times higher than levels found in birds from a control area.

## Control

The construction of artificial wetlands that are likely to attract bird use in areas of selenium-rich soils should be carefully evaluated for the potential for bioaccumulation of selenium in food items. It is preferable not to create wetlands where toxic concentrations of selenium can be expected. For existing wetlands, control measures should be directed at providing sources of clean water and at preventing environmental contamination by selenium through carefully disposing of selenium-containing wastes, including irrigation drainwater and sewage. The use of scare devices and other methods to prevent birds from using heavily contaminated areas should be considered.

## Human Health Considerations

The ingestion of high levels of selenium can result in poisoning in humans. One should wear gloves when handling carcasses, but birds suspected of having died of selenium poisoning present no special hazard, because residues are biologically bound within tissues.

*J. Christian Franson*

## Supplementary Reading


Eisler, R., 1985, Selenium hazards to fish, wildlife, and invertebrates: a synoptic review: Fish and Wildlife Service Biological Report 85(1.5), 57 p.

Heinz, G.H., 1996, Selenium in birds, *in* Beyer, W.N., and others, eds., Environmental contaminants in wildlife: interpreting tissue concentrations: Boca Raton, Fla., Lewis Publishers, p. 447–458.

Ohlendorf, H.M., and Hothem, R.L., 1995, Agricultural drainwater effects on wildlife in central California, *in* Hoffman, D.J., and others, eds., Handbook of ecotoxicology: Boca Raton, Fla., Lewis Publishers, p. 577–595.

Ohlendorf, H.M., 1996, Selenium, *in* Fairbrother, A., and others, eds., Noninfectious diseases of wildlife (2nd ed.): Ames, Iowa, Iowa State University Press, p. 128–140.

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.

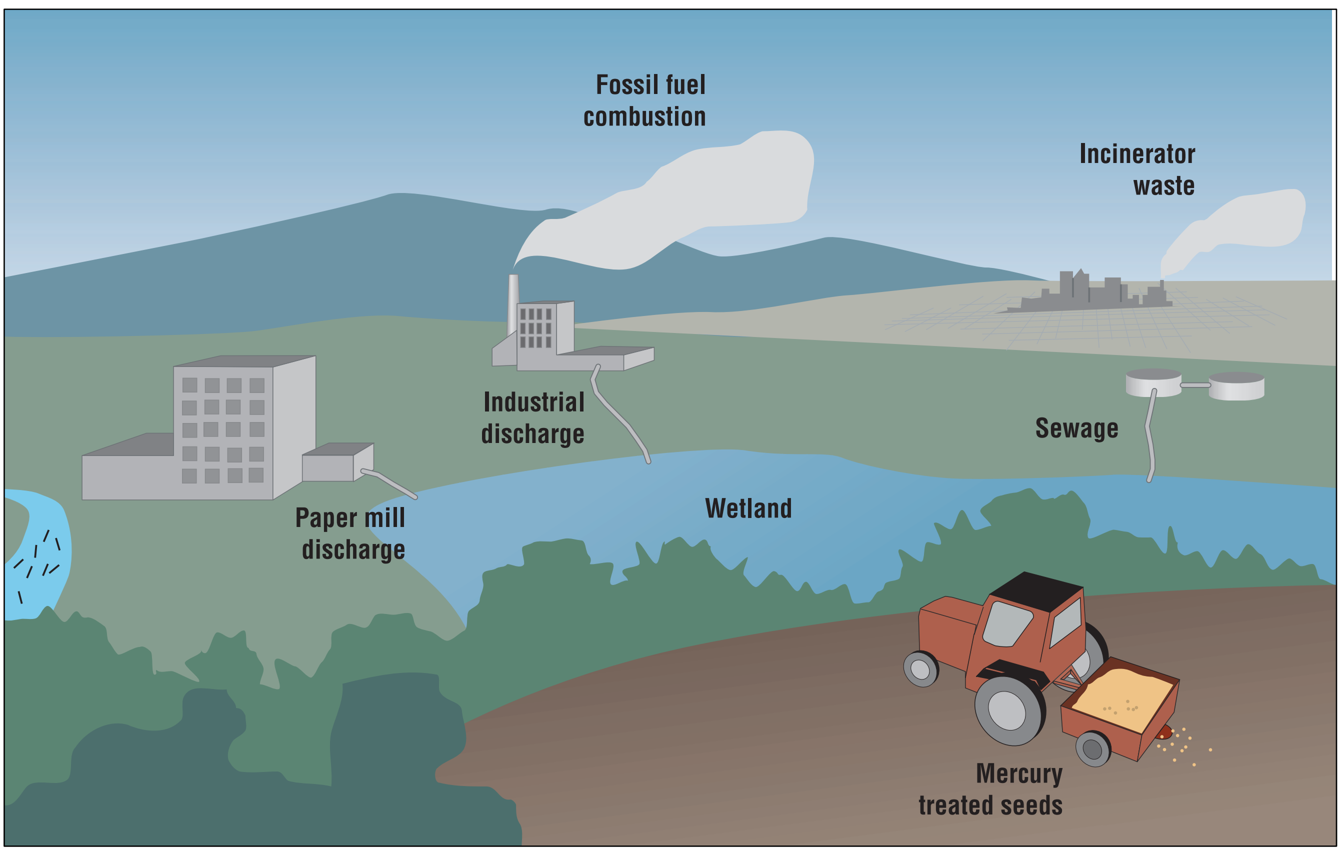
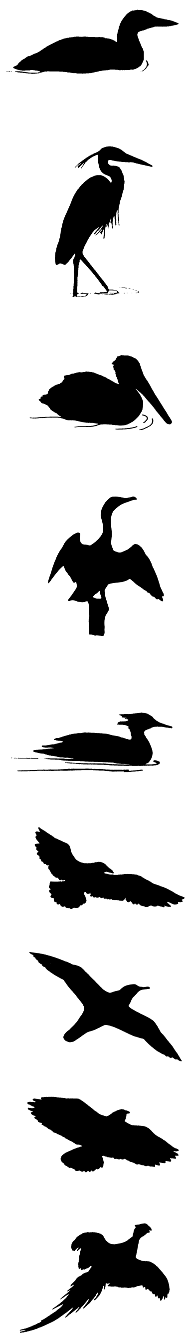



***Figure 45.1*** *Sources of mercury contamination.*



**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

***Figure 45.2*** *Avian species most likely to be exposed to mercury.*

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 point-sources 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 sparrowhawk, 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.

*J. Christian Franson*

## 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.

Hecky, R.E., Ramsey, D.J., Bodaly, R.A., and Strange, N.E., 1991, Increased methylmercury contamination in fish in newly formed freshwater reservoirs, *in* Suzuki, T., and others, Advances in mercury toxicology: New York, N.Y., Plenum Press, p. 33–52.

Heinz, G.H., 1996, Mercury poisoning in wildlife, *in* Fairbrother, A., and others, eds., Noninfectious diseases of wildlife (2nd ed.): Ames, Iowa, Iowa State University Press, p. 118–127.

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.

Chapter 46

# Cyanide

### Synonyms

*Hydrocyanic acid poisoning, Prussic acid poisoning*

## Cause

Cyanide poisoning of birds is caused by exposure to cyanide in two forms: inorganic salts and hydrogen cyanide gas (HCN). Two sources of cyanide have been associated with bird mortalities: gold and silver mines that use cyanide in the extraction process and a predator control device called the M-44 sodium cyanide ejector, which uses cyanide as the toxic agent.

Most of the cyanide mortality documented in birds is a result of exposure to cyanide used in heap leach and carbon-in-pulp mill gold or silver mining processes. At these mines, the animals are exposed when they ingest water that contains cyanide salts used in mining processes or, possibly, when they inhale HCN gas. In heap leach mining operations, the ore is placed on an impermeable pad over which a cyanide solution is sprayed or dripped. The cyanide solution dissolves and attaches to or "leaches out" the gold. The cyanide and gold solution is then drained to a plastic-lined pond, which is commonly called the pregnant pond. The gold is extracted, and the remaining solution is moved into another lined pond, which is commonly called the barren pond. The cyanide concentration in this pond is increased so that the solution is again suitable for use in the leaching process, and the solution is used again on the ore heap (Fig. 46.1). Bird use of the HCN-contaminated water in the ponds (Fig. 46.2) or contaminated water on or at the base of the heap leach pads (Fig. 46.3) can result in mortality.

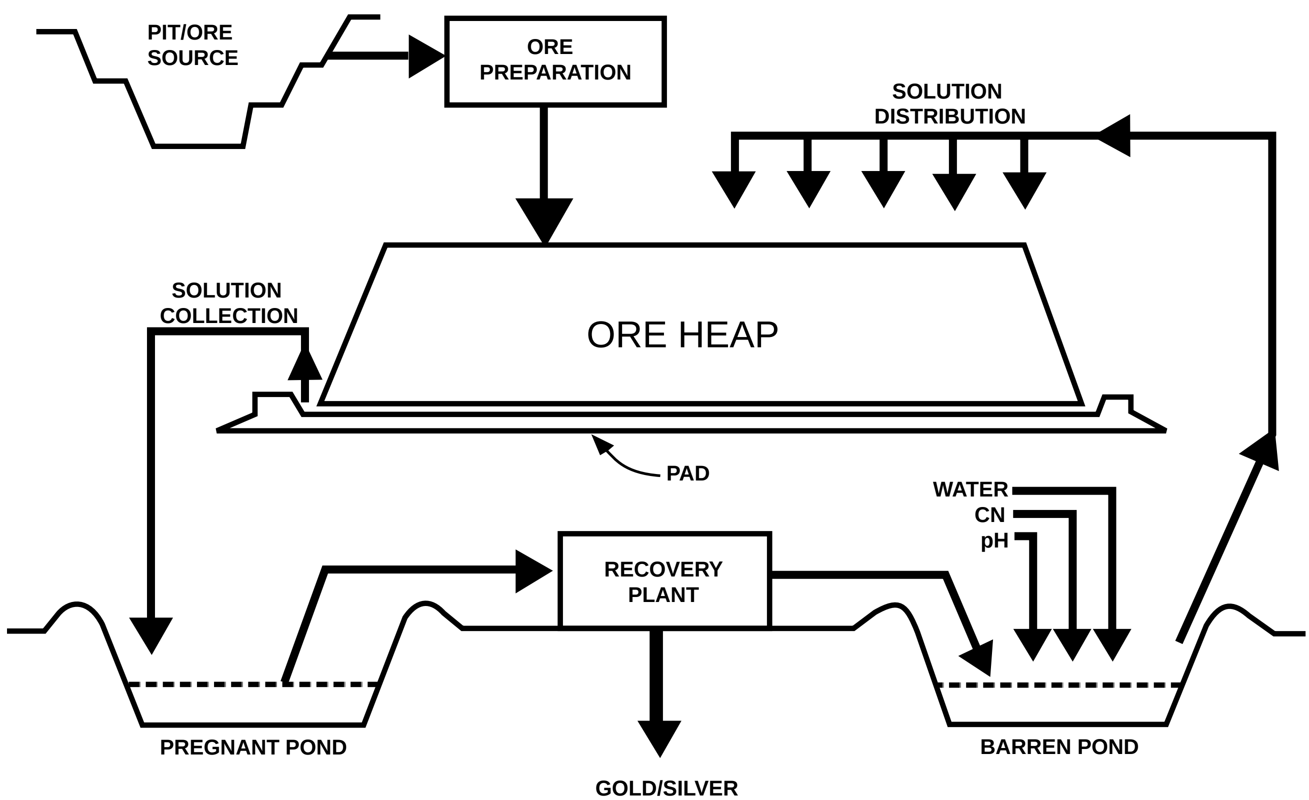



***Figure 46.1*** *Schematic of a typical heap leach system (Graphic by Robert Hallock, U.S. Fish and Wildlife Service).*

Mill tailings ponds produced by mines using the carbon-in-pulp mill process have also been responsible for migratory bird mortalities. In this process, crushed ore, cyanide solution, and carbon are placed together in a large vat. The cyanide solution extracts the gold from the ore, and the gold then adheres to the surface of the carbon. After the gold is extracted from the ore, the spent ore and the cyanide solution slurry are discharged to a mill tailings pond. The cyanide solution from the pond is drained, recharged, and reused in the extraction process. Tailings ponds range from 10 to several hundred surface acres and, in addition to open water, frequently have “mud flats” that are attractive to a wide variety of migratory birds. Cyanide concentrations are typically greatest near the spigots where mill slurry is discharged into the pond and are lowest in the solution reclamation areas.

The M-44 is a mechanical device designed to kill mammalian predators, specifically coyotes, by ejecting sodium cyanide into the animal’s mouth (Fig. 46.4). Cyanide from M-44s has occasionally been documented as the cause of mortality in nontarget bird species, such as eagles and other scavengers, that are attracted by the bait and trigger the M-44 device.

## Species Affected

Both birds and mammals can be killed by cyanide. From 1986–95, more than 3,000 cyanide-related mortalities involving about 75 species of birds representing 23 families were reported to the National Wildlife Health Center (NWHC). Waterbirds and passerines represented the greatest number of species affected (Fig. 46.5). Exposure to cyanide used in gold mining accounted for almost all of the mortalities; only one bird in these submissions, a bald eagle, was killed by an M-44.

## Distribution

Mines that use cyanide in the gold-or silver-extraction process are located in many areas of the United States. However, most mines are concentrated in western States, particularly in arid areas (Fig. 46.6). Because water is limited in these areas, birds are often attracted to the water sources created by the mining operations. Bird mortality associated with mining operations in six States has been reported to the NWHC (Fig. 46.7).

The M-44 is used more commonly in the Western states, and its use is restricted by the Environmental Protection Agency and individual State regulations.

## Seasonality

Cyanide toxicosis can occur at any time of the year. However, most mortalities associated with exposure to cyanide at mines are reported in the spring and fall months when birds are migrating through areas where mines are located.

Photo by Diane Fries, U.S. Fish and Wildlife Service

***Figure 46.2*** *Aerial view of a heap leach mine. Note the open ponds of water (arrows).*

Photo by Diane Fries, U.S. Fish and Wildlife Service

***Figure 46.3*** *Heap leach pads at a mine that uses cyanide in the gold-extraction process. The water puddling at the base of the pad in the foreground contains cyanide.*

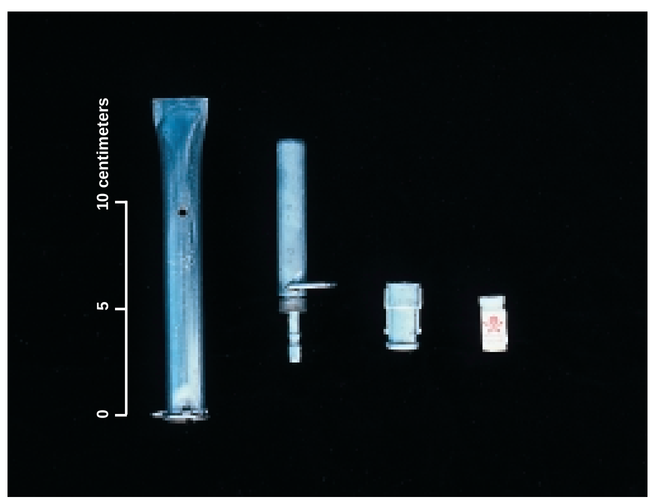



Photo by Guy Connolly, U.S. Department of Agriculture
National Wildlife Research Center

***Figure 46.4*** *The M-44 device consists of a stake (left), an ejector, a top, and a capsule containing cyanide.*

Photo by Diane Fries, U.S. Fish and Wildlife Service

***Figure 46.5*** *All of these birds were killed by cyanide-contaminated water at a heap leach gold mine. Note the diversity of the species present.*

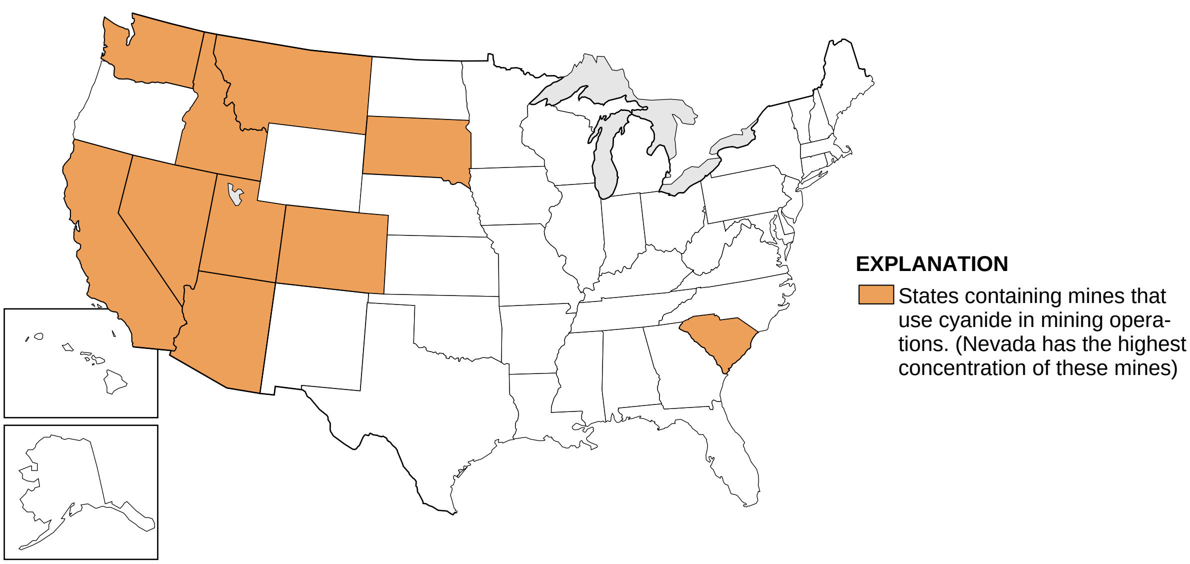



***Figure 46.6*** *States containing mines that use cyanide in leaching operations.*

## Field Signs

Cyanide acts rapidly, and affected birds are most often found dead. Cyanide interferes with the body's ability to utilize oxygen in the blood. Although the blood is well oxygenated, this oxygen cannot be released to the tissues and the animal dies from lack of oxygen or anoxia.

## Gross Lesions

Animals that die from cyanide toxicosis have bright red, oxygenated blood, and their tissues or organs, particularly the lungs, may appear congested with blood. The lungs of affected animals may also be hemorrhagic and edematous (Fig. 46.8). A yellow Day-Glo® fluorescent particle marker is used in the M-44 chemical mixture and animals exposed to cyanide through the M-44 device may have fluorescent yellow staining in the mouth or on the feathers or fur around the face. Visualization of this staining can be enhanced with ultraviolet light.

Photo by James Runningen

***Figure 46.8*** *Lungs from a cyanide-poisoned bird. Note the congestion and edema.*

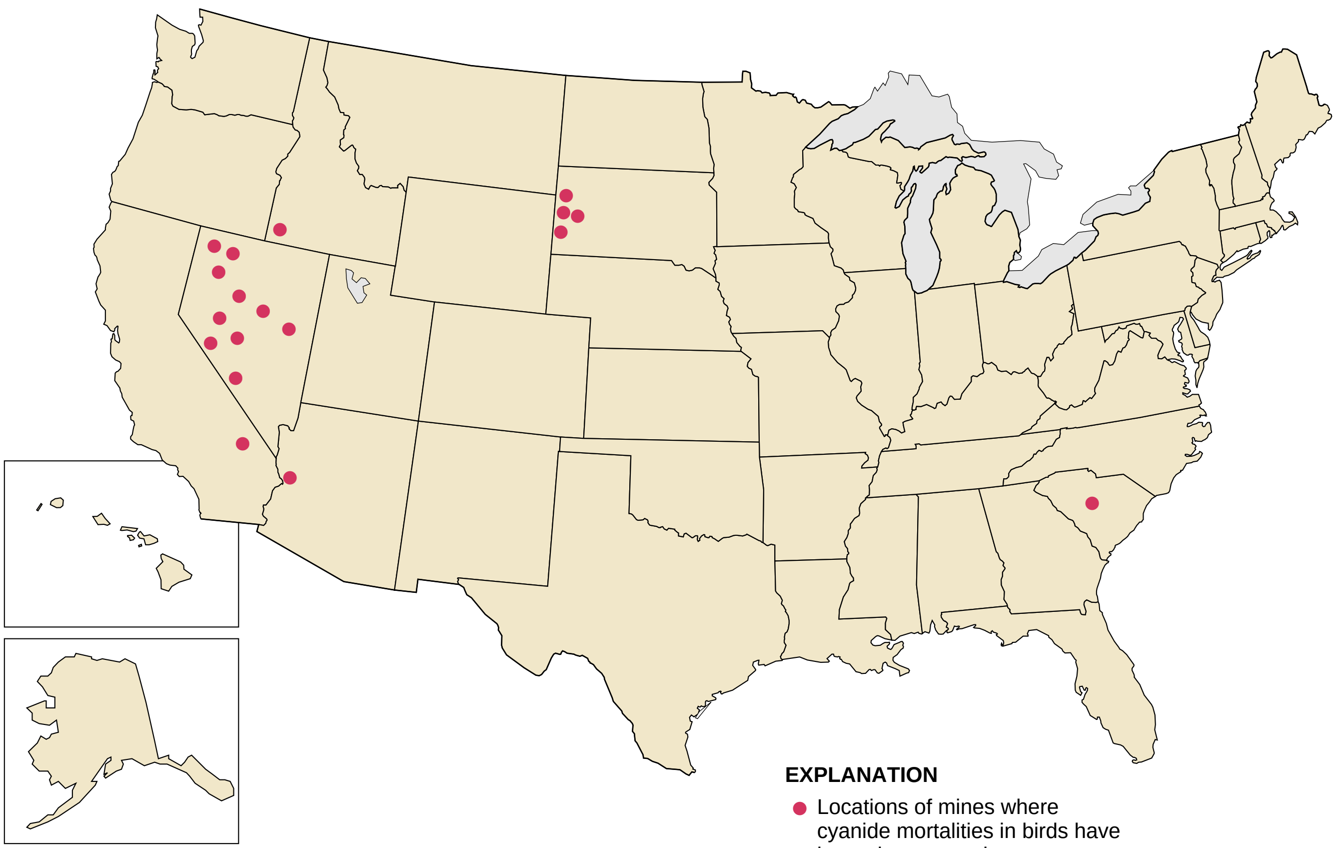



***Figure 46.7*** *Locations of mines where cyanide mortalities in birds have been documented.*

## Diagnosis

Diagnosis is based on the field history; by the lack of gross lesions other than those described above; and by chemical analysis of tissues, such as the blood, heart, liver, and brain, to detect cyanide. Proper carcass handling is very important for meaningful chemical analysis results because cyanide levels in tissues can diminish rapidly after death unless the carcass or tissues are frozen. Consequently, the best sample to submit to the diagnostic laboratory is the whole carcass of a bird found freshly dead and frozen immediately after retrieval. The carcass should remain frozen during shipment to the diagnostic laboratory; this is one instance in which dry ice is recommended. Contact a diagnostic laboratory for advice on carcass handling and shipment.

