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December 1999

### Biotoxins (Field Manual of Wildlife Diseases)

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## Section 6

# **Biotoxins**

**Algal Toxins**

**Mycotoxins**

**Avian Botulism**

*Aerial view of a large dinoflagellate bloom in near-shore ocean waters*

Photo by Peter Frank, Scripps Institute of Oceanography

# Introduction to Biotoxins

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“Ecological toxicology is the study of all toxicants produced by living organisms and of the ecological relationships made possible by these poisons.”  
(Hayes)

“In all communities chemical interrelations are important aspects of the adaptation of species to one another; in some communities chemical relations seem to be the principal basis of species niche differentiation and community organization.” (Whittaker and Feeny)

“Undoubtedly there is much to be learned from finding out how the battle [between toxicants produced by living organisms and host defenses developed in response to these toxicants] has been fought for the last several million years.”  
(Hayes)

Biotoxins are usually defined as poisons that are produced by and derived from the cells or secretions of living organisms. These natural poisons include some of the most toxic agents known and they are found within a wide variety of life forms. Organisms that produce such toxins are generally classified as being venomous or poisonous. The classification of venomous is usually associated with animal life forms such as poisonous reptiles and insects that have highly developed cellular mechanisms for toxin production and that deliver their toxins during a biting (rattlesnake) or stinging (black widow spider) act. Poisonous organisms are generally thought of as those that deliver toxins by being ingested or by their secretions being ingested by another organism. Therefore, these toxins are essentially forms of food poisoning. Readers should appreciate that virtually all venomous organisms are poisonous but many poisonous organisms are not venomous. This Section will address poisonous, but not venomous, organisms, and it includes the perspective of biotoxins as products of plants and lower life forms.

Birds become poisoned by a broad array of biotoxins. The chapter about avian botulism involves microbial toxins produced within replicating *Clostridium botulinum* bacteria. The potency of toxins that are produced by the disease-causing Clostridia are legendary, and the toxins include such human

diseases as tetanus and lethal botulism food poisoning. Avian botulism is currently the most important disease of waterfowl and shorebirds, nationally and internationally, and outbreaks of this disease commonly kill tens of thousands of birds during a single event. Up to a million birds have recently been lost within a single location during the course of a protracted outbreak.

Because many avian botulism die-offs occur on the same wetlands year after year, one of the primary areas of research on this disease has focused on identifying and understanding the microenvironmental characteristics that contribute to a mortality event. The development of wetland-specific risk assessment tools will enable wildlife disease specialists and natural resource managers to more effectively manage avian botulism.

Fungi are an additional source of microbial biotoxins that cause the death of free-ranging wild birds. Mycotoxins, which are toxins produced by fungi, have received considerable study because of their effects on food animals and humans. In poultry, for example, many types of mycotoxins are known to cause problems that include mortality, decreased growth, impaired reproduction, immunosuppression, and pathologic effects on a variety of other organ systems. Although these toxins have received little study in wildlife, a growing body of literature documents similar effects of mycotoxins in a variety of free-ranging species. The chapter about mycotoxins illustrates the capabilities of aflatoxins and trichothecenes to cause large-scale bird losses as the result of bird ingestion of food contaminated by molds that produce these toxins. As more becomes known about the occurrence of mycotoxins

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All quotes from:

Hayes, Wayland, J., Jr., 1991, in Hayes, Wayland J., Jr., and Laws, Edward R., Jr., eds., Handbook of pesticide toxicology, v. 1, General principles: New York, Academic Press, p. 7–8.

in the natural environment, and as analytical techniques for the specific toxins become more commonly available, it is likely that more and more cases of mycotoxicosis will be reported in wildlife.

The range of living organisms that cause poisoning in wild birds is further illustrated by plant toxins in the chapter about algal toxins. Less is known about poisoning of birds from toxic plants than is known about poisoning from bacterial and fungal toxins. Plant toxins other than algal toxins that have caused bird mortality have rarely been reported. Choke cherry seeds contain chemical compounds that release cyanide upon digestion if the seed capsule is broken during digestion. Songbirds have been killed by cyanide poisoning from eating these seeds. Waterfowl mortality has been attributed to ingestion of castor beans, which results in intoxication from ricin, the active ingredient within the seed that causes poisoning. A small number of other reports of plant toxins causing wild bird mortality also exist.

The so-called algal toxins are produced by a variety of organisms, including true algae, dinoflagellates (aquatic protozoa), and blue-green algae, and are the least understood of the biotoxins covered in this Section. Algal blooms, especially red tides and blue-green blooms, wreak aesthetic and

economic havoc in many freshwater and marine environments because of the potential for toxins to be present. Perhaps one of the most widely recognized toxins in this group is saxitoxin, the agent of paralytic shellfish poisoning, which causes occasional human deaths and renders many tons of shellfish inedible throughout the world.

Algal toxins are likely to become increasingly recognized as a cause of waterbird mortality. Eutrophication of inland waterbodies due to nutrient loads is causing more algal blooms within those waters, many of which are used by large numbers of water birds. Enhanced technology and increased study are needed to better understand the ecology of algal blooms and the production of toxic components that are hazardous to bird life. With the exception of avian botulism, biotoxins as a cause of disease in wild birds have received little study. However, there should be no debate regarding the need for study since disease caused by biotoxins extends beyond direct mortality. Impaired immune system function or immunosuppression and cancers caused by biotoxins have both been documented in animals and humans. Other effects on wildlife are also likely because of the diversity of disease impacts seen in humans and domestic animals.



## Chapter 36

# Algal Toxins

### Synonyms

*Red tide toxins, phycotoxins*

Periodic blooms of algae, including true algae, dinoflagellates, and cyanobacteria or blue-green algae have been reported in marine and freshwater bodies throughout the world. Although many blooms are merely an aesthetic nuisance, some species of algae produce toxins that kill fish, shellfish, humans, livestock and wildlife. Pigmented blooms of toxin-producing marine algae are often referred to as “red tides” (Fig. 36.1). Proliferations of freshwater toxin-producing cyanobacteria are simply called “cyanobacterial blooms” or “toxic algal blooms.” Cyanobacterial blooms initially appear green and may later turn blue, sometimes forming a “scum” in the water (Fig. 36.2).

Although algal blooms historically have been considered a natural phenomenon, the frequency of occurrence of harmful algae appears to have increased in recent years. Agricultural runoff and other pollutants of freshwater and marine wetlands and water bodies have resulted in increased nutrient loading of phosphorus and nitrogen, thus providing conditions favorable to the growth of potentially toxic algae. The detrimental impact of red tides and cyanobacterial blooms on wetland, shore, and pelagic species has long been suspected but not often been substantiated because information on the effects of these toxins in fish and wildlife species is lacking and diagnostic tools are limited.

### Cause

Some dinoflagellates and cyanobacteria produce toxins that can affect domestic animals and humans. Some of these toxins such as domoic acid, saxitoxin (paralytic shellfish poisoning or PSP toxin), brevetoxin, and cyanobacterial toxins (including anatoxins, microcystins, and nodularins) have been suspected, but they have rarely been documented, as the cause of bird mortality (Table 36.1). Marine algal toxins such as domoic acid, saxitoxin, and brevetoxin that bioaccumulate or are magnified in the food chain by fish and shellfish, and anatoxins from freshwater cyanobacteria, affect the nervous system; cyanobacteria that contain microcystins or nodularin cause liver damage.

