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Chemical Toxins (Field Manual of Wildlife Diseases)

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Section 7

Chemical Toxins

Organophosphorus and Carbamate Pesticides

Chlorinated Hydrocarbon Insecticides

Polychlorinated Biphenyls

Oil

Lead

Selenium

Mercury

Cyanide

Salt

Barbiturates

Miscellaneous Chemical Toxins

Oiling disrupts normal feather structures and function

Photo by Nancy J. Thomas

Introduction to Chemical Toxins

"Dosage alone determines poisoning."
(Translation of Paracelsus)

Many kinds of potentially harmful chemicals are found in environments used by wildlife. Some chemicals, such as pesticides and polychlorinated biphenyls (PCBs), are synthetic compounds that may become environmental contaminants through their use and application. Other materials, such as selenium and salt, are natural components of some environments, but contaminants of others. Natural and synthetic materials may cause direct poisoning and death, but they also may have adverse effects on wildlife that impair certain biological systems, such as the reproductive and immune systems. This section provides information about some of the environmental contaminants and natural chemicals that commonly cause avian mortality; microbial and other biotoxins are addressed in the preceding section.

Direct poisoning and mortality of wildlife caused by exposure to chemical toxins are the focus of this section. However, the indirect effects of chemicals may have significantly greater impacts on wildlife populations than the direct effects. Behavioral changes that affect survival, reproductive success and the survival of young, and that impair the functioning of the immune system are examples of indirect chemical toxicity that are known to occur but that are beyond the scope of this publication. For additional information readers are directed to more comprehensive treatments of environmental toxicology and to publications that focus on specific chemicals and their effects on wildlife.

The diagnosis of chemical poisoning as the cause of wildlife mortality is a challenging task because of the vast array of chemicals that wildlife may be exposed to (Table 1), the variable biological responses following concurrent exposure to multiple chemicals, the absence of tissue residues for some chemical toxins, and the lack of specific pathological changes associated with most chemical toxins in tissues. The diagnostic process can be greatly facilitated by a thorough field observation record, comprehensive background information about the circumstances of a mortality event, and by properly collecting, handling, and preserving samples submitted to the diagnostic laboratory (see Section 1). Sources of assistance for the investigation of wildlife mortality, when toxins are suspected, are listed in Appendix B.

Areas Covered

The chapters that follow address chemical toxins that are recurrent causes of avian mortality. The chapters discuss chemicals that cause frequent and sometimes large-scale mortality events, as well as some chemicals that are less significant, because they are restricted to certain geographic areas or have been recently recognized as emerging problems.

- Pesticides
  - Organophosphorus and carbamate compounds
  - Chlorinated hydrocarbons
- Polychlorinated biphenyls
- Oil
- Lead
- Selenium
- Mercury
- Cyanide
- Salt
- Barbiturates

Quote from:

Philipus Aureolus Paracelsus, a German-Swiss physician and alchemist who lived from 1493 to 1541.

Table 1 *Examples of chemical toxins to which wildlife may be exposed.*

Pesticides

This group includes chemicals that are used to kill or repel organisms that are unwanted in particular situations. Insecticides are generally the best known pesticides but others, their target organisms, and examples of compounds within those groups include the following:

Pesticide type	Target organisms	Compounds
Acaricides	Mites, ticks, spiders	Permethrin, Phosmet, Methiocarb, Bomyl®, Carbofuran, Demeton (Systox®)
Algacides	Algae	Copper sulfate, Potassium bromide, Chlorine
Antibiotics	Bacteria	Phenol, Nitrapyrin
Avicides	Birds	Avitrol®, Fenthion, Compound 1080, Starlicide®
Fungicides	Fungi	Thiram, Ziram, Captan, Hexaconazole
Herbicides	Plants	Diquat®, Alachlor (Lasso®), Atrazine
Molluscicides	Snails and slugs	Bayluscide®, Methiocarb, Zectran®
Nematocides	Nematodes (worms)	Terbufos (Counter®), Isazofos (Triumph®), Aldicarb (Temik®), Carbofuran, Diazinon
Piscicides	Fish	Rotenone, Antimycin
Repellents	Mammals Birds	Thiram Methiocarb
Rodenticides	Rodents	Warfarin, Diphacinone, Brodifacoum (Talon®), Chlorophacinone

Metals

Wildlife may be exposed to metals when they are components of pesticides, such as mercury and cadmium in fungicides, or through other routes, such as aquatic food chains with high mercury levels.

Metal	Source
Arsenic	Used as an insecticide and preservative; present in wastes from metal smelting and glass manufacturing.
Cadmium	Used as a fungicide; waste from electroplating and production of plastics and batteries.
Chromium	Industrial effluents from ore refinement, chemical processing.
Copper	Used as a fungicide, an algicide, and in agriculture.
Lead	Mine tailings, ingestion of particulate lead deposited during sporting activities.
Mercury	Used as a fungicide in paper mills and other industrial and agricultural uses; combustion of fossil fuels.
Selenium	Irrigation drain water from soils with high selenium concentrations; combustion of fossil fuel; sewage sludge.
Zinc	Found throughout the environment; higher levels in areas of industrial discharge.

Petroleum

Wildlife may be exposed to many forms of petroleum, ranging from crude oils to highly refined forms, such as fuel oil.

Others

Many manufactured compounds, such as antifreeze (ethylene glycol) and certain drugs (such as euthanasia agents), present hazards to exposed wildlife.

Chapter 39

Organophosphorus and Carbamate Pesticides

Synonyms

Organophosphates, OPs

The insecticidal properties of organophosphorus (OP) and carbamate compounds were first discovered in the 1930s, and the compounds were developed for pesticide use in the 1940s. They have been used increasingly since the 1970s when environmentally persistent organochlorine pesticides, such as DDT and dieldrin, were banned for use in the United States. Organophosphorus and carbamate pesticides are generally short-lived in the environment (usually lasting only days to months instead of years) and, generally, chemical breakdown is accelerated as temperatures or pH or both increase.

Cause

The toxicity of OP and carbamate pesticides is due to the disruption of the nervous system of an invertebrate or a vertebrate through the inhibition of cholinesterase (ChE) enzymes. These enzymes are involved in transmitting normal nerve impulses throughout the nervous system. An acute pesticide dose reduces the activity of ChEs, and nerve impulses cannot be transmitted normally. This can paralyze the nervous system, and it may lead to death, usually from respiratory failure.

Species Affected

It is possible for a wide variety of vertebrate species to be affected by OP or carbamate pesticides. However, birds appear to be more sensitive than other vertebrates to the toxic effects of OP and carbamate pesticides. More than 100 avian species have been poisoned by these pesticides. Waterfowl, passerines, and raptors are the species most commonly identified in reported OP- and carbamate-related mortalities in the United States (Fig. 39.1). Raptors and other bird species become victims of secondary poisoning when they scavenge dead animals poisoned by pesticides or when they feed on live animals or invertebrates that are unable to escape predation because of pesticide intoxication.

Age, sex, diet, and body condition all are factors that affect a bird's susceptibility to pesticide poisoning. Generally, embryos and young birds, particularly the dependant or altricial birds, appear to be more sensitive to OP or carbamate compounds than adults. Dietary deficiencies, low fat reserves, poor physiological condition, and high energy needs, such as migration or high metabolic rates, may increase vulnerability to these compounds. Behavioral traits may also increase the potential for exposure to OP or carbamate compounds. Species at increased risk are those that congregate in areas of treated habitats, gorge on a food source (like geese), forage in treated substrates, or feed on target organisms shortly after applications of these compounds.

Common routes of exposure of birds to OP and carbamate pesticides include:

- Consumption of:
  - Treated seeds
  - Vegetation with pesticide residues
  - Dead or struggling poisoned insects
  - Granular formulations as grit, food, or coincidentally with other food items
  - Carrion killed by a pesticide
  - Food intentionally baited with pesticide
  - Live animals intoxicated with pesticide
  - Water contaminated with pesticide from runoff or irrigation
- Inhalation
- Absorption through the skin

Also, there can be considerable variability in the sensitivity of individual species to these pesticides (Table 39.1).

Table 39.1 *Toxicity for birds of organophosphorus pesticides and carbamate pesticides.*
[Modified from Hoffman and others, 1995. LD_{50} is the single oral dose of pesticide in milligrams per kilogram of body weight that is required to kill 50 percent of the experimental population]

Compound	Class	Species LD_{50}		
		Mallard duck	Ring-necked pheasant	Red-winged blackbird
Aldicarb	Carbamate	3.4	5.3	1.8
Carbaryl	Carbamate	>2,000	707	56
Carbofuran	Carbamate	0.5	4.1	0.4
Methiocarb	Carbamate	13	270	4.6
Mexacarbate	Carbamate	3.0	4.6	10
Azinphos-methyl	OP	136	75	8.5
Dimethoate	OP	42	20	6.6
Ethion	OP	>2,000	1,297	45
Phorate	OP	0.6	7.1	1.0
Temephos	OP	79	35	42

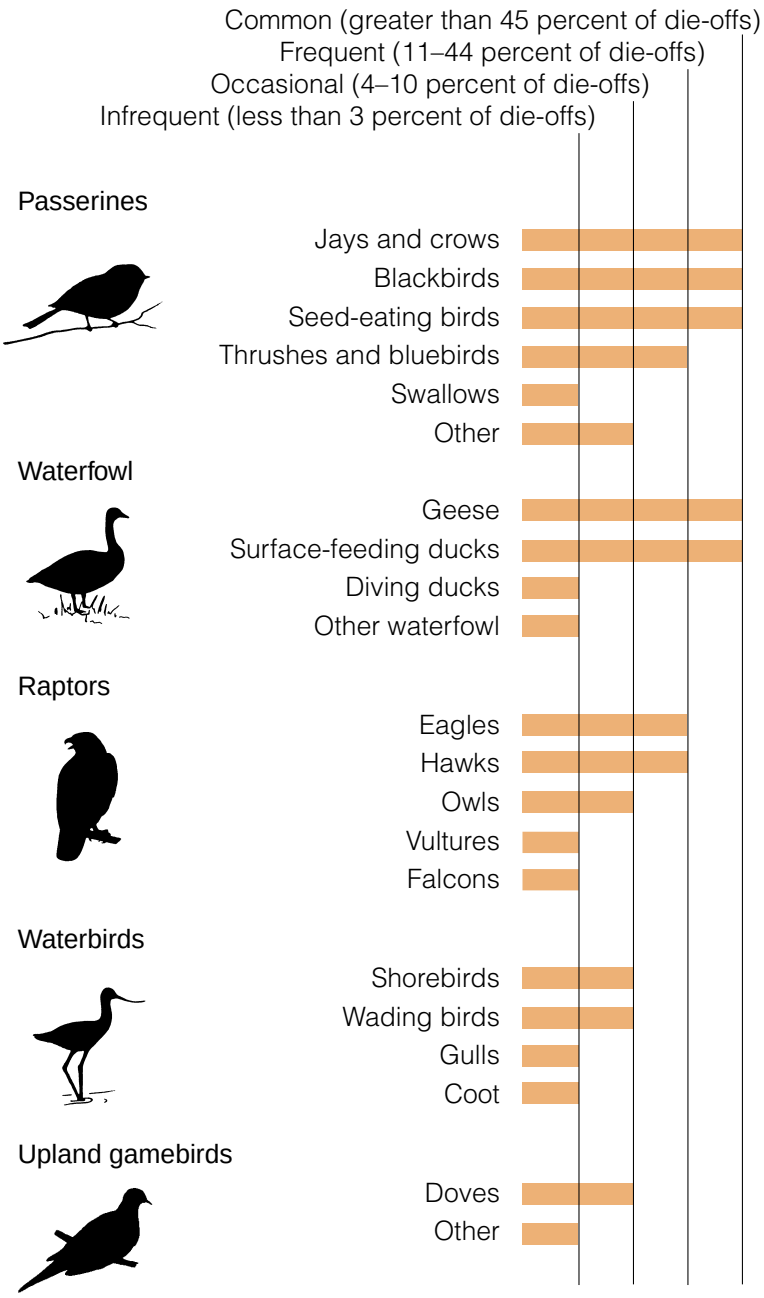

Figure 39.1 *Frequency of occurrence of major groups of birds in documented organophosphorus and carbamate pesticide mortality events from 1986–95 (National Wildlife Health Center data base).*

Distribution

Organophosphorus and carbamate compounds are used throughout the world as insecticides, herbicides, nematocides, acaricides, fungicides, rodenticides, avicides, and bird repellants. These compounds are applied in a wide variety of habitats including agricultural lands, forests, rangelands, wetlands, residential areas, and commercial sites. Wild bird deaths from OP and carbamate poisoning have been reported throughout the United States (Fig. 39.2). In more than half of these mortality incidents, the pesticide source is unknown (Fig. 39.3). Known applications of these compounds fall into five groups: approved applications in 1) agricultural land uses such as field and row crops, pastures, orchards, and forests; 2) residential and urban sites for turf in parks, golf courses, yards, and other urban pest control uses; 3) livestock uses such as pour-ons or feed products; 4) vertebrate pest control; and 5) malicious pesticide use, such as baiting to intentionally harm wildlife (Fig. 39.3).

