

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 vulner-

ability 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. LD₅₀ 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
Mexacarbamate	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

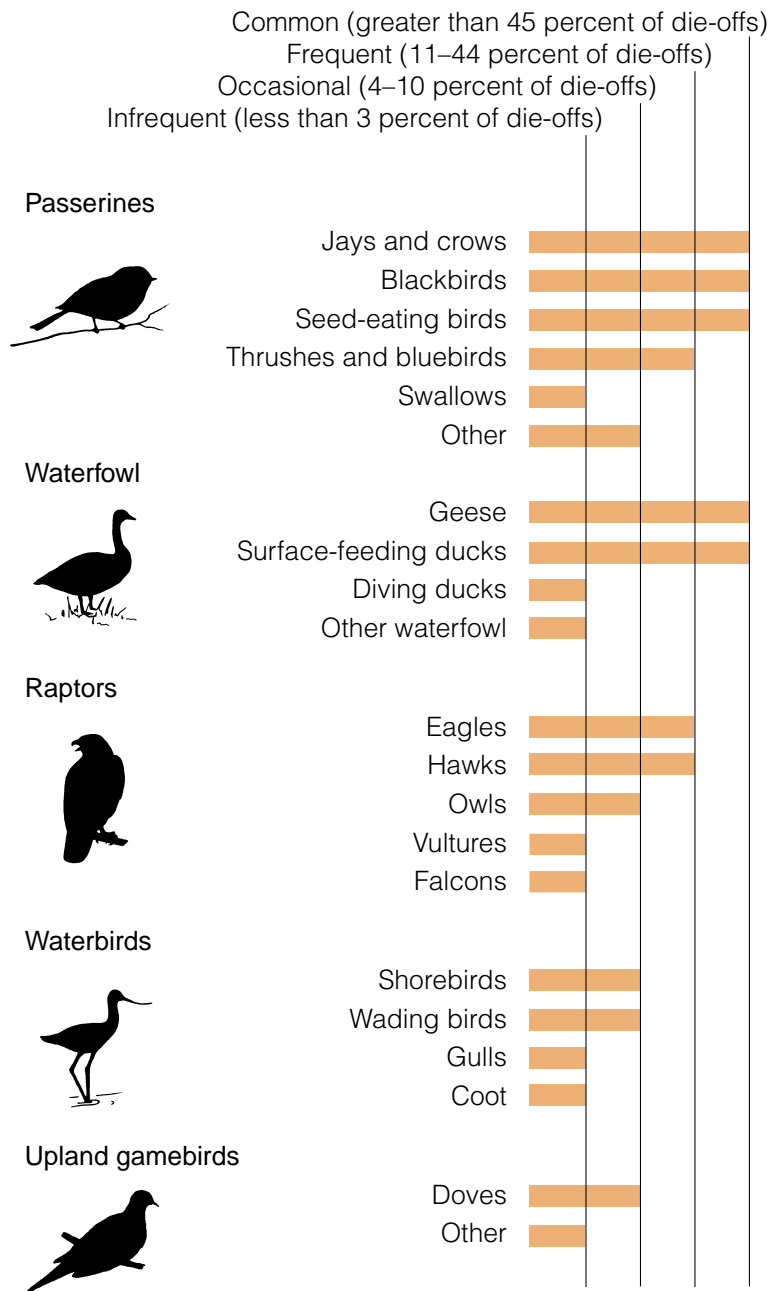


Figure 39.1 Frequency of occurrence of major groups of birds in documented organophosphorus and carbamate pesticide mortality events from 1986–95 (National Wildlife Health Center data base).

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).