## Control

The primary method for preventing cyanide toxicosis at heap leach and carbon-in-pulp mill mining sites is to deny birds access to cyanide-contaminated water. This may or may not be difficult (or even possible) depending upon the size and configuration of a particular site. Successful methods used include netting over the solution ponds, covering heap leach collection channels, and designing mines that have no exposed solution ponds. Prevention of puddling in association with the heaps or netting over problem areas where puddling occurs are also beneficial. Detoxification or dilution have been the only successful means of preventing wildlife mortality at mill tailings ponds due to their large size and changing shapes. Hazing has not been very successful in preventing bird mortality at heap leach pads or heap leach and mill tailings ponds.

M-44s should be placed and baited to target only the intended species. Proper use of the M-44 lessens potential exposure of nontarget birds and mammals (Fig. 46.9).

## Human Health Considerations

Cyanide gas can cause death in humans; therefore, care should be taken when visiting mining sites. Alkaline cyanide solutions that are allowed to become acidic release cyanide gas. Abandoned sites where the cyanide solutions are no longer monitored and the proper pH maintained pose the greatest risk. In some instances, protective equipment may be necessary for site inspection or carcass pick-up. Untrained persons should not handle the M-44 sodium cyanide ejector. An antidote is provided with the device, and the people authorized to handle the device should be trained to administer the antidote quickly in the case of an accident.

*Lynn H. Creekmore*


*Photos by Guy Connolly, U.S. Department of Agriculture National Wildlife Research Center*

***Figure 46.9*** ***(A)*** *Closeup of a set M-44 device and* ***(B)*** *a completed M-44 set with a cow chip cover (arrow). Notice the warning sign. These signs are required at main entrances to areas in which M-44 devices are set and within 25 feet of each device.*

## Supplementary Reading


Connolly, G., 1988, M-44 sodium cyanide ejectors in the animal damage control program, 1976–1986, *in* Crabb, A.A., and March, R.E., eds., Proceedings of the vertebrate pest conference (v. 13): Davis, Calif., University of California, p. 220–225.

Eisler, Ronald, 1991, Cyanide hazards to fish, wildlife, and invertebrates: A synoptic review: U.S. Fish and Wildlife Service Contaminant Hazard Reviews Report 23, Biological Report 85(1.23). 55 p.

Henny, C.J., Hallock, R.J., and Hill, E.F., 1994, Cyanide and migratory birds at gold mines in Nevada, USA: Ecotoxicology, v. 3, p. 45–58.

Proceedings of the Nevada wildlife/mining workshop, Reno, Nevada, March 27–29, 1990: Reno, Nev., Nevada Mining Association, 233 p.

Wiemeyer, S.N., Hill, E.F., Carpenter, J.W., and Krynitsky, J.A., 1986, Acute oral toxicity of sodium cyanide in birds: Journal of Wildlife Diseases, v. 22, no. 4, p. 538–546.

Chapter 47

# Salt

### Synonyms

*Water deprivation, salt encrustation*

## Cause

Animals become victims of salt poisoning or toxicosis when toxic levels of sodium and chloride accumulate in the blood after they ingest large amounts of salt or, in some species, are deprived of water. For birds, salt sources may include saline water and road salt.

Normally, the salt glands of birds (Fig. 47.1) excrete sodium and chloride to maintain the proper physiologic chemical balance. However, when there has been insufficient time for acclimation of the salt gland to the saline environment, or when salt gland function is compromised by exposure to certain pesticides or oil, the electrolyte balance of the blood may be upset by the excess sodium and chloride, resulting in toxicosis. Salt accumulation on the outside of the body, or salt encrustation, is a greater problem for waterbirds that use very saline waters than is salt toxicosis. Salt encrustation can lead to exertion, acute muscle degeneration, and eventual drowning during the struggle to escape entrapment.

## Species Affected

This infrequently reported toxicosis has affected gallinaceous birds, such as pheasants, and rock doves that consumed road salt and migratory waterbirds forced to use highly saline water. Mortality from salt encrustation most often involves diving ducks.

## Distribution

Salt poisoning and salt encrustation can occur anywhere that birds use saline environments. However, salt poisoning may be more likely in northern latitudes where saline lakes remain open while nearby freshwater habitats freeze over and where salt is used for removing ice from roadways.

## Seasonality

Salt poisoning and salt encrustation may affect birds at any time of the year. In winter or early spring, terrestrial birds may consume road salt for grit and mineral content. Migratory waterbirds are more likely to be poisoned during late autumn migration after they have spent several months on freshwater nesting grounds. Cold snaps that freeze freshwater areas along the migratory route may force birds to use more saline waters that remain open because of the high salt content. High winds can contribute to salt encrustation by continually covering birds with salt-laden water.

## Field Signs

Clinical signs of salt poisoning may include muscle weakness, partial paralysis, and difficult breathing, all of which can be caused by a variety of other toxicoses. Carcasses may or may not be covered with salt (Fig. 47.2).

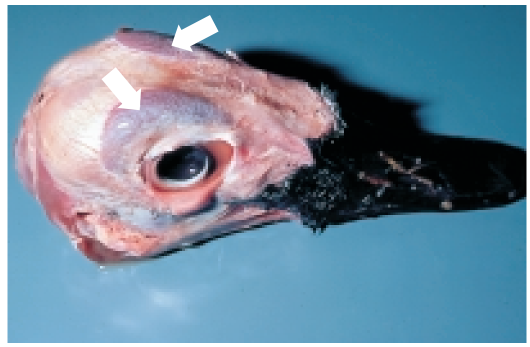
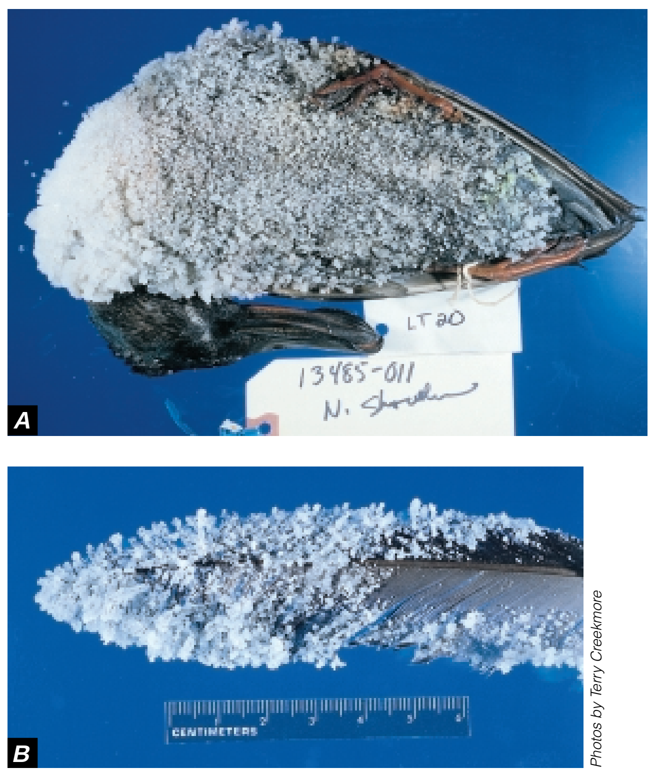


Photo by James Runningen

***Figure 47.1*** *The salt glands of birds are located just above the eyes (arrows).*




Photos by Terry Creekmore

***Figure 47.2*** *Salt encrustation may completely cover the bird with salt* ***(A)****, or salt may accumulate on margins of feathers* ***(B)****.*

## Gross Lesions

Gross lesions are nonspecific, and they may include reddening of the brain surface (Fig. 47.3), visceral gout (Fig. 47.4), fluid accumulation in the lungs, small hemorrhages on the viscera, and erosions on the surfaces of the eyes.

## Diagnosis

Finding a source of salt exposure lends support to a sometimes difficult diagnosis of salt poisoning. Salt on the feathers provides further evidence, but is not in itself diagnostic. Refrigerated blood and frozen as well as formalin-fixed brain are the best tissues to collect for laboratory analysis. Because the body maintains a constant internal environment or homeostasis, sodium concentrations in these tissues normally deviate very little. Therefore, a comparison of sodium concentrations between suspect and reference specimens can be used to support a diagnosis of salt poisoning. Microscopic examination of formalin-fixed brain tissue is also useful when salt poisoning is suspected.

## Control

Birds that are on highly saline lakes can be hazed to freshwater areas, if such areas exist nearby. Road salt should be used sparingly and should be stored out of reach of wildlife. Management practices that may expose birds to compounds that interfere with salt gland function, such as applications of organophosphorus and carbamate pesticides, should be done only when necessary and should be scheduled to allow arriving birds maximum time to adapt to saline environments.

## Human Health Considerations

None.

*J. Christian Franson and Milton Friend*

## Supplemental Reading


Friend, M., and Abel, J.H., Jr., 1976, Inhibition of mallard salt gland function by DDE and organophosphates, *in* Page, L.A., ed., Wildlife Disease: New York, N.Y., Plenum Press, p. 261–269.

Trainer, D.O., and Karstad, L., 1960, Salt poisoning in Wisconsin wildlife: Journal of the American Veterinary Association v. 136, p. 14–17.

Windingstad, R.M., Kartch, F.X., Stroud, R.K., and Smith, M.R., 1987, Salt toxicosis in waterfowl in North Dakota: Journal of Wildlife Diseases, v. 23, p. 443–446.

Wobeser, G.A., 1997, Salt and saline water, *in* Diseases of wild waterfowl (2nd ed): New York, N.Y., Plenum Press, p. 204–207.

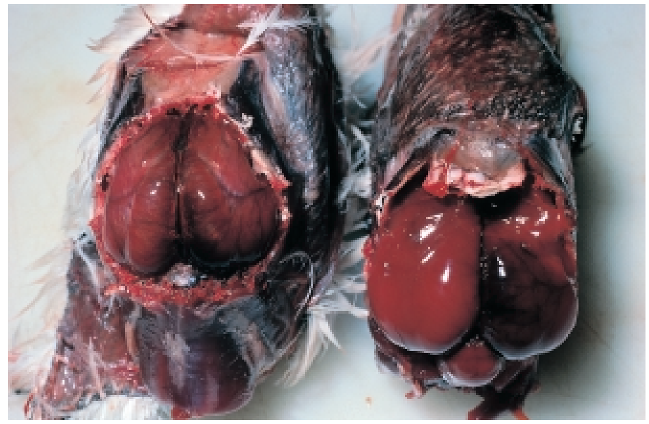



Photo by James Runningen

***Figure 47.3*** *The brains of salt-poisoned birds are sometimes very red and congested.*

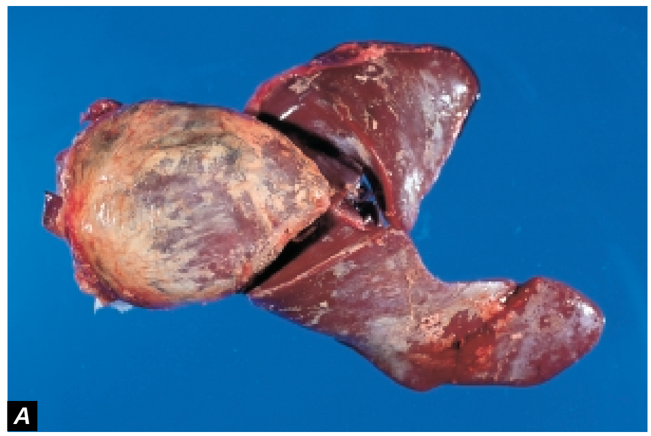




Photo by J. Christian Franson



Photo by James Runningen

***Figure 47.4*** ***(A and B)*** *Visceral gout, or accumulation of gritty uric acid deposits on visceral surfaces, is a nonspecific lesion that is sometimes associated with salt poisoning.*

Chapter 48

# Barbiturates

### Synonyms

*Pentobarbital poisoning, sodium pentobarbital poisoning*

## Cause

Barbiturate products are commonly used to euthanize domestic animals. The primary active component in euthanasia solutions is sodium pentobarbital, but some products also contain other minor ingredients (Fig. 48.1).

Euthanasia solutions are generally injected intravenously in domestic animals; therefore, after death, the solutions will be most concentrated in the blood and the highly vascularized organs, such as the liver or spleen, of the euthanized animal.

Euthanized carcasses that are available as carrion pose a hazard to scavenging birds and mammals. Large domestic animal carcasses, such as horses, that are not used for food or rendering but that are sufficiently valuable (monetarily or psychologically) to warrant veterinary services and euthanasia drugs are the most common sources of barbiturate poisoning in scavengers. In one instance in British Columbia, a single cow carcass was responsible for poisoning 29 bald eagles.

Circumstances that interfere with burial, such as frozen winter soil or bulky carcasses, result in euthanized carcasses being available for scavenger species. This problem could increase in the future if more stringent air-quality standards restrict carcass incineration.


Photo by Nancy Thomas

***Figure 48.1*** *The active agent in most injectable euthanasia solutions is sodium pentobarbital.*

## Species Affected

Bald and golden eagles are the only free-living wildlife species that have been reported to have died of barbiturate poisoning. Raptors generally have a narrow tolerance for barbiturate compounds; therefore, an anesthetic dose is often close to a fatal dose in these species.

## Distribution

As of 1997, the National Wildlife Health Center database contained records of 17 cases of barbiturate poisoning in eagles from six States (Fig. 48.2). Additional cases have been reported by other investigators.

## Seasonality

Cases of barbiturate poisoning have been more frequent in late winter and early spring, but they are not confined to that period. Cases of barbiturate poisoning may be correlated with the spring thaw in northern climates, when carcasses thaw, and the internal organs become more readily available to scavengers. Residues in those carcasses become available to scavenger species at that time. Food supplies are often limited at this time, so scavenging is more common.

## Field Signs

The most useful and specific field sign is the proximity of dead or moribund birds to a euthanized animal carcass that shows evidence of scavenging. In lieu of that, the proximity of dead or moribund birds to a domestic animal carcass of unknown origin is a less specific sign, but under that circumstance, barbiturates should be considered along with other poisons, such as pesticides.

Barbiturate-poisoned birds have been found near landfills in which euthanized animal carcasses were discarded. Landfills are legal disposal sites for carcasses in some States or locales.

Barbiturate poisoning may take hours to develop; therefore, poisoned birds can be found distant from the poison source. Eagles have been found beneath their roost trees without evident sources of poisoning.

Barbiturate-intoxicated birds are sedated, drowsy, sluggish, or comatose; have varying degrees of consciousness; and have slow heart and respiration rates. Although they may struggle to right themselves if they fall from a perch as toxicity progresses, signs of prolonged or violent struggling are unlikely. They are more likely to be found on undisturbed substrate. If more than one bird is exposed, the dose ingested and susceptibility to the poison may vary with each bird;


***Figure 48.2*** *Seventeen cases of barbiturate poisoning in bald and golden eagles have been reported by the National Wildlife Health Center from six States as of 1997.*

therefore, a range of signs from sublethal sedation to coma to death may be observed. Birds that are sedated or even comatose can recover if they are given supportive care until they metabolize the drug. Recovery may take several days.

## Gross Lesions

There are no specific lesions. Ingesta may be present in the upper gastrointestinal tract as in other acute poisonings. The ingesta may be recognizable as domestic animal in origin. Barbiturate-poisoned birds are often in good body condition, thus reflecting the acute nature of this toxicosis.

## Diagnosis

Analysis of liver or upper gastrointestinal contents detects pentobarbital and, sometimes, other components of euthanasia drugs. Liver analysis is more definitive for determining that a bird absorbed drug from the ingesta. Blood samples from live birds can be analyzed, but the clinical and field signs and the bird's recovery may be sufficient for a presumptive diagnosis.

Samples of blood-engorged organs, blood clots, or other tissue from scavenged sites in the suspect domestic animal carcass can assist in tracing the source of the poison.

## Control

### *Treatment*

Birds found alive in the field are often hypothermic (exhibiting low body temperature); warming of less affected birds, in itself, may result in recovery. A veterinarian can provide supportive care, administer cardiac and respiratory stimulants, and remove the undigested crop contents so that no further drug is absorbed.

### *Management*

State agricultural departments in the United States generally regulate carcass disposal to assure that carcasses are not available to scavengers. Circumstances such as frozen ground that prevents burial, poor compliance with regulations, or shallow burial may circumvent these regulations. Landfill regulations or policy can guarantee that carcasses are covered before scavenging is likely.

Cases of barbiturate poisoning are generally inadvertent. Prevention can be greatly enhanced by increasing awareness of the hazard among the public and veterinary community.

## Human Health Considerations

None. Euthanized carcasses are not for human consumption. Barbiturates are not absorbed through the skin.

*Nancy J. Thomas*

## Supplemental Reading

Langlier, K.M., 1993, Barbiturate poisoning in twenty-nine bald eagles, *in* Redig, P.T., and others, eds., Raptor Biomedicine: Minneapolis, Minn., University of Minnesota Press, p. 231–232.

Chapter 49

# Miscellaneous Chemical Toxins

The previous chapters provide information about some of the chemical toxins that have lethal effects on wild birds. The material presented in Section 7, Chemical Toxins, is far from comprehensive because wild birds are poisoned by a wide variety of toxic substances. Also, monitoring of wild bird mortality is not yet organized so that diagnostic findings can be extended to reflect the relative impacts among the types of toxins, within populations, or among species, geographic areas, and time. The data that are available are not collectively based on random sampling, nor do specimen collection and submission follow methodical assessment methods. Instead, most data simply document individual bird poisoning events. The inherent biases in this information include the species of birds observed dead (large birds in open areas are more likely to be observed dead than small forest birds); the species of birds likely to be submitted for analysis (bald eagles are more likely to be submitted than house sparrows); collection sites (agricultural fields are more likely to be observed than urban environments); geographic area of the country; season; reasons for submissions; and other variables. Nevertheless, findings from individual events reflect the causes of mortality associated with those events and collectively identify chemical toxins that repeatedly cause bird mortalities which result in carcass collection and submission for diagnostic assessment.

The tables that follow illustrate the relative occurrence of poisoning by different types of toxic substances for wild bird carcasses evaluated at the National Wildlife Health Center during the period of 1984 through 1995. This information was compiled to reflect the relative frequency of poisoning in different groups of birds as a function of the number of years that mortality occurred, the number of multiple-death events, and the number of years that had multiple-species deaths.

As noted above, biases in collecting and submitting carcasses prevent extrapolating these data to population impacts. The specimens that were evaluated depend on submissions from field personnel who had detected avian mortality events, and, for various reasons, had sought a diagnosis of the causes of mortality. Therefore, the tables simply reflect a relative accounting of what types of toxins were found most commonly to be the cause of death of the species that were submitted for evaluation. These data are not without meaning, because they clearly identify specific causes of poisoning in various groups of wild birds.

Carbofuran stands out as a frequent cause of mortality of a variety of bird species (Table 49.1). Diazinon was the most frequently diagnosed pesticide-induced cause of mortality in waterfowl, and famphur and carbofuran had similar prominence for eagles (Tables 49.1 and 49.2). As should be expected, chlorinated hydrocarbon pesticides were not frequently determined to be the cause of wild bird mortality (Table 49.3) now that these pesticides have been replaced by organophosphates, carbamates, and other compounds. Strychnine was a frequent cause of eagle mortality among compounds used as rodenticides and repellents (Table 49.4).

More than 30 different toxic substances were diagnosed as the cause of bird mortalities in specimens submitted (Tables 49.l through 49.5). The substances included naturally occurring materials such as selenium and sodium as well as synthetic products such as insecticides, and data in the tables are limited to those substances that caused direct lethal effects. As previously noted, there are many possible impacts of chemical toxins in addition to immediate toxicity that cause illness and death; some of these impacts involve interactions with other chemical or biological agents.

Residue analyses by themselves are often insufficient determinants of cause of mortality from chemical toxins be-

***Table 49.1*** *Relative occurrence of carbamate-caused mortality in free-ranging birds, 1984–95.*

*[Frequency of occurence: ● frequent, ● common, ● occasional, ○ infrequent or not reported]*

| Compound | Species | | | | | | |
|---|---|---|---|---|---|---|---|
| | Eagles | Hawks | Waterfowl | Gulls/terns | Crows[1] | Songbirds | Doves |
| Aldicarb | common | infrequent | infrequent | infrequent | occasional | infrequent | infrequent |
| Carbofuran | frequent | common | common | occasional | common | common | occasional |
| Methiocarb | infrequent | infrequent | infrequent | infrequent | occasional | infrequent | infrequent |
| Unspecified | occasional | infrequent | common | infrequent | infrequent | occasional | infrequent |

[1] Includes vultures, ravens, magpies, and crows.