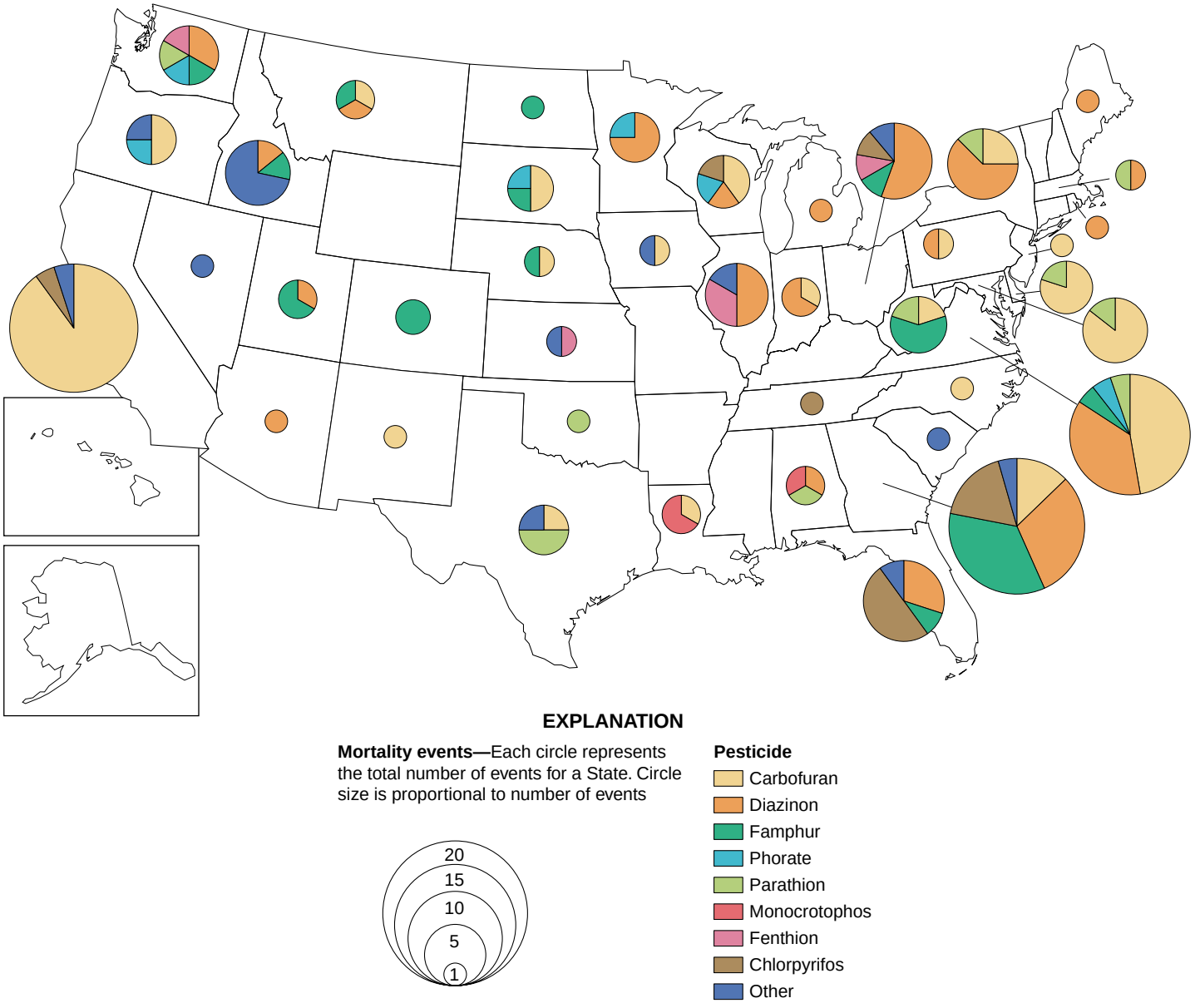

Figure 39.2 *Distribution of 181 avian mortality events caused by organophosphorus and carbamate pesticides, 1986–1995 (National Wildlife Health Center data base).*

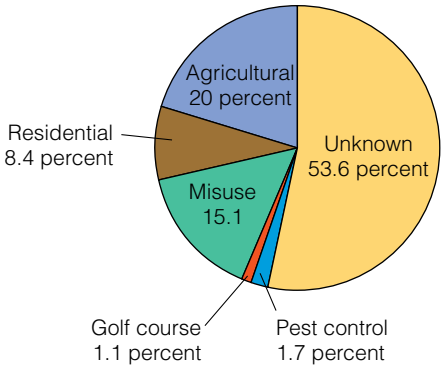

Figure 39.3 *Applications associated with avian mortality caused by organophosphorus and carbamate pesticides from 1986–95 (National Wildlife Health Center data base).*

Seasonality

Because OP and carbamate pesticides are typically short-lived in the environment, seasonality of avian mortality is generally associated with pesticide applications (Fig. 39.4). In documented mortality events in the United States, February was the peak month for the onset of bird die-offs, and most of these die-offs occurred in the southern United States, where the growing season starts early in the year.

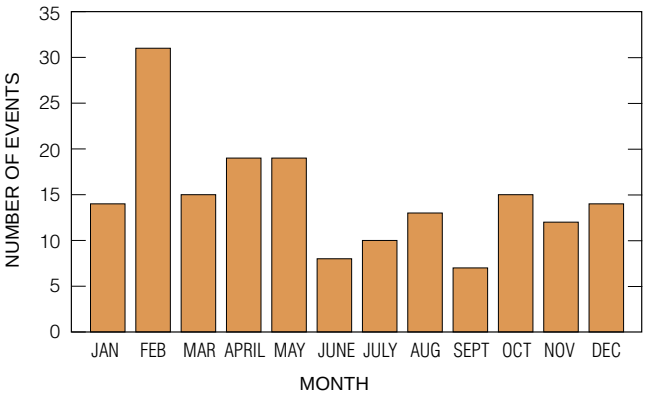

Figure 39.4 *Avian mortality events due to organophosphorus and carbamate pesticides by date of onset, 1986–95 (National Wildlife Health Center data base).*

Field Signs

Mortality can be the first sign noted in a pesticide poisoning, but the observer may find other clues at the scene of a mortality event. Live affected birds may exhibit convulsions, lethargy, paralysis, tremors, or other nonspecific neurological signs.

Clinical signs and bird behaviors that are commonly associated with acute exposure to cholinesterase-inhibiting pesticides

[Modified from Mineau, 1991]

Convulsions
Hyperexcitability
Incoordination of muscular action (ataxia)
Muscular weakness (myasthenia)
Difficult breathing (dyspnea)
Rapid breathing (tachypnea)
Vomiting
Defecation
Diarrhea
Spasmodic contraction of anal sphincter (tenesmus)
Lethargy
Induced tranquility
Head and limbs arched back (opisthotonos)
Slight paralysis (paresis)
Blindness
Contraction of pupils (miosis)
Dilation of pupils (mydriasis)
Drooping of eyelid (ptosis)
Protrusion of eyes (exopthalmia)
Excessive tear formation (lacrimation)
Excessive thirst (polydypsia)
Bleeding from nares (epistaxis)
Erection of contour feathers (piloerection)

Birds that die rapidly with pronounced neurological signs may leave evidence of their struggle even after death, such as vegetation clenched in their talons (Fig. 39.5) or vegetation that they disturbed during thrashing or convulsions. Animals may not have time to disperse before the toxin takes effect, and carcasses of multiple species, especially predators and granivorous or insectivorous wildlife, may be found within the same area following OP or carbamate exposure.

Birds can also be affected by a sublethal dose of an OP or carbamate pesticide. Sublethal exposure may contribute to other causes of mortality in birds, such as trauma. In some instances when birds have died due to trauma from a vehicle impact, a building strike, or predation, decreased brain ChE has been demonstrated, which indicates pesticide exposure. The sublethal dose of pesticide likely impaired the nervous system enough to alter behavior, thus making the animal more vulnerable to a traumatic cause of death. Special studies that evaluated sublethal OP or carbamate compound exposure in birds have found other effects to birds, including a reduced ability to regulate body temperature; impaired reproduction; and reduced tolerance to cold stress, which can cause reduced activity, leading to decreased feeding and weight loss. Altered behaviors such as reduced nest attentiveness and changes in singing by passerines have also been observed.

Photo by James Runningen

Figure 39.5 *Vegetation clenched in the talons of a bald eagle, which is a finding consistent with organophosphorus or carbamate poisoning in raptors.*

Gross Findings

Granular material or the presence of dye or both in the gastrointestinal tract are conspicuous findings that implicate pesticide ingestion. The necropsy finding of freshly ingested food in the upper gastrointestinal tract of a carcass is a good indicator of death by intoxication, especially when a large amount of a uniform food item is present. Feathers, flesh, hair, or other animal parts in the stomachs of raptors or of scavengers are common in secondary poisoning, whereas ingested grain is often found in waterfowl and passerines (Fig. 39.6). The food item may indicate the pesticide source, and the food can then be analyzed for specific chemical compounds.

The gross lesions that are associated with acute mortality from pesticide poisoning in birds are nonspecific and are usually minimal. Reddening of the intestinal wall, or even hemorrhage (Fig. 39.7), is observed occasionally with ingestion of certain pesticides. Redness and excess fluid in the lungs may be observed; these findings are consistent with respiratory failure. However, these changes are not unique to pesticide poisonings; they can be found in animals that died from other causes.

Diagnosis

A diagnostic evaluation is essential. A diagnosis of pesticide poisoning in birds is based on evidence of ChE inhibition in the brain or the blood and identification of pesticide residues in gastrointestinal contents. In many instances, depressed ChE activity will be the first indication that OP or carbamate pesticides caused a mortality event. A necropsy is necessary to rule out other causes of mortality or to identify contributing causes.

Brain ChE activity is a reliable indicator of OP and carbamate exposure in dead birds, but the absence of ChE depression does not reliably rule out poisoning. Brain ChE activity is measured and compared to normal brain ChE activity of the same species to determine the decrease in enzyme activity from normal levels (Appendix D). A decrease in brain ChE activity of 25 percent or more from normal indicates exposure to a cholinesterase-inhibiting compound (OP or carbamate pesticide); a decrease of 50 percent or more from normal is evidence of lethal exposure. Because of the variation in results between laboratories and the variability even between methods and procedures within a lab, it is important to compare results with controls from the same laboratory using the same method and not interpret analytical results from two or more laboratories or from two or more analytical methods.

Analyses can be carried one step further to differentiate the effect of OP from carbamate compounds by measuring the enzyme activity of a sample after incubation at 37–40 °C and comparing it to the initial measurement. Enzyme activity that returns toward a normal level after incubation, or that reactivates, indicates that carbamate poisoning is likely because carbamates tend to release their bond with ChE over time at increased temperatures or in aqueous environments. Because reactivation can occur with some pesticides, depressed brain ChE activity in a pesticide-poisoned bird may be difficult to document if the carcass has remained in a warm environment for an extended period of time. Another method that is used to differentiate an OP from a carbamate compound exposure is reactivation analysis, during which 2-PAM, a cholinesterase regenerating agent, is added to the sample and the change in brain ChE activity is then measured. Reactivation of ChE activity using 2-PAM occurs only when an OP compound is bound to the enzyme.

When a pesticide die-off is suspected, it is important to chill carcasses immediately. If diagnostic evaluation cannot be initiated within 24–48 hours, carcasses should be frozen as soon as possible to prevent further change in brain ChE activity. Also, when normal brain ChE activity values are not known for a particular species, control samples collected from normal birds of the same species are needed in order to compare ChE values.

In birds that recover from OP or carbamate poisoning, brain ChE activity will typically increase but it may remain below normal levels for up to 3 weeks, depending on the compound and on the dose received. Cholinesterase activity in blood from live birds may be used as an indicator of pesticide exposure; however, blood ChE activity is more variable than brain ChE activity. Cholinesterase enzymes in the blood are more sensitive than brain ChE to OP and carbamate pesticides; therefore, pesticide exposure quickly and dramatically depresses blood ChE activity, which then rapidly returns to normal levels.

One advantage of measuring blood ChE activity is that a nonlethal sample can be taken to provide evidence of OP or carbamate pesticide exposure in live birds. A disadvantage of measuring blood ChE activity is that interpretation is difficult because normal blood ChE activity varies among spe-

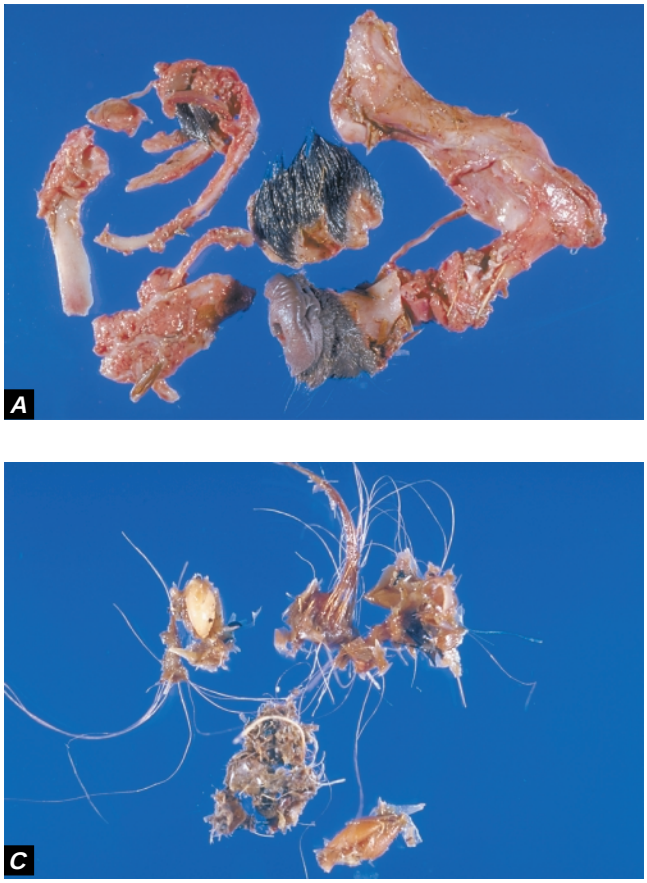

Figure 39.6 *Examples of food items found in the gastrointestinal tracts of birds that died from organophosphorus or carbamate poisoning.* ***(A)*** *Pig remains from the crop of a bald eagle.* ***(B)*** *Bovine skin from the stomach of a bald eagle.* ***(C)*** *Bovine hair from the stomach of a magpie.* ***(D)*** *Corn in the esophagus of a mallard.*

cies, age, sex, and body condition, and because a diurnal ChE variation may occur in some species. The reactivation analysis described above, which is used to differentiate an OP- from a carbamate-induced intoxication when measuring brain ChE activity, can also be used to evaluate blood ChE activity. In live animals, a presumptive diagnosis can also be made by reversing the neurological signs with proper medical treatment.