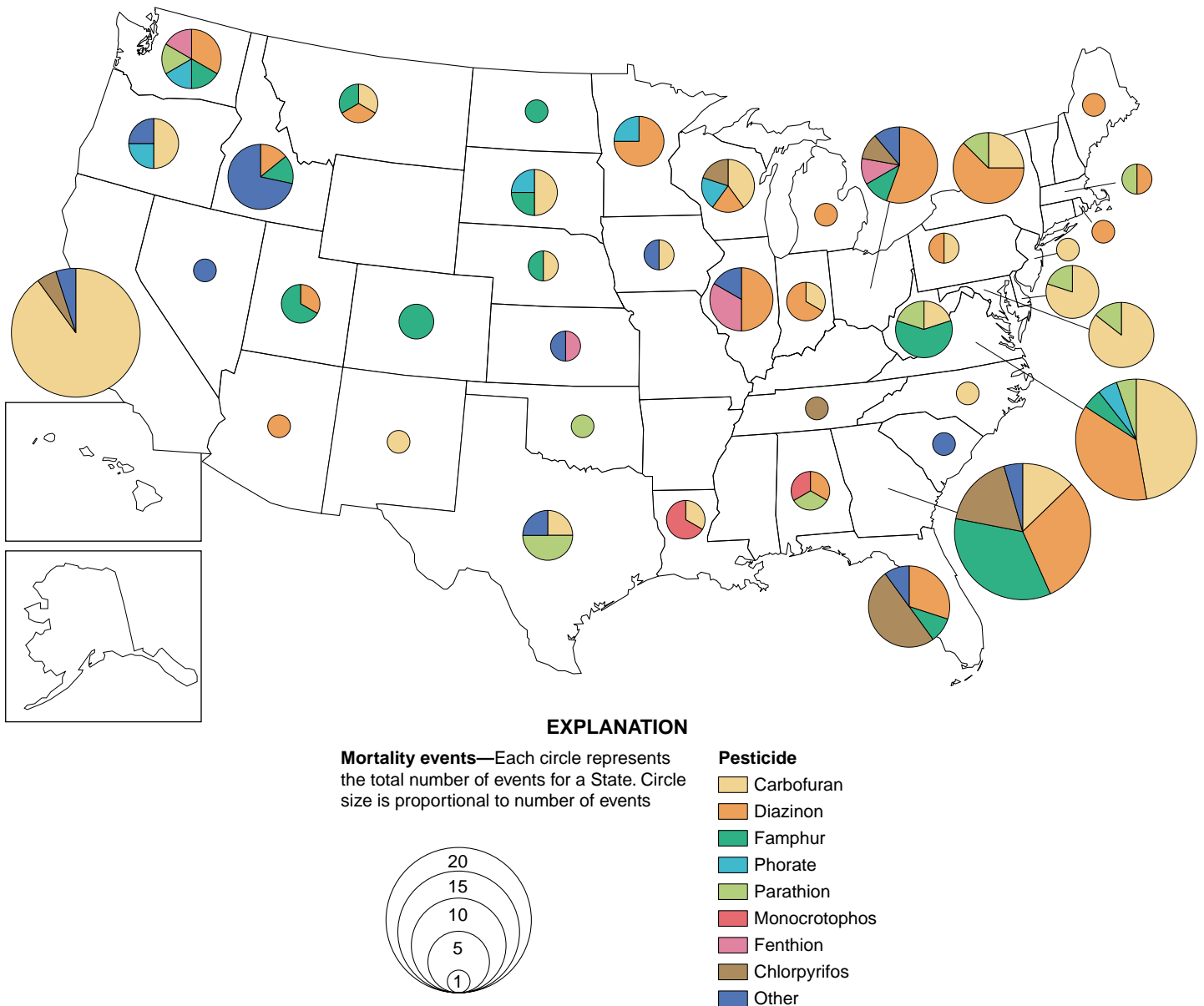


Figure 39.2 Distribution of 181 avian mortality events caused by organophosphorus and carbamate pesticides, 1986–1995 (National Wildlife Health Center data base).

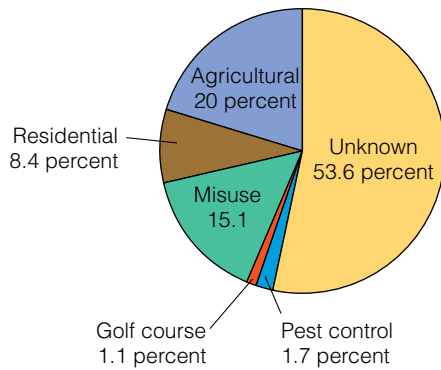


Figure 39.3 Applications associated with avian mortality caused by organophosphorus and carbamate pesticides from 1986–95 (National Wildlife Health Center data base).

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.

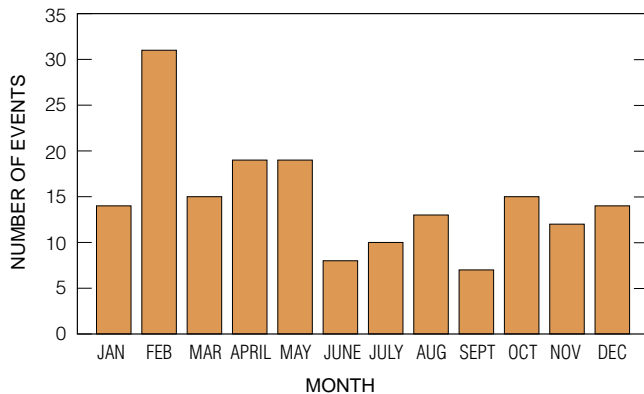


Figure 39.4 Avian mortality events due to organophosphorus and carbamate pesticides by date of onset, 1986–95 (National Wildlife Health Center data base).

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 (exophthalmia)
- 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.



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-



Photo by J. Christian Franson



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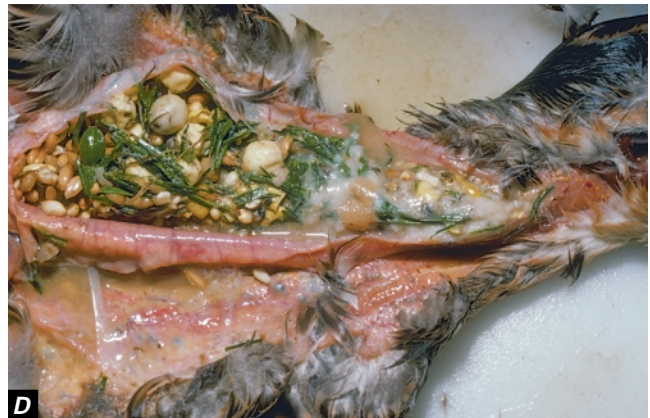


Photo by James Rummigen

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.

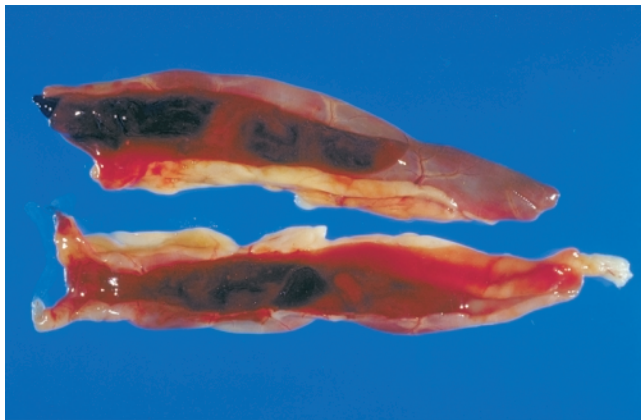


Photo by Paul Soler

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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