***Table 49.2*** *Relative occurrence of organophosphorus-caused mortality in free-ranging birds, 1984–95.*

*[Frequency of occurence; ● frequent, ◐ common, ◔ occasional, ○ infrequent or not reported]*

| | Species | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Compound | Eagles | Hawks | Owls | Waterfowl | Cranes | Shorebirds | Crows[1] | Songbirds | Doves |
| Chlorpyrifos | ○ | ○ | ○ | ○ | ○ | ○ | ◔ | ○ | ○ |
| Coumaphos | ◔ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Diazinon | ○ | ○ | ○ | ● | ○ | ○ | ◔ | ◐ | ○ |
| Dimethoate | ○ | ○ | ○ | ◔ | ○ | ○ | ○ | ○ | ○ |
| Disulfoton | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ◐ | ○ |
| Famphur | ● | ◐ | ◔ | ○ | ○ | ○ | ◐ | ◐ | ◔ |
| Fenthion | ◐ | ◐ | ◔ | ○ | ○ | ○ | ○ | ◔ | ○ |
| Fonofos | ○ | ○ | ○ | ◔ | ○ | ○ | ○ | ○ | ○ |
| Monocrotophos | ○ | ○ | ○ | ◔ | ○ | ○ | ○ | ◐ | ◔ |
| Parathion | ◔ | ◔ | ○ | ◐ | ○ | ◔ | ○ | ◔ | ○ |
| Phorate | ◔ | ○ | ○ | ◐ | ○ | ○ | ○ | ○ | ○ |
| Terbufos | ◔ | ○ | ○ | ○ | ○ | ○ | ○ | ◔ | ○ |
| Unspecified | ◐ | ◐ | ◔ | ○ | ◔ | ○ | ○ | ○ | ○ |

[1] Includes vultures, ravens, magpies, and crows.

***Table 49.3*** *Relative occurrence of chlorinated-hydrocarbon-caused mortality in free-ranging birds, 1984–95.*
*[Frequency of occurence: ● frequent, ◐ common, ◔ occasional, ○ infrequent or not reported]*

| | Species | | |
|---|---|---|---|
| Compound | Eagles | Owls | Songbirds |
| Dieldrin | ◔ | ◔ | ◐ |
| Heptachlor | ◔ | ○ | ○ |

***Table 49.4*** *Relative occurrence of rodenticides and repellents as causes of mortality in free-ranging birds, 1984–95.*

*[Frequency of occurence: ● frequent, ◐ common, ◔ occasional, ○ infrequent or not reported]*

| | Species | | | | |
|---|---|---|---|---|---|
| Compound | Eagles | Hawks | Waterfowl | Crows[1] | Songbirds |
| Avitrol® | ○ | ○ | ○ | ○ | ◔ |
| Brodifacoum | ◔ | ○ | ◔ | ○ | ○ |
| 1080 | ◔ | ○ | ○ | ○ | ○ |
| Strychnine | ● | ◔ | ◔ | ◔ | ◐ |
| Thallium | ◐ | ○ | ○ | ○ | ○ |
| Zinc phosphide | ○ | ○ | ◐ | ○ | ○ |

[1] Includes vultures, ravens, magpies, and crows.

***Table 49.5*** *Relative occurrence of miscellaneous toxicants as causes of mortality in free-ranging birds, 1984–95.*
*[Frequency of occurence: ● frequent, ◍ common, ◌ occasional, ○ infrequent or not reported]*

| Species | Compound | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Chloride | Cyanide | Ethylene glycol | Fluorine | Hydrogen sulfide | Penta-barbitol | Selenium | Sodium |
| Eagles | ○ | ◌ | ○ | ○ | ○ | ● | ○ | ○ |
| Hawks | ○ | ◌ | ○ | ○ | ○ | ○ | ○ | ○ |
| Owls | ○ | ○ | ○ | ○ | ◌ | ○ | ○ | ○ |
| Waterfowl | ◌ | ◍ | ○ | ◌ | ○ | ○ | ◍ | ◍ |
| Cranes | ○ | ◌ | ○ | ○ | ○ | ○ | ○ | ○ |
| Grebes | ○ | ◌ | ○ | ○ | ○ | ○ | ◍ | ◌ |
| Pelicans | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ◌ |
| Gulls/terns | ○ | ◌ | ○ | ○ | ○ | ○ | ○ | ○ |
| Shorebirds | ○ | ◍ | ○ | ○ | ○ | ○ | ○ | ○ |
| Egrets[1] | ○ | ○ | ○ | ○ | ○ | ○ | ◍ | ○ |
| Crows[2] | ○ | ○ | ◌ | ○ | ○ | ○ | ○ | ○ |
| Songbirds | ○ | ◍ | ○ | ○ | ○ | ○ | ○ | ○ |
| Doves | ○ | ◌ | ○ | ○ | ○ | ○ | ○ | ◌ |
| Swallows | ○ | ◌ | ○ | ○ | ○ | ○ | ○ | ○ |
| Quail | ○ | ○ | ○ | ○ | ◌ | ○ | ○ | ○ |

[1] Includes long-legged wading birds such as herons and egrets.
[2] Includes vultures, ravens, magpies, and crows.

cause of species variations, lack of residue for some types of compounds, and other variables. Similarly, the often-quoted 16th Century statement that, "Dosage Alone Determines Poisoning" is modified by such factors as route of exposure and other important factors.

Chemical toxins are, and will continue to be, important causes of wildlife mortality. Documentation of mortality from chemical toxins requires rigorous diagnostic work. Determination of wildlife impacts will best be accomplished through methodical monitoring programs that allow sound evaluations of changes in the status and trends of specific compounds and their impacts on wild bird populations by geographic area.

*Milton Friend*

Section 8
# Miscellaneous

## Electrocution

## Miscellaneous Diseases

*Vertebral column deformity (scoliosis) in a bald eagle*
*Photo by James Runningen*

# Introduction to Miscellaneous Diseases

"Nature is far from benign; at least it has no special sentiment for the welfare of the human versus other species." (Lederberg)

The fact that "Nature is far from benign" is clearly evident from the preceding chapters of this Manual. The diseases and other conditions described are the proverbial "tip of the iceberg" relative to the number of specific causes of ill health and death for free-ranging wild birds, but the wild bird health problems described account for most major wild bird disease conditions seen within the United States. However, the full toll from disease involves many other causes of illness and death that individually may cause substantial die-offs. Two examples of these other causes of die-offs are the deaths of Canada geese that ingest dry soybeans, which then expand and cause lethal impactions within the moist environment of digestive tract, and the poisoning of ducks from rictin, a naturally occurring toxic component of castor beans. Some of these lesser-known causes of disease and mortality may become increasingly important in the future because landscape and other changes could result in environmental conditions that may enhance the interface between specific disease agents and susceptible bird species.

This final Section of the Manual includes some of the lesser-known causes of avian mortality. The first chapter provides an overview of electrocution in birds, with a special emphasis on eagles. The second chapter is a miscellaneous chapter that highlights a significant disease of domestic ducklings not yet known to exist in wild birds, disease caused by stress due to improper handling of birds, and several other conditions that might be encountered by biologists who work with birds. These other conditions include tumors, traumatic injuries, weather, nutritional factors, and drowning as causes of avian illness and death. These two chapters expand the scope of disease presented in the previous chapters and provide additional perspectives of the diverse causes of avian mortality. It is our hope that the collective information provided in this Manual will stimulate those interested in the conservation and well-being of avian species to give greater consideration to disease in the management strategies employed for the conservation of these species.

Quote from:

Lederberg, J., 1993, Viruses and humankind: intracellular symbiosis and evolutionary competition, *in* Morse, S.S., ed., Emerging viruses: Oxford, England, Oxford University Press, p. 3.

Chapter 50

# Electrocution

## Cause

Power lines and power poles present a potential electrocution hazard to wild birds. Many birds, especially raptors, select power poles for perching, and, sometimes, for nesting (Figs. 50.1–3). If a bird's appendages bridge the gap between two energized parts or between an energized and a grounded metal part, electricity flows through the "bridge" that is filling the gap and the bird is electrocuted.

Most commonly, birds are electrocuted where conducting wires (conductors) are placed closer together than the wingspan of birds that frequent the poles (Fig. 50.2). Feathers are poor electrical conductors, but if contact is made between points on the skin, talons, or beak, or if the feathers are wet, conduction can occur. Common anatomical sites of contact include conduction between the wrists of each wing or between the skin of one wing and a foot or leg. The resulting shock causes severe, usually fatal, cardiovascular injury.

Because conductors on distribution lines are placed closer together than high voltage transmission lines, birds are more frequently electrocuted on distribution lines despite their lower voltage.

In addition to one to three conductors, power poles may also carry ground wires, transformers, or grounded metal crossarm braces. Complicated wiring configurations that put multiple energized and grounded metal parts near attractive perching or nesting sites are the most hazardous configurations (Fig. 50.3).

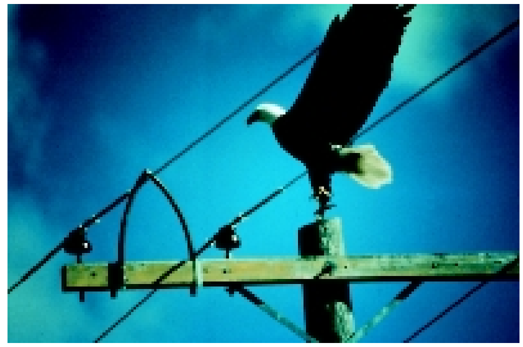


Photo by Brian Woodbridge, U.S. Fish and Wildlife Service, Macdoel, CA

***Figure 50.1*** *A bald eagle using a power pole as a perch.*

Photo by Morlan Nelson, PacifiCorp, Boise, ID

***Figure 50.2*** *This is a hazardous situation because the eagle's wings can contact two conductors at once.*

## Species Affected

Electrocution is primarily a problem of large raptors in open habitat, particularly treeless areas. Golden eagles are by far at greatest risk, but other eagles, large buteos, falcons, and the largest owls, such as the great horned owl, are also susceptible. The large wingspan of these birds appears to be the single most important factor in their susceptibility.

In addition to their size, the perching behavior of these bird species puts them at greater risk. Species that prefer exposed high perches are more likely to be attracted to power poles, as are the species that use a "still hunting" technique in which they perch and visually search the landscape for prey rather than hunting in flight.

Immature and subadult raptors are more commonly electrocuted. This predisposition is presumably related to their inexperience and awkwardness in taking off and landing.

Photo by Monte Garrett, PacifiCorp

***Figure 50.3*** *An eagle nest on the top of a power pole.*

## Distribution

Bird electrocutions are most common in the western plains of the United States where open shrub and grassland habitats are common, and are less prevalent in forested habitat (Fig. 50.4). However, birds may be electrocuted wherever electrical lines are above ground.

Generally, electrocutions are more prevalent in sites where a susceptible species' prey base is present and where suitable perches, other than power structures, are lacking. In the western plains, elevated perches are at a premium, and the more susceptible raptor species are abundant. The combination of golden eagles, jackrabbits, grassland habitat, and dangerous power pole configurations can be expected to be lethal. Similar conditions exist on the Russian steppes. Electrocution is a major cause of mortality for the Russian steppe eagle and for other raptors that nest on power poles and use them for perches in this largely treeless area (Fig. 50.5).

Photo by Milton Friend

***Figure 50.5*** *Power lines that are not designed to prevent electrocution and that cross largely treeless areas, such as this line on the Russian steppes, pose a significant hazard for large raptors that use the poles as perches for hunting and as nesting platforms.*

## Seasonality

Birds can be electrocuted during any season, but there can be seasonal fluctuations in electrocution frequency that are related to weather conditions or bird behavior. Electrocutions are more frequent during periods of rain and snow because of the increased conductivity of wet feathers. Inclement wet weather may also combine with windy conditions so that birds are less stable while landing and taking off. Where distribution lines are oriented with crossarms perpendicular or diagonal to the prevailing wind, more electrocutions occur.

Golden eagles may make greater use of power poles as night roosts during migration and wintering. This habit may make them more prone to electrocution as they stretch out to dry their wings in the morning sun.

Inattentiveness during seasonal mating behaviors or territorial conflicts have also been reported to predispose birds to electrocution.

## Field Signs

Electrocuted birds often die immediately, so they are found near a power pole or beneath a power line.

The electrical hazard may be apparent in the configuration of the nearby pole. The conductors and other electrical hardware on the pole may be close together. The greatest hazards may be at corner poles where extra wires (jumpers) are required to provide a change in direction, or at poles with transformers or grounded metal equipment near the conductors (Fig. 50.6).

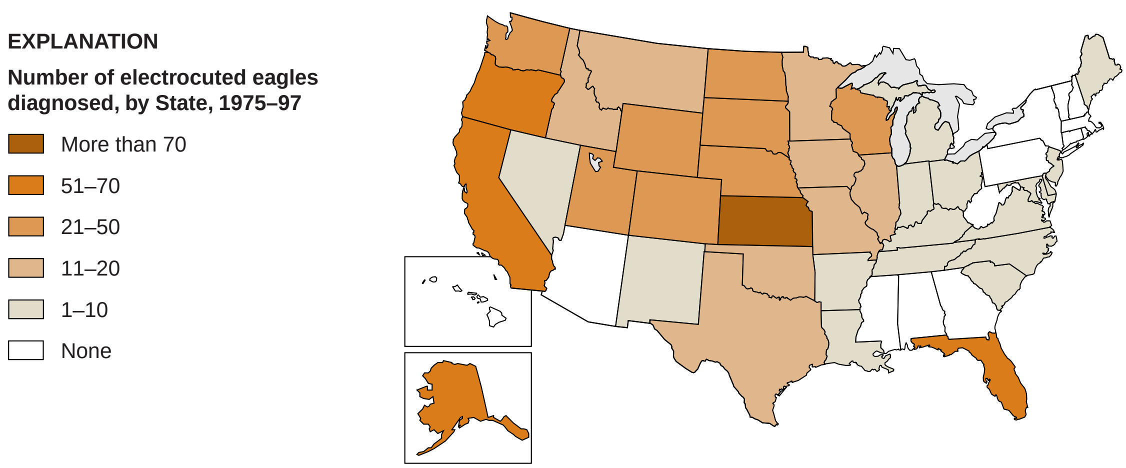



***Figure 50.4*** *Number of electrocuted eagles diagnosed per State from 1975–95. (From unpublished data from the National Wildlife Health Center.)*

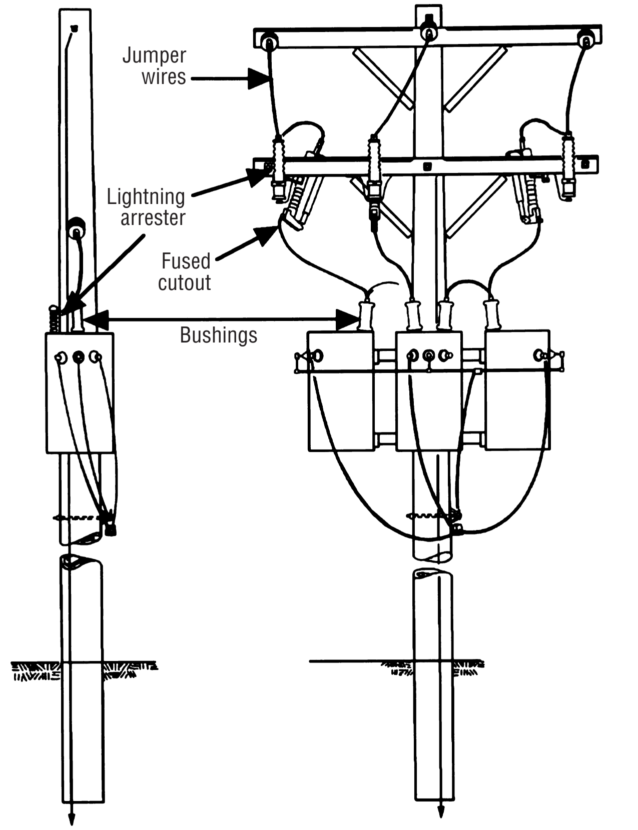



*__Figure 50.6__ Complicated wiring that is configured with transformers, jumpers, and additional hardware is often responsible for raptor electrocutions. (Graphic provided by Monte Garrett, PacifiCorp)*

Bird electrocutions can cause power outages; therefore, a history of electrical power disruption can help confirm the diagnosis and fix the location and time of electrocution.

Electrocuted birds may catch on fire and ignite vegetation beneath the power structures.

If a bird is electrocuted because the prey item or wet nest material it is carrying comes in contact with an energized part, then these items may be found with the carcass or clutched in its talons.

## Gross Lesions

The hallmark of electrocution is burn marks. Burns are generally confined to the sites of body contact with the electrical source; however, if the feathers are ignited then the entire carcass may be charred (Fig. 50.7). Burn marks from fatal electrocutions can have a remarkable range in appearance from very subtle feather disruption to limb amputation. Burns cause the feather edges to curl or twist (Fig. 50.8), and light-colored feathers may be discolored brown or charred. Burns on avian skin appear as dry blisters, particularly on the scales of the feet or legs (Fig. 50.9A and B). The margins of these blisters may be brown or charred. Severe, deep burns can extend through the skin, cauterize muscles and tendons, liquefy fat, and even fracture bones.

Sublethal bird electrocutions are uncommon. In these cases, a single limb is usually affected. Initially, burns may be seen on the skin or the feathers at the contact site. Later, the only evidence may be the loss of blood supply to a wing or foot and eventual gangrene. If the damage can be removed by surgical amputation, some electrocuted birds can recover and be kept permanently in captivity.

## Diagnosis

A diagnosis of electrocution is based on the presence of burns and an absence of evidence of other causes of death. Hemorrhages in the subcutaneous tissue and internal organs suggest cardiovascular injury and can support the diagnosis.

A field history that includes proximity to an electrical line is helpful but not sufficient in itself. Birds may collide with

Photo by James Runningen

*__Figure 50.7__ An electrocuted bald eagle that is charred over most of its body.*

Photo by James Runningen

*__Figure 50.8__ Electrical burns on the wing feathers of a bald eagle. Note also the fracture and charring of nearby bones.*


*__Figure 50.9__ __(A)__ A large burn on a golden eagle's foot. __(B)__ Multiple small, subtle burns in the scales on a bald eagle's foot.*

power lines, be shot while perching, or fall from perches after poisoning or illness; therefore, location is not definitive for electrocution.

## Control

Raptor electrocutions generally can be reduced by adopting safe electrical pole and line configurations or managing raptor perching. Safe wiring configurations separate the wires and the grounded metal parts so that raptors cannot simultaneously touch two of them at once (Fig. 50.10). Existing installations that contain hazardous configurations can be modified by insulating or reconfiguring the wiring. Rather than comprehensive modifications, an economical but effective approach is to modify selected poles based on field observations of bird use and mortality. If reconfiguring or insulating the wires is not feasible, then access to the hazardous perch can be blocked and safer, alternate perches can be provided. Despite the inherent equipment costs of modification, electrical power companies are often proactive in preventing bird electrocution. Power companies benefit by reducing costly power outages, by avoiding liability for migratory bird mortalities, and by the positive public image that is generated by control projects.

When new electrical installations are planned, the design can take into consideration the likelihood of raptor electrocution. The risk can be evaluated in advance by considering raptor concentrations and behavior along the installation route. Structures in raptor migratory corridors, as well as nesting and wintering ranges, may pose a risk.

## Human Health Considerations

Under normal circumstances, there is no exposure.

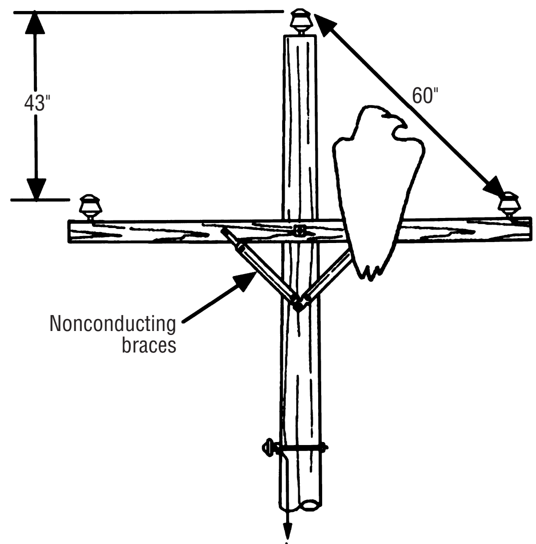



*__Figure 50.10__ A safe wiring configuration separates the conductors and other energized hardware so that large raptors are unable to touch two pieces of hardware simultaneously. (Graphic provided by Monte Garrett, PacifiCorp)*

*Nancy J. Thomas*

## Supplementary Reading

Avian Power Line Interaction Committee, 1996, Suggested practices for raptor protection on power lines: The state of the art in 1996: Edison Electric Institute/Raptor Research Foundation, Washington, D.C., 125 p.

Bevanger, K., 1994, Bird interactions with utility structures: Ibis, v. 136, p. 412–425.

Chapter 51

# Miscellaneous Diseases

This concluding chapter is intended to further inform the reader of the broad spectrum of causes affecting the health of wild birds by illustrating a variety of disease conditions that are not described elsewhere in this Manual. The information in this chapter is not intended to represent a comprehensive description of other causes for ill-health and death in wild birds. Instead, examples are provided of some less commonly reported conditions that, in some instances, illustrate larger health issues. Too little is known about these conditions to currently assess their biological significance as mortality factors in wild birds.