Specific compound residues may be identified in gastrointestinal contents. Mass spectrometry and gas chromatography are the usual analytical methods. Table 39.3 lists the compounds that were identified as the cause of mortality in the documented wild bird mortality events illustrated in Fig. 39.2.

Table 39.3 *Specific organophosphorus and carbamate pesticides known to cause wild bird mortality events.*

Carbamates	Organophosphorus compounds	
Carbofuran	Chlorpyrifos	Fenthion
Methiocarb	Diazinon	Fonofos
Oxamyl	Dicrotophos	Methamidophos
Aldicarb	Dimethoate	Monocrotophos
	Disulfoton	Parathion
	Famphur[1]	Phorate
	Fenamiphos	Phosphamidon
	Fensulfothion	Terbufos

[1] Famphur is regulated by the Food and Drug Administration as a drug.

Control

When a die-off with a confirmed pesticide poisoning diagnosis has occurred, birds should be denied use of the pesticide-affected area. Carcass pickup is necessary to prevent secondary toxicity to scavengers and prevent mortality from other causes related to decomposing carcasses, such as botulism. Any remaining pesticide in bags, on treated seed, bait, or grain must be removed to prevent further mortality.

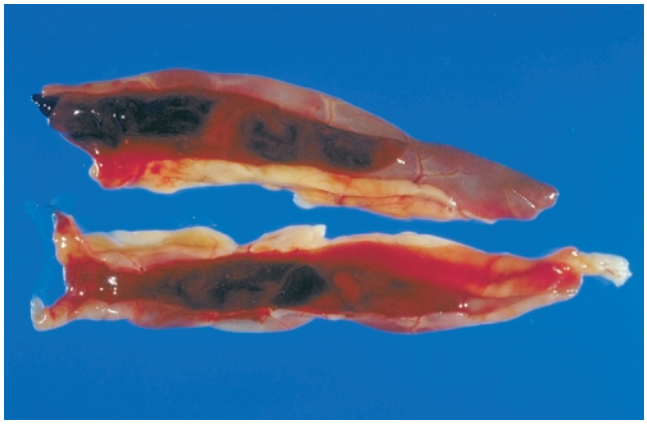

Photo by Paul Soler

Figure 39.7 *Hemorrhage in the intestine is an occasional finding in waterfowl that died of exposure to pesticides, particularly organophosphorus compounds.*

Followup to wildlife mortality incidents due to pesticide poisoning is important for determining the source and the use of a chemical. Documented wildlife mortality from approved pesticide applications is considered by regulatory authorities for developing label use restrictions and for licensing pesticide formulations. Malicious use of pesticides to kill unwanted wildlife is against the law, and legal means can be employed to stop illegal use.

Persons who apply pesticides need to consider wildlife use and environmental conditions when they apply the chemicals. Migration patterns of the wildlife that use the area, the presence of nesting and breeding species, and weather conditions, such as the potential for aerial drift or runoff into wetlands or ponds, are among the factors that should be considered. Pesticides should be applied only as directed; the use of alternate chemicals or formulations that pose less risk to nontarget species should also be considered. Buffer zones at crop perimeters will provide more protection to areas used by wildlife. Agricultural land planted adjacent to wetlands should be plowed parallel to a wetland to minimize runoff.

Human Health Considerations

Human exposure to OP or carbamate pesticides can result in serious illness or even death. Exposure can occur through inhalation, absorption through the skin, or by ingestion. When pesticides that may be associated with wildlife mortality incidents are investigated, field procedures should be scrutinized to avoid inadvertent exposure of personnel to pesticides. Persons who collect carcasses or field samples must prevent their exposure by wearing nonpermeable gloves, rubber boots, or other appropriate clothing that will prevent skin absorption, and respirators should be used if chemical inhalation is possible.

Poisoning in humans should be treated as a serious medical emergency. When someone seeks medical attention for exposure to an OP or carbamate compound, the attending physician should be informed that the person may have been exposed to these chemicals. Patients can be monitored by blood sampling to evaluate their blood ChE levels. Aggressive treatment of acute intoxication does not protect against the possibility of delayed onset neurotoxicity or persistent neurological defects. Certain compounds have been documented to cause delayed effects in humans. An intermediate syndrome that occurs within 24–96 hours after exposure has recently been described with intoxications of fenthion, dimethoate, monocrotophos, and methamidophos. Muscles of the limbs and those innervated by cranial nerves are affected, causing palsies, respiratory depression, and distress. Another delayed neurotoxicity from some OP compounds can occur 1–2 weeks after exposure. Initially, incoordination develops, and it can progress to moderate to severe muscle weakness and paralysis. This delayed effect was documented with some OP compounds that were rarely used as pesticides, but the effect may be a potential risk with similar compounds that are more commonly used today if sufficient exposure to the compound occurs. These delayed effects could be a problem in wildlife, but they have not been recognized yet in any wildlife species.

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Supplementary Reading

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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.]

	LD_{50} (mg/kg)								
Species	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.

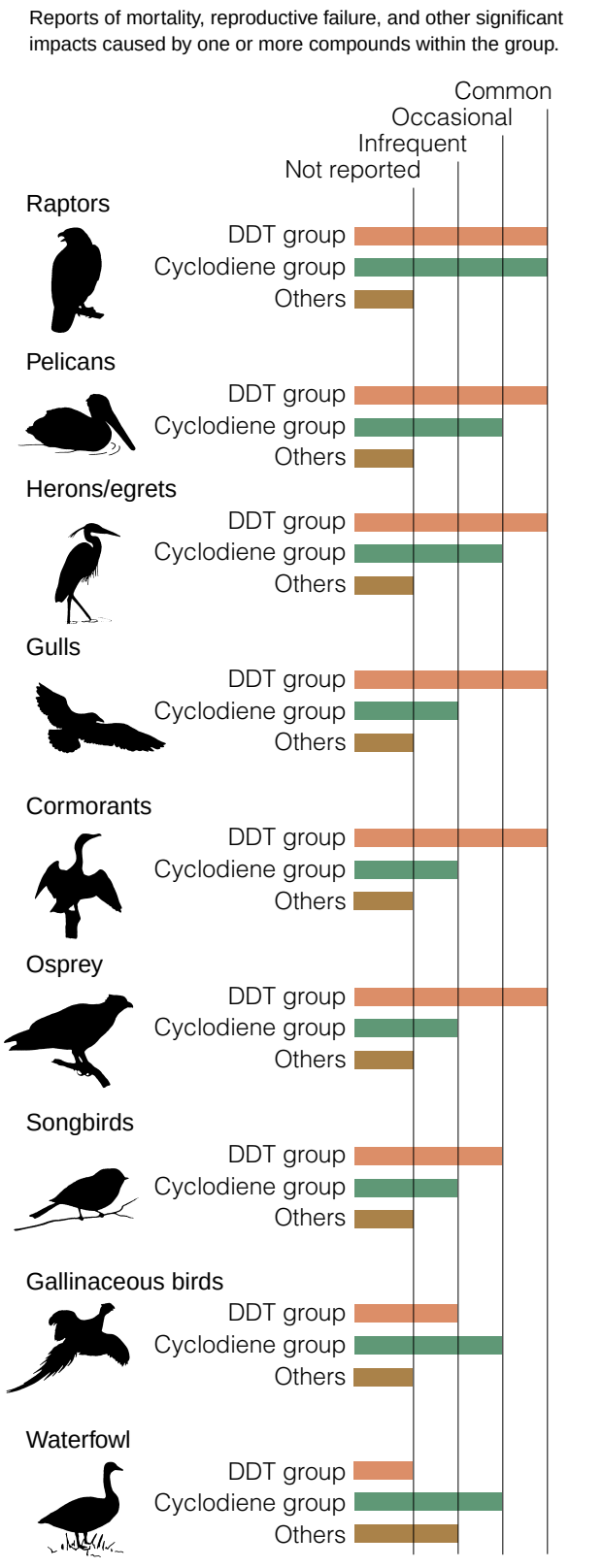

Figure 40.1 *Relative importance of health effects caused by chlorinated hydrocarbon insecticides in selected free-living birds.*

Photo by Milton Friend

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

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.*

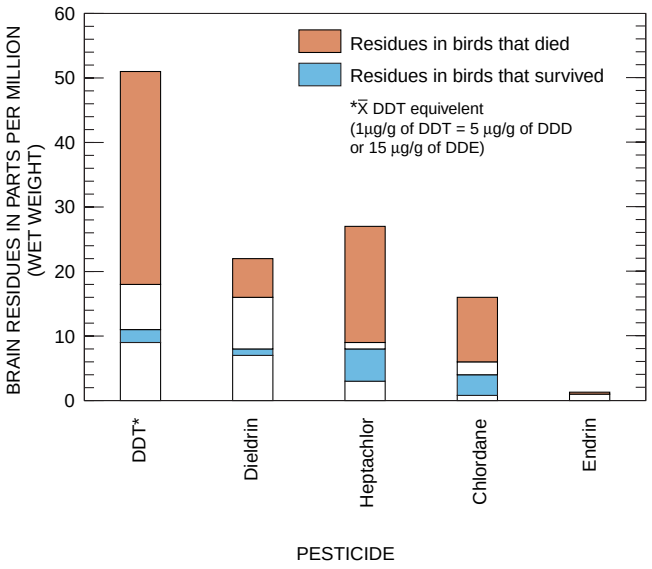

Figure 40.5 *Mean chlorinated hydrocarbon insecticide residues in brains of experimentally dosed passerines.*

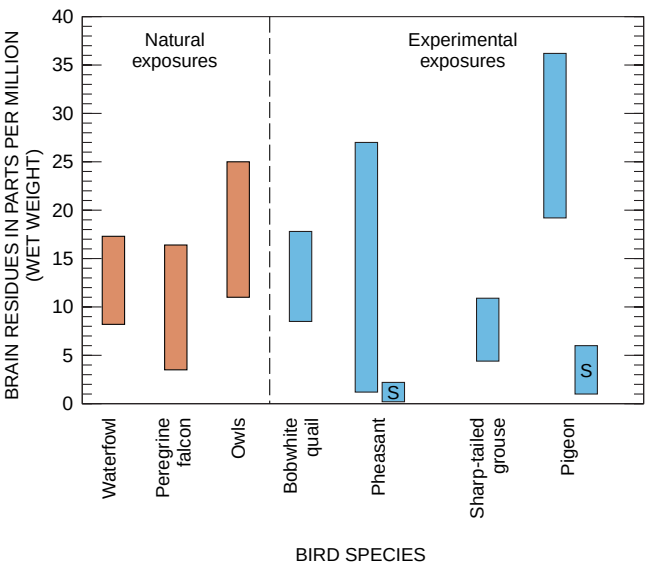

Figure 40.6 *Range of dieldrin residues in brains of selected avian species. [S is the range of concentration within which some of the birds survived.]*

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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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.]*

Species	LD_{50} (mg/kg of Aroclor®)			
	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. Knowl-

edge 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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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.

Photo by Milton Friend

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.

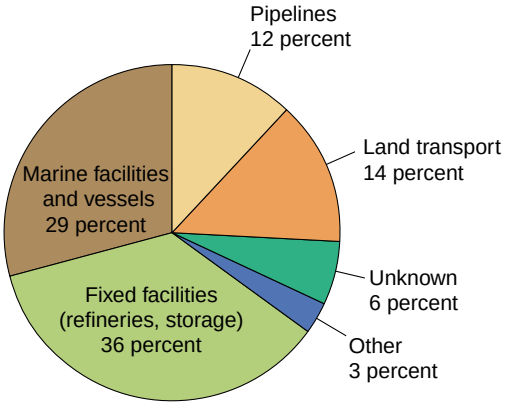

Figure 42.1 *Origin of petroleum spills, 1987–94.*

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 se-

questered 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).

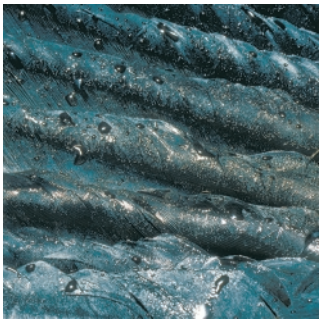

Photo by Nancy J. Thomas

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 nonnavigable 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-

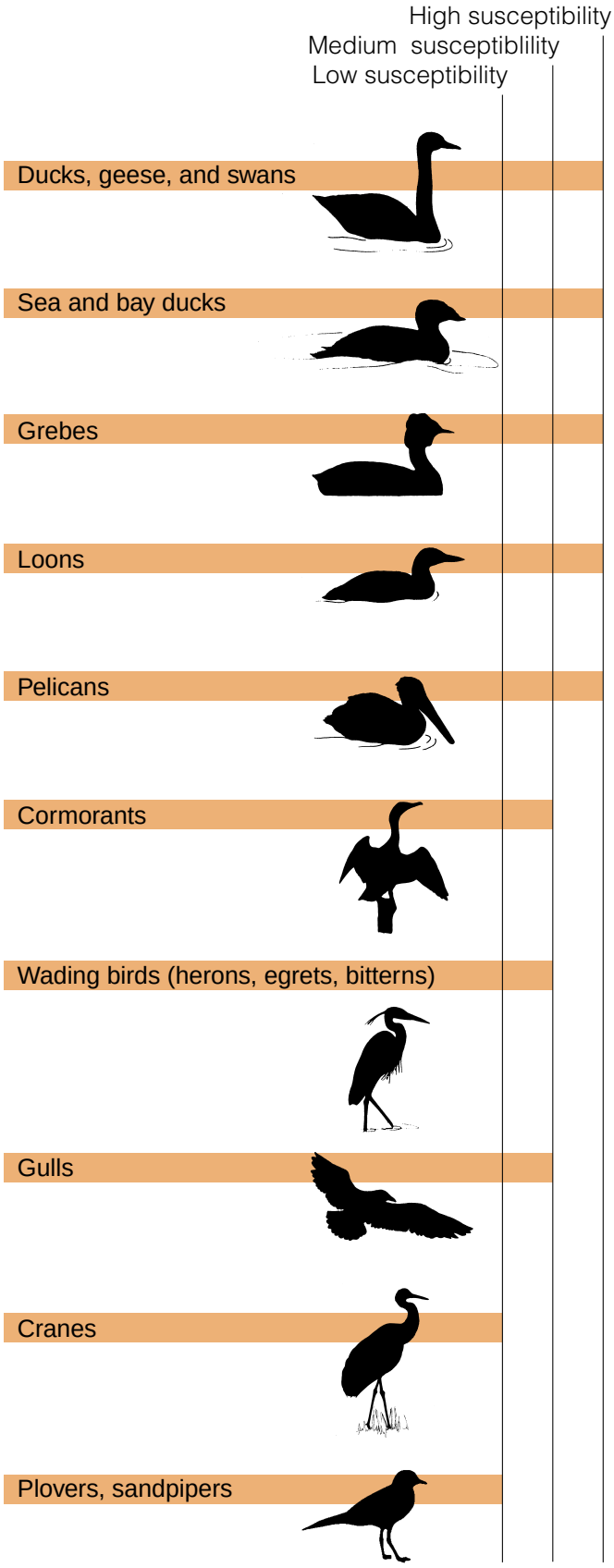

Figure 42.4 *Relative susceptibility of birds to oiling.*

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

Photo by James Runningen

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

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.*

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).