The effects of some harmful algae are not related to toxin production but rather are related to depleted dissolved-oxygen concentrations in water caused by algal proliferation, death, and decay, or night respiration. Other harmful effects include occlusion of sunlight by large numbers of algae and physical damage to the gills of fish caused by the structure of some algal organisms. All of these effects can

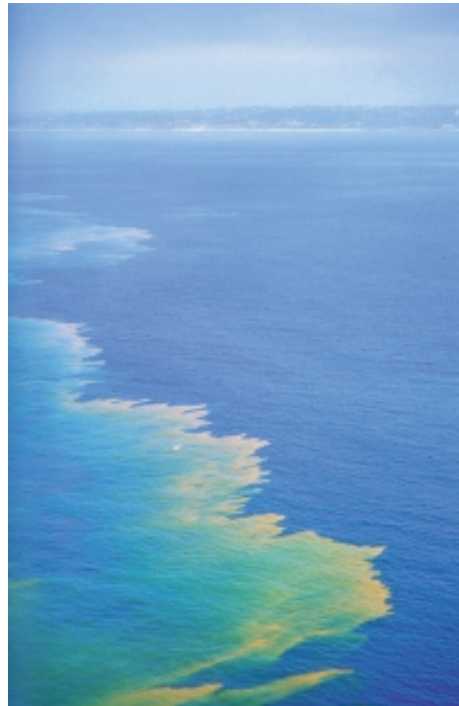


Photo by Peter Frank, Scripps Institute of Oceanography

**Figure 36.1** Aerial view of a large dinoflagellate bloom in near-shore ocean waters. The organism responsible for this bloom is not a toxin producer; however, toxic blooms may have a similar appearance.



Photo by Wayne Carmichael, Wright State University

**Figure 36.2** A cyanobacterial or blue-green algal bloom.

**Table 36.1** Documented instances of wild bird mortality caused by algal toxins.

| Toxin  | Algal species   | Toxin type(s)   | Migratory bird species affected  | Route of exposure    |
|--|---|---|--|----------------------|
| Cyanobacterial                                     | <i>Microcystis</i> sp.,<br><i>Anabaena</i> sp.,<br><i>Aphanizomenon</i> sp.,<br><i>Nodularia</i> sp.,<br>and<br><i>Oscillatoria</i> sp. | Hepatotoxins<br>(microcystins<br>and nodularin)<br>Neurotoxins<br>(anatoxin-a and<br>anatoxin-a(s)) | Unidentified ducks,<br>geese, and songbirds,<br>Franklin's gull, American<br>coot, mallard, American<br>wigeon                     | Oral<br>(water)      |
| Domoic acid<br>(amnesic shellfish<br>poisoning)    | <i>Pseudonitzschia</i> sp.  | Neurotoxin  | Brown pelican,<br>Brandt's cormorant   | Oral<br>(food items) |
| Saxitoxin<br>(paralytic<br>shellfish<br>poisoning) | <i>Alexandrium</i> sp.  | Neurotoxin  | Shag, northern fulmar,<br>great cormorant,<br>herring gull, common<br>tern, common murre,<br>Pacific loon, and sooty<br>shearwater | Oral<br>(food items) |
| Brevetoxin   | <i>Gymnodinium</i> sp.  | Neurotoxin  | Lesser scaup   | Oral<br>(food items) |

lead to mortality of aquatic invertebrates, aquatic plants, or fish and may produce an environment conducive to botulism. Other marine algal toxins (okadaic acid, neosaxitoxin, ciguatoxin, and *Pfiesteria* exotoxin) and cyanobacterial toxins (saxitoxin, neosaxitoxin, and cylindrospermopsin) have not yet been identified as causes of bird mortality events, but increased awareness and further research may establish a relationship.

### Species Affected

Many bird and mammal species can be affected by algal toxins. Most reports of mortality in birds are of die-offs that occur in conjunction with a bloom. Sometimes algal toxins are found in potential food items; however, there have been very few instances in which the algal toxin has been isolated from the ingesta or tissues of affected birds. Domoic acid poisoning caused mortality in brown pelicans and Brandt's cormorants on the central California coast. Brevetoxin has been suspected as the cause of mortality in lesser scaup, and saxitoxin has been strongly suspected as the cause of mor-

tality in sea birds (common terns, shags, great cormorants, northern fulmars, herring gulls, common murre, Pacific loons, sooty shearwaters, and others). Cyanobacterial toxicosis has been suspected in mortalities of free-ranging ducks, geese, eared grebes, gulls, and songbirds.

### Distribution

Many of the organisms responsible for red tides are widely distributed and, in recent years, the organisms seem to be markedly spreading. Natural events such as hurricanes can disperse organisms, and it is suspected that some organisms may be transported long distances in ship ballast waters. Another factor that may encourage algal proliferation in both marine and freshwater systems is increased nutrient loading. Certain algae occur more commonly in some areas than others and it is useful to know which ones are problems in specific locations. Good sources of information about algal blooms are the State public health department or the State division of marine resources or marine fisheries.

## Seasonality

There have not been enough confirmed instances of wild bird mortality caused by red tides and cyanobacterial blooms to establish seasonal patterns of occurrence.

## Field Signs

Field signs reported are variable and they depend on the toxin involved. Domoic acid poisoning of brown pelicans caused neurologic signs that included muscle tremors, a characteristic side-to-side head movement, pouch scratching, awkward flight, toe clenching, twisting of the head over the back, vomiting, and loss of the righting reflex just before death. Brandt's cormorants that also were involved in this mortality event were easily approached and handled, but they did not exhibit the neurologic signs seen in the pelicans. Sea birds suspected of having been poisoned by saxitoxin exhibited paralysis and vomiting. Clinical signs observed in lesser scaup suspected of having been poisoned by brevetoxin included lethargy, weakness, reluctance or inability to fly, head droop, and excessive ocular, nasal, and oral discharge.

White Pekin ducklings that were experimentally exposed to brevetoxins exhibited lethargy, loss of muscle coordination or ataxia, spastic head movements, head droop to one side, and leg extension to the rear during rest. Clinical signs in muscovy ducks dosed with anatoxin-a(s) included excessive salivation, regurgitation of algae, diarrhea, tremors, reduced responsiveness and activity, incoordination, difficulty breathing, excessive thirst, congestion in foot webs, wing and leg weakness, and recumbency and intermittent seizures prior to death.

## Gross Lesions

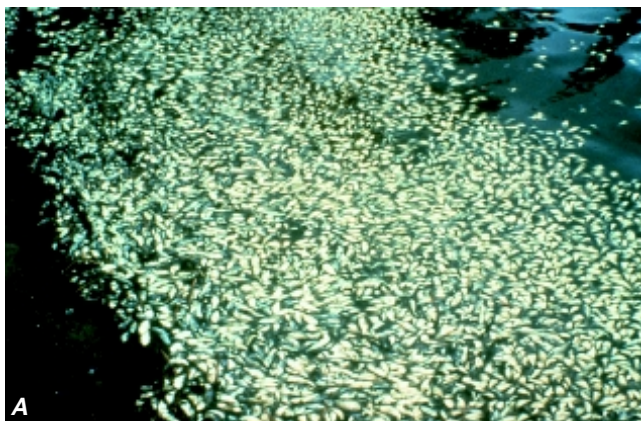
No characteristic or diagnostic gross lesions have been described for most types of algal toxin poisonings of wild

birds. Many of the toxins, particularly the neurotoxins, have a chemical effect that does not produce a grossly observable lesion. Birds that ingest toxic blooms of *Microcystis* may have notable lesions of necrosis or tissue death and hemorrhage in the liver. These lesions have been reported in domestic mammals and birds, including ducks, that died as a result of exposure to a toxic *Microcystis* algal bloom or that were experimentally dosed with microcystin.