## Disease in Hatchlings and Young

Much of what is known about disease in free-ranging wild birds is the result of observations and investigations of fully grown birds. Nevertheless, the knowledge gained from domestic poultry and captive-reared wild birds has often demonstrated great disease impacts for young birds. Loss of young can have significant impacts on population levels (see Trichostrongylidosis in Chapter 35); therefore, special vigilance is needed to prevent the introduction of disease into free-ranging populations that have the potential for high mortality of young.

Duck hepatitis is an example of a disease of domestic ducks that could cause mortality of young free-ranging birds if it were to spread to free-ranging populations (Figs. 51.1–3). This highly fatal, rapidly spreading viral disease is found worldwide and is economically important to all duck-raising operations because of the high potential of mortality if it is not controlled. Young pheasants, goslings, and young guinea fowl have all suffered high mortality following experimental infection with duck hepatitis virus, thereby illustrating a greater host range than waterfowl. Mallard ducklings are also killed by this virus, and adult mallards have been reported to serve as mechanical or noninfected transport hosts for the movement of duck hepatitis virus between commercial duck-raising operations. Clinical signs and mortality in mallards have been confined to ducklings less than 3-weeks old. However, birds that recovered from infection have been reported to shed the virus in their feces for up to 8 weeks postinfection.

## Plastic Debris

Improper disposal of several types of products made from plastic causes problems for birds. Some of these problems can result in mortality. They can frequently be reduced by educating people about the problems and by other means (Figs. 51.4–6).

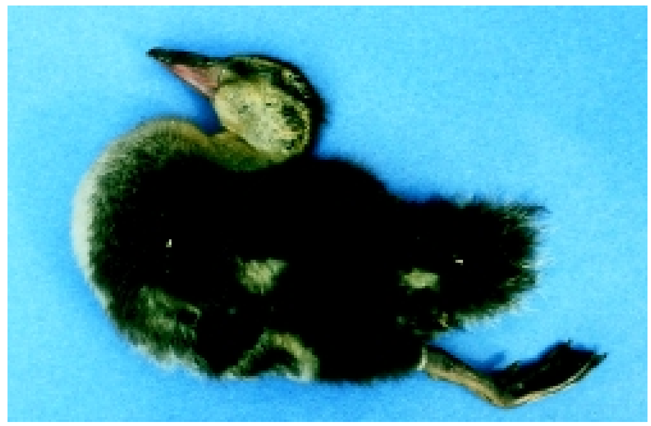


Photo by Milton Friend

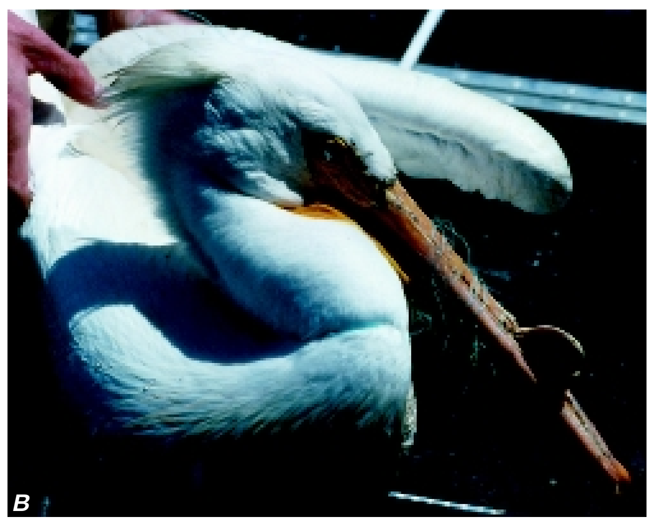
***Figure 51.1*** *Typical terminal position of ducklings that die from duck hepatitis. This posture is referred to as opisthotonos, and it is characterized by the body being somewhat bowed forward with the head and bottom of the feet bent backward.*


Photo by Milton Friend

***Figure 51.2.*** *Typical liver lesions of mallard ducklings that died from duck hepatitis. Note the color change and enlargement of the two infected livers **(A** and **B)** compared with the liver from an uninfected duckling of the same age **(C)**. The principal lesions, in addition to the greatly enlarged liver, are hemorrhages over varying amounts of the surface area. The more discrete areas of hemorrhage are referred to as petechia (for the very small isolated areas) or punctate (dotlike), and the broader areas of hemorrhage as ecchymotic.*

Photo by Milton Friend

***Figure 51.3*** *Mallard duckling infected with duck hepatitis. The livers of infected birds generally become so swollen that they fill much of the bird's abdominal cavity.*



Photos by Milton Friend

***Figure 51.4*** ***(A)*** *Improperly discarded fishing line carried to the top of this tree by a double-crested cormorant became a "hangman's noose" and strangled the bird in this photograph. The line tangled around the tree top and it also looped around the bird's neck when it attempted to fly from its perch above a small urban lake.* ***(B)*** *Discarded fishing line wrapped around the bill of this white pelican would have resulted in death by starvation had the bird not been captured and the line removed. Note also the constricted areas of the pouch caused by the line.*

National Wildlife Health Center photo files

***Figure 51.5*** *A Canada goose with a plastic 6-pack ring entangle around its neck (arrow). Birds accidentally acquire these rings when they place their heads through them as they feed on the ground.*

Photo by Lou Sileo

***Figure 51.6*** *These discarded plastic materials were found in the stomach of an albatross chick. Items such as these are ingested as food by adult birds when they feed at sea and reach the chick when the adult regurgitates food to feed its young. Fortunately, most debris of this type is voided by the chicks without causing them harm. However, birds can suffer intestinal blockages and other ill effects.*

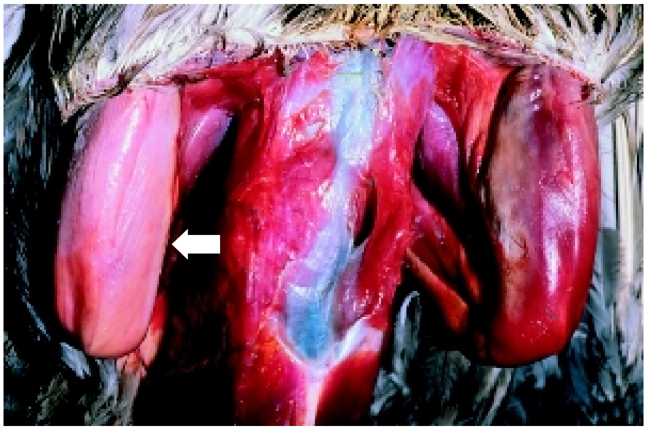


Photo by James Runningen

***Figure 51.7*** *Light colored muscle of leg (arrow) represents capture myopathy in a sandhill crane.*

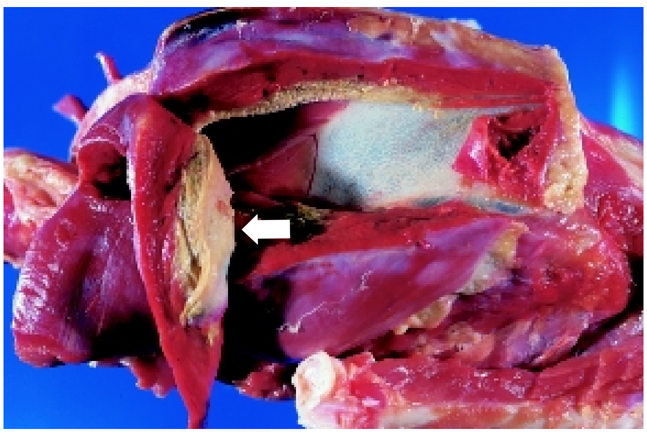


Photo by Nancy J. Thomas

***Figure 51.8*** *Light colored area in breast muscle (arrow) of a peregrine falcon with capture myopathy.*

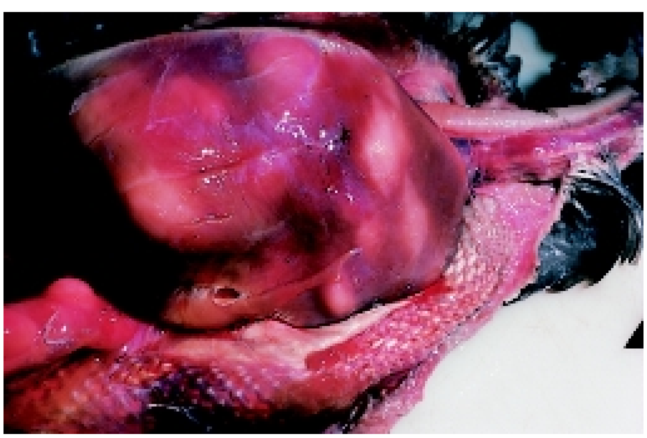


Photo by James Runningen

***Figure 51.9*** *Tumors in breast muscle of a Canada goose.*

## Disease Due To Handling

Improper judgements and procedures by humans while they pursue, handle, and transport wild animals, including birds, during wildlife management activities can induce capture myopathy (Figs. 51.7–8). More descriptive names include over-straining disease, transport myopathy, exertional myopathy, muscle necrosis, white muscle disease, and stress myopathy. These names convey that improper handling or stress can cause a bird to overexert and result in stress-related injury to its muscles. Tissue damage is a result of complex physiological processes, not physical trauma such as bruising from impact. Mortality has been reported in a wide variety of bird species including flamingos, cranes, waterfowl, raptors, gulls, wild turkey, and other species. This disease of overexertion results in severe damage to striated muscles including the heart. Birds may die hours or even several days after they have been released, thereby leaving their human captors and handlers unaware of the damage that was done. The potential for inducing this disease should be carefully considered during the planning phases of wildlife capture, handling, and transportation, and measures should be taken to minimize risks. Warm environmental temperatures are often a risk factor as are the duration of pursuit, the method and duration of restraint, placement of birds in unfamiliar surroundings, and noise associated with human activities. Situations that have induced capture myopathy in birds include trapping and handling operations involving drop nets and rocket nets; drive-trapping, handling, and translocation of flightless birds; and handling birds so that marking devices, including radio transmitters, can be placed on them. All of these needed activities can be done safely if proper consideration is given to capture myopathy and the steps that can be taken to avoid inducing this disease.

## Tumors

Neoplasms or tumors are infrequent findings in free-ranging wild birds, but they are found (Figs. 51.9–12). Tumors are formed by the abnormal progressive multiplication of cells into uncontrolled (by the body) new tissue that appears as various growths within tissues and organs. These growths may be noninvasive or benign, or they may spread to other tissues and parts of the body and be malignant. Tumors result from multiple causes. Virus-induced tumors, such as the herpesvirus that causes an important infectious poultry disease known as Marek's disease, are transmissible. Tumors formed due to other than infectious agents have been reported from all major body systems of birds, the reproductive, digestive, respiratory, nervous, and endocrine systems, in addition to the skin surfaces.

Less than 1 percent of the wild birds for which postmortem examinations were done at the National Wildlife Health Center (NWHC) over a span of more than 20 years (1975–1998) had tumors. These findings are consistent with those of other disease diagnostic laboratories that process large numbers of free-ranging wildlife. A notable exception at the NWHC has been a high prevalence of tumors in Mississippi sandhill cranes received from the wild (Figs. 51.11, 12). The cause(s) of the tumors in this endangered species remains undetermined.

## Trauma

Many wild birds are injured and killed each year from impacts with buildings, wires, and other products of the human environment (Figs. 51.13, 14). Birds that have large wing spans, such as cranes and eagles, are among those commonly found with fractured wings and other injuries from collisions with power lines and wire fences. Road kills of raptors that feed on carrion are common. Whenever it is feasible, bird flight patterns and bird use of local habitat should be considered in the routing of power transmission lines, wind power generation units, and roads. Protective measures against bird strikes should be employed when they are warranted if less hazardous alternative routings cannot be accomplished. Monitoring for road kills of birds and observations of birds feeding on carcasses can indicate food shortages for species such as eagles and can be mitigated by establishing short-term feeding stations that move the birds from the roadways to safer locations during the period of food scarcity.

## Other

Wild birds are subject to major direct losses from weather. Waterfowl and other species have been frozen to the ice by their feet and feathers (Fig. 51.15), and strong winds associated with hurricanes have filled coastal beaches with large numbers of birds with fractured wings. Heavy snows and storms that coat vegetation with a thick layer of ice deprive

Photo by James Runningen

***Figure 51.10*** *Tumor on the leg of a ruffed grouse.*

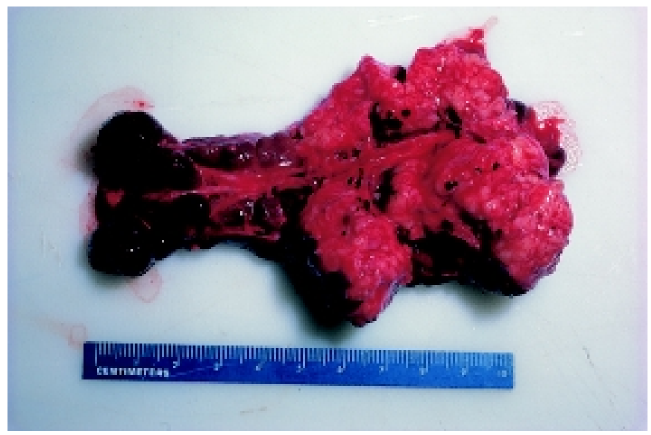


Photo by James Runningen

***Figure 51.11*** *Tumor attached to the kidneys of a Mississippi sandhill crane.*

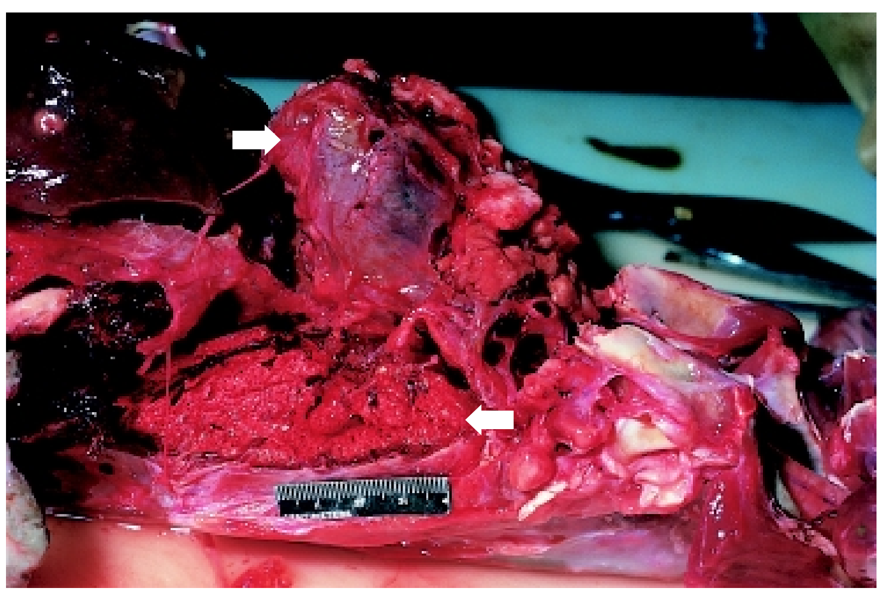


Photo by James Runningen

***Figure 51.12*** *A malignant tumor covering the heart (top arrow) and lungs (bottom arrow) of a Mississippi sandhill crane.*

Photo by Rod Drewien, University of Idaho

***Figure 51.13*** *Collision with fences, power lines, and other structures is a significant mortality factor for birds. This whooping crane died after striking a fence.*

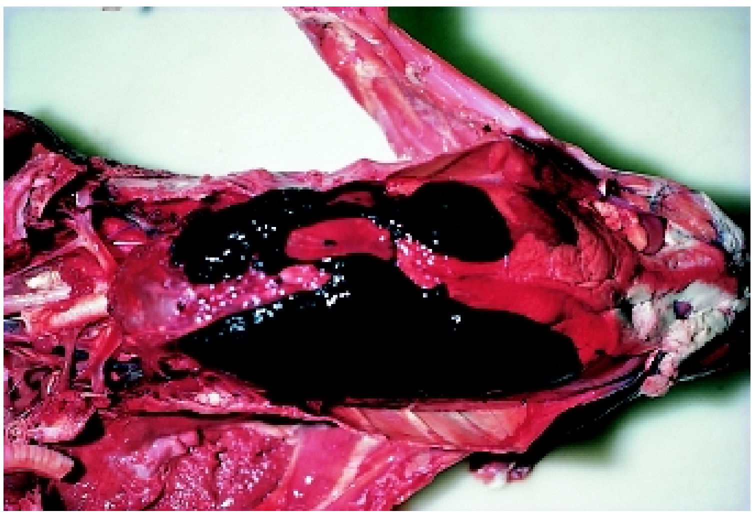


Photo by Michael Coffey

***Figure 51.14*** *Trauma from collision often results in massive internal hemorrhage.*

Photo by James Hurt, Nebraska Game and Parks Commission

***Figure 51.15*** *Severe weather can cause large losses of wildlife. These Canada geese became entrapped by ice when high winds accompanied by temperatures that rapidly dropped below freezing during a spring storm quickly turned this shallow wetland into a frozen body of water. The high winds prevented flight, and the water splashing over the birds froze them in place. Severe traumatic injuries resulted as the birds tried to free themselves from the ice.*

wild birds and other wildlife of access to food and can result in starvation (Fig. 51.16). Numerous other weather-related situations also affect bird health.

Malnutrition resulting in starvation is but one aspect of nutritional diseases that may affect birds. Nutritional diseases are a complex subject area that is beyond the scope of this Manual, and they are mentioned only to make the reader aware of them. Nutritional diseases involve excess intake as well as deficiencies. Changes in bird diets associated with landscape changes due to agriculture can contribute to nutritional diseases. For example, excesses of dietary protein and vitamin deficiency may occur due to extensive feeding on agricultural grains rather than natural food sources. Visceral gout may result (Fig. 51.17). Under experimental conditions, substances that are toxic to the kidneys (nephrotoxic agents) and diets deficient in Vitamin A and high in calcium have caused avian gout.

Wild birds also drown. Drowning may be an outcome of extreme weather conditions that aquatic birds are sometimes subject to; exhaustion of passerines during migration, which causes them to drop into water bodies that they may be traversing at the time; and as a result of other factors, such as the feathers of aquatic birds becoming waterlogged from oil contamination or nonfunctioning preen glands that prevent birds from “waterproofing” their feathers.

Various deformities due to a variety of causes are also seen in wild birds (Fig. 51.18). Some deformities result from exposure to excess levels of selenium; others may result from exposure to synthetic compounds, nutritional disorders, or injury to tissues during early developmental stages of the bird; they may be of genetic origin; or result from other causes. Deformities are not commonly observed because birds that are afflicted with such conditions are likely to be more vulnerable to factors that reduce their chance for survival. Therefore, clusters of observations of deformities should be viewed as an indication of a larger problem and warrant investigation to determine the underlying cause.

*Milton Friend and Nancy J. Thomas*

## Supplementary Reading


Fairbrother, A., Locke, L.N., and Hoff, G.L., 1996, Noninfectious diseases of wildlife, (2d ed.): Ames, Iowa, Iowa State University Press, 219 p.

Wallach, J.D., and Cooper, J.E., 1982, Nutritional diseases of wild birds, *in* Hoff, G.L., and others, eds., Noninfectious diseases of wildlife: Ames, Iowa, Iowa State University Press, p. 113–126.



***Figure 51.16*** *Ice that coats vegetation may prevent access to food, resulting in starvation.*

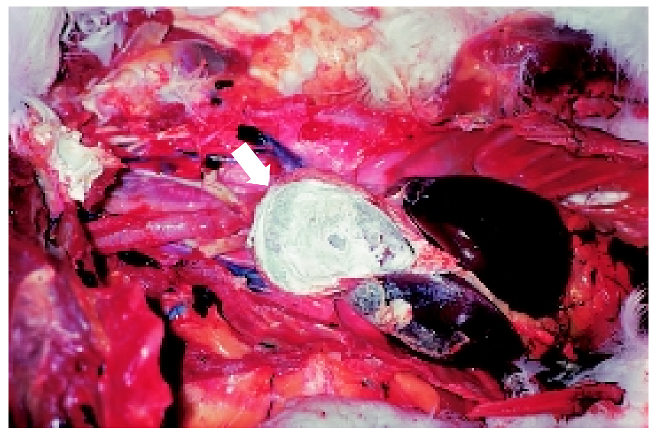



***Figure 51.17*** *Dietary protein imbalances can cause visceral gout, exhibited by an accumulation of white, gritty deposits on surfaces of organs, such as the heart (arrow).*

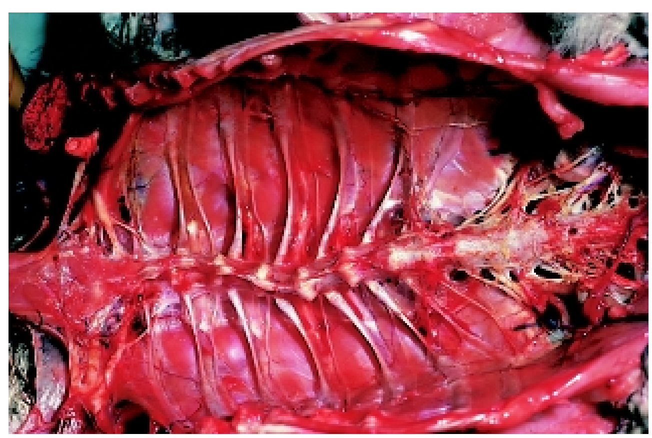



***Figure 51.18*** *Vertebral column deformity (scoliosis) in a bald eagle.*

# Appendix A

## Sample specimen history form

Submitter's name: Affiliation:

Address: Telephone: E-mail:

Date collected:

Method of collection: [found dead, euthanized (describe method) etc.]