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.

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

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

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

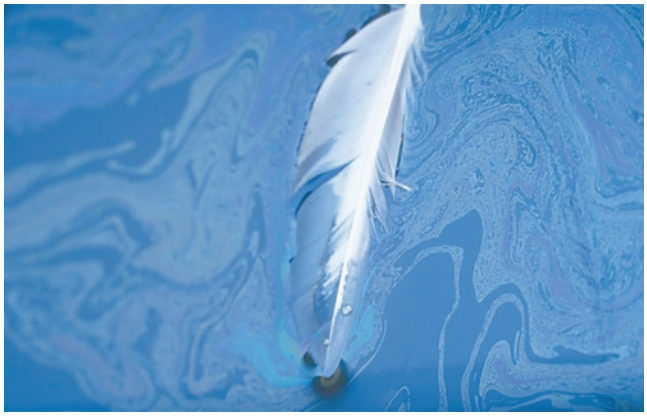

Photo by James Runningen

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

Photo by J. Christian Franson

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

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,

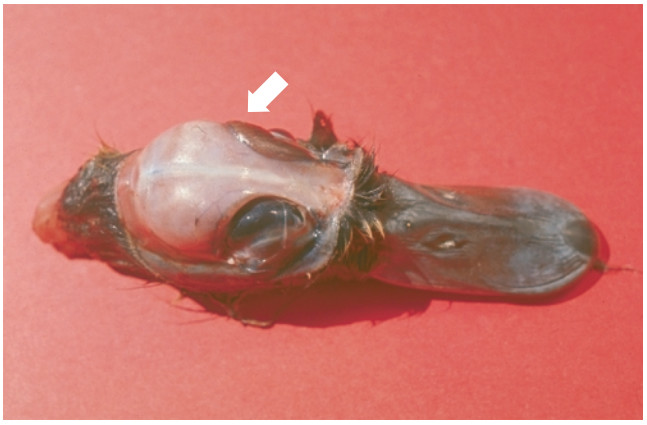

Photo by Milton Friend

Figure 42.10 *Swollen salt glands.*

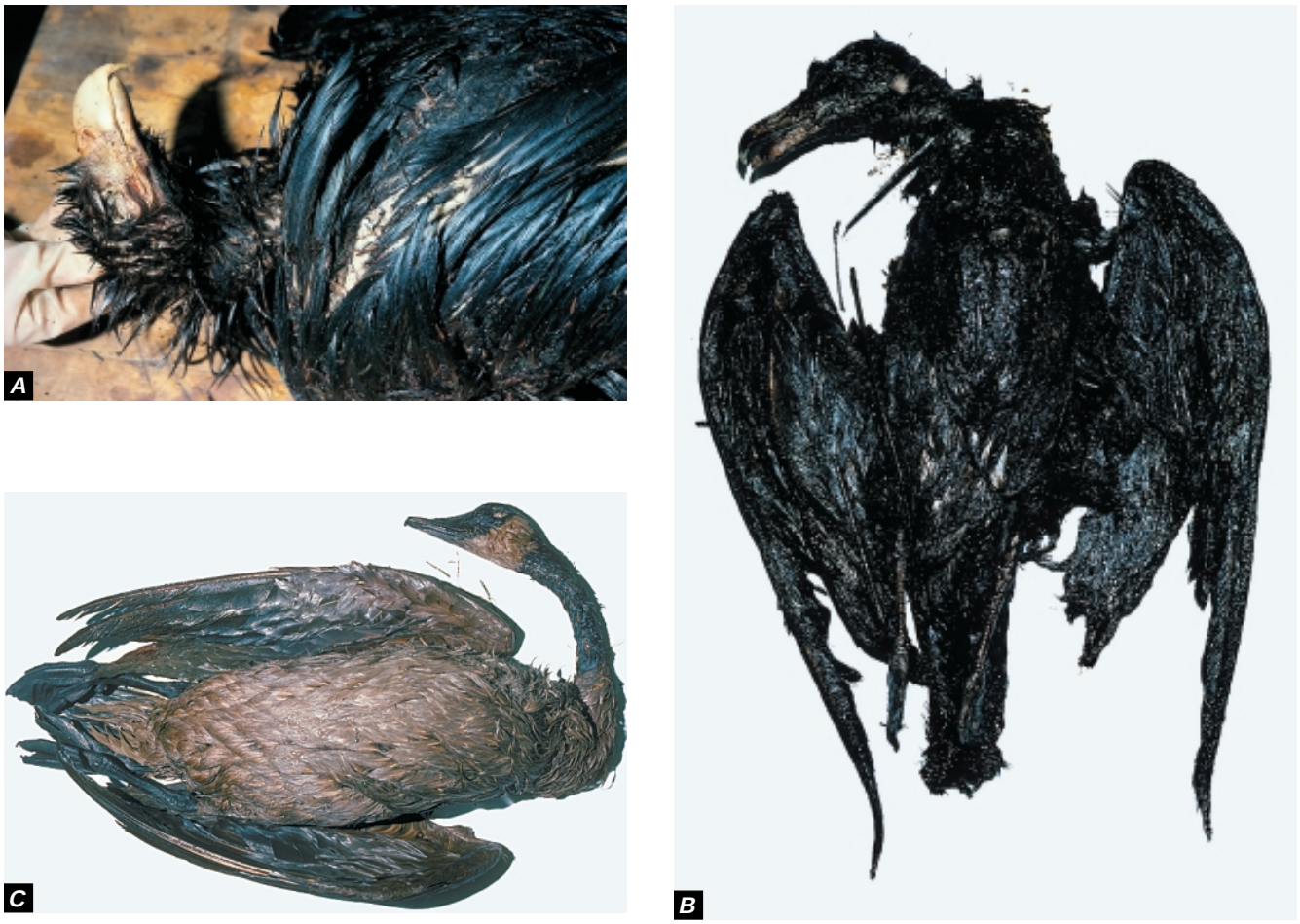

Figure 42.11 *Diagnosis of oiling is facilitated when oil is plainly visible on the carcass, such as on this bald eagle* ***(A)****, herring gull* ***(B)****, and Canada goose* ***(C)****.*

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

Tonie E. Rocke

Supplementary Reading

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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

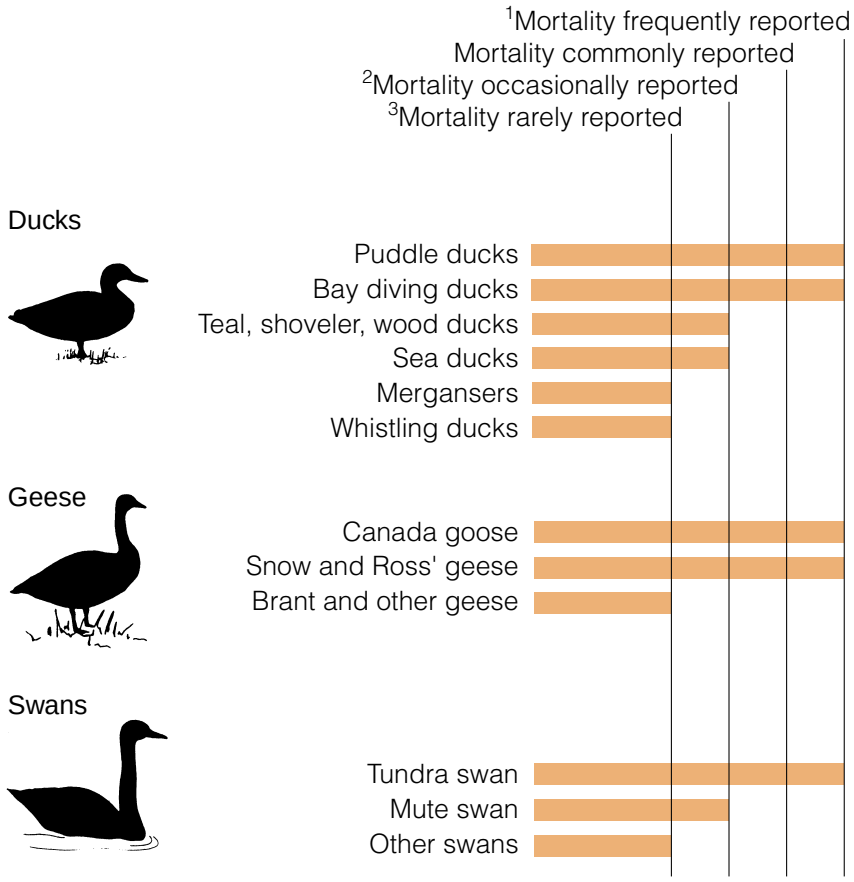
Lead poisoning has affected every major species of waterfowl in North America and has also been reported in a wide variety of other birds. The annual magnitude of lead poisoning losses for individual species cannot be precisely determined. However, reasonable estimates of lead-poisoning losses in different 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

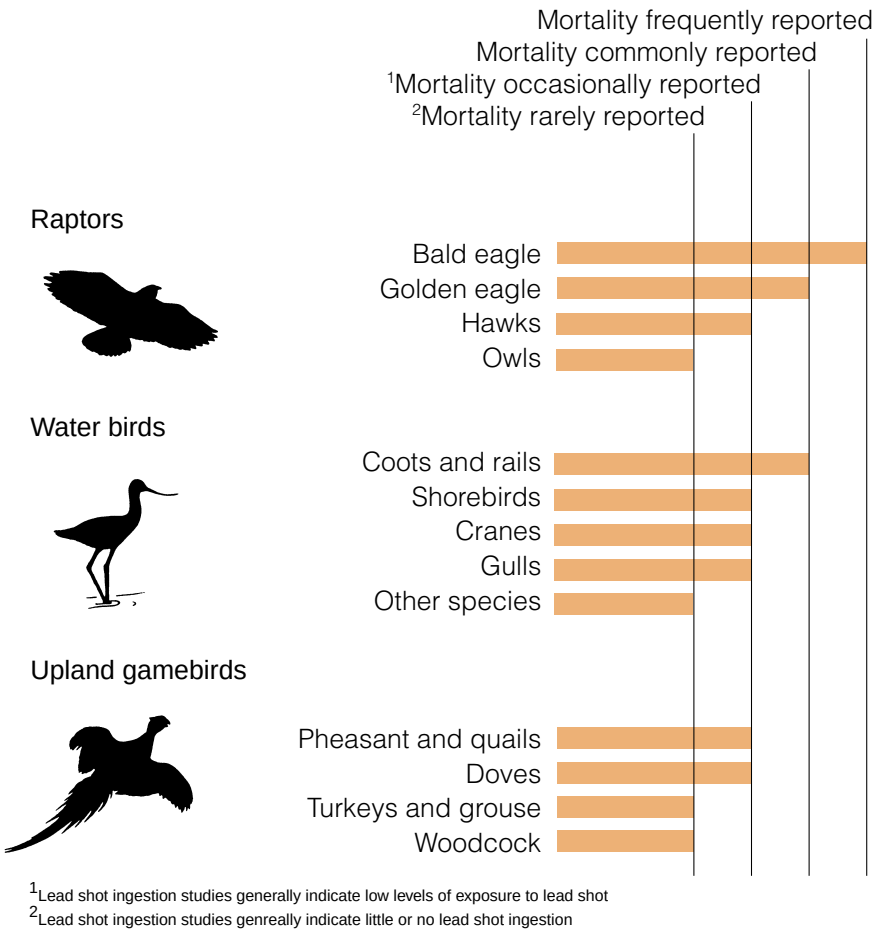


Figure 43.2 *Relative occurrence of lead shot poisoning in groups of birds other than waterfowl.*

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:

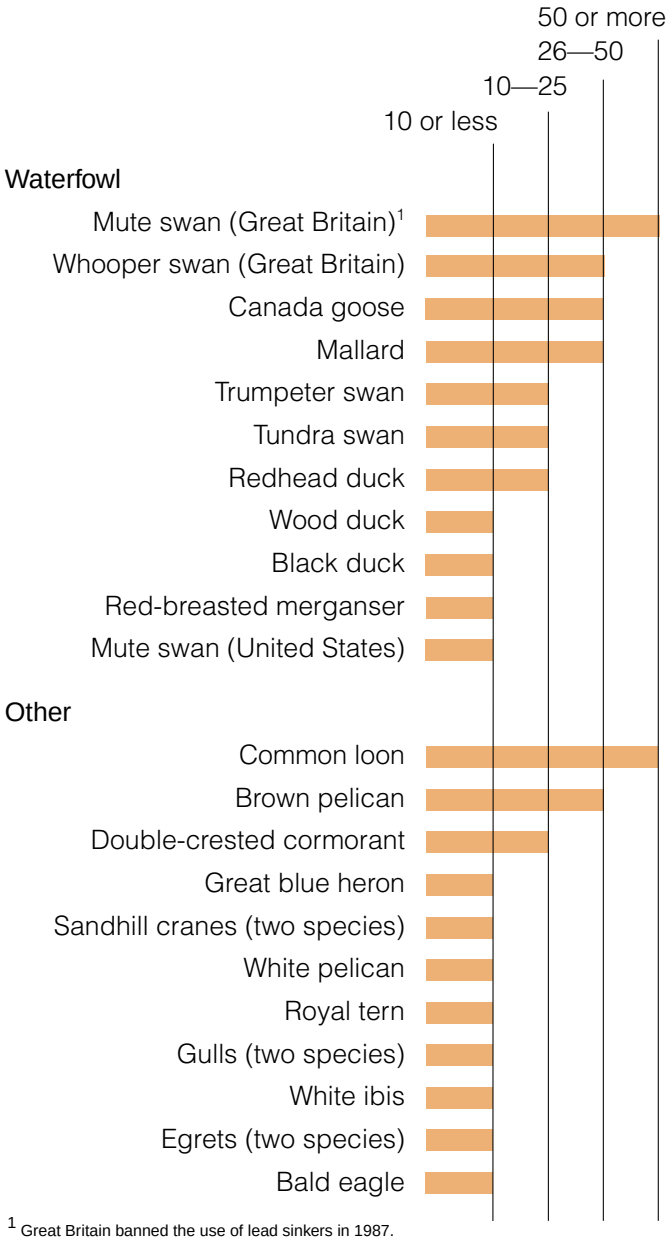
1. Severe wasting of the breast muscles (Fig. 43.11).
2. Absent or reduced amounts of visceral fat (Fig. 43.12).
3. Impactions of the esophagus or proventriculus in approximately 20–30 percent of affected waterfowl. These impactions may contain food items, or combinations of food, sand, and mud. The 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.*

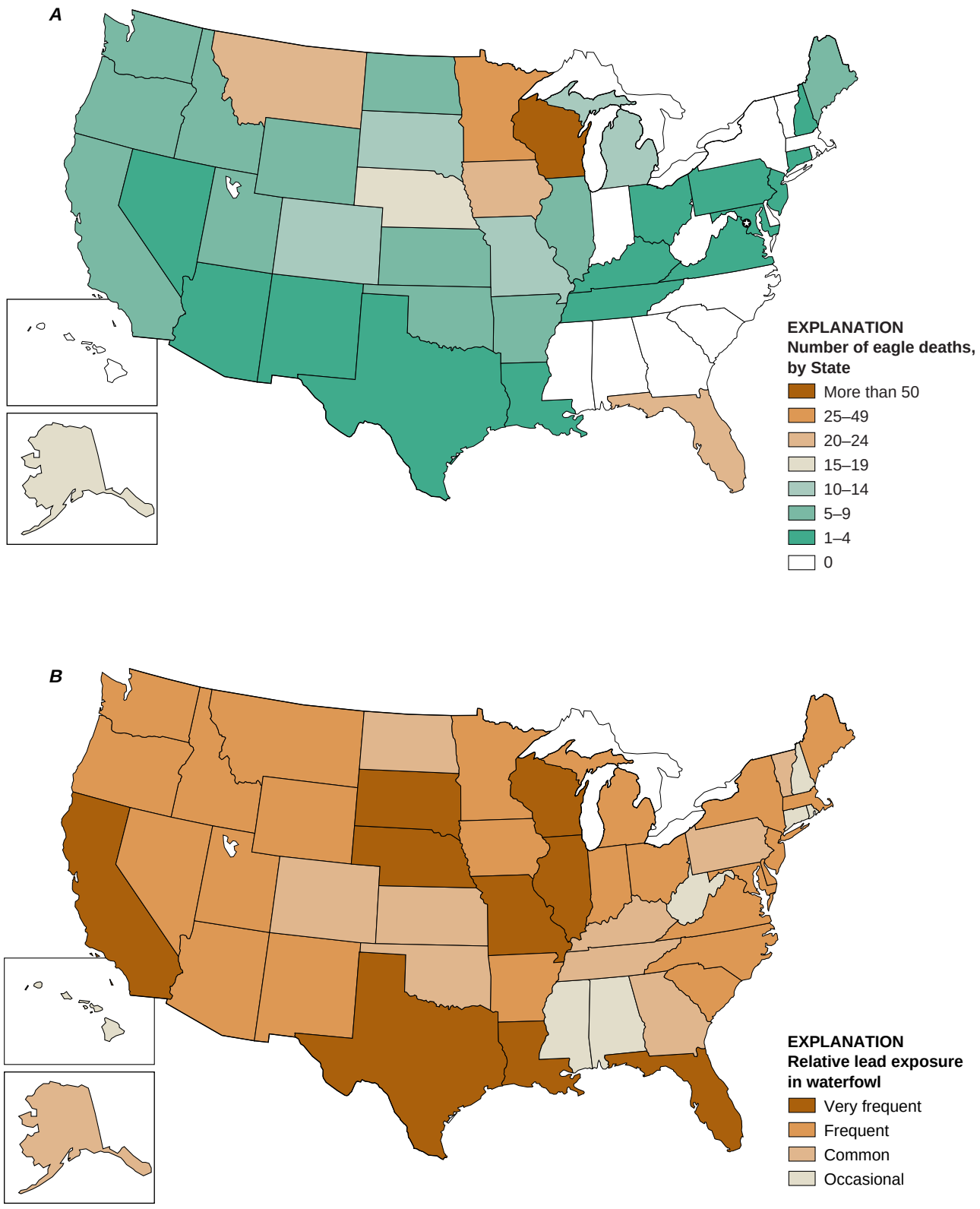


Figure 43.5 ***(A)*** *Diagnosed cases of lead poisoning in bald eagles though mid-April, 1996.* ***(B)*** *Relative occurrence of lead exposure in waterfowl prior to the 1991 ban on use of lead shot for waterfowl hunting. Evaluation is based on gizzard analysis and reported mortality.*