## Diagnosis

Definitive diagnosis of algal toxicosis is difficult. Circumstantial evidence, such as the occurrence of a marine red tide or freshwater cyanobacterial bloom in conjunction with a die-off, and supportive clinical and pathologic findings, such as a lack of evidence of the presence of other types of toxins or infectious disease, are often used to reach a presumptive diagnosis. Analysis of the upper gastrointestinal tract contents or tissues of affected birds for algal toxins is possible but the tests are not yet widely available. In addition, there are no established toxic thresholds for wildlife species. Even when levels of particular toxins can be measured it may be difficult to assess their significance. Recently developed methods permit detection of microcystins in animal tissues and gastrointestinal contents by using enzyme linked immunosorbent assay (ELISA) technologies. Also, it is now possible to detect saxitoxin in urine and blood samples from affected animals by using highly sensitive neuroreceptor assays.

A sample of organisms from the bloom may be useful or necessary for diagnosis. Because of the ephemeral nature of blooms, collect algal samples during the die-off event as soon as possible after carcasses are found. Contact a diagnostic laboratory for advice on appropriate sample collection.



Photos by JoAnn Burkholder, North Carolina State University

**Figure 36.3 (A and B)** These fish were killed by *Pfiesteria* sp., an organism that has caused neurological problems, including prolonged amnesia, in people exposed by aerosols in a laboratory. This organism has only been fully described recently, and it has not been reported to cause mortality in birds; however, it may be encountered by biologists investigating concurrent bird and fish kills.



## Control

Because it is difficult to identify algal toxins as the cause of wildlife mortalities, there has been little opportunity to consider control measures. Currently, there is much interest in algal toxins and their threat to human water and food supplies. Identification of the conditions that trigger harmful algal blooms may aid in developing strategies to prevent red tides or freshwater cyanobacterial blooms and associated wildlife mortality. Controlling nutrient loading through reduced fertilizer use, improved animal waste control, and improved sewage treatment may reduce the number, or likely locations, of toxic algal blooms. Careful monitoring and early detection of potentially toxic algal blooms could allow time to initiate actions to prevent or reduce bird mortality.

## Human Health Considerations

Most red tide and toxic freshwater cyanobacteria are not harmful unless they are ingested. However, some organisms irritate the skin and others release toxic compounds into the water and, if aerosolized by wave action, these compounds may cause problems when people inhale them (Fig. 36.3). When investigating wildlife mortality that is occurring in conjunction with a known red tide or cyanobacterial bloom, contact the local public health department or a diagnostic laboratory for information on precautions you may need to take. As in the investigation of all wildlife mortality events, wear rubber or latex gloves when handling carcasses.

Lynn H. Creekmore

## Supplementary Reading

- Anderson, D.A., 1994, Red tides: Scientific American, August, p. 62–68.
- \_\_\_\_\_, 1994, The toxins of cyanobacteria: Scientific American, January, p. 78–86.
- Forrester, D.J., Gaskin, J.M., White, F.H., Thompson, N.P., Quick, J.A., Jr., Henderson, G.E., Woodard, J.C., and Robertson, W.D., 1977, An epizootic of waterfowl associated with a red tide episode in Florida: Journal of Wildlife Diseases, v. 13, p. 160–167.
- Nisbet, I.C.T., 1983, Paralytic shellfish poisoning: Effects on breeding terns; Condor, v. 85, p. 338–345.
- Work, T.M., Barr, B., Beale, A. M., Quilliam, M.A., and Wright, J.L.C., 1993, Epidemiology of domoic acid poisoning in brown pelicans (*Pelecanus occidentalis*) and Brandt's cormorants (*Phalacrocorax penicillatus*) in California: Journal of Zoo and Wildlife Medicine, v. 24, p. 54–62.

## Chapter 37

# Mycotoxins

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Mycotoxins are toxins produced by molds (fungi) that, when they are ingested, can cause diseases called mycotoxicosis. These diseases are not infectious. The effects on the animal are caused by fungal toxins in foods ingested, usually grains, and are not caused by infection with the fungus. Many different molds produce mycotoxins and many corresponding disease syndromes have been described for domestic animals. However, only two types of mycotoxin poisoning, aflatoxicosis and fusariotoxicosis, have been documented in free-ranging migratory birds.

Until recently, sickness or death caused by mycotoxins were rarely reported in migratory birds. Identification of mycotoxins as the cause of a mortality event can be difficult for a number of reasons. The effects may be subtle and difficult to detect or identify, or the effects may be delayed and the bird may have moved away from the contaminated food source before becoming sick or dying. Also, grain containing toxin-producing molds can be difficult or impossible to recognize because it may not appear overtly moldy.

Techniques to detect and quantify a variety of mycotoxins important to domestic animal and human health are available through many diagnostic laboratories that serve health needs for those species. These same techniques are applicable for wildlife. Further study and improved diagnostic technology is likely to result in identification of additional types of mycotoxins as causes of disease and death in waterfowl and other wildlife.

## Aflatoxin Poisoning

### Synonyms

*Aflatoxicosis*

### Cause

Aflatoxins are a group of closely related toxic compounds produced by the fungi *Aspergillus flavus* or *A. parasiticus*. Four types of aflatoxins commonly are found in grains contaminated by these fungi: aflatoxin B1, aflatoxin B2, aflatoxin G1, and aflatoxin G2. These compounds become more toxic when they are metabolized after they are ingested. Aflatoxin B1 is the most commonly occurring and the most toxic. Aflatoxins present in very low concentrations, in parts per billion (ppb), can cause toxicosis.

Domestic ducklings are quite sensitive and the effects of aflatoxin exposure have been studied extensively in this species. In one study using 1-day-old ducklings, the LD<sub>50</sub>, which is the dose of toxin required to produce death in 50 percent

of the test animals via a single dose or single day's feeding, of aflatoxin B1 was 360 ppb aflatoxin.

Aflatoxins are often associated with groundnuts (peanuts) and corn, but they also have been found in other grains and nuts. *Aspergillus* sp. fungi can proliferate in improperly stored grain that has a moisture content of greater than 14 percent, relative humidity greater than 70 percent, and temperature greater than 70 °F. These fungi also can invade grains in the field, especially when there is drought stress, insect damage, or mechanical damage.

### Species Affected

Aflatoxins can affect humans, many species of warm-blooded domestic and wild animals, and fish, most notably rainbow trout. Animals that consume grain are more likely to be affected than those that do not. Susceptibility depends on species, age, and diet. In general, birds are more susceptible than mammals, and young birds are more susceptible than adult birds. Mortality events caused by exposure to aflatoxins have been reported in free-ranging birds including a variety of duck species (mallard, black duck, lesser scaup, gadwall, and blue- and green-winged teal), Canada geese, snow geese, and sandhill cranes.