Collector's name:

Specific die-off location:

State: County: Latitude/longitude:

Environmental factors: (Record conditions such as storms, precipitation, temperature changes, or other changes that may contribute to stress.)

Disease onset: (The best estimate of when the outbreak started.)

Species affected: (The diversity of species affected may provide clues to the disease involved.)

Age/sex: (Any selective mortality related to age and sex.)

Morbidity/mortality: (Ratio of sick animals to dead animals.)

Known dead: (Actual pickup figures.)

Estimated dead: (Consider removal by scavengers or other means.)

Clinical signs: (Any unusual behavior and physical appearance.)

Population at risk: (Number of animals in the area that could be exposed to the disease.)

Population movement: (Recent changes in the number of animals on the area and their source or destination, if known.)

Problem area description: (Land use, habitat types, and other distinctive features.)

Comments: (Additional information/observations that may be of value such as past occurrences of disease in area.)

# Appendix B

## Sources of wildlife diagnostic assistance in the United States

Assistance in obtaining a diagnosis of wildlife illness or death is available from a variety of sources. However, it is advisable to make inquiries before the need arises about available services, the estimated response time for completing work, and who to contact when assistance is required.

The following wildlife disease programs can offer information, assistance, and services.

*Wildlife Disease Programs*

1. U.S. Department of Interior,
   U.S. Geological Survey,
   Biological Resources Division
   National Wildlife Health Center
   6006 Schroeder Road
   Madison, WI 53711
   Telephone (608) 270-2400
   Web site: http://www.emtc.usgs.gov/nwhchome.html

2. State fish and game agencies. Several States have wildlife disease programs. Among those are Alaska, California, Colorado, Florida, Idaho, Michigan, New Jersey, New York, Wisconsin, and Wyoming. Contact the State fish and game agency headquarters to inquire about assistance.

3. Regional wildlife disease programs. Two regional programs are presently affiliated with universities:

   Southeastern Cooperative Wildlife Disease Study
   College of Veterinary Medicine
   University of Georgia
   Athens, GA 30602
   Telephone (706) 542-1741

   Northeastern Research Center for Wildlife Diseases
   University of Connecticut
   Department of Pathobiology
   Storrs, CT 06269-3089
   Telephone (860) 486-4000

4. University programs. Several other universities, for example, the University of Florida—Gainesville and Virginia Polytechnical Institute and State University—Blacksburg, are involved in wildlife disease activities. Inquiries at schools of veterinary medicine and departments of veterinary or animal science at universities throughout the United States will reveal additional sources of wildlife disease diagnostic assistance.

5. Private sector. Some private consultants also deal with wildlife disease problems.

*Domestic Animal Disease Programs*

1. U.S. Department of Agriculture,
   Animal and Plant Health Inspection Service
   National Veterinary Services Laboratories
   P.O. Box 844, Ames, Iowa 50010
   Telephone (515) 239-8600.

   This facility accepts diagnostic specimens that have been referred to it through appropriate State or Federal channels.

2. State departments of agriculture. Animal disease diagnostic laboratories exist to serve domestic animal needs, but will often accept wildlife specimens.

3. Private sector. Veterinarians in private practice often have both interest and expertise in wildlife diseases and may become involved with these problems.

### Additional sources of assistance for investigating wildlife mortality events when chemical toxins are suspected.

#### *Federal Government*

*U.S. Fish and Wildlife Service (FWS)*

The Division of Environmental Contaminants (DEC) is the FWS focal point for issues associated with chemical toxins. Information on contaminants can be obtained from the Central Office in Washington, D.C.

Telephone: (703) 358-2148
Web site: http://www.fws.gov/~r9dec/ecprog.html and from DEC staff in the seven FWS Regional Offices.

DEC biologists are assigned to field offices throughout the FWS Regions. They work on specific contaminant issues in each Region and are available to provide information and assistance regarding mortality event investigations.

Toxic spill coordinators are located in each Regional Office, providing a focal point for response actions.

FWS Regional Offices are located in:
Portland, Ore.; Albuquerque, N. Mex.; Fort Snelling, Minn.; Atlanta, Ga.; Hadley, Mass.; Denver, Colo.; and Anchorage, Alaska.

*U.S. Geological Survey, Biological Resources Division (BRD)*

The National Wildlife Health Center, which is the BRD Science Center in Madison, Wis., provides information about and assists in investigating wildlife mortality events.

Telephone: (608) 270-2400
Web site: http://www.emtc.usgs.gov/nwhchome.html

Research on chemical toxins is carried out at several BRD Science Centers. Those Centers maintain in-depth technical knowledge regarding the fate and impacts of chemicals in the environment. The Patuxent Wildlife Research Center in Laurel, Md. is an internationally recognized source of information on the effects of contaminants, particularly on avian species.

Telephone: (301) 497-5500
Web site: http://www.pwrc.usgs.gov/

*U.S. Environmental Protection Agency (EPA)*

Office of Solid Waste and Emergency Response

Telephone: (703) 308-8413
Web site: http://www.epa.gov/epaoswer/

*The Office of Pollution Prevention and Toxics (OPPT)* assesses the hazards and risks posed by industrial chemicals to human health and the environment. The Environmental Effects Branch in OPPT can provide information on the toxicity of chemicals to aquatic and terrestrial organisms.

Telephone: (202) 260-1268

*Office of Pesticide Programs* maintains the Ecological Incident Information System, which is a data base on mortality of non-target organisms caused by pesticides.

Telephone: (703) 305-5392

### State Government

Many State natural resource agencies have environmental contaminant programs that provide a mechanism to report suspected chemical toxin problems. Some States have groups that investigate mortality events associated with chemicals, and that may be able to provide field assistance and chemical analysis.

Natural resource agencies in several States maintain wildlife health programs, which respond to wildlife mortality events.

State veterinary diagnostic laboratories often have toxicologists on staff who have specific knowledge of toxic problems within the region.

### Poison Control Centers

*The National Animal Poison Control Center* at the University of Illinois College of Veterinary Medicine provides a fee-based service directed to prevention and treatment of adverse effects of chemical exposures in animals. This service is staffed by veterinary health professionals who have access to a wide range of information specific to animal poisoning.

Telephone (800) 548-2423, (900) 680-0000
Web site: http://www.cvm.uiuc.edu/NAPCC/NAPCC.html

*The American Association of Poison Control Centers (AAPCC*) has certified about 40 regional poison information centers throughout the U.S. that focus on human exposure to chemical toxins. These centers function to provide poison information, telephone management and consultation, collect pertinent data, and deliver professional and public education. The national AAPCC office is in Washington, D.C.
Telephone: (202) 362-7217. A directory of the regional centers is available at Web site: http://www.pitt.edu/~martint/pages/rpiclist.htm

### Colleges of Veterinary Medicine

Most colleges of veterinary medicine have toxicology departments staffed with experts in the area of animal toxicology.

### Analytical Laboratories

Choosing an analytical laboratory requires attention to methods used, quality assurance/quality control (QA/QC), and cost. Laboratories should be using methods that are appropriate to the analysis required in the matrix (material being analyzed) that is submitted. Minimum quality control data provided by the laboratory should include:

(1) The results of analysis of spiked samples, or recovery. A known amount of the compound being analyzed for is added to the appropriate matrix. The recovery is the amount of the compound that was recovered in the analysis, and it is expressed as a percentage of the amount of compound added.
(2) A replication of results, or an agreement of analyses of duplicate samples.
(3) The results of blank samples, or an absence of the compound being analyzed for in a “clean” sample of the appropriate matrix.
(4) The results of analysis of standard reference samples. A sample with a known quantity of the compound is prepared by an independent laboratory, and this sample is then analyzed by the laboratory being evaluated. Although good QA/QC adds to the expense of analytical work, the alternative may be an incorrect diagnosis.

Some of the analytical laboratories that have been used by the FWS DEC and others include:

*For inorganic analyses:*

Environmental Trace Substance Laboratory
University of Missouri - Rolla
101 USBM Bldg., 1300 North Bishop Ave.
Rolla, MO 65409-0530
Telephone: (314) 341-6607

Research Triangle Institute
3040 Cornwallis Road, Bldg. 6
Research Triangle Park, NC 27709-2194
Telephone: (919) 541-6896

Geochemical & Environmental Research Group
833 Graham Road
College Station, TX 77845
Telephone: (409) 690-0095

*For organic analyses:*

Geochemical & Environmental Research Group (see above)

Mississippi State Chemical Laboratory
Mississippi State University
Hand Chemical Lab, Rm 201, Morrill Road
Mississippi State, MS 39762
Telephone: (601) 325-3251

The above listing is not intended to be comprehensive, nor does it constitute endorsement by the Federal government. Rather, it illustrates the diversity of possible sources of assistance. Individual circumstances and events dictate which of these sources will be most useful in specific situations.

# Appendix C

**Sources of supplies used for collecting, preserving, and shipping specimens**

| Company | Address and telephone | Item |
|---|---|---|
| Scientific Products | 319 West Ontario<br>Chicago, IL 60610<br>Tel: (800)323-4515 | Whirl-Pak® bags, formalin, wide-mouth, plastic jars, indelible markers |
| Curtis Matheson Scientific, Inc. | P.O. Box 1546<br>Houston, TX 77251<br>Tel: (713)820-9898 | Whirl-Pak® bags, wide-mouth, plastic jars, indelible markers |
| Fisher Scientific Co. | 711 Forbes Ave.<br>Pittsburgh, PA 15219<br>Tel: (800)766-7000 | Whirl-Pak® bags, formalin, indelible markers |
| Thomas Scientific | P.O. Box 99<br>Swedesboro, NJ 08095<br>Tel: (609)467-2000 | Whirl-Pak® bags, wide-mouth, plastic jars, indelible markers |
| VWR Scientific | P.O. Box 66929<br>O'Hare Amp<br>Chicago, IL 60666<br>Tel: (800)932-5000 | Whirl-Pak® bags, indelible markers |
| Local hospital and medical supply businesses | | Whirl-Pak® bags, wide-mouth, plastic jars |
| Some pharmacies | | formalin |
| Freund Can Co. | 167 W. 84th Street<br>Chicago, IL 60620<br>Tel: (312)224-4230 | metal paint cans with lids |
| U.S. General Services (gov't agencies only) | GSA Customer<br>Supply Center<br>Administration (GAS)<br>5619 W. 115th St.<br>Worth, IL 60482<br>Tel: (800)262-0570 | filament strapping tape, plastic bags, indelible markers |
| GSA Federal Supply Service | 1500 E. Bannister Rd.<br>Kansas City, MD 64131<br>Tel: (816)926-7315 | filament strapping tape, plastic bags, indelible markers |

| Company | Address and telephone | Item |
|---|---|---|
| Local hardware, sports, and discount stores | | Styrofoam®, shipping coolers, indelible markers, ice packs, filament strapping tape |
| Polyfoam Packers Corp. | 2320-T Foster Avenue<br>Wheeling, IL 60090<br>Tel: (800)225-7443 | biomedical shippers and mailers |

Protective clothing, gloves, and disinfectants can also be obtained from many of the sources listed above. The above list is not intended to be comprehensive, nor does it constitute endorsement by the Federal government.

# Appendix D

**Normal brain cholinesterase activity values**

| Species | Mean[1] | Standard deviation | Sample size |
|---|---|---|---|
| Avocet, American | 19.4 | 2.9 | 5 |
| Blackbird, Red-winged | 24.5 | 1.2 | 5 |
| Bobwhite, Northern | 16.3 | 2.1 | 7 |
| Brant, Black | 14.4 | 0.8 | 5 |
| Coot, American | 20.5 | 5.0 | 12 |
| Cormorant, Double-crested | 29.3 | 2.4 | 5 |
| Cowbird, Brown-headed | 19.7 | 2.6 | 5 |
| Crane, Mississippi Sandhill | 16.6 | 2.3 | 15 |
| Crane, Sandhill | 17.5 | 1.4 | 8 |
| Crane, Whooping | 15.1 | 1.5 | 9 |
| Dove, Mourning | 22.8 | 3.3 | 11 |
| Duck, American Wigeon | 10.5 | 1.0 | 12 |
| Duck, Blue-winged Teal | 19.5 | 3.6 | 6 |
| Duck, Green-winged Teal | 13.5 | 2.0 | 17 |
| Duck, Kola | 11.0 | 1.2 | 9 |
| Duck, Mallard | 11.0 | 1.6 | 75 |
| Duck, Muscovy | 11.6 | 3.5 | 8 |
| Duck, Northern Shoveler | 14.7 | 1.2 | 5 |
| Duck, Pintail | 11.6 | 1.3 | 24 |
| Duck, Ruddy | 13.4 | 1.4 | 8 |
| Duck, Wood | 10.2 | 1.5 | 9 |
| Eagle, Bald | 16.0 | 2.6 | 156 |
| Eagle, Golden | 16.0 | 2.2 | 57 |
| Egret, Common | 17.4 | 1.9 | 5 |
| Egret, Snowy | 25.0 | 1.3 | 5 |
| Falcon, Peregrine | 18.6 | 3.2 | 27 |
| Goldfinch, American | 17.8 | 1.5 | 10 |
| Goose, Canada | 11.9 | 2.0 | 36 |
| Goose, Canada (Aleutian) | 14.0 | 3.6 | 8 |
| Goose, Ross | 14.2 | 2.3 | 9 |
| Goose, Snow | 13.6 | 2.6 | 42 |
| Goose, White-fronted | 12.1 | 1.2 | 9 |
| Grebe, Eared | 14.7 | 1.4 | 17 |
| Grosbeak, Evening | 20.3 | 3.1 | 5 |
| Gull, Ring-billed | 23.9 | 6.5 | 8 |
| Hawk, Red-tailed | 17.5 | 1.4 | 7 |
| Hawk, Sharp-shinned | 21.5 | 2.0 | 6 |
| Heron, Black-crowned Night | 15.6 | 3.0 | 5 |
| Heron, Great Blue | 13.3 | 2.1 | 8 |
| Loon, Common | 17.3 | 4.4 | 5 |
| Owl, Great-horned | 15.5 | 2.0 | 7 |
| Owl, Screech | 18.7 | 1.4 | 5 |
| Owl, Spotted | 14.6 | 2.0 | 9 |

| Species | Mean[1] | Standard deviation | Sample size |
|---|---|---|---|
| Parrot, Puerto Rican | 19.4 | 2.0 | 6 |
| Pelican, American White | 13.0 | 1.3 | 13 |
| Pelican, Brown | 11.2 | 1.2 | 16 |
| Sandpiper, Semipalmated | 14.1 | 1.1 | 5 |
| Siskin, Pine | 16.9 | 2.3 | 16 |
| Stork, Wood | 18.7 | 4.2 | 8 |
| Swan, Trumpeter | 11.3 | 1.3 | 10 |
| Swan, Tundra | 11.6 | 1.7 | 14 |
| Tern, Least California | 44.1 | 9.1 | 5 |
| Woodcock, American | 16.8 | 1.1 | 5 |
| Woodpecker, Red-cockaded | 38.4 | 4.8 | 5 |

[1] Cholinesterase activity is expressed as micromoles acetylthiocholine hydrolyzed per minute per gram of wet weight brain tissue (Hill, E.F. and Fleming, W.J., 1982, Environmental toxicology and chemistry 1:27–38).

*Milton Smith*

# Appendix E

**Common and scientific names of birds in text**

Albatrosses, shearwaters, and petrels (Order Procellariiformes)
- Albatrosses (Family Diomedeidae)
  - Black-footed albatross (*Diomedia nigripes*)
  - Laysan albatross (*Diomedia immutabilis*)
- Shearwaters and petrels (Family Procellariidae)
  - Northern fulmar (*Fulmarus glacialis*)
  - Petrels (*Pterodroma* sp, *Oceanodroma* sp, *Oceanites* sp)
  - Sooty shearwater (*Puffinus griseus*)

Cormorants, pelicans, and tropicbirds (Order Pelecaniformes)
- Cormorants and Shags (Family Phalacrocoracidae)
  - Brandt's cormorant (*Phalacrocorax penicillatus*)
  - Cape cormorant (*Phalacrocorax capensis*)
  - Double-crested cormorant (*Phalacrocorax auritus*)
  - Great cormorant (*Phalacrocorax carbo*)
  - Shags (*Phalacrocorax* sp)
- Gannets and boobies (Family Sulidae)
  - Gannet (*Morus bassanus*)
- Pelicans (Family Pelecanidae)
  - Brown pelican (*Pelecanus occidentalis*)
  - White pelican (*Pelecanus erythrorhynchos*)
- Tropicbirds (Family Phaethontidae)
  - Red-tailed tropicbird (*Phaethon rubricauda*)
  - White-tailed tropicbird (*Phaethon lepturus*)

Cranes, coots, and rails (Order Gruiformes)
- Cranes (Family Gruidae)
  - Brolga crane (*Grus rubicunda*)
  - Common crane (*Grus grus*)
  - Demoiselle crane (*Anthropoides virgo*)
  - East African crowned crane (*Balearica regulorum gibbericeps*)
  - Greater sandhill crane (*Grus canadensis tabida*)
  - Hooded crane (*Grus monacha*)
  - Lesser sandhill crane (*Grus canadensis canadensis*)
  - Manchurian crane (red-crowned crane) (*Grus japonensis*)
  - Mississippi sandhill crane (*Grus canadensis pulla*)
  - Sarus crane (*Grus antigone*)
  - Stanley crane (blue crane) (*Anthropoides paradisea*)
  - White-naped crane (*Grus vipio*)
  - Whooping crane (*Grus americana*)
- Rails, coots, and gallinules (Family Rallidae)
  - American coot (*Fulica americana*)
  - Common moorhen (*Gallinula chloropos*)
  - Sora (*Porzana carolina*)

Emus and ostriches (Order Struthioniformes)
  - Emu (Family Casuariidae, *Dromaius novaehollandiae*)
  - Ostrich (Family Struthionidae, *Struthio camelus*)

Grebes (Order Podicipediformes, Family Podicipedidae)
  - Eared grebe (*Podiceps nigricollis*)
  - Western grebe (*Aechmophorus occidentalis*)

Grouse, quail, and partridges (Order Galliformes, Family Phasianidae)
- Grouse (Subfamily Tetraoninae)
  - Blue grouse (*Dendragapus obscurus*)
  - Ptarmigan (*Lagopus* sp)
  - Red grouse (*Lagopus lagopus scoticus*)
  - Ruffed grouse (*Bonasa umbellus*)
  - Sage grouse (*Centrocercus urophasianus*)
  - Sharp-tailed grouse (*Tympanuchus phasianellus*)
- Quail (Subfamily Odontophorninae)
  - Bobwhite quail (Northern) (*Colinus virginianus*)
  - California quail (*Callipepla californica*)
  - Coturnix quail (*Coturnix coturnix*)
  - Japanese quail (*Coturnix japonica*)
  - Scaled quail (*Callipepla squamata*)
- Partridges and pheasants (Subfamily Phasianinae)
  - Chukar partridge (*Alectoris chukar*)
  - Gray partridge (Hungarian partridge) (*Perdix perdix*)
  - Common peafowl (*Pavo cristatus*)
  - Ring-necked pheasant (*Phasianus colchicus*)
- Turkeys (Subfamily Meleagridinae)
  - Wild turkey (*Meleagris gallopavo*)
- Guinea fowl (Subfamily Numidinae, *Numida* sp)

Hawks, falcons, and vultures (Order Falconiformes)
- Hawks, eagles, and kites (Family Accipitridae)
  - Eurasian sparrowhawk (*Accipiter nisus*)
  - Bald eagle (*Haliaeetus leucocephalus*)
  - Booted eagle (*Hieraaetus pennatus*)
  - Cooper’s hawk (*Accipiter cooperii*)
  - Common buzzard (*Buteo buteo*)
  - Ferruginous hawk (*Buteo regalis*)
  - Golden eagle (*Aquila chrysaetos*)
  - Northern goshawk (*Accipiter gentilis*)
  - Imperial eagle (*Aquila heliaca*)
  - Northern harrier (marsh hawk) (*Circus cyaneus*)
  - Red-shouldered hawk (*Buteo lineatus*)
  - Red-tailed hawk (*Buteo jamaicensis*)
  - Rough-legged hawk (*Buteo lagopus*)
  - Sharp-shinned hawk (*Accipiter striatus*)
  - Steppe eagle (*Aquila nipalensis*)
  - White-tailed eagle (*Haliaeetus albicilla*)
  - Osprey (*Pandion haliaetus*)
- Falcons and caracaras (Family Falconidae)
  - American kestrel (sparrowhawk) (*Falco sparverius*)
  - Gyrfalcon (*Falco rusticolus*)
  - Peregrine falcon (*Falco peregrinus*)
  - Prairie falcon (*Falco mexicanus*)
  - Red-headed falcon (*Falco chicquera*)
  - Saker falcon (*Falco cherrug*)
- Vultures (Family Cathartidae)
  - California condor (*Gymnogyps californianus*)
  - King vulture (*Sarcoramphus papa*)
  - Turkey vulture (*Cathartes aura*)

Herons, ibises, and storks (Order Ciconiiformes)
- Flamingos (Family Phoenicopteridae)