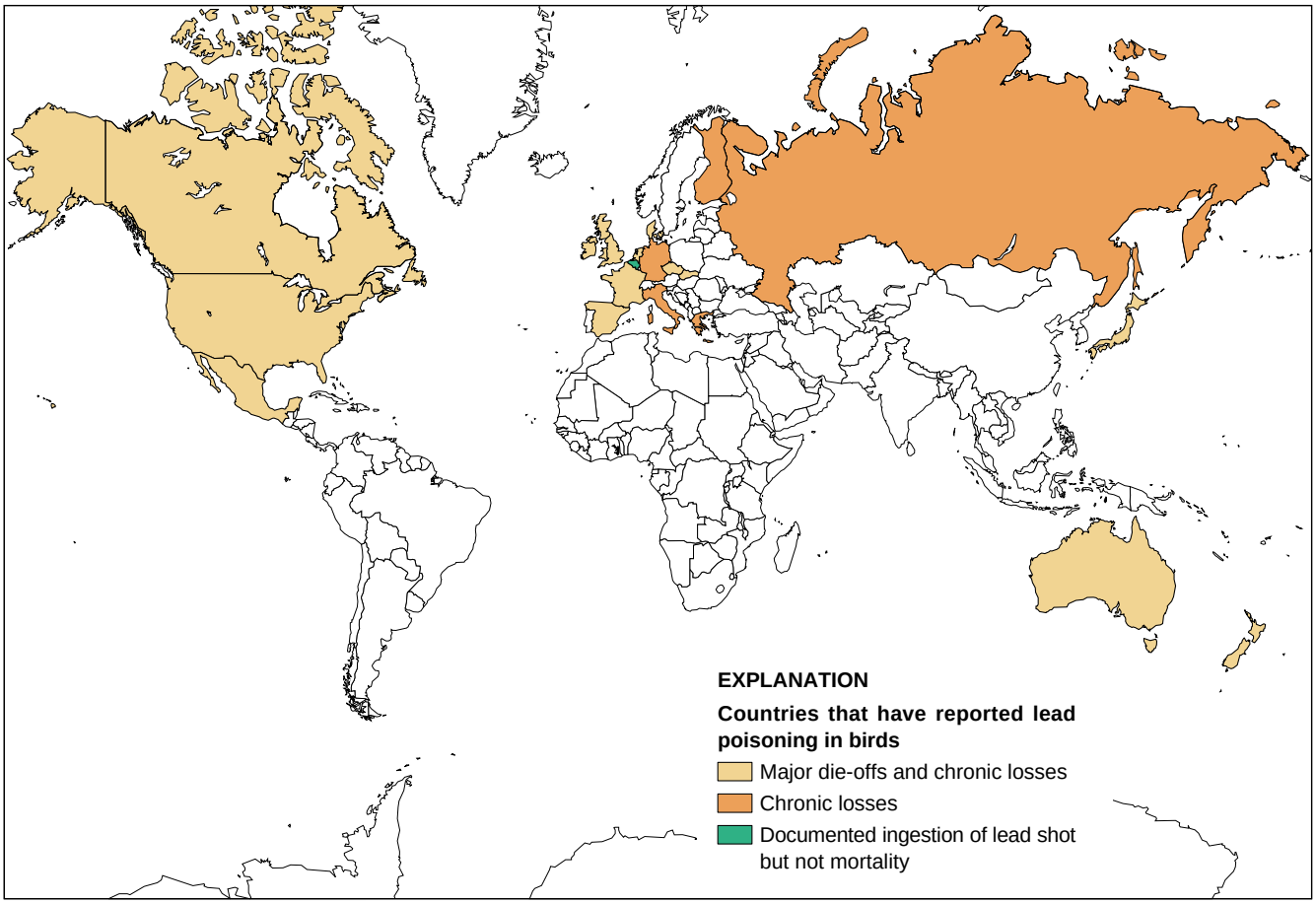


Figure 43.6 *Countries that have reported lead poisoning in birds.*

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

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

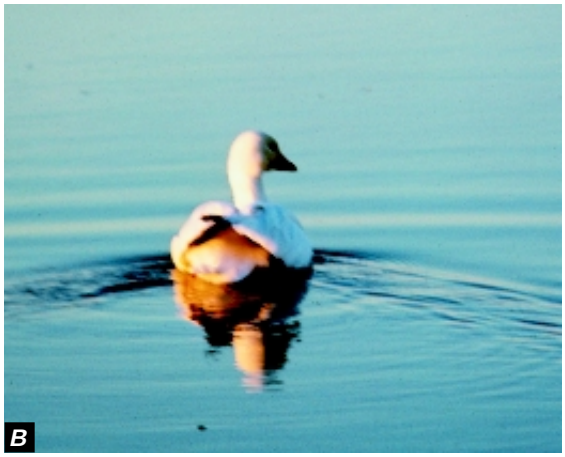


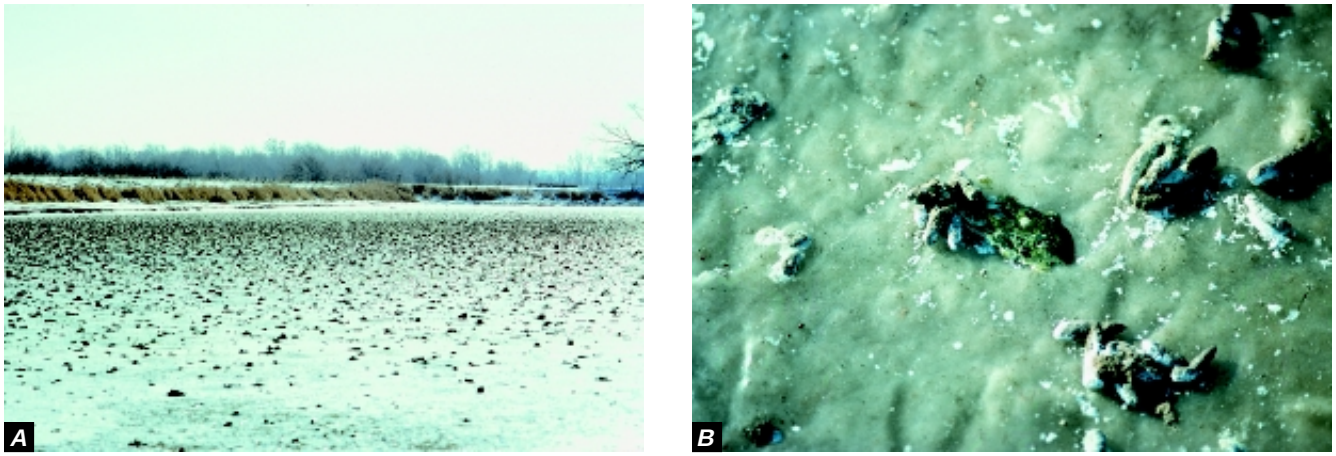
Photo by Milton Friend

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.*

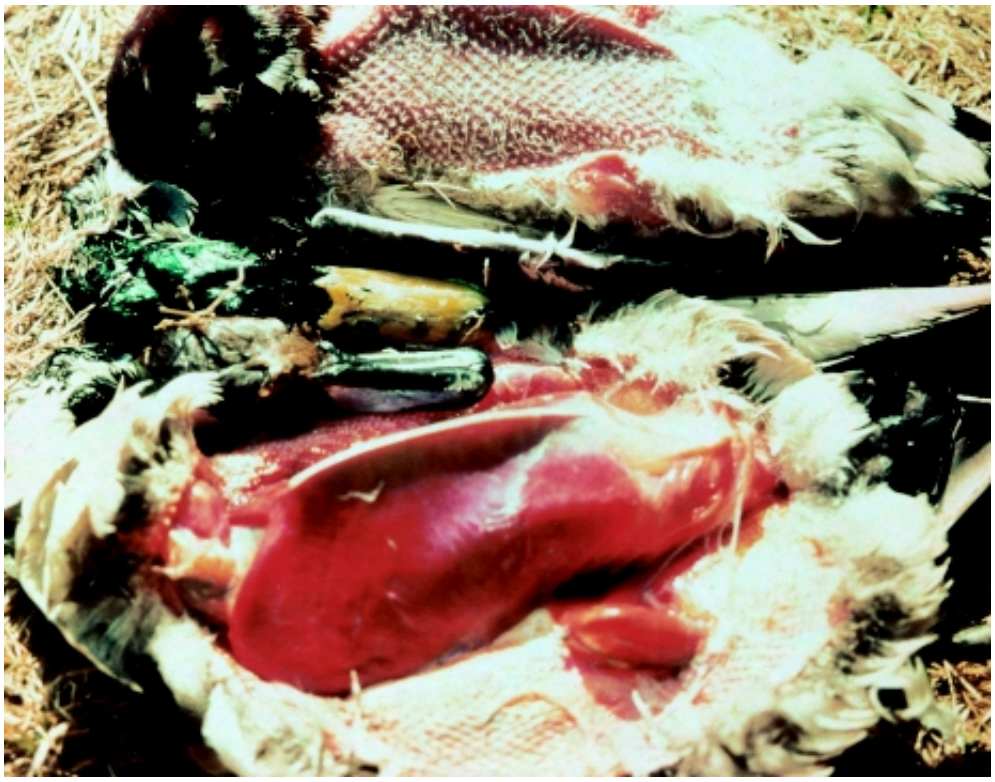
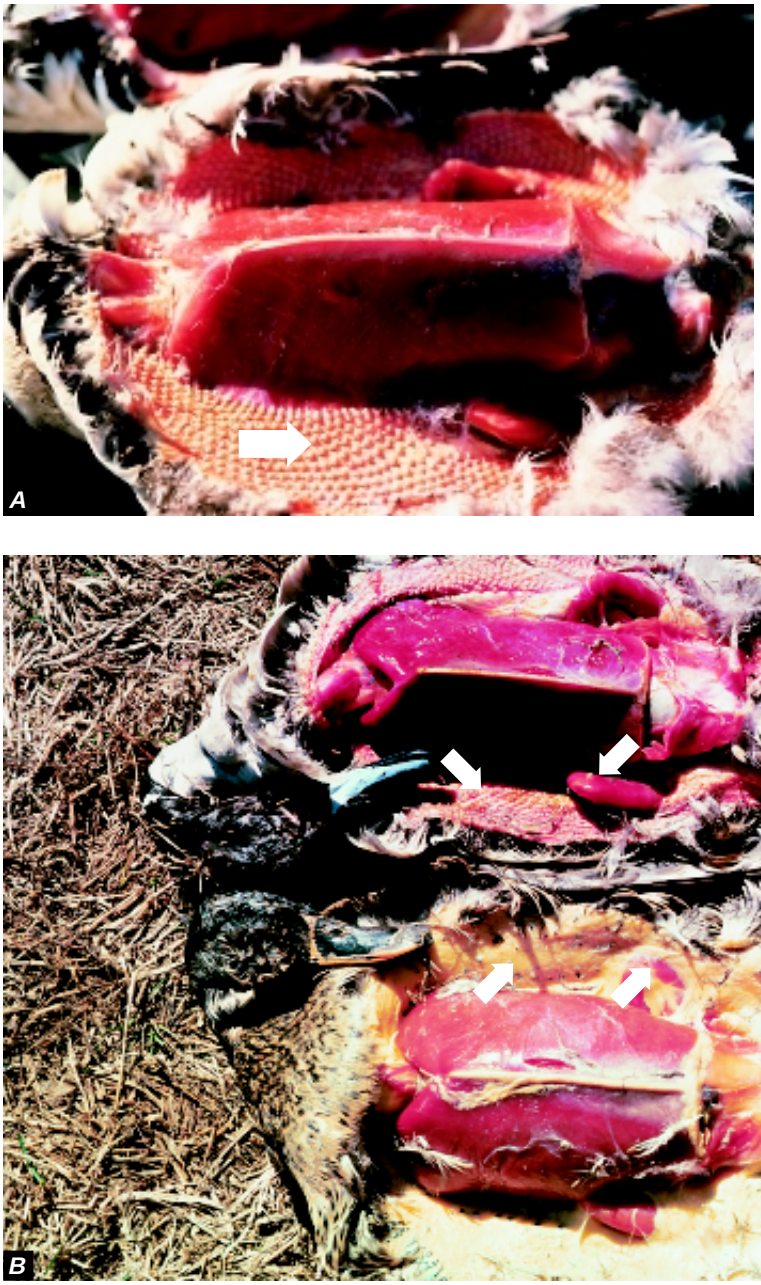


Photo by Milton Friend

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

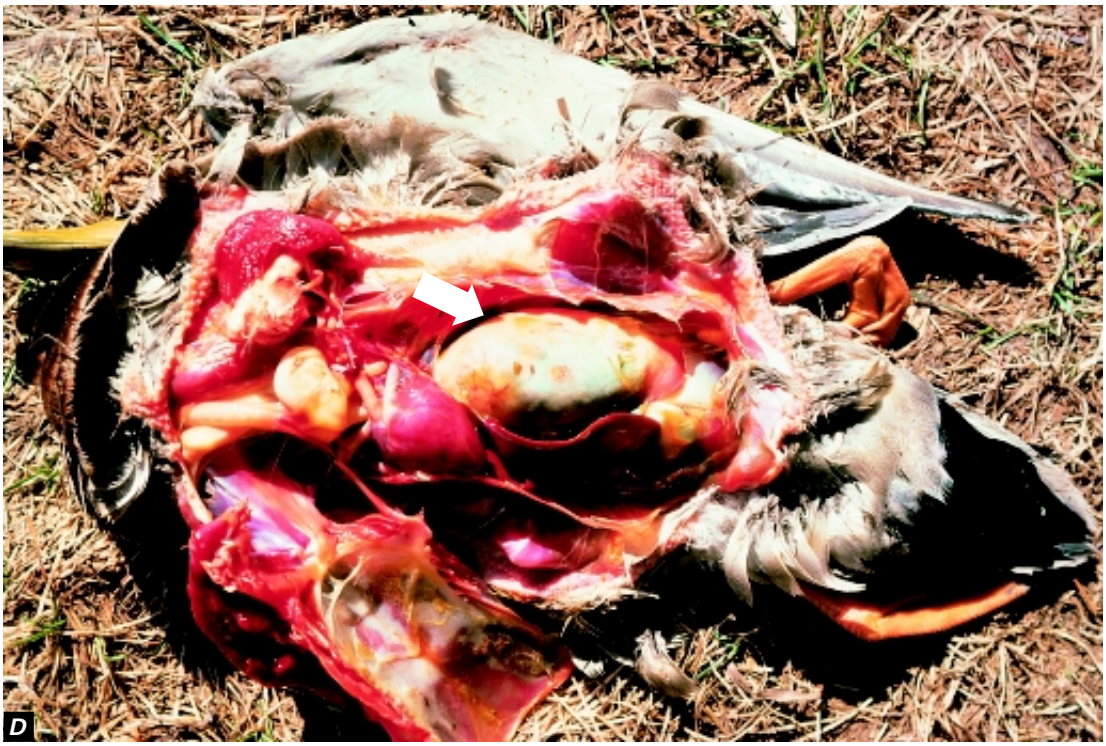

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Photo by Milton Friend

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.*

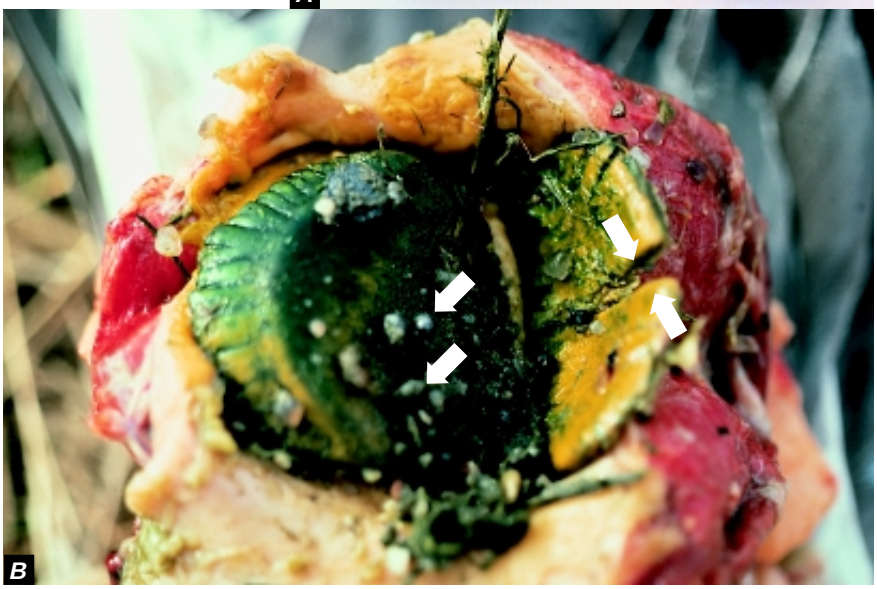

Photo from U.S. Fish and Wildlife Service files

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.*

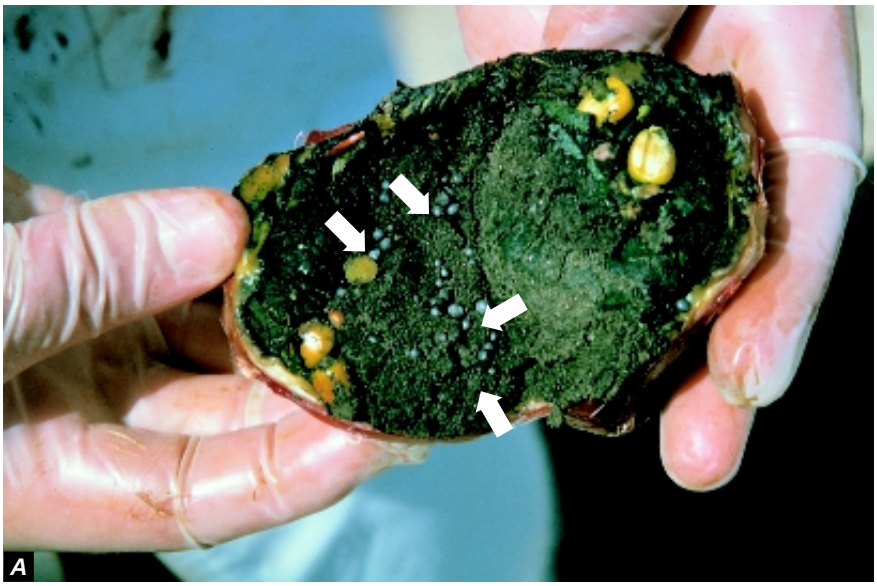

Photo by Milton Friend

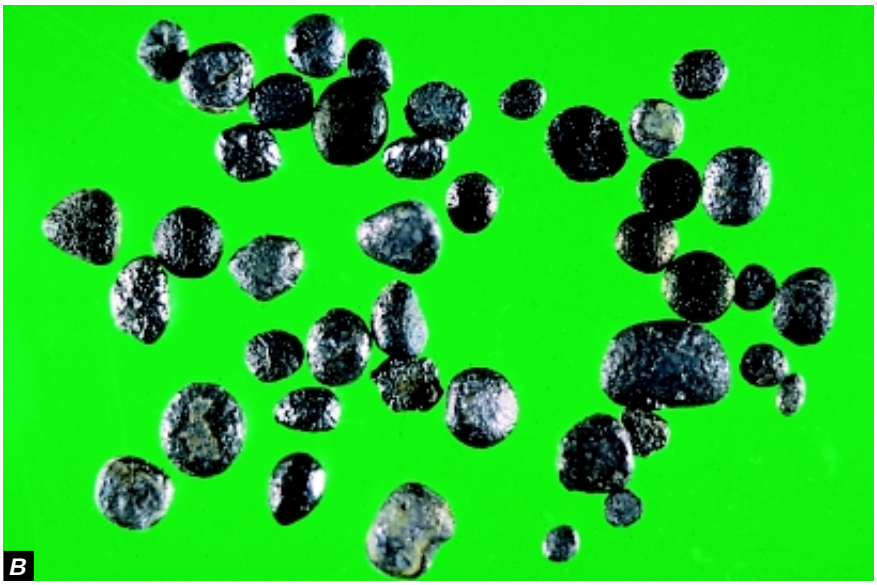

Photo by James Runningen

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

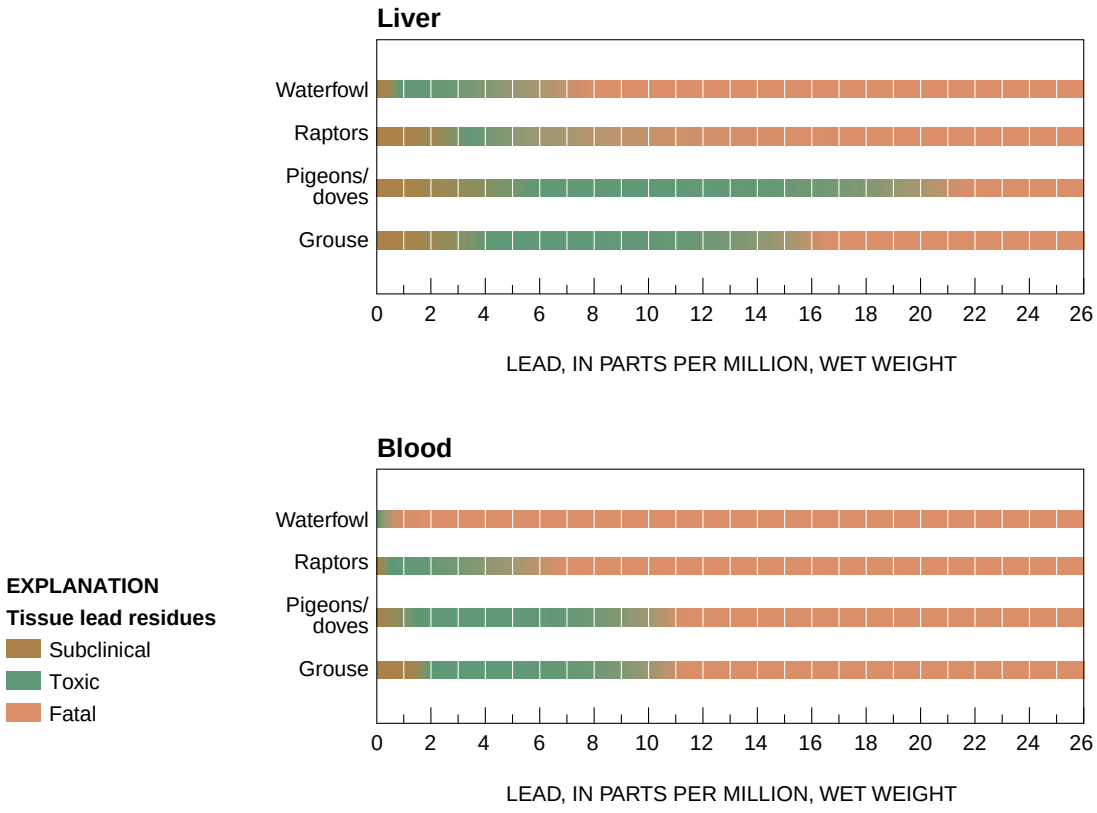

Figure 43.19 *Lead residues in the liver and blood associated with subclinical, toxic, and mortality findings in several groups of birds. Variability within groups occurs because of differences in species and a variety of factors affecting toxicity within species.*