### Distribution

Within the United States, the problem of aflatoxin-contaminated grain as a cause of disease in domestic animals and humans has been associated with the Southeastern and Gulf Coast States. Documented wildlife mortality events caused by aflatoxicosis are few; of those reported in wild birds, most occurred in Texas. However, the major fungi that produce aflatoxins, *A. flavus* and *A. parasiticus*, are widespread in temperate and tropical environments.

### Seasonality

Most mortalities caused by exposure to acutely toxic levels of aflatoxins are reported in the fall and winter and coincide with times during migration and wintering when cranes and waterfowl are consuming waste grain in fields (Fig. 37.1). Mortality can occur at any time of the year when contaminated grain is provided at birdfeeding stations.

### Field Signs

Field signs of aflatoxicosis reported in waterbirds vary from depression and lethargy to blindness, lack of awareness of surroundings, inability to fly, tremors, and wing flapping. Often the birds are simply found dead.



National Wildlife Health Center file photo

**Figure 37.1** Canada geese feeding on standing waste corn in a winter field.

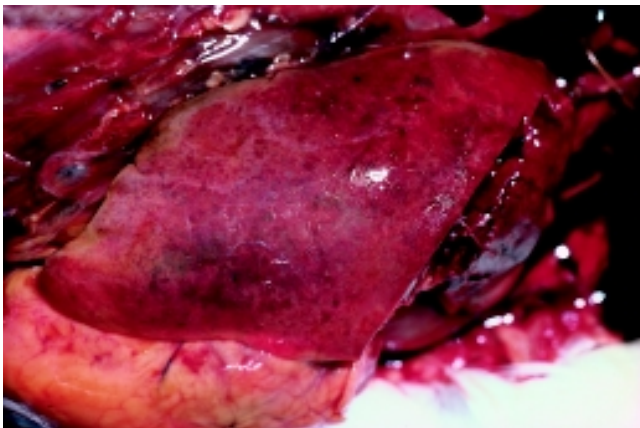


Photo by James Rumminger

**Figure 37.2** Pale, enlarged liver with multiple, focal hemorrhages from a bird with acute aflatoxicosis.

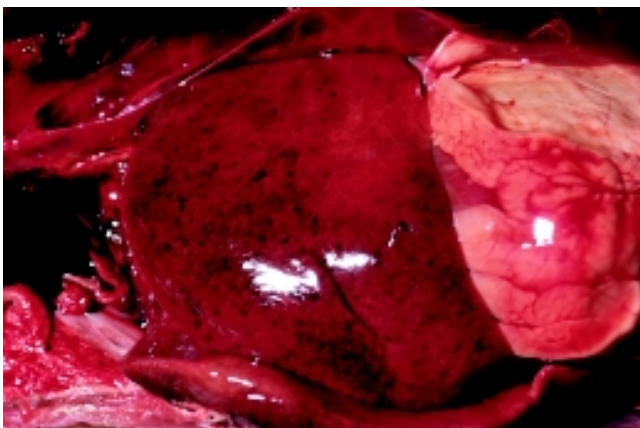


Photo by James Rumminger

**Figure 37.3** Hemorrhagic liver from a bird with acute aflatoxicosis.

## Gross Lesions

Lesions of aflatoxicosis can be variable, depending on the amount of aflatoxin ingested and the length of time the animal is exposed. Birds exposed to high levels over a short period of time may have an enlarged, swollen pale liver (Fig. 37.2). Liver hemorrhages may be found in multiple focal areas, or may be diffuse and involve most of the liver tissue (Fig. 37.3). Hemorrhages and fluid may be observed in many organs in the chest and abdomen. Inflammation and bleeding of the gastrointestinal tract lining, which may cause the intestines to appear blackish-red throughout their entire length, may also be observed.

Birds that are exposed to small amounts of aflatoxin over a long period of time may not die suddenly, but rather may have chronic health problems. Chronic effects, which include appetite loss, weight loss, and general ill health, can be more insidious and difficult to definitively relate to aflatoxin exposure. Chronic exposure also may produce a shrunken, fibrous liver with regenerative nodules or tumors. In laboratory tests, aflatoxin B1 has been shown to cause genetic mutations, liver cancers, and, possibly, fetal defects. Chronic low level aflatoxin exposure also is known to suppress the immune system, which may predispose animals to infectious diseases.

## Diagnosis

Diagnosis is made by examining tissues for gross lesions and typical microscopic lesions, which include liver tissue death or necrosis and proliferation of lesions in the bile duct. Measurement of aflatoxin levels in ingesta and tissues collected from affected birds and from the grain suspected of being contaminated is also crucial for confirming the diagnosis. The samples of choice include whole refrigerated carcasses for necropsy as well as grain that affected birds have been eating. Because mycotoxin occurrence can vary widely within an agricultural field, it is important to try to obtain a representative sample of the suspect grain. If possible, transport the grain frozen, and ensure that the sample remains frozen so that fungal growth and toxin production secondary to improper postcollection storage does not occur.

## Control

Wildlife should not be fed grain that has levels of aflatoxins in excess of those allowed for use in human or domestic animal food (20 ppb for consumption by humans or young animals and dairy cattle; 100 ppb for mature poultry). In years when aflatoxins are a problem, grain from fields that are frequently used by wildlife should be checked for aflatoxin levels. If the fields are aflatoxin-contaminated, deep plowing of the contents can make the grain unavailable to wildlife. If the fields cannot be plowed, hazing wildlife from the area can lessen their exposure.

## Human Health Considerations

Handling aflatoxin-poisoned sick or dead birds does not pose a human health risk. However, birds known to have died from acute aflatoxicosis should not be consumed.

## Fusariotoxin Poisoning

### Synonyms

*Fusariomycotoxicosis*, *trichothecene mycotoxicosis*, *T-2 toxicosis*, *vomitoxicosis*, *zearalenone toxicosis*

### Cause

Fusariotoxin poisoning is caused by toxins produced by fungi of the genus *Fusarium*. There are two classes of toxins produced by these organisms: metabolites that have properties similar to the hormone estrogen such as zearalenone (F-2 toxin), and the trichothecenes. Zearalenone has been linked to hyperestrogenic or feminizing syndromes in domestic animals, but similar effects have not yet been observed in wild species. More than 50 trichothecene toxins have been identified from *Fusarium* cultures and field samples. However, the trichothecenes have rarely been documented as the cause of mortality in free-ranging birds.

The most frequently occurring trichothecene toxin in the United States is deoxynivalenol, commonly called vomitoxin. Others include T-2 toxin, diacetoxyscirpenol (DAS), neosolaniol and iso-neosolaniol. The feedstuffs involved in *Fusarium* sp. toxin production include corn, wheat, barley, oats, peanuts, and sometimes forages. *Fusarium* toxins differ from other mycotoxins in that they tend to be produced during the colder seasons of the year.

### Species Affected

Poisoning caused by trichothecene toxins has been documented in domestic mammals, poultry, and waterfowl. However, reports of fusariotoxicosis in free-ranging waterfowl and other migratory birds are rare and poorly documented. Mortality attributed to trichothecenes has occurred on several occasions in free-ranging sandhill cranes. It is suspected that wild waterfowl could be affected by trichothecenes because these toxins tend to be produced in low temperature conditions when waterfowl make heavy use of waste grain as food.

### Distribution

*Fusarium* sp. are widespread in the environment and they commonly occur as plant pathogens or contaminants in stored or waste grain and other plant parts. Toxin production from *Fusarium* sp. is most commonly a problem in the North-Central United States and Canada. However, the only documented locations of wild bird mortality caused by *Fusarium* toxins are in Texas and New Mexico, and these die-off events involved sandhill cranes that fed on *Fusarium*-contaminated peanuts.