Greater flamingo (*Phoenicopterus ruber*)
Herons, egrets, and bitterns (Family Ardeidae)
Bitterns (*Botaurus* sp*, Ixobrychus* sp)
Black-crowned night heron (*Nycticorax nycticorax*)
Great blue heron (*Ardea herodias*)
Great egret (common or american egret) (*Casmerodius albus*)
Snowy egret (*Egretta thula*)
Ibises and spoonbills (Family Threskiornithidae)
Glossy ibis (*Plegadis falcinellus*)
White ibis (*Eudocimus albus*)
White-faced ibis (*Plegadis chihi*)
Storks (Family Ciconiidae)
Wood stork (*Mycteria americana*)
Loons (Order Gaviiformes, Family Gaviidae)
Common loon (*Gavia immer*)
Pacific loon (*Gavia pacifica*)
Red-throated loon (*Gavia stellata*)
Owls (Order Strigiformes)
Typical owls (Family Strigidae)
Barred owl (*Strix varia*)
Eagle owl (*Bubo bubo*)
Eastern screech owl (*Otus asio*)
Great-horned owl (*Bubo virginianus*)
Long-eared owl (*Asio otus*)
Short-eared owl (*Asio flammeus*)
Snowy owl (*Nyctea scandiaca*)
Spotted owl (*Strix occidentalis*)
Barn-Owls (Family Tytonidae)
Barn owl (*Tyto alba*)
Parrots, parakeets, and macaws (Order Psittaciformes)
Parrots (Family Psittacidae)
Budgerigar (*Melopsittacus undulatus*)
Cockatiel (*Nymphicus hollandicus*)
Hawk-headed parrot *(Deroptyus accipitrinus*)
Lories (*Lorius* sp)
Lorikeets (*Trichoglossus* sp)
Lovebirds (*Agapornis* sp)
Macaws (*Ara* sp)
Puerto Rican parrot (*Amazona vittata*)
Rosellas (*Platycercus* sp)
Yellow-naped parrot (*Amazona auropalliata*)
Perching birds (Order Passeriformes)
Finches (Family Fringillidae)
American goldfinch (*Carduelis tristis*)
Apapane (*Himatione sanguinea*)
Cassin's finch (*Carpodacus cassinii*)
Eurasian bullfinch (*Pyrrhula pyrrhula*)
Evening grosbeak (*Coccothraustes vespertinus*)
Goldfinches (*Carduelis* sp)
House finch (*Carpodacus mexicanus*)
Pine siskin (*Carduelis pinus*)
Purple finch (*Carpodacus purpureus*)
Wood-warblers, tanagers, grosbeaks, sparrows, and blackbirds (Family Emberizidae)
Brown-headed cowbird (*Molothrus ater*)

Chipping sparrow (*Spizella passerina*)
Common grackle (*Quiscalus quiscula*)
Dusky seaside sparrow (*Ammospiza nigrescens*)
Northern cardinal (*Cardinalis cardinalis*)
Orioles (*Icterus* sp)
Red-winged blackbird (*Agelaius phoeniceus*)
Rufous-sided towhee (*Pipilo erythrophthalmus*)
Chickadees ( Family Paridae)
Chestnut-backed chickadee (*Parus rufescens*)
Crows, jays, and magpies (Family Corvidae)
American Crow (*Corvus brachyrhychos*)
Blue jay (*Cyanocitta cristata*)
Eurasian jay (*Garrulus glandarius*)
Jackdaw (*Corvus monedula)*
Magpie (*Pica* sp)
Raven (*Corvus corax)*
Rook (*Corvus frugilegus*)
Steller's jay (*Cyanocitta stelleri*)
Waxwings (Family Bombycillidae)
Cedar waxwing (*Bombycilla cedrorum*)
Weavers (Family Ploceidae, *Ploceus* sp)
Old world sparrows (Passeridae)
House sparrow (English sparrow) (*Passer domesticus*)
Larks (Family Alaudidae)
Horned lark (*Eremophila alpestris*)
Mockingbirds and thrashers (Family Mimidae)
Northern mockingbird (*Mimus polyglottos*)
Thrashers (*Toxostoma* sp)
Nuthatches (Family Sittidae, *Sitta* sp)
Shrikes (Family Laniidae, *Lanius* sp)
Starlings (Family Sturnidae)
European starling (*Sturnus vulgaris*)
Mynas (*Acridotheres* sp*, Gracula* sp)
Swallows (Family Hirundinidae, *Hirundo* sp*, Tachycineta* sp*, Riparia* sp*, Stelgidopteryx* sp)
Martins (*Progne* sp)
Thrushes, solitaires, and bluebirds (Family Muscicapidae)
American robin *(Turdus migratorius*)
Bluebirds (*Sialia* sp)
Eurasian blackbird (*Turdus merula*)
Thrushes (*Turdus* sp, *Ixoreus* sp, *Hylocichla* sp, *Catharus* sp)
Estrildid finches (Family Estrildidae)
Java finch (*Padda oryzivora*)
Penguins (Order Sphenisciformes, Family Spheniscidae)
Blackfooted penguin (*Spheniscus demersus*)
Pigeons and doves (Order Columbiformes, Family Columbidae)
Band-tailed pigeon (*Columba fasciata*)
Mourning dove (*Zenaida macroura*)
Ringed turtle dove (*Streptopelia risoria*)
Rock dove (common pigeon) (*Columba livia*)
White-winged dove (*Zenaida asiatica*)
Wood pigeon (*Columba palumbus*)
Shorebirds (Order Charadriiformes)
Gulls, terns, skuas, and skimmers (Family Laridae)
California gull (*Larus californicus*)

Common tern (*Sterna hirundo*)
Franklin's gull (*Larus pipixcan*)
Glaucous-winged gull (*Larus glaucescens*)
Herring gull (*Larus argentatus*)
Kittiwakes (*Rissa* sp)
Laughing gull (*Larus atricilla*)
Least California tern (*Sterna antillarum*)
Ring-billed gull (*Larus delawarensis*)
Royal tern (*Sterna maxima*)
Skua (*Catharacta skua*)

Auks, murres, and puffins (Family Alcidae)
Common murre (*Uria aalge*)
Guillemots (*Cepphus* sp)
Murres (*Uria* sp)
Puffins (*Fratercula* sp)
Razorbill (*Alca torda*)

Plovers (Family Charadriidae, *Charadrius* sp*, Pluvialis* sp)

Sandpipers, turnstones, surfbirds, and phalaropes (Family Scolopacidae)
American woodcock (*Scolopax minor*)
Curlews (*Numenius* sp)
Long-billed dowitcher (*Limnodromus scolopaceus*)
Marbled godwit (*Limosa fedoa*)
Pectoral sandpiper (*Calidris melanotos*)
Ruddy turnstone (*Arenaria interpres*)
Semipalmated sandpiper (*Calidris pusilla*)
Spotted sandpiper (*Actitis macularia*)
Western sandpiper (*Calidris mauri*)

Stilts and avocets (Family Recurvirostridae)
American avocet (*Recurvirostra americana*)
Black stilt (*Himantopus novaezelandiae*)
Black-necked stilt (*Himantopus mexicanus*)

Waterfowl (Order Anseriformes, Family Anatidae)

Dabbling ducks
American wigeon (*Anas americana*)
American black duck (*Anas rubripes*)
Blue-winged teal (*Anas discors*)
Gadwall (*Anas strepera*)
Green-winged teal (*Anas crecca*)
Koloa duck (*Anas wyvilliana*)
Laysan duck (*Anas laysanensis*)
Mallard (*Anas platyrhynchos*)
Mottled duck (*Anas fulvigula*)
Muscovy duck (*Cairina moschata*)
Northern pintail (*Anas acuta*)
Northern shoveler (*Anas clypeata*)
White Pekin duck (*Anas platyrhynchos*)
Wood duck (*Aix sponsa*)

Diving ducks
Bufflehead (*Bucephala albeola*)
Canvasback (*Aythya valisineria*)
Common goldeneye (American goldeneye) (*Bucephala clangula*)
Greater scaup (*Aythya marila*)
Harlequin duck *(Histrionicus histrionicus*)
Lesser scaup (*Aythya affinis*)

Oldsquaw (*Clangula hyemalis*)
Redhead (*Aythya americana*)
Ring-necked duck (*Aythya collaris*)
Ruddy duck (*Oxyura jamaicensis*)

Sea ducks

Black scoter (common scoter) (*Melanitta nigra*)
Common eider (*Somateria mollissima*)
Common merganser (*Mergus merganser*)
Mergansers (*Mergus* sp, *Lophodytes* sp)
Red-breasted merganser *(Mergus serrator*)
Spectacled eider (*Somateria fischeri*)
Surf scoter (*Melanitta perspicillata*)
White-winged scoter (*Melanitta fusca*)

Geese (Tribe Anserini)

Aleutian Canada goose (*Branta canadensis leucopareia*)
Bean goose (*Anser fabalis*)
Black brant (*Branta bernicla nigricans*)
Brant (*Branta bernicla*)
Canada goose (*Branta canadensis*)
Hawiian goose (nene goose) (*Nesochen sandvicensis*)
Ross' goose (*Chen rossii*)
Snow goose (*Chen caerulescens*)
White-fronted goose (*Anser albifrons*)

Swans (Tribe Cygnini)

Bewick's swan (*Cygnus columbianus bewickii*)
Black swan (*Cygnus atratus*)
Mute swan (*Cygnus olor*)
Trumpeter swan (*Cygnus buccinator*)
Tundra swan (whistling swan) (*Cygnus columbianus*)
Whooper swan (*Cygnus cygnus*)

Whistling ducks (Tribe Dendrocygnini)

Fulvous whistling duck (*Dendrocygna bicolor*)

Woodpeckers (Order Piciformes, Family Picidae)

Red-cockaded woodpecker (*Picoides borealis*)

## Sources

American Ornithologists' Union. 1983. Check-list of North American birds, (6th ed.): Lawrence, Kansas, Allen Press, 877 p.

Howard, R. and Moore, A. 1991. A complete checklist of the birds of the world, (2nd ed.): San Diego, California, Academic Press, 622 p.

Monroe, B. L. Jr., and Sibley, C. G. 1993. A world checklist of birds: New Haven, Connecticutt, Yale University Press, 393 p.

# Appendix F

### Common and scientific names other than birds

Mammals

- African lion (*Panthera leo*)
- Bears (*Ursus americanus*; *U. arctos*; *U. maritimus*)
- Beaver (*Castor canadensis*)
- Bighorn sheep (*Ovis canadensis*)
- Bison (*Bison bison*)
- Bobcat (*Felis rufus*)
- Caribou (New World and Siberia) (*Rangifer tarandus*)
- Chipmunks (*Tamias striatus* and *Eutamias* sp.)
- Cottontail rabbit (*Sylvilagus floridanus*)
- Coyote (*Canis latrans*)
- Deer (*Odocoileus* sp.)
- Elk (*Cervus elaphus*)
- Foxes (*Vulpes* sp., *Urocyon* sp., and *Alopex lagopus*)
- Fur seal (*Callorhinus ursinus*)
- Jackrabbits (*Lepus* sp.)
- Lynx (*Felis lynx*)
- Mink (*Mustela vison*)
- Muskrat (*Ondatra zibethicus*)
- Nutria (*Myocaster coypus*)
- Pronghorn antelope (*Antilocapra americana*)
- Puma (Mountain lion) (*Felis concolor*)
- Raccoon (*Procyon lotor*)
- Rats (*Rattus* sp.)
- Reindeer (Old World and Greenland) (*Rangifer tarandus*)
- Sea lions (*Zalophus californianus* and *Eumetopias jubata*)
- Voles - small, mouse-like mammals primarily of the genera *Clethrionomys* and *Microtus*.
- Weasels (*Mustela* sp.)

Invertebrates

- Asian tiger mosquito (*Aedes albopictus*)
- Black widow spider (*Latrodectus mactans*)
- Caecal worm (*Heterakis gallinarum*)

Fish

- Rainbow trout (*Salmo gairdneri*)

Plants

- Choke cherry (*Prunus virginiana*)

# Appendix G

## Chemical names

| Common Names | Chemical Names |
|---|---|
| 1080 | Sodium monofluoracetate |
| 2-PAM | Pralidoxime chloride |
| Alachlor (Lasso®) | 2-Chloro-N-(2,6-diethylphenyl)-N-(methoxymethyl)acetamide |
| Aldicarb (Temik®) | 2-Methyl-2-(methylthio)propanal O-[(methylamino)carbonyl]oxime |
| Aldrin | 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-hexahydro-1,4:5,8-dimethanonaphthalene |
| Antimycin | 3-Methylbutanoic acid 3-[3-(formylamino)-2-hydroxybenzoyl]amino-8-hexyl-2,6-dimethyl-4,9-dioxo-1,5-dioxonan-7-yl ester |
| Aroclor® | Group of polychlorinated biphenyls |
| Atrazine | 6-Chloro-N-ethyl-N'-(1-methylethyl)-1,3,5-triazine-2,4-diamine |
| Avitrol® | 4-Aminopyridine |
| Bayluscide® | 5-Chloro-N-(2-chloro-4-nitrophenyl)-2-hydroxybenzamide compound respectively with 2-aminoethanol(1:1) |
| Beuthanasia-D-Special® | Sodium pentobarbital |
| Bomyl® | 3-[(Dimethoxyphosphinyl)oxy]-2-pentenedioic acid dimethyl ester |
| Brodifacoum (Talon®) | 3-[3-(4'-Bromo[1-1'-biphenyl]-4-yl)-1,2,3,4-tetrahydro-1-napthalenyl]-4-hydroxy-2H-1-benzopyran-2-one |
| Captan | 3a,4,7,7a-Tetrahydro-2- [(trichloromethyl)thio]-1H-isoindole-1,3(2H)-dione |
| Carbofuran | 2,3-Dihydro-2,2-dimethyl-7-benzofuranol methylcarbamate |
| Chlorophacinone | 2-[(4-Chlorophenyl)phenylacetyl]-1H-indene-1,3(2H)-dione |
| Chlorpyrifos | Phosphorothioic acid O,O-diethyl O-(3,5,6-trichloro-2-pyridinyl) ester |
| Chlordane | 1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methano-1H-indene |
| Clophens® | Group of polychlorinated biphenyls |
| Coumaphos | Phosphorothioic acid O-(3-chloro-4-methyl-2-oxo-2H-1-benzopyran-7-yl) O,O-diethyl ester |
| DDD | Dichlorodiphenyl dichloroethane |
| DDE | Dichlorodiphenyldichloroethylene |
| DDT | Dichloro diphenyl trichloroethane |
| Demeton (Systox®) | Phosphorothioic acid O,O-diethyl O-[2-(ethylthio)ethyl] ester mixture with O,O- diethyl S-[2-(ethylthio)ethyl]phosphorothioate |
| Diazinon | Phosphorothioic acid O,O-diethyl O-[6-methyl-2-(1-methylethyl)-4-pyrimidinyl] ester |
| Dicrotophos | Phosphoric acid 3-(dimethylamino)-1-methyl-3-oxo-1-propenyl dimethyl ester |
| Dieldrin | (1aα,2β,2aα,3β,6β,6aα,7β,7aα)-3,4,5,6,9,9-Hexachloro-1a,2,2a,3,6,6a,7,7a-octahydro-2,7:3,6-dimethanonaphth[2,3-b]oxirene |

| Common Names | Chemical Names |
|---|---|
| Dimethoate | Phosphorodithioic acid O,O-dimethyl S-[2-(methylamino)-2-oxoethyl] ester |
| Diphacinone | 1,3-Inandione, 2-diphenylacetyl |
| Diquat® | Dipyrido(1,2-a:2',1'-c)pyrazinediium, 6,7-dihydro |
| Disulfoton | Phosphorodithioic acid O,O-diethyl S-[2-(ethylthio)ethyl] ester |
| EDTA | Ethylenediaminetetraacetic acid |
| Endosulfan | 6,7,8,9,10,10-Hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzodioxathiepin 3-oxide |
| Endrin | (1aα,2β,2aβ,3α,6α,6aβ,7β,7aα)-3,4,5,6,9,9-Hexachloro-1a,2,2a,3,6,6a,7,7a-octahydro-2,7:3,6-dimethanonaphth[2,3-b]oxirene |
| Enflurane | 2-Chloro-1-(difluoromethoxy)-1,1,2- trifluoroethane |
| Famphur | Phosphorothioic acid O-[4-[(dimethylamino)sulfonyl]phenyl] O,O-dimethyl ester |
| Fenamiphos | (1-Methylethyl)phosphoramidic acid ethyl 3-methyl-4-(methylthio)phenyl ester |
| Fenbendazole | [5-(Phenylthio)-1H-benzimidazol-2-yl]carbamic acid methyl ester |
| Fenclors® | Group of polychlorinated biphenyls |
| Fensulfothion | Phosphorothioic acid O,O-diethyl O-[4-methylsulfinyl)phenyl] ester |
| Fenthion | Phosphorothioic acid O,O-dimethyl O-[3-methyl-4-(methylthio)phenyl ester |
| Fonofos | Ethylphosphonodithioic acid O-ethyl S-phenyl ester |
| Formalin | Formaldehyde solution |
| Halothane | 2-bromo-2-chloro-1,1,1-trifluoroethane |
| HCN | hydrogen cyanide gas |
| Heptachlor | 1H-1,4,5,6,7,8,8-Heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene |
| Hexaconazole | (RS)-2-(2,4-dichlorophenyl)-1-(IH-1,2,4,-triazol-1-yl)-hexan-2-ol |
| Hexane | $C_6H_{14}$ |
| Isazophos (Triumph®) | Phosphorothioic acid O-[5-chloro-1-(1-methylethyl)-1H-1,2,4-triazol-3-yl] O,O-diethyl ester |
| Isoflurane | 2-Chloro-2-(difluoromethoxy)-1,1,1-trifluoroethane |
| Ivermectin | 22,23-Dihydroabamectin |
| Kanechlors® | Group of polychlorinated biphenyls |
| Lindane | (1α,2α,3β,4α,5α,5β)-1,2,3,4,5,6-Hexachlorocyclohexane |
| Methamidophos | Phosphoramidothioic acid O,S-dimethyl ester |
| Methiocarb | 3,5-Dimethyl-4-(methylthio)phenyl methylcarbamate |
| Methoxyflurane | 2,2-Dichloro-1,1-difluoro-1-methoxyethane |
| Mirex | 1,1a,2,2,3,3a,4,5,5,5a,5b,6-Dodecachlorooctahydro-1,3,4-metheno-1H-cyclobuta[cd]pentalene |
| Monocrotophos | (E)-Phosphoric acid dimethyl [1-methyl-3-(methylamino)-3-oxo-1-propenyl] ester |
| Nitrapyrin | 2-Chloro-6-(trichloromethyl)pyridine |
| OC | Organochlorine |
| OP | Organophosphate |

| Common Names | Chemical Names |
|---|---|
| Oxamyl | 2-(Dimethylamino)-N-[[(methylamino)-carbonyl]oxy]-2-oxoethanimidothioic acid methyl ester |
| PAH | Polycyclic aromatic hydrocarbon |
| Parathion | Phosphorothioic acid O,O-diethyl O-(4-nitrophenyl) ester |
| PCB | Polychlorinated biphenyl |
| Permethrin | 3-(2,2-Dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid (3-phenoxyphenyl)methyl ester |
| Phenoclors® | Group of polychlorinated biphenyls |
| Phenol | $C_6H_6O$ |
| Phorate | Phosphorodithioic acid O,O-diethyl S- [(ethylthio)methyl] ester |
| Phosmet | Phosphorodithioic acid S-[(1,3-dihydro-1,3- dioxo-2H-isoindol-2-yl)methyl] O,O-dimethyl ester |
| Phosphamidon | Phosphoric acid 2-chloro-3-(diethylamino)-1- methyl-3-oxo-1-propenyl dimethyl ester |
| Pyralenes® | Group of polychlorinated biphenyls |
| Rotenone | [2R-(2α,6aα,12aα)]-1,2,12,12a-Tetrahydro-8,9-dimethoxy-2-(1-methylethenyl)[1]benzopyrano[3,4-b]furo[2,3-h]benzopyran-6(6aH)-one |
| Sleepaway® | Sodium pentobarbital |
| Starlicide® | 3-Chloro-p-toluidine hydrochloride |
| Strychnine | $C_{21}H_{22}N_2O_2$ |
| Terbufos (Counter®) | Phosphorodithioic acid S-[[(1,1-dimethylethyl)thio]methyl] O,O-diethyl ester |
| Thiabendazole | 2-(4-Thiazoyl)-1H-benzimidazole |
| Thiram | Tetramethylthioperoxydicarbonic diamide |
| Toxaphene | Chlorinated camphene |
| Warfarin | 4-Hydroxy-3-(3-oxo-1-phenylbutyl)-2H-1-benzopyran-2-one |
| Zectran® | 4-(Dimethylamino)-3,5-dimethylphenol methylcarbamate (ester) |
| Ziram | (T-4)-Bis(dimethylcarbamodithioato-S,-S$^1$)zinc |

## Sources


Howard, P.H., and Neal, M., 1992, Distionary of chemical names and synonyms: Boca Raton, Florida, Lewis Publishers, 1,394 p.

Budavari, S., O'neil, M.J., Smith, A., and others, (eds.),1996, The Merck index, 12th ed.): Whitehouse Station, New Jersey, Merck and Co., 1,741 p.

Meister, R.T. (ed.), 1996, Farm chemicals handbook '96: Willoughby, Ohio, Meister Publishing Co., 446 p.

# Appendix H

## Conversion table

The units of measurement that are used by laboratories for recording the results of scientific or diagnostic tests are reported in this Manual in the metric or SI units (from the French “Le System International d’Unites”) that are customary for those results. Other units of measurement, such as those that are used in the field for reporting area or length, are reported in the inch-pound units that are common in the United States. The lists of conversion factors and abbreviations below are for those who are interested in converting the basic units of measurement that are used in this Manual to the system of choice. For temperature, use the equations provided.