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

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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

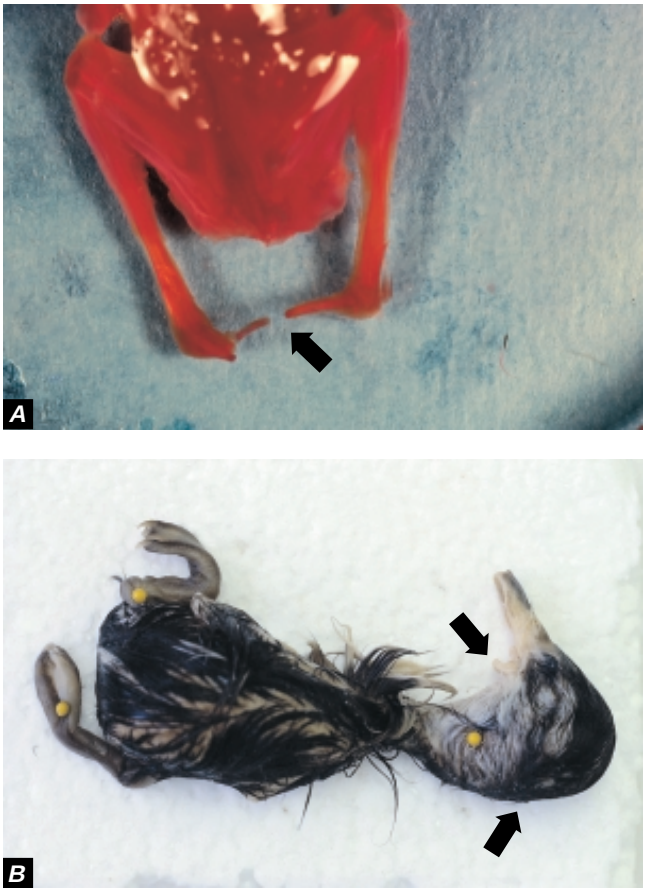
The seasonality of selenium poisoning depends on when birds use habitats that have high selenium concentrations.

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

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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.

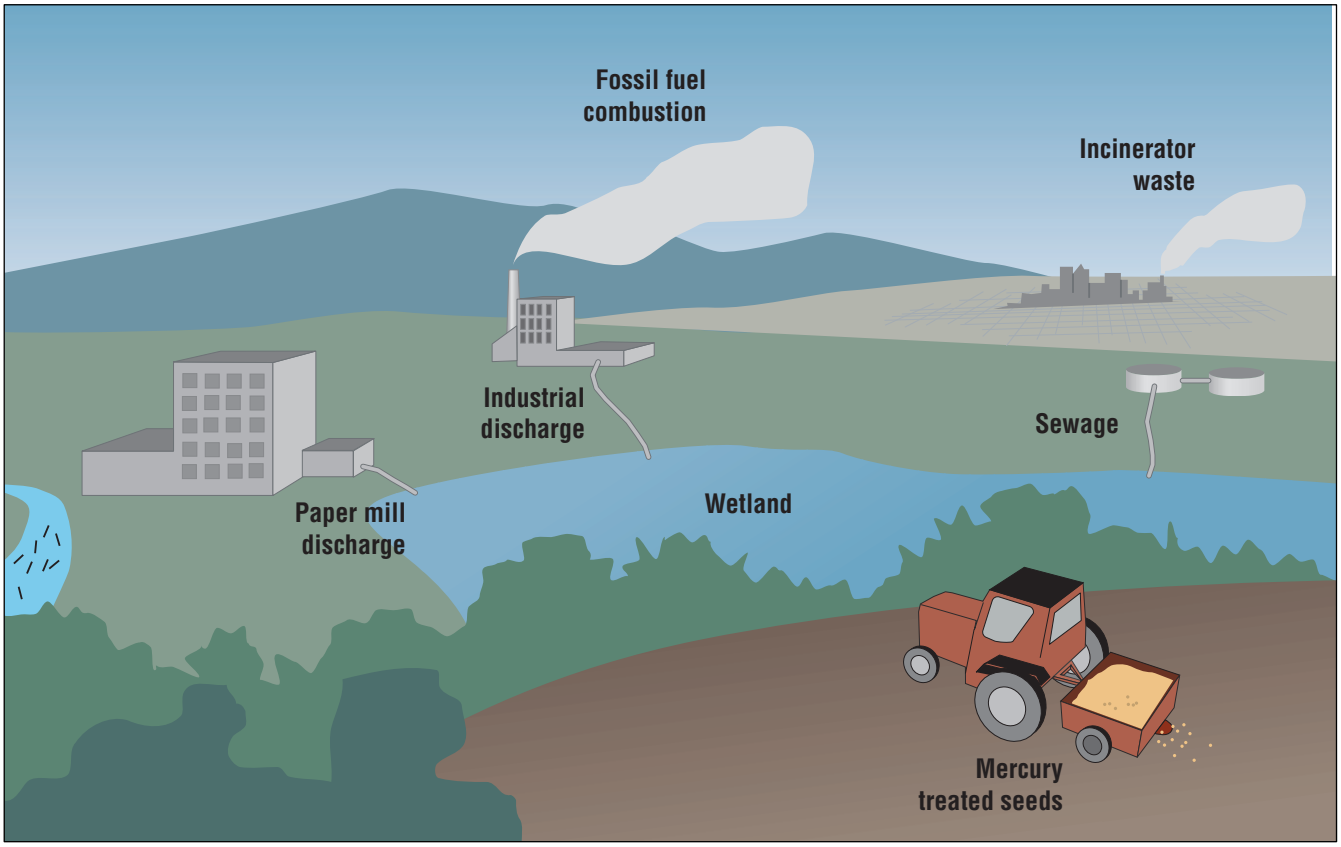

Figure 45.1 *Sources of mercury contamination.*

	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.

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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.

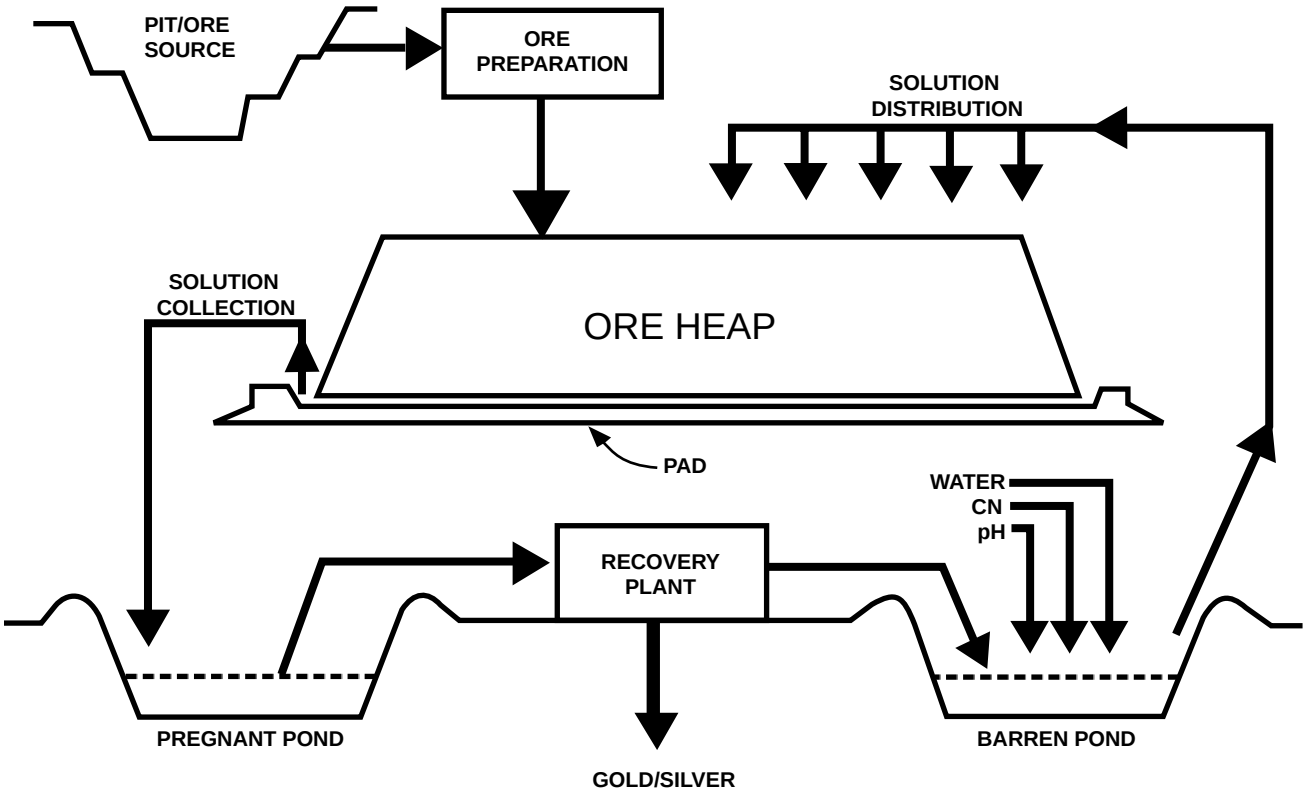


Figure 46.1 *Schematic of a typical heap leach system (Graphic by Robert Hallock, U.S. Fish and Wildlife Service).*

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.*

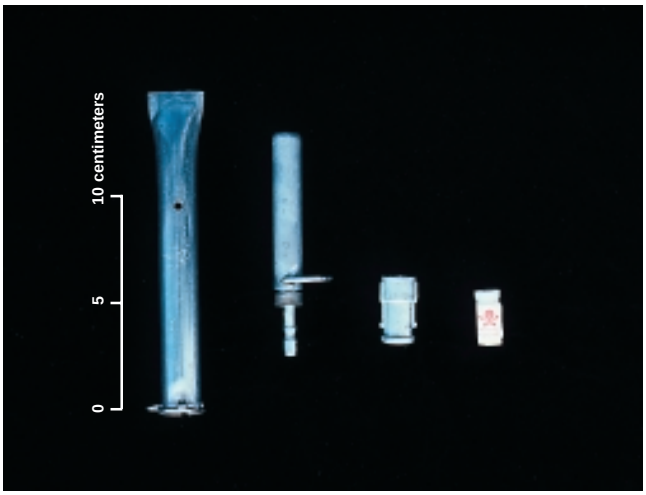


Photo by Guy Connolly, U.S. Department of Agriculture
National Wildlife Research Center

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.*

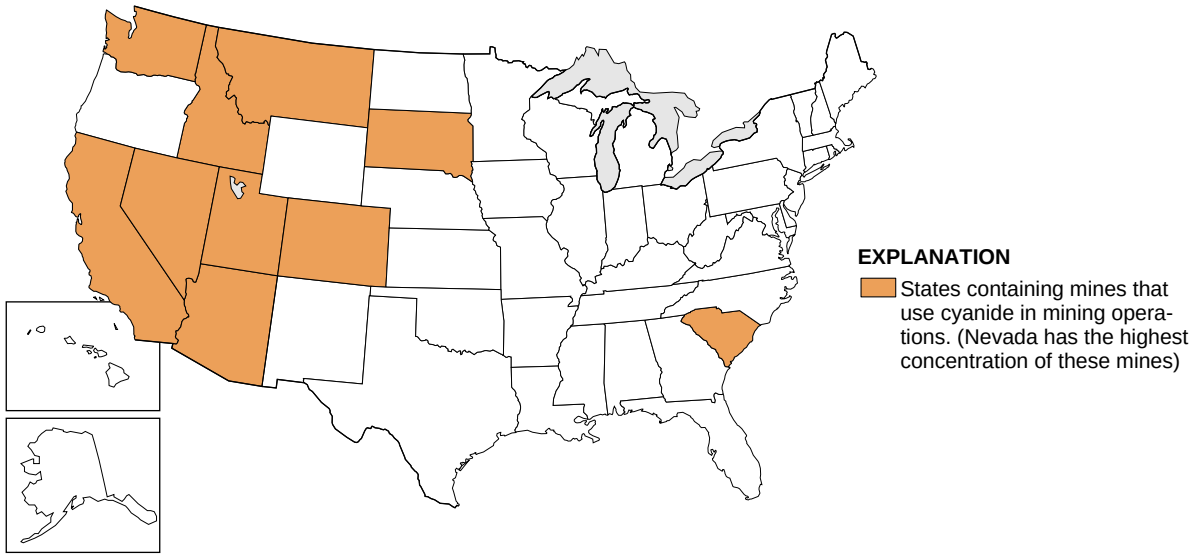


Figure 46.6 *States containing mines that use cyanide in leaching operations.*

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.