## Seasonality

*Fusarium* invasion often occurs during wet conditions in the summer and fall while crops are in the field. However, the organism also can grow in stored grain. The optimal temperatures for mold growth and toxin production differ. Temperatures that support growth of the vegetative form of the fungus are between 64 and 77 °F, but actual toxin production tends to occur at temperatures between 40 and 65 °F and, in some cases, has even been documented at near-freezing temperatures. Colder temperatures favoring toxin production coincide with times that cranes and waterfowl are using waste grain in fields during their fall and winter migration.

## Field Signs

Different trichothecenes have different effects. In general, clinical signs in domestic poultry and geese include feed refusal, vomiting, and gastrointestinal bleeding. Some birds have been described as having neurologic abnormalities. Free-ranging sandhill cranes diagnosed with trichothecene toxicosis had difficulty keeping their balance and had flaccid paralysis or weakness of the neck and wing muscles. This created a stance characterized by a drooping head and wings (Fig. 37.4). The more toxic trichothecenes cause immune suppression and may predispose birds to secondary infections.

## Gross Lesions

Inflammation and ulceration of the skin and mucosal surfaces of the oral cavity and upper gastrointestinal tract are the most commonly reported lesions in domestic animals and were observed in affected sandhill cranes (Fig. 37.5). Gross lesions described in sandhill cranes also included subcutaneous fluid over the head and neck (Fig. 37.6) and multiple hemorrhages and pale areas in skeletal muscle.



Photo by Ronald M. Windingstad

**Figure 37.4** A sandhill crane suffering from fusariotoxinosis. Notice the wing and head droop.



Photo by James Rumminger

**Figure 37.5** Inflammation and ulceration of the mucosal surface of the esophagus in a sandhill crane with fusariotoxigenesis.



Photo by J. Christian Franson

**Figure 37.6** Fluid beneath the skin of the head and neck of a sandhill crane with fusariotoxigenesis.



Photo by Ronald M. Windingstad

**Figure 37.7** Plowing of this field made these peanuts unavailable to the cranes and ended a fusariotoxigenesis mortality event.

## Diagnosis

Diagnosis is made through observing the appropriate field signs, finding gross as well as microscopic tissue lesions, and detecting the suspected toxin in grains, forages, or the ingesta of affected animals. However, the tests required to detect these toxins are complex and few diagnostic laboratories offer tests for multiple trichothecenes. The samples of choice include both refrigerated and frozen carcasses for necropsy examination and a representative sample of the suspected contaminated grain source. Because the toxin is produced under cold conditions, the grain sample should be frozen rather than refrigerated for shipment to the diagnostic laboratory.

## Control

Wildlife should not be fed grain with levels of fusariotoxins in excess of those recommended for use in domestic animal food. In years when fusariotoxins are a problem, grain from fields that are frequently used by wildlife should be checked for toxins. If the fields are significantly contaminated, deep plowing of the contents of the field can make the grain unavailable to wildlife (Fig. 37.7). If the fields cannot be plowed, hazing wildlife from the area can lessen their exposure.

## Human Health Considerations

Handling fusariotoxin-poisoned sick or dead birds does not pose a human health risk, but birds known to have died from acute fusariotoxigenesis should not be consumed. The more potent trichothecenes in grain or forages may present a health hazard when the fungal spores and contaminated plant parts are inhaled or when they contact the skin.

Lynn H. Creekmore

## Supplementary Reading

- Pier, A.C., 1981, Mycotoxins and animal health, *in* Cornelius, C.E., and Simpson, eds., *Advances in veterinary science and comparative medicine*: New York, Academic Press, p. 186–243.
- Robinson, R.M., Ray, A.C., Reager, J.C., and Holland, L.A., 1982, Waterfowl mortality caused by aflatoxicosis in Texas: *Journal of Wildlife Diseases*, v. 18, p. 311–313.
- Roffe, T.J., Stroud, R.K., and Windingstad, R.M., 1989, Suspected fusariomycotoxicosis in sandhill cranes (*Grus canadensis*): clinical and pathological findings: *Avian Diseases*, v. 33, p. 451–457.
- Windingstad, R.M., Cole, R.J., Nelson, P.E., Roffe, T.J., George, R.R., and Dorner, J. W., 1989, *Fusarium* mycotoxins from peanuts suspected as a cause of sandhill crane mortality: *Journal of Wildlife Diseases*, v. 25, p. 38–46.
- Wobeser, G.A., 1997, Mycotoxins, *in* *Diseases of wild waterfowl* (2nd ed): New York, N.Y., Plenum Press, p. 189–193.

## Chapter 38

# Avian Botulism

### Synonyms

*Limberneck, Western duck sickness, duck disease, alkali poisoning*

### Cause

Avian botulism is a paralytic, often fatal, disease of birds that results when they ingest toxin produced by the bacterium, *Clostridium botulinum*. Seven distinct types of toxin designated by the letters A to G have been identified (Table 38.1). Waterfowl die-offs due to botulism are usually caused by type C toxin; sporadic die-offs among fish-eating birds, such as common loons and gulls, have been caused by type E toxin. Type A botulinum toxin has also caused disease in birds, most frequently in domestic chickens. Types B, D, F, and G are not known to cause avian botulism in North America.

**Table 38.1** *Botulinum toxins and primary species affected.*

| Toxin type | Animals affected                    | Risk for humans |
|------------|-------------------------------------|-----------------|
| A          | Poultry, occasionally               | High            |
| B          | Horses                              | High            |
| C          | Wild birds, cattle, horses, poultry | Low             |
| D          | Cattle                              | Low             |
| E          | Fish-eating birds                   | High            |
| F          | *                                   | Unknown         |
| G          | *                                   | Unknown         |

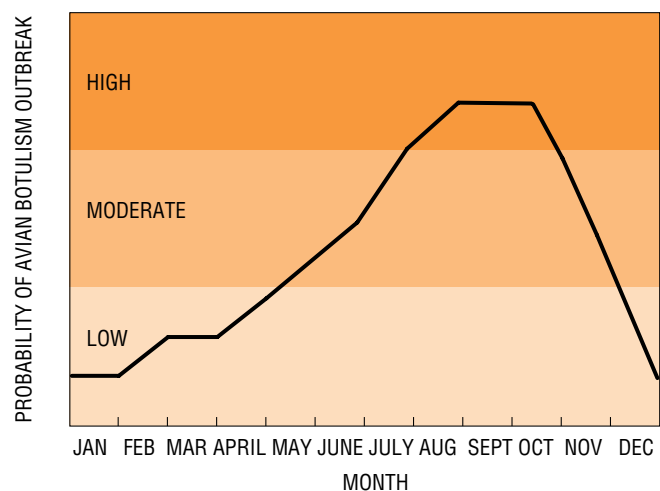
\* Rarely detected in nature; too little information for species evaluations.