**To convert SI to inch-pound**

| *Multiply* | *By* | *To obtain* |
|---|---|---|
| Length | | |
| millimeters (mm) | 0.039 | inch (in.) |
| centimeters (cm) | 0.39 | inch (in.) |
| meter (m) | 3.28 | foot (ft) |
| meter (m) | 1.09 | yard (yd) |
| kilometer (km) | 0.62 | mile (mi) |
| Weight | | |
| kilogram (kg) | 2.21 | pound (lb) |
| metric ton | 1.10 | short ton |
| Volume or capacity | | |
| milliliters (mL) | 0.03 | fluid ounce (fl. oz) |
| microliters (µL) | 0.003 | fluid ounce (fl. oz) |

Temperature

°C, degrees Celsius, to °F, degrees Fahrenheit 9/5 x °C + 32 = °F

**To convert inch-pound to SI**

| *Multiply* | *By* | *To obtain* |
|---|---|---|
| Length | | |
| inch (in.) | 25.4 | millimeters (mm) |
| foot (ft) | 2.54 | centimeters (cm) |
| mile (mi) | 0.3048 | kilometer (km) |
| Area | | |
| acre | 4,047 | square meters ($m^2$) |
| Weight | | |
| pound (lb) | 0.4356 | kilogram (kg) |
| short ton | 0.907 | metric ton |

Temperature

°F, degrees Fahrenheit, to °C, degrees Celsius

(°F - 32) X 5/9 = °C

**Other abbreviated units of measurement used in this Manual:**

cc cubic centimeter, a unit of measurement that indicates the dosage of a drug or substance that is administered intravenously or by injection.

µg/g micrograms per gram

mg/kg milligrams per kilogram

**Conversion table for units of measurement that are frequently used with the toxicology of pesticides:**

| Concentration of compound in tissue, food, or water, in SI units | Concentration of compound in tissue, food, or water, in parts per million (ppm) |
|---|---|
| 1 nanogram per gram (1 ng/g) | 0.001 |
| 1 microgram per 100 grams (1 µg/100 g) | 0.01 |
| 1 milligram per kilogram (1 mg/kg) | 1 |
| 1 microgram per gram (1 µg/g) | 1 |
| 1 microgram per 100 milligrams (1 µg/100 mg) | 10 |
| 1 milligram per 100 grams (1 mg/100 g) | 10 |
| 1 milligram per gram (1 mg/g) | 1,000 |

| Concentration of compound in air, in SI units | Concentration of compound in air, in parts per million (ppm) |
|---|---|
| 1 microgram per liter (1 µg/L) | 1 |

# Glossary

**Abdomen** — the portion of the body that lies between the thorax and the pelvis.

**Abdominal cavity** — the space that contains the abdominal viscera (the liver, spleen, intestines, etc.).

**Abdominal wall** — the layers of muscles lying between the skin and the abdominal cavity.

**Absorption** — to take in a substance through the pores or cells of a tissue. The substance must pass through the tissue to be absorbed.

**Acanthocephalans** — cylindrical, unsegmented worms that attach to the host by a retractable proboscis with sharp hooks.

**Acaracides** – substances, such as pesticides, that kill mites.

**Acariasis** — infestation of the body by mites.

**Accipiters** — short winged, long-tailed hawks; North American species are goshawk, Cooper's hawk, and sharp-shinned hawk.

**Acute** — sharp or severe, such as an illness with a sudden onset and a relatively short course.

**Air sacs** — thin-walled sacs that communicate with the lungs and are part of the avian respiratory system.

**Airsacculitis** — inflammation of the air sacs in birds.

**Alcids** — typically, pelagic colonial nesting seabirds, including species such as auklets, guillemots, murres, murrelets, and puffins.

**Algae** — a special form of plant life that lacks true roots, stems, or leaves, and that ranges in size from microscopic single cells to multicellular structures, such as seaweeds.

**Alimentary canal** — the digestive tract.

**Allergic disease** — development of a hypersensitivity of the host to substances foreign to the body, primarily antigens and other proteins.

**Altricial** — refers to newly hatched birds that require care in the nest for some period of time.

**Ambient temperature** — room or environmental temperature.

**Amino acids** — organic compounds of specific composition from which proteins are synthesized.

**Amphibians** — coldblooded animals characterized by moist, smooth skin that live both on land and in water at various life stages and that have gills at some stage of development, that is, frogs, toads, salamanders.

**Amplification host** — a host in which disease agents, such as viruses, increase in number.

**Amyloid deposit** — a complex protein material that grossly resembles starch and that in certain abnormal conditions accumulates in various body tissues causing cellular damage and injury to the affected organ.

**Anaerobic** — absence of oxygen; often refers to an organism that grows, lives, or is found in an environment devoid of oxygen, such as the cellular form of `Clostridium botulinum`, which causes avian botulism.

**Analgesia** — the absence of normal sensitivity to pain, typically, being in a semiconscious state induced through an anesthetic.

**Anemia** — a reduction in the normal number of red blood cells, or erythrocytes, in the body.

**Anesthetic** — a drug used to temporarily deaden pain.

**Anesthetic induction time** — the time between administering an anesthetic chemical and the actual time when target nerves are deadened.

**Animal pathogens** — organisms such as viruses, bacteria, and parasites, that are capable of invading and infecting animal hosts and causing disease.

**Annelids** — a group of invertebrates characterized by the segmented worms, including those in marine and freshwater and earthworms in addition to leeches.

**Anorexia** — lack of appetite.

**Anoxia** — a total lack of oxygen caused by several mechanisms that prevent oxygen from reaching the mitochondria of cells. Anoxia indicates a level of oxygen in animal tissues that is below normal in the presence of an adequate blood supply.

**Antibody** — a specialized serum protein produced by the immune system in response to an antigen in an attempt to counteract the effects of the antigen; antibodies in the blood indicate exposure to specific antigens or disease agents.

**Antidote** — substances that counteract or prevent the action of a poison.

**Antigen** — any foreign substance (generally proteins) to which the body reacts by producing antibodies. Antigens may be soluble substances such as toxins, particulate matter such as pollen, or microorganisms such as bacteria and viruses.

**Antiserum** — a serum containing antibodies to specific antigens; can be used to test biological samples for the presence of specific antigens.

**Antitoxin** — any substance that counteracts the action of a toxin or poison; generally, a specific type of antibody produced in experimental animals as a result of exposure to a specific toxin. Botulism antitoxin, for example, can be produced by exposing an animal to low levels of botulism toxin over a long period of time and then harvesting serum from that animal to treat other animals.

**Arbovirus** — a virus that is transmitted by invertebrates of the phylum Arthropoda [insects, arachnids (spiders, mites, ticks, etc.) and crustaceans].

**Arthropod** — members of the phylum Arthropoda (insects, arachnids, and crustaceans).

**Ascites** — accumulation of fluid in the abdominal cavity.

**Aseptic** — free from infection; sterile.

**Asexual reproduction** — the formation of new individuals without the union with cells of the opposite sex and usually by an individual.

**Asymptomatic** — without visible signs of illness; an asymptomatic carrier is an organism that harbors a disease agent, but that shows no outward signs.

**Ataxia** — incoordination.

**Avicides** — chemical substances used to kill or repel birds.

**Avirulent** — not virulent, does not cause disease.

**Bacterin** — a vaccine consisting of killed bacteria that is used for protection against infection by a specific bacterial disease.

**Bacteriophage** — a virus that infects a bacterium.

**Bacterium** — singular for bacteria. Any of a group of microscopic, unicellular organisms that have distinct cell membranes and that lack a distinct nucleus surrounded by a nuclear membrane.

**Barbiturate** — a type of sedative or anesthetic that is chemically derived from barbituric acid.

**Bay diving ducks** — typically, ducks that feed in deep bodies of water, usually in coastal bays and deep lakes. Species include canvasback, goldeneyes, redhead, and scaup.

**Benign** — noninvasive, that is, tumors that do not spread to other parts of the body; not malignant.

**Big game** — hunted species of large mammals (from deer to elephants).

**Bile** — yellow-brown to greenish liquid secreted by the liver and stored in the gallbladder before excretion by way of the intestine. Bile is composed of metabolic breakdown products derived from hemoglobin and other metabolic waste products.

**Bioaccumulation** — the accumulation of long-lived toxins, such as chlorinated hydrocarbons, as a result of repeated exposure or of exposure from a variety of sources.

**Biomagnification** — an increase in concentrations of long-lived contaminants in animals at higher positions in the food chain.

**Biota** — the plant and animal life of an area.

**Biotoxins** — poisons produced by and derived from the cells or secretions of a living organism, either plant or animal.

**Birds of prey** — synonymous with raptors; includes eagles, hawks, falcons, kites, and owls.

**Biting louse** — see Hippobascid flies.

**Black flies** — small, bloodsucking, biting flies of the genus *Simulium*; vectors for *Leucocytozoon* infections.

**Blood flukes** — trematode parasites that are found in the blood cells of the host.

**Brine flies** — species of flies whose larvae live in brine.

**Brooding** — care of young birds by the adult.

**Buffered formalin** — a 3.7 percent solution of formaldehyde (equal to 10 percent formalin) to which sodium phosphate buffers have been added. Buffered formalin is the best overall fixative for tissue for later microscopic study.

**Bumblefoot** — an inflammation and, often, swelling of the foot of birds as the result of a bacterial infection.

**Bursa of Fabricious** — a saclike outgrowth of the cloaca of birds that is part of the avian immune system.

**Buteos** — a subfamily of the hawks characterized by soaring behavior, broad, rounded wings, and a broad, fanned tail, such as the red-tailed hawk.

**Caecum** (British; plural caeca) or cecum (American; plural ceca) — a large, blind pouch or sac (often a pair) at the junction of the small intestine and the large intestine.

**Calcification** — the process by which tissues become hardened by the deposition of calcium salts.

**Canidia** — fungal spores.

**Canker** — synonymous with trichomoniasis in doves and pigeons.

**Capture myopathy** — a state of immobility resulting from damage to skeletal and cardiac muscles caused by extreme physical exertion, struggle, or stress; may occur in wildlife as they are chased in capture attempts; may appear later when captured wildlife are under physical restraint; or may appear after they have been released.

**Cardiac muscle** — heart muscle.

**Cardiovascular system** — the heart and blood vessels by which blood is pumped and circulated through the body.

**Carnivores** — refers to flesheating mammals in the Order Carnivora and includes dogs, skunks, weasels, cats, raccoons, etc.

**Carrion** — dead and decaying flesh.

**Caseous** — resembling cheese or curd.

**Central nervous system** — the brain and spinal cord.

**Ceratopogonid flies** — very small, bloodsucking gnats commonly known as punkies, no-see-ums, or sand flies.

**Cercaria** — the final free-swimming larval stage of a trematode parasite.

**Cestodes** — flattened, usually segmented, parasitic worms; tapeworms.

**Chelating chemical** — a chemical that combines with a metal ion in a firm, ringlike band and that prevents the metallic ion from having any further biochemical effect.

**Chlorinated hydrocarbons** — organic compounds characterized by the presence of chlorine; commonly refers to persistent chemicals with insecticidal properties; DDT and dieldrin are common examples.

**Choana** — one of the paired openings on the inner side of the maxilla (upper beak), near the back of the oral cavity, that opens into the nasal cavity.

**Cholinesterase enzymes** — enzymes that are particularly important in the transmission of nerve impulses; the activity of these enzymes is inhibited by exposure to organophosphorus and carbamate compounds, and death results when activity is greatly reduced.

**Chronic** — persisting for a relatively long time.

**Chronic losses** — mortality of attrition; small numbers of continual losses over extended periods of time.

**Clinical sign** — an abnormal physiological change or behavior pattern that is indicative of illness. Signs are externally observable, as contrasted with symptoms, which are subjective.

**Cloaca** — a common passage for the fecal, urinary, and reproductive discharges of most lower vertebrates (birds, reptiles, and amphibians).

**Coalescence** — the fusion or growing together of tissue damage from a disease agent.

**Coldblooded vertebrates** — species such as fishes and reptiles, which have blood that varies in temperature to approximately that of the surrounding environment.

**Colibacillosis** — infection with the bacterium *Escherichia coli*.

**Colon** — the large intestine.

**Colonial nesters** — birds that nest in large groups.

**Comatose** — in a coma or comalike state; an abnormal state of continuous deep unconsciousness.

**Congener** — a member of the same taxonomic grouping, such as polychlorinated biphenyls, that possess similar chemical structures.

**Congenital abnormality** — usually an anatomical malformation that results from incomplete growth during embryonic development. Also refers to an abnormal biochemical pathway caused by a genetic factor.

**Congestion** — the abnormal accumulation of blood in a tissue or organ; often causes a reddening of the affected area.

**Contagious** — capable of being transmitted from animal to animal, such as a contagious disease.

**Coccidiasis** — the presence of coccidia, protozoa of the subphylum Sporozoa.

**Coccidiosis** — a disease caused by coccidia, protozoa of the subphylum Sporozoa.

**Cornea** — the transparent tissue on the front of the eyeball that covers the iris and pupil, through which light passes to the interior.

**Coronary band** — a fatty band encircling the heart; in hooved animals, the germinal layer beneath skin at the junction of the skin and hoof.

**Cracker shell** — a shotgun shell that is loaded to produce a visible burst and loud sound in order to frighten animals.

**Crop** — a dilation of the esophagus at the base of the neck of some birds.

**Crustacea** — a specialized group of invertebrates that includes such diverse species as lobster, shrimp, barnacles, wood lice, and water fleas.

**Cygnet** — a young swan.

**Cyanobacteria** — a genus of bacteria composed of the blue-green algae; like the dinoflagellates, cyanobacteria are important sources of environmental toxins that can cause illness and death in humans and wildlife.

**Cyanosis** — a bluish discoloration of the skin and mucous membranes due to an excessive concentration of deoxygenated hemoglobin in the blood.

**Cystocanth** — an infective juvenile stage of thorny-headed worms (acanthocephalan parasites).

**Cytoplasm** — the aqueous part of the cell that is outside of the nucleus but that is contained within the cell wall. The cytoplasm is the site of most of the chemical activities of the cell.

**Dabbling ducks** — Ducks that feed on the surface or in shallow water, including mallard, American black duck, gadwall, American wigeon, northern pintail, northern shoveler, and teal. Also referred to as puddle ducks.

**Definitive host** — an organism in which sexually mature stages of a parasite occur.

**Dehydration** — a condition that results from excessive loss of body fluids.

**Depopulation** — the destruction of an exposed or infected group of animals.

**Dermatophytosis** — a fungal infection of the skin.

**Dessication** — the act or process of drying a substance.

**Digestive tract (alimentary canal)** — the organs associated with the ingestion, digestion, and absorption of food, such as the esophagus, stomach, and intestines.

**Dinoflagellates** — aquatic protozoa that are an important component of plankton. These single-celled organisms may be present in vast numbers, causing discoloration of the water referred to as "red tide." Some species secrete powerful neurotoxins.

**Dioxins** — a chemical component of defoliants, such as agent orange, that are considered to be carcinogenic (cause cancer), teratogenic (cause fetal abnormalities), and mutagenic (cause abnormal mutation rate).

**Direct life cycle** — a parasitic life cycle that requires only a single host for its completion.

**Diurnal** — active during the day.

**Diving ducks** — synonymous with bay diving ducks.

**Domestic duck** — ducks typically raised for market, such as the white Pekin.

**Drive-trapping** — capture of flightless birds during the molt and of other animals by herding them into a netted or fenced containment area.

**Drop nets** — suspended nets used to capture animals by remote release of the nets or triggering mechanisms at the net site.

**Dyspnea** — labored breathing.

**Ecchymotic** — a hemorrhagic, irregular-shaped area in tissues that is bruise-like in appearance and, often, in color.

**Ecology** — the study of the interrelationships between living organisms and their environment.

**Ectoparasite** — a parasite that lives on the external surface, or in the integument, of its host.

**Ectotherms** — species that rely on sources of heat outside themselves (i.e., coldblooded species).

**Edematous** — swelling of tissues due to abnormal accumulation of fluid in the intercellular tissue spaces; seepage of these fluids may result in accumulations within the body cavity.

**EDTA** — ethylenediamine tetra-acetic acid; a chelating agent that binds with lead and that is used in the treatment of lead poisoning.

**EEE** — eastern equine encephalomyelitis; a viral disease.

**ELISA** — a molecular-based enzyme-linked immonosorbent assay; a type of test used to detect either antigen or antibody.

**Emaciation** — a wasted condition of the body; excessive leanness.

**Emasculatome** — a veterinary instrument designed for bloodless castration of cattle or sheep; has been used for euthanasia of birds by cervical dislocation.

**Encrustation** — forming a crust or a covering; for example, salt encrustation.

**Endemic** — a disease that commonly is present within a population or a geographical area.

**Endogenous phase** — developmental phase of the life cycle of a parasite that occurs within the host.

**Endoparasite** — a parasite that lives within the body of its host.

**Endotherms** — warmblooded vertebrates; species able to internally regulate their body temperatures.

**Enteritis** — inflammation of the intestine.

**Enzootic** — an animal disease that commonly is present within a population or geographical area.

**Epicardium** — the outer covering of the heart.

**Epidemic** — the presence of a disease in a population or in an area in a higher than expected prevalence, or rate.

**Epithelial cell**s — cells that cover the external and internal surfaces of the body.

**Epizootic** — a disease affecting a greater number of animals than normal; typically, occurrences involving many animals in the same region at the same time.

**Epizootiology** — the study of the natural history of disease in animal populations.

**Erosion** — wearing away; gradual disintegration.

**Erythrocytes** — red blood cells; serve to transport oxygen throughout the body.

**Esophagus** — the passage extending from the mouth to the stomach.

**Estrogenic** — possessing characteristics of the hormone estrogen; estrogenic compounds may elicit the development of feminine characteristics in male animals.

**Etiologic agent** — any living or nonliving thing, power, or substance capable of causing a disease.

**Eutrophication** — the excessive growth, caused by an oversupply of nutrients, of plants and algae in bodies of water.

**Exotic disease** — a disease that normally does not occur within a particular area.

**Exotoxin** — a toxin formed and excreted by bacterial cells.

**Exsanguination (bleeding out)** — the draining of blood from an animal.

**Fastidious** — refers to the very specific requirements for the culture of some bacteria.

**Fauna** — the animals of an area.

**"Feather edge"** — a long, shallow edge of a body of water that gradually deepens offshore.

**Femur** — the thigh bone of humans; the upper legbone in hooved mammals and birds. The bone between the pelvis and the knee.

**Feral pigeon** — rock dove.

**Fibrin** — an insoluble protein that forms a network of fibers during clotting of the blood.

**Fibrinoperitonitis** — fibrin-coated inflammation of the surfaces of the peritoneal cavity.

**Fibrinous** — a pathologic term referring to a threadlike sheet of material that may occur on surfaces of organs in some disease conditions; clotting factors in blood contribute to the structure of this material.

**Flatworms** — the common name for parasites of the phylum Platyhelminthes, flukes or trematodes.

**Flukes** — parasitic flatworms; also referred to as trematodes.

**Flylarvae** — maggots.

**Fomite** — an object that is not in itself harmful, such as a wooden object or article of clothing, but that may harbor pathogenic microorganisms and serve to transmit an infection to a living organism.

**Food chain** — ascending trophic levels within an ecosystem in which species at the lower level are the primary food base for the species at the next highest level.

**Formalin** — a liquid solution of formaldehyde that is used as a tissue fixative, usually to prepare tissues for microscopic examination.

**Fossorial** — refers to digging animals that live in burrows.

**Frounce** — synonymous with trichomoniasis in raptors.

**Fungicides** — chemicals that kill fungi.

**Gallinaceous birds** — heavy-bodied, chickenlike land birds. Includes ring-necked pheasant, quails, grouse, and wild turkey.

**Gamete** — one of two cells produced by a gametocyte; the union of male and female gametes initiates the development of a new individual during sexual reproduction

**Gametocyte** — an undifferentiated cell that develops into a gamete.

**Gangrene** — tissue death due to a failure of the blood supply to that tissue area followed by bacterial invasion and putrefication.

**Gapes** — see gapeworm.

**Gapeworm** — parasites of the trachea of birds; synonym for tracheal worms.

**Gastrointestinal tract** — the tubular organs that form a digestive pathway from the mouth to the vent, including the stomach and intestines.

**Geographic information system** — a specialized computer system for storage, manipulation, and presentation of layers of geographical information.

**Gizzard** — the enlarged muscular ventriculus (stomach) of many birds.

**Granuloma** — refers to a tumorlike mass or nodule; often associated with a response to an infection.

**Haemoproteus** — blood parasites transmitted by louse flies of the family Hippoboscidae and midges of the family Ceratopogonidae.

**Hatchet-breast** — a common term to describe the prominent, protruding breast keel seen as the result of the atrophy of the breast muscles. "The keel appears as sharp and as prominent as the back of a hatchet."

**Hawaiian forest birds** — native and introduced avifauna of the forested areas of the Hawaiian Islands. Includes such species as sparrows, finches, cardinals, honeycreepers, and thrushes.

**Helminths** — parasitic worms.

**Hemosporidia** — protozoan blood parasites.

**Hemoglobin** — the oxygen-carrying pigment of red blood cells.

**Hemozoin** — a dark pigment produced from the hemoglobin in the host's red blood cells by malarial parasites that collect in tissues, such as the spleen and liver, causing those organs to appear grayish to dark brown or black.