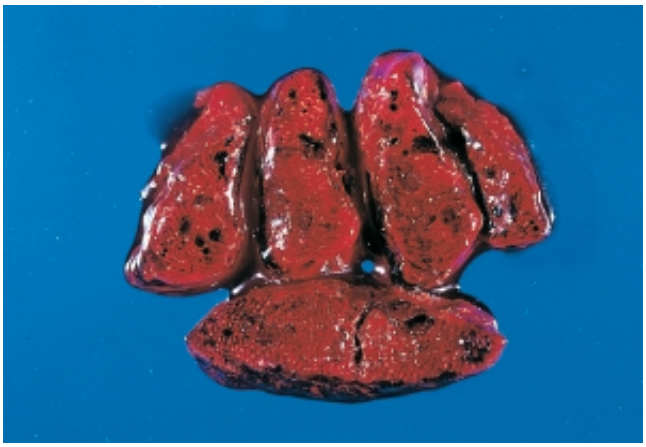


Photo by James Runningen

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

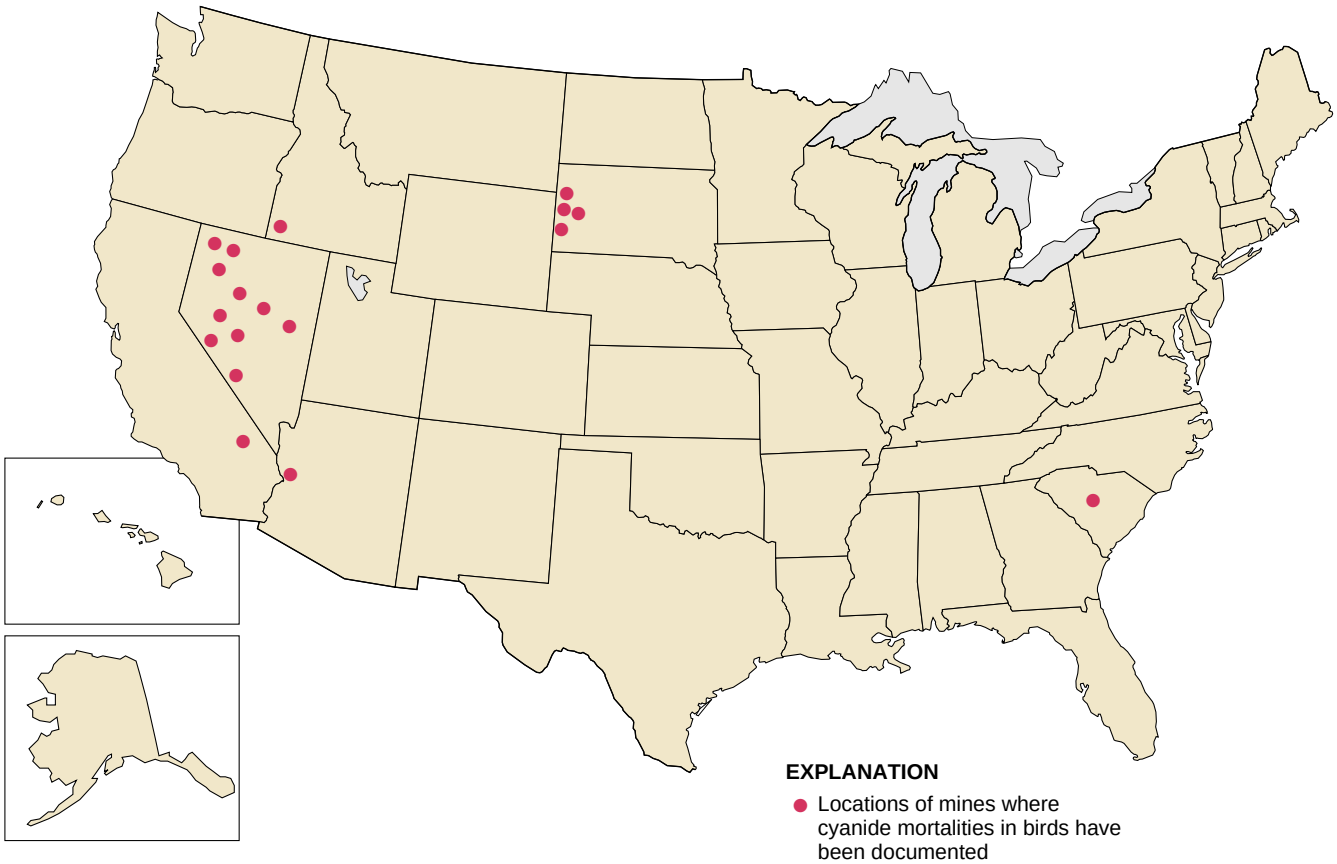


Figure 46.7 *Locations of mines where cyanide mortalities in birds have been documented.*

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

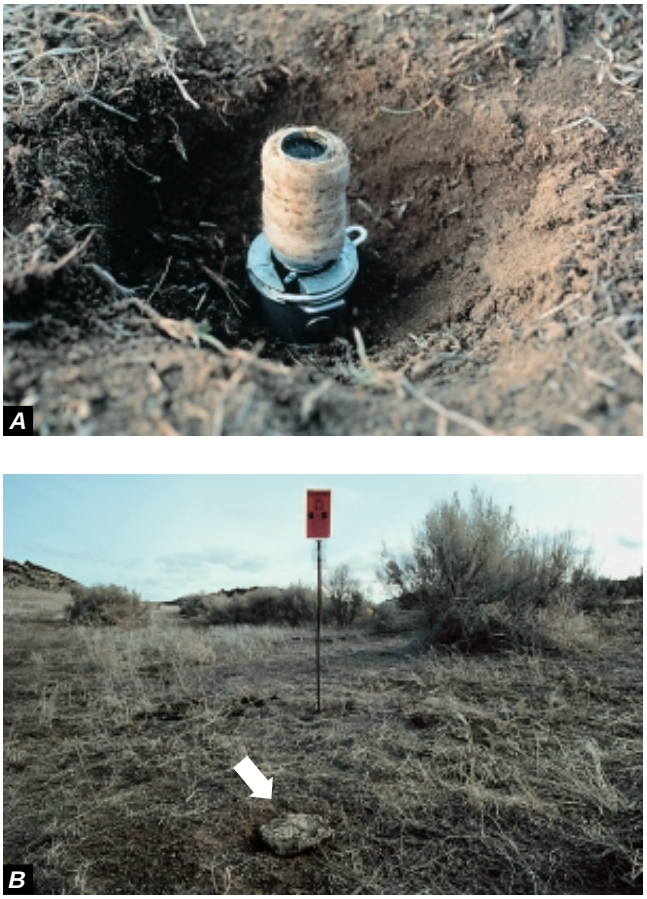
The primary method for preventing cyanide toxicosis at heap leach and carbon-in-pulp mill mining sites is to deny birds access to cyanide-contaminated water. This may or may not be difficult (or even possible) depending upon the size and configuration of a particular site. Successful methods used include 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

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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).

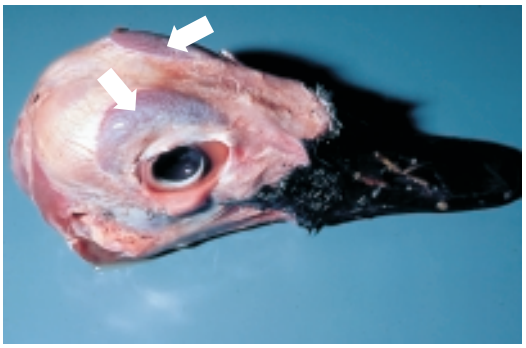
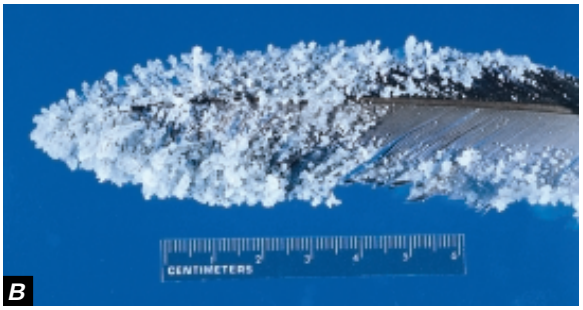


Photo by James Runningen

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

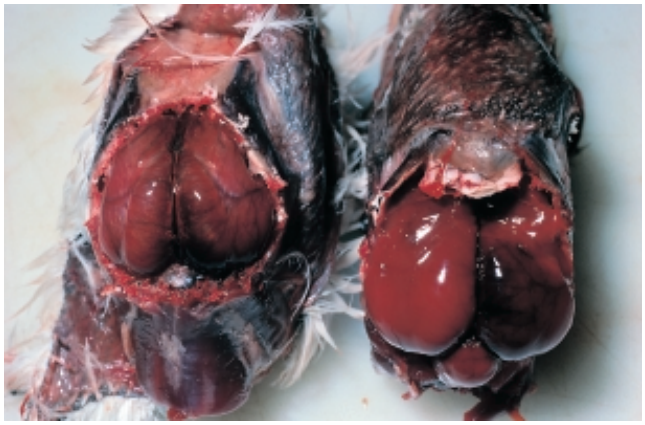


Photo by James Runningen

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

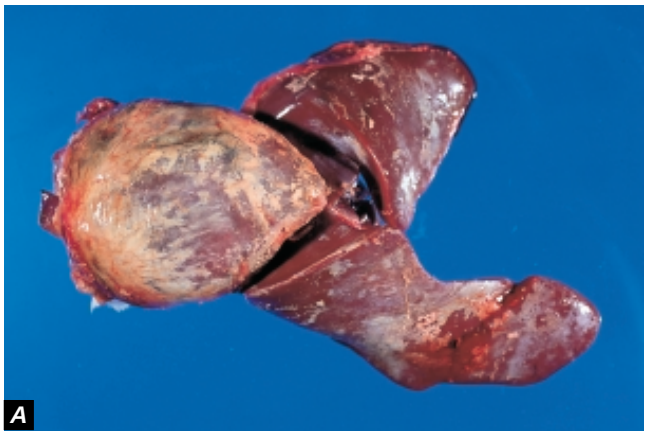


Photo by J. Christian Franson

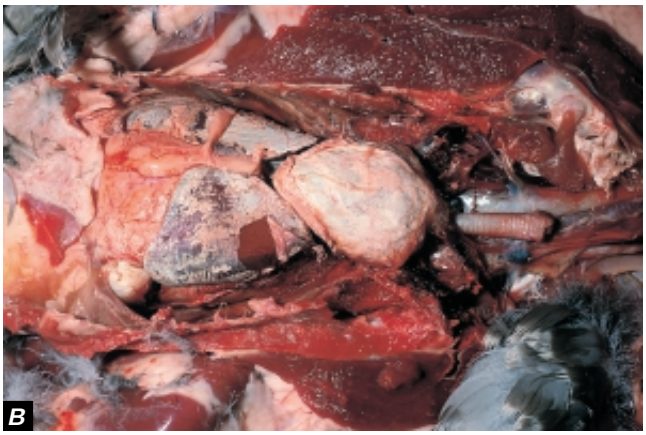


Photo by James Runningen

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.*

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.

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.

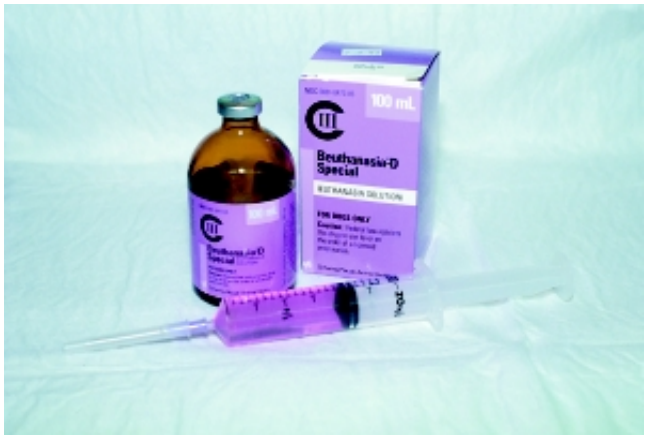


Photo by Nancy Thomas

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;

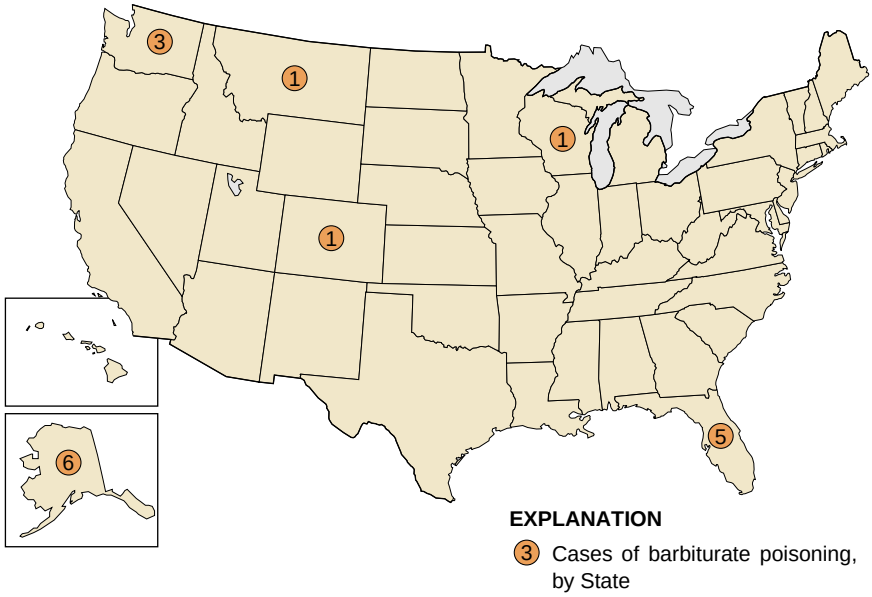


Figure 48.2 *Seventeen cases of barbiturate poisoning in bald and golden eagles have been reported by the National Wildlife Health Center from six States as of 1997.*

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	●	○	○	○	●	○	○
Carbofuran	●	●	●	●	●	●	●
Methiocarb	○	○	○	○	●	○	○
Unspecified	●	○	●	○	○	●	○

[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