*C. botulinum* is an oxygen-intolerant or anaerobic bacterium that persists in the form of dormant spores when environmental conditions are adverse. The spores are resistant to heating and drying and can remain viable for years. Spores of type C botulism strains are widely distributed in wetland sediments; they can also be found in the tissues of most wetland inhabitants, including aquatic insects, mollusks, and crustacea and many vertebrates, including healthy birds. Botulinum toxin is produced only after the spores germinate, when the organism is actively growing and multiplying. Although the bacteria provide the mechanism for toxin production, the gene that encodes for the toxin protein is actually carried by a virus or phage that infects the bacteria. Unfortunately, little is known about the natural factors that control phage infection and replication within the bacteria, but several factors may play a role, including the bacterial

host strain and environmental characteristics, such as temperature and salinity.

Because botulinum spores and the phages that carry the toxin gene are so prevalent in wetlands, they are not considered to be a limiting factor in the occurrence of outbreaks in waterbirds. Other factors are thought to be more critical in the timing and location of botulism outbreaks; these include optimal environmental conditions for spore germination and bacterial growth, suitable material or substrates that provide energy for bacterial replication, and a means of toxin transfer to birds. It is likely that toxin production, toxin availability to birds and, subsequently, botulism outbreaks in birds are largely controlled by these ecological factors.

As with other bacteria, temperature plays a critical role in the multiplication of *C. botulinum*, with optimal growth in the laboratory occurring between 25 ° and 40 °C. Most botulism outbreaks take place during the summer and fall when ambient temperatures are high (Fig. 38.1). Winter botulism outbreaks have been documented in some locations, but these are generally thought to be due to residual toxin produced during the previous summer. Conditions that elevate wetland sediment temperatures and decrease dissolved oxygen, including the presence of decaying organic matter and shal-



**Figure 38.1** *Relative seasonal probability of type C botulism in North American water birds.*

low water, may increase the risk of botulism outbreaks (Fig. 38.2). However, these conditions are not prerequisite to an outbreak because botulism has occurred in large river systems and in deep, well-oxygenated wetlands, which suggests that other environmental conditions may be more critical. In studies conducted by the National Wildlife Health Center, several environmental factors, including pH, salinity (Fig. 38.3), temperature, and oxidation-reduction potential in the sediments and water column, appeared to significantly influence the likelihood of botulism outbreaks in wetlands.

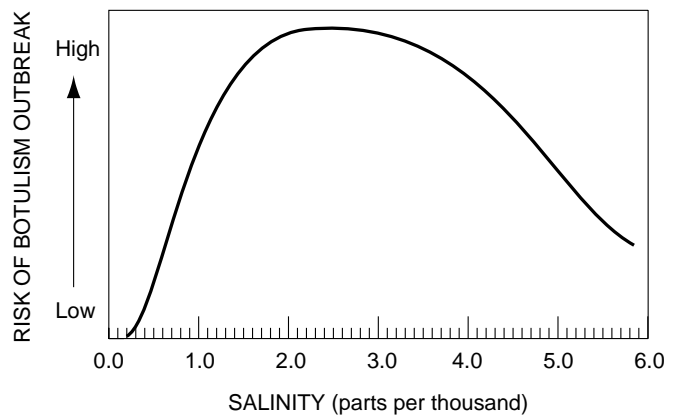
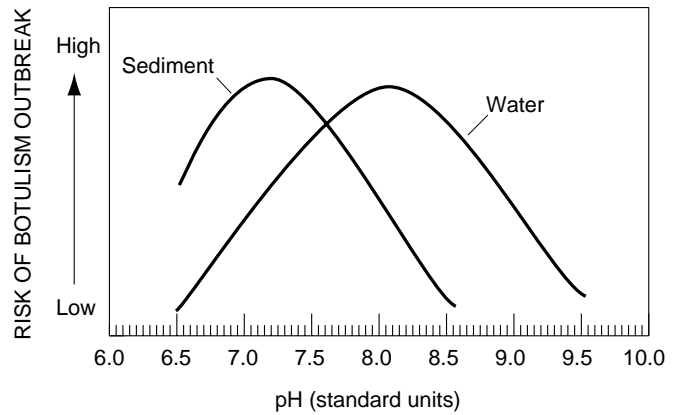
In addition to permissive environmental conditions, *C. botulinum* also requires an energy source for growth and multiplication. Because it lacks the ability to synthesize certain essential amino acids, the bacterium requires a high protein substrate; it is essentially a “meat lover.” The most important substrates for toxin production in natural wetlands have never been identified, but there are many possibilities, including decaying organic matter or any other protein particulates. Decomposing carcasses, both vertebrate and invertebrate, are well known to support toxin production. Human activities can also increase the available substrate for toxin production in wetlands (Table 38.2). For example, wetland flooding and draining, pesticides, and other agricultural pollutants may kill aquatic life, thereby providing more substrate for toxin production. Raw sewage and rotting vegetation are other potential sources of energy.

Although many substrates are suitable for botulinum toxin production, in order for a botulism outbreak to occur the toxin must be in a form that is available to birds. In some cases, decaying organic matter may be directly ingested, but in other cases there must be some means of toxin transfer from the substrate to the birds, presumably through zooplankton or invertebrate food items that inadvertently consumed toxin. Invertebrates are unaffected by the toxin and, because they feed on decaying matter, they can effectively act to concentrate toxin. Although most waterfowl will not directly consume a vertebrate carcass, they will readily ingest any maggots that fall off of it. In this way, botulism outbreaks often become self-perpetuating. This has become known as the carcass-maggot cycle of avian botulism (Fig. 38.4).