**Hepatitis** — inflammation of the liver.

**Hepatomegaly** — enlargement of the liver.

**Herbicides** — chemicals used to kill unwanted vegetation.

**Hermaphroditic** — organisms that possess both male and female functional reproductive organs.

**Herpesvirus** — one of the major groups of related viruses that have DNA nucleic acids and that are further characterized by similar size, shape, and physiochemical reactions.

**Herpetologists** — those who study the natural history and biology of reptiles.

**Heterogenous organism** — one that is derived from a combination of different types of parent organisms.

**Hippoboscid flies** — a group of wingless and winged parasitic flies found on birds and mammals.

**Histoplasmosis** — a disease of humans caused by inhalation of the fungus *Histoplasma capsulatum*.

**History** — as it refers to wildlife disease investigations, a record of background information and chronological events associated with a die-off.

**Homeostasis** — the tendency toward equilibrium; refers to the capacity of living organisms to maintain internal body environmental conditions necessary for survival.

**Husbandry practice** — the care and maintenance of animals.

**Hydropericardium** — an excessive amount of fluid within the sac surrounding the heart.

**Hypersensitivity** — greater than normal sensitivity to stimuli or to biological agents.

**Hypothermia** — greatly reduced body temperature.

**Hypovalemic shock** — shock resulting from insufficient blood volume to maintain adequate cardiac output and blood pressure; caused by acute hemorrhage or excessive fluid loss.

**Icthyologists** — those who study the natural history and biology of fishes.

**Immune** — being resistant to a disease.

**Immunosuppressive therapy** — a medical treatment that suppresses the normal immune response.

**Impaction** — an abnormal accumulation of food or other ingested materials that become lodged in a section of the digestive tract.

**Immune system** — the combination of host body defenses that guard against infectious disease.

**Inapparent** — an infection in which the infectious agent exists within the host but that causes no recognizable signs of illness; the infectious agent may or may not be shed at irregular times.

**Incidence** — the number of new cases of a disease occurring in a population within a certain time period.

**Inclusion body** — a structure within the cytoplasm or nucleus of a cell; a characteristic of some viral diseases, inclussion bodies occur in only a few species.

**Incubation period** — the time interval required for the development of disease; the time between the invasion of the body by a disease agent and the appearance of the first clinical signs.

**Indigenous** — native to a particular area.

**Indirect life cycle** — a life cycle that requires more than one host for its completion.

**Infection** — the invasion and multiplication of an infectious agent in host body tissues.

**Infectious agent** — a living organism capable of invading another.

**Infective** — capable of producing infection.

**Infestation** — parasitic invasion of external surfaces of a host.

**Insecticides** — pesticides used to kill insects.

**Intermediate host** — an organism in which a parasite undergoes a stage of asexual development.

**Intracellular parasite** — a parasitic organism, usually microscopic, that lives within the cells of the host animal.

**Involuntary muscle** — muscle that is not under the control of the individual.

**Isolate** — refers to microorganisms; the separation of a population of organisms that occur in a particular sample (verb); for example, to isolate a bacterial or viral organism from a sample. As a noun, refers to the organism that was isolated; for example, a bacterial isolate was obtained from a sample.

**Isopods** — crustaceans with flattened bodies, such as sowbugs, pillbugs, and wood lice.

**Joint capsule** — the thick, fibrous capsule surrounding a joint, as around the knee.

**Keel** — the narrow middle portion of a bird's sternum.

**Kites** — hawk-like birds.

**Lacrimal discharge** — a discharge from the tear glands near the eye.

**Laparotomy** — a surgical procedure in which an incision is made into the abdominal cavity, often to determine the sex of birds for which plumage and other characteristics cannot be used for that purpose.

**Larva** — an immature parasitic life cycle stage; typically, the form of the parasite is unlike the mature stage.

**Larynx** — the musculocartilaginous structure at the upper part of the trachea; it guards the entrance to the trachea and secondarily serves as the organ of voice.

**Latent** — dormant or concealed; a latent infection refers to the situation in which a disease condition is not apparent.

**Lentogenic** — refers to a form of Newcastle disease virus that is mildly virulent as measured in chickens.

**Leucocytozoon** — blood parasites transmitted by black flies of the family Simulidae.

**Lesion** — an abnormal change in tissue or an organ due to disease or injury.

**Lethargy** — abnormal drowsiness or stupor.

**Lousefly** — see Hippoboscid flies.

**Lyme disease** — an infectious disease that is caused by the spirochete *Borrelia burgdorferi* and transmitted by ticks.

**Lipophilic** — having an affinity for fat; such as chemicals that accumulate in fat and fatty tissues.

**Livestock** — domestic animals raised for food and fiber commonly refers to animals such as hogs, sheep, cattle, and horses.

**M-44** — a predator-control device that uses cyanide as the toxic component.

**Macrocyst** — a large cyst; a large spore case (fungi); an encapsulated reproductive cell of some slime molds.

**Macrogamete** — the female sexual form of the malaria parasite that is found in the gut of the mosquito vector.

**Maggot** — a soft-bodied larva of an insect, especially a form that lives in decaying flesh.

**Malarias** — infectious diseases caused by protozoan parasites that attack the red blood cells.

**Malignant** — spread from the location of origin to other areas; that is, tumors that are invasive and that spread throughout the body.

**Marek's disease** — an important infectious disease of poultry, that is caused by infection with a herpesvirus.

**Marine birds** — birds of the open ocean, typically pelagic, and often colonial nesters, such as alcids, shearwaters, storm petrels, gannets, boobies, and frigatebirds.

**Meningoencephalitis** — inflammation of the transparent covering (meninges) of the brain.

**Mergansers** — a group of waterfowl that are commonly referred to as "fish ducks" due to their food habits.

**Meront** — an asexual stage in the development of some protozoan parasites that gives rise to merozoites.

**Merozoite** — a stage in the life cycle of some protozoan parasites.

**Mesogenic** — refers to a form of Newcastle disease virus that is moderately virulent as measured in chickens.

**Metabolic rate** — an expression of the rate at which oxygen is used by cells of the body.

**Metacercaria** — the encysted resting or maturing stage of a trematode (fluke) parasite in the tissues of an intermediate host.

**Microgamete** — the male sexual form of the malaria parasite found in the gut of the mosquito vector.

**Migratory birds** — all birds listed under the provisions of the Migratory Bird Treaty Act.

**Minamata disease** — mercury poisoning of humans; named after an incident resulting from contamination within Minamata Bay, Japan.

**Miracidium** — the first larval stage of a trematode parasite, which undergoes further development in the body of a snail.

**Mobilization** — refers to the tendency of lipophilic chemicals [environmental contaminants, such as chlorinated hydrocarbons, that have an affinity for storage in adipose (fat) tissue] to be released into the bloodstream as fat stores are depleted.

**Mollusks** — species of the phylum Mollusca; includes snails, slugs, mussels, oysters, clams, octopuses, nautiluses, squids, and similar species.

**Molt** — the normal shedding of hair, horns, feathers, and external skin before replacement by new growth.

**Moribund** — a visible, debilitated state resulting from disease; appearing to be suffering from disease and close to death.

**Motility/motile/nonmotile** — these terms refer to whether or not a bacterial organism moves on a particular culture medium; such movement reflects the presence of flagellae. Thus, the absence or presence of motility is a useful characteristic for identifying bacteria.

**Motor paralysis** — paralysis of the voluntary muscles.

**Mucosa** — a mucous membrane.

**Mucous membrane** — the layer of tissue that lines a cavity or the intestinal tract and that secretes a mixture of salts, sloughed cells, white blood cells, and proteins.

**Mucosal surface** — a layer of cells lining the inside of the intestinal tract or other body part that secretes mucus.

**Myocarditis** — inflammation of heart muscle.

**Myocardium** — the middle and thickest layer of the heart wall; composed of cardiac muscle.

**Mycosis** — fungal infection.

**Mycotoxin** — a poison produced by various species of molds (fungi).

**Myiasis** — infestation of the body by fly maggots.

**Nares** — the external openings on the top of the bill of birds; the external orifices of the nose; the nostrils.

**Nasal gland** — a specialized gland of birds and some other species that serves to concentrate salt and secrete it from the body.

**Nasal cavity** — the forward (proximal) portion of the passages of the respiratory system, extending from the nares to the pharynx and separated from the oral cavity by the roof of the mouth.

**NDV** — Newcastle disease virus.

**Necropsy** — the methodical examination of the internal organs and tissues of an animal after death to determine the cause of death or to observe and record pathological changes.

**Necrosis** — the death of cells in an organ or tissue.

**Necrotic** — dead; exhibiting morphological changes indicative of cell death; in this Manual, necrotic lesions refer to areas of dead tissue.

**Nematocides** — chemicals used to kill nematode worms.

**Nematodes** — unsegmented, cylindrical parasitic worms; roundworm.

**Neoplasm** — see tumor.

**Nervous system** — specialized components of vertebrates, and, to a lesser extent invertebrates, that control body actions and reactions to stimuli and the surrounding environment.

**Neurotoxin** — toxins that cause damage to or destroy nerve tissue.

**Nictitating membrane** — the so-called third eyelid, a fold of tissue connected to the medial (side closest to the midline) side of the eye, which moves across the eye to moisten and protect it.

**Nocturnal** — species that are active during evening (nondaylight) hours.

**Nodule** — a small mass of tissue that is firm, discrete, and detectable by touch.

**Nontoxic shot** — shotshells with shotpellets that are not made of lead or other toxic metals; typically, soft iron is used, and is referred to as steel shot.

**No-see-ums** — see Ceratopognid flies.

**Occlusion** — a blockage or obstruction; the closure of teeth.

**Oligochaetes** — the earthworms and aquatic forms of the class Oligochaeta.

**Oocyst** — the encysted or encapsulated zygote in the stage of some protozoan parasites; often highly resistant to environmental conditions.

**Opisthotonos** — abnormal spasm of the neck and back muscles resulting in a body position in which the head and heels are involuntarily thrown back and the body is arched forward.

**Osmoregulatio**n — adjustment of osmotic pressure in relation to the surrounding environment.

**Osteoporosis** — loss of bone structure.

**PAH** — an acronym for polycyclic aromatic hydrocarbons.

**Panzootic** — a disease involving animals within a wide geographic area such as a region, continent, or globally.

**Parasitism** — an association between two species in which one (the parasite) benefits from the other (the host), often by obtaining nutrients.

**Paratenic host** — a host that has been invaded by a parasite, but within which no morphological or reproductive development of the parasite takes place; a “transport” host.

**Paresis** — partial paralysis.

**Passerines** — small- to medium-sized perching birds.

**Pathogenic** — the ability to cause disease.

**Pathological** — an adjective used to describe structural or functional changes that have occurred as the result of a disease.

**PCB** — acronym for polychlorinated biphenyls, a group of chlorinated aromatic hydrocarbons used in a variety of commercial applications. These compounds have long environmental persistence and have been a source for various toxic effects in a wide variety of fauna.

**Pelagic** — refers to living in or near large bodies of water, such as oceans or seas; typically, this term refers to avian species that only come to land areas during the breeding season.

**Pericardium** — the fibrous sac surrounding the heart.

**Pericarditis** — inflammation and thickening of the sac surrounding the heart.

**Peritoneal cavity** — the abdominal cavity, which contains the visceral organs.

**Phage** — a virus that has been isolated from a prokaryote (an organism without a defined nucleus, having a single double-standard DNA molecule, a true cell wall, and other characteristics). Most phages are bacterial viruses.

**Pharynx** — the musculomembranous passage between the mouth and the larynx and esophagus.

**Pigeon milk** — the regurgitated liquid that an adult pigeon feeds its young.

**Pinnipeds** — aquatic mammals that include the sea lions, fur seals, walruses, and earless seals.

**Plaque** — a patch or a flat area, often on the surface of an organ.

**Plasmodium** — blood parasites transmitted by mosquitos of the family Culicidae.

**Plumage** — the feather covering of birds.

**Postmortem** — examination and dissection of animal carcasses performed after the death of the animal. Also, changes that occur in tissues after death.

Poultry — domestic avian species, such as chickens and domestic ducks, geese, and turkeys.

Prefledglings — birds of the current hatch year that have not become feathered enough to fly.

**Prevalence** — the number of cases of a disease occurring at a particular time in a designated or defined area; rate.

**Proboscis** — a tubular process or structure of the head or snout of an animal, usually used in feeding; in this Manual, the tubular process of Acanthocephalan parasites is used for attachment to the host and feeding from it.

**Protoporphyrin** — a component of hemoglobin; useful in the diagnosis of exposure to lead.

**Protozoan** — a one-celled animal with a recognizable nucleus, cytoplasm, and cytoplasmic structures.

**Psittacines** — parrots, parakeets, and other species within the family Psittacidae.

**Puddle ducks** — see dabbling ducks.

**Proventriculus** — the first, or "glandular," stomach of a bird.

**Puddle ducks** — synonymous with dabbling or surface-feeding ducks.

**Punkies**— small, biting midges of the genus Culicoides; vectors for Haemoproteus infections. See ceratopognid flies.

**Purulent** — containing pus, as in a purulent discharge.

**Range** — the geographic distribution of a population or the area within which an individual animal moves (as in home range).

**Raptors** — synonymous with birds of prey. Birds, including hawks, owls, falcons, and eagles, that feed on flesh.

**Reactivation** — refers to the process by which cholinesterase enzyme activity returns to normal after carbamate exposure.

**Rendering** — a process by which animal carcasses are converted into fats and fertilizer.

**Reptiles** — coldblooded vertebrates that belong to the class Reptiles; such as., snakes, turtles, lizards.

**Reservoir host** — the host that maintains the disease agent in nature and that provides a source of infection to susceptible hosts.

**Respiratory system** — the collection of organs that provide oxygen to the organism and result in the release of carbon dioxide; typically, the trachia and lungs.

**Rice breast disease** — synonym for sarcocystis.

**Rocket nets** — remotely triggered, weighted firing devices that are propelled through the air by an explosive force carrying the netting to which they are attached over the birds or other animals being captured.

**Rodenticides** — toxic substances used to kill rodents.

**Rodents** — mammals that have chisel-like, ever growing incisor teeth that are used for gnawing; i.e., mice to beavers.

**Rookery** — a nesting area for some colonial birds, such as herons and egrets.

**Roost sites** — typically, locations where birds congregate at night in trees and other locations.

**Rough fish** — a term given to bottom-feeding freshwater fish with large scales, such as carp, buffalo, and similar species.

**Roundworms** — see nematodes.

**Ruminants** — hooved mammals possessing a rumen or first stomach, from which food or a cud is regurgitated for further chewing. Includes deer, elk, sheep, cattle, etc.

**Salivary glands** — the glands of the mouth that produce saliva.

**Salt gland** — see nasal gland.

**Sand flies** — see punkies.

**Scavengers** — animals that feed on dead, sick or injured prey. Includes crows, vultures, gulls, eagles, hawks, etc.

**Schizogony** — a type of asexual reproduction in some protozoan parasites in which daughter cells are produced by multiple nuclear divisions of the parasite (schizont).

**Schizonts** — the multinucleate, intermediate parasite stage that develops into merozoites within a host cell.

**Scoliosis** — an abnormal lateral curvature of the spine.

**Sea ducks** — ducks that frequent open ocean, although some species may be found on coastal bays or inland waters. Includes oldsquaw, eiders, scoters, and harlequin duck.

**Secondary poisoning** — intoxication of an animal as a result of eating a poisoned animal; for example, the poisoning of an eagle after it has fed on a duck that was poisoned by a chemical in treated grain. This differs from biomagnification, which involves increasing concentrations of toxic compounds within the body of organisms at increasing higher levels of a food chain.

**Section 7 consultations** — the Endangered Species Act requires discussion and evaluation of any proposed Federal activity, program, or permit that might affect an endangered species.

**Sedated** — chemically quieted.

**Septicemia** — the presence of pathogenic microorganisms or toxins in the blood.

**Serosa** — refers to the outside layer of an organ, such as the serosal surface of the intestine, or the lining of a body cavity.

**Serosal surface** — the external surface of an organ or a tissue within the body.

**Serotype** — a taxonomic subdivision of a microorganism, based on characteristic antigens or proteins.

**Serovar** — a taxonomic subdivision of a microorganism similar to serotype (above) but usually more specific.

**Shorebirds** — birds that feed at the edge of shallow water, along mudflats, and in shallow wetlands. Typically, these birds feed on invertebrates and include such species as American avocet, black-necked stilt, curlews, plovers, phalaropes, sandpipers, yellowlegs, and sanderling.

**Signs** — observable evidence of disease in animals (similar to symptoms in humans).

**Sloughing** — shedding of dead cells or dead tissue from living structures or tissues.

**Slugs** — terrestrial, snail-like mollusks that have a long, fleshy body and only a rudimentary shell.

**Small mammals** — mice to rabbits, racoons etc.; a general term used in wildlife management to group species of small to moderate size.

**Small rodents** — see rodents; rodents of small size, such as rats and mice.

**Songbirds** — small perching and singing birds, typically of the order Passeriformes, including sparrows, finches, and cardinals.

**Sowbugs** — see isopods.

**Splenomegaly** — enlargement of the spleen.

**Spore** — refers to a resistant stage, usually of bacteria or fungi, by which some microorganisms survive unfavorable environmental conditions and then develop into active life forms during favorable environmental conditions.

**Sporogony** — sporulation that involves multiple fission of a sporont (schizogony), resulting in the production of a sporocysts and sporozoites.

**Sporont** — a zygote of coccidian protozoa.

**Sporozoite** — the elongate nucleated infective stage of coccidian protozoan parasites.

**Sporulation** — the formation or libertion of spores.

**Squab** — a nestling pigeon that has not fledged.

**Sternum** — the breastbone.

**Subcutaneous** — under the skin.

**Systemic** — affecting the entire body.

**Tapeworms** — segmented parasitic flatworms; also referred to as cestodes.

**Teal** — small, swift-flying waterfowl of the genus Anas.

**Tegument** — the covering of an organ or the body.

**Tenosynovitis** — inflammation of the tendon sheath.

**Teratogenic** — causing embryonic deformities due to abnormal differentiation and development of cells.

**Thermoregulation** — regulation of the internal temperature of the body by various physiological processes.

**Thorax** — the part of the body between the neck and the respiratory diaphragm (in mammals), encased by the ribs.

**Thorny-headed worms** — acanthocephalan parasites.

**Thymus gland** — a lymph-gland-like organ involved in cellular immunity, located in the neck or upper thoracic cavity.

**Torticollis** — twisting or rotation of the neck causing an unnatural position of the head.

**Toxic** — poisonous.

**Toxicosis** — the condition of being poisoned.

**Trematodes** — flat, unsegmented parasitic worms; flatworms, flukes.

**Trichomonids** — protozoan parasites of the genus Trichomonas.

**Trophic level** — refers to an animal's position in the food chain. Species at higher trophic levels are, to a greater or lesser extent, dependent upon species in preceding trophic levels as sources of energy.

**Tumor (neoplasm)** — growths within organs and tissues of the body that result from the abnormal progressive multiplication of cells in a manner uncontrolled by the body.

**Ubiquitous** — found everywhere.

**Ulceration** — crater-like lesions in the skin and other tissues.

**Ungulate**s — hoofed mammals.

**Unthrifty appearance** — an expression used in animal husbandry to describe an animal that is unkempt and dirty. Usually hair or feathers are soiled by excrement.

**Upland gamebirds** — game birds found in terrestrial habitats. Includes species such as ring-necked pheasant, quails, grouse, wild turkey, etc.

**Upper digestive tract** — the portion of the gastrointestinal tract that extends from the anterior opening of the esophagus in the region of the mouth to the stomach, but not including the intestines.

**Ureter** — the tubular structure that transports urates from the kidneys to the cloaca of birds.

**Vascular system** — blood circulation system.

Vector — an insect or other living organism that carries and transmits a disease agent from one animal to another.

**Vegetative form** — in bacteria, an active, growing, multiplying stage of development as opposed to a "spore," or a resistant resting stage.

**Velogenic** — refers to highly virulent strains of Newcastle disease virus that are capable of producing severe disease in the host.

**Ventriculus** — the stomach of a bird.

**Verminous peritonitis** — inflammation of the peritoneal cavity caused by parasites, usually nematodes.

**Vertebrates** — animals with backbones.

**Viremia** — the presence of virus in the blood.

**Virulence** — the disease-producing ability of a microorganism, generally indicated by the severity of the infection in the host and the ability of the agent to invade or cause damage or both to the host's tissues.

**Virulent** — the degree to which an infectious agent produces adverse effects on the host; a highly virulent organism may produce severe disease, including death.

**Virus shedding** — discharge of virus from body openings by way of exudate, excrement, or other body wastes or discharges.

**Viscera** — the internal organs, particularly of the thoracic and abdominal cavities.

**Viscerotropic** — possessing an affinity for visceral organs; a disease that acts primarily on the soft internal tissues of the body such as the heart, lungs, liver, and digestive tract.

**Voluntary muscle** — muscle normally under control of the individual.

**Voucher specimen** — specimens deposited in scientific collections that are representative of a species or a subgrouping of a species.

**Wading birds** — long-necked, long-legged birds that feed by wading in wetlands and catching prey with their bills. Includes egrets, herons, ibises, roseate spoonbills, flamingos, and bitterns.

**Waterbirds** — birds that require aquatic habitat.

**Waterfowl** — species of the Family Anatidae; ducks, geese, and swans. Does not include American coot.

**Whistling ducks** — the fulvous whistling duck or the tropical black-bellied tree duck.

**Yeasts** — single-celled, usually rounded fungi that produce by budding.

**Zooplankton** — minute animal organisms that in combination with counterparts from the plant kingdom constitute the plankton (minute free-floating organisms) of natural waters.

**Zygote** — a cell resulting from the union of a male and a female gamete, until it divides; the fertilized ovum.

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