Photo by Milton Friend

**Figure 38.2** Decaying organic matter may increase the risk of avian botulism outbreaks.

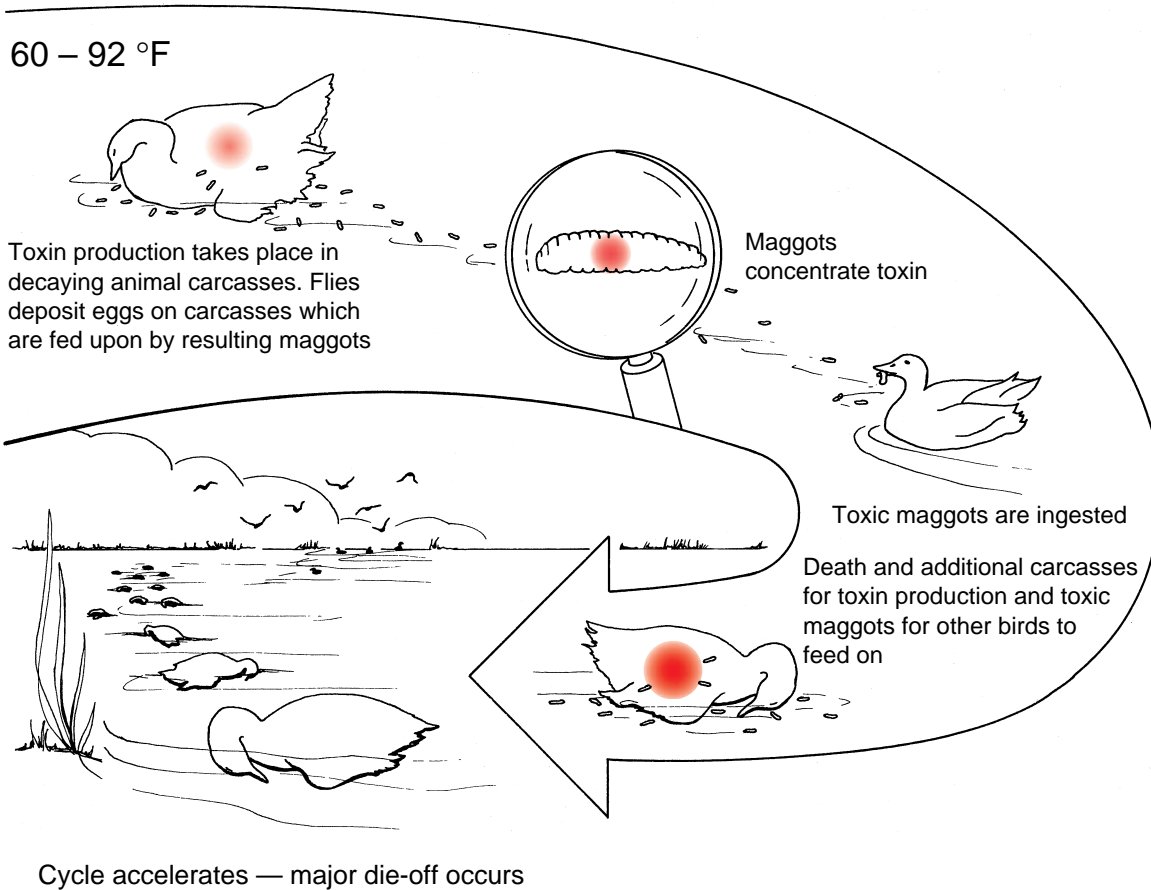


**Figure 38.3** Relationship of pH and salinity to avian botulism outbreaks.

**Table 38.2** Human activities speculated to contribute to avian botulism outbreaks in wetlands.

| Action  | Consequences of action   |
|---|--|
| Fluctuating water levels for flooding and drying                    | Deaths of terrestrial and aquatic invertebrates and fish   |
| Pesticides and other chemical inputs into wetlands from agriculture | Deaths of aquatic life   |
| Raw sewage discharges into wetlands                                 | Nutrient enhancement resulting in "boom and bust" invertebrate populations and oxygen depletion causing deaths of aquatic and plant life |

### Carcass-maggot cycle of avian botulism



**Figure 38.4** Carcass-maggot cycle of avian botulism.

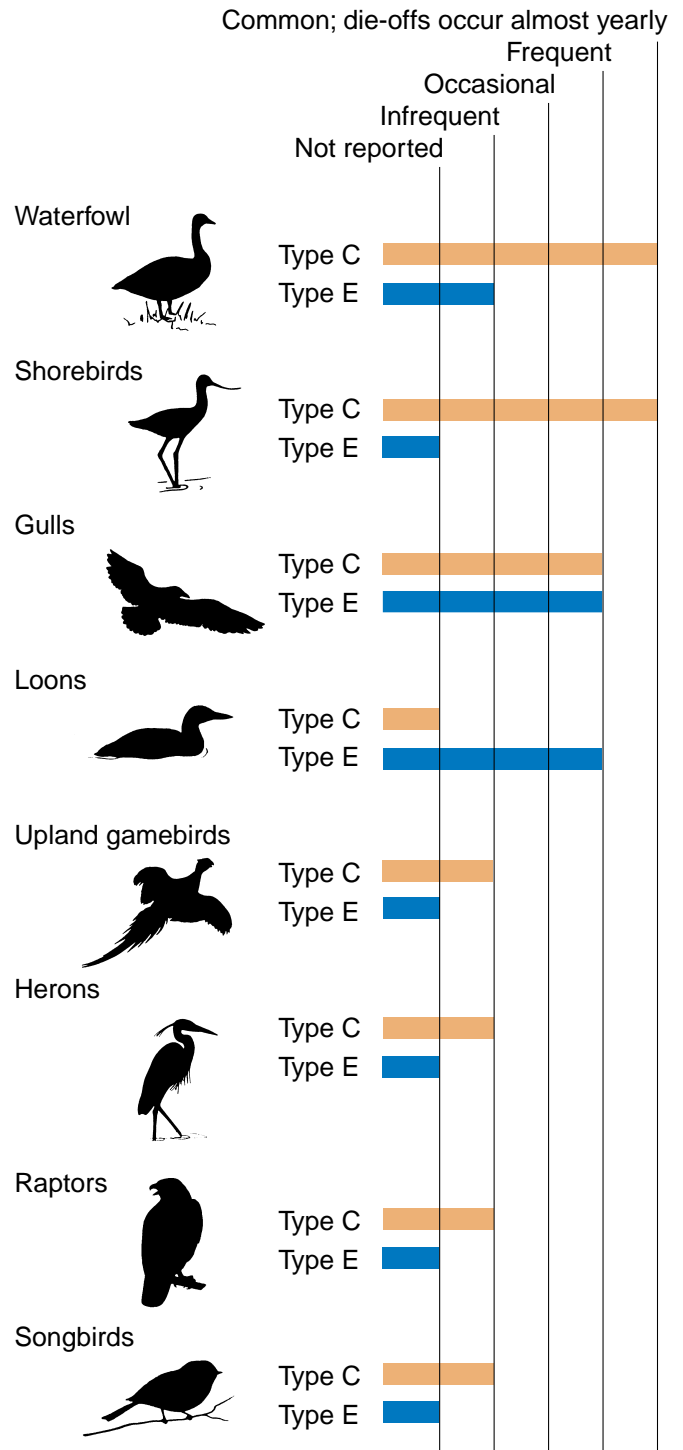


## Species Affected

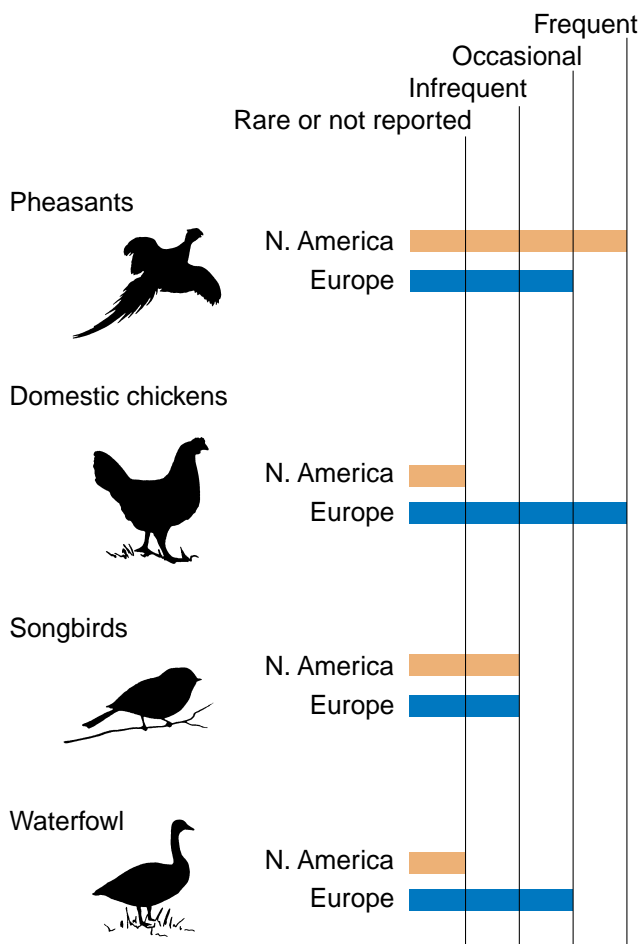
Many species of birds and some mammals are affected by type C botulism. In the wild, waterbirds suffer the greatest losses, but almost all birds are susceptible to type C botulism. The exception is vultures, which are highly resistant to type C toxin. Foraging behavior is probably the most significant host determinant for botulism. Filter-feeding and dabbling waterfowl and probing shorebirds appear to be among the species at greatest risk (Fig. 38.5). Mortality of wild raptors from botulism has been associated with improper disposal of poultry carcasses. Among captive and domestic birds, pheasants, poultry, and waterfowl are the most frequently affected (Fig. 38.6). Cattle, horses, and ranch mink are also susceptible to type C botulism. Although dogs and cats are usually regarded as being resistant to type C toxin, a few cases have been reported in dogs, which is a factor to consider when dogs are used to retrieve carcasses during outbreaks. Also, type C botulism occurred in captive African lions which were fed toxin-laden chickens.

Losses vary a great deal from year to year at site-specific locations and from species to species. A few hundred birds may die one year and tens of thousands or more the following year. More than a million deaths from avian botulism have been reported in relatively localized outbreaks in a single year, and outbreaks with losses of 50,000 birds or more are relatively common (Table 38.3).

On a worldwide basis, avian botulism is probably the most important disease of migratory birds.



**Figure 38.5** Frequency of botulism in major groups of wild birds.



**Figure 38.6** Frequency of botulism in captive birds.

**Table 38.3** Major waterfowl botulism outbreaks in the United States and Canada.

| Location                 | Year | Estimated loss      |
|--------------------------|------|---------------------|
| Utah and California      | 1910 | “Millions”          |
| Lake Malheur, Oregon     | 1925 | 100,000             |
| Great Salt Lake, Utah    | 1929 | 100,000–<br>300,000 |
| Tulare Basin, California | 1941 | 250,000             |
| Western United States    | 1952 | 4–5 million         |
| Montana                  | 1978 | 50,000              |
| Montana                  | 1979 | 100,000             |
| Great Salt Lake, Utah    | 1980 | 110,000             |
| Canada (Alberta)         | 1995 | 100,000             |
| Canada (Manitoba)        | 1996 | 117,000             |
| Canada (Saskatchewan)    | 1997 | 1 million           |
| Great Salt Lake, Utah    | 1997 | 514,000             |

## Distribution

Outbreaks of avian botulism have occurred in the United States and Canada since the beginning of the century, if not earlier. Outbreaks have also been reported in many other countries; most of these reports are recent, usually within the past 30 years (Fig. 38.7). Most type C botulism outbreaks within the United States occur west of the Mississippi River; however, outbreaks have occurred from coast-to-coast and border-to-border, and the distribution of the disease has greatly expanded since the early 1900s (Fig. 38.8). Type E outbreaks in birds are much less frequent and, within the United States, have been confined to the Great Lakes region.

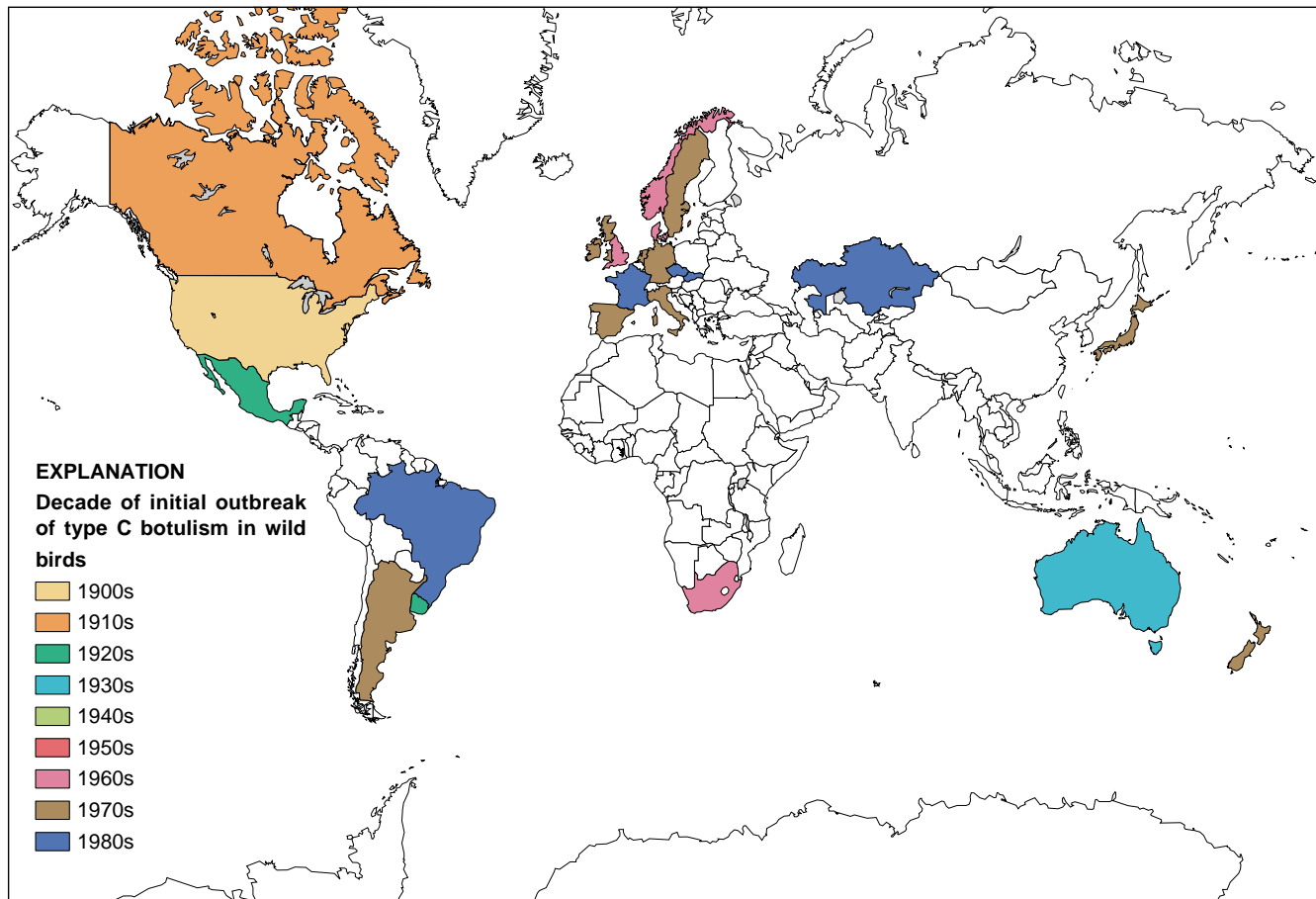
## Seasonality

July through September are the primary months for type C avian botulism outbreaks in the United States and Canada. However, outbreaks occur as late as December and January and occasionally during early spring in southern regions of the United States and in California. Type E outbreaks have occurred during late fall and spring.

## Field Signs

Lines of carcasses coinciding with receding water levels generally typify the appearance of major botulism die-offs, although outbreaks have also occurred in impoundments containing several feet of water, lakes with stable water levels, and in large rivers. When receding water conditions are involved, botulism is typically a disease of the water's edge and seldom are sick or dead birds found very far from the edge of vegetation bordering the water or the original water's edge (Fig. 38.9). In impoundments where water levels are relatively stable, affected birds are likely to be found in areas of flooded vegetation. Botulism-affected birds also tend to congregate along vegetated peninsulas and islands (Fig. 38.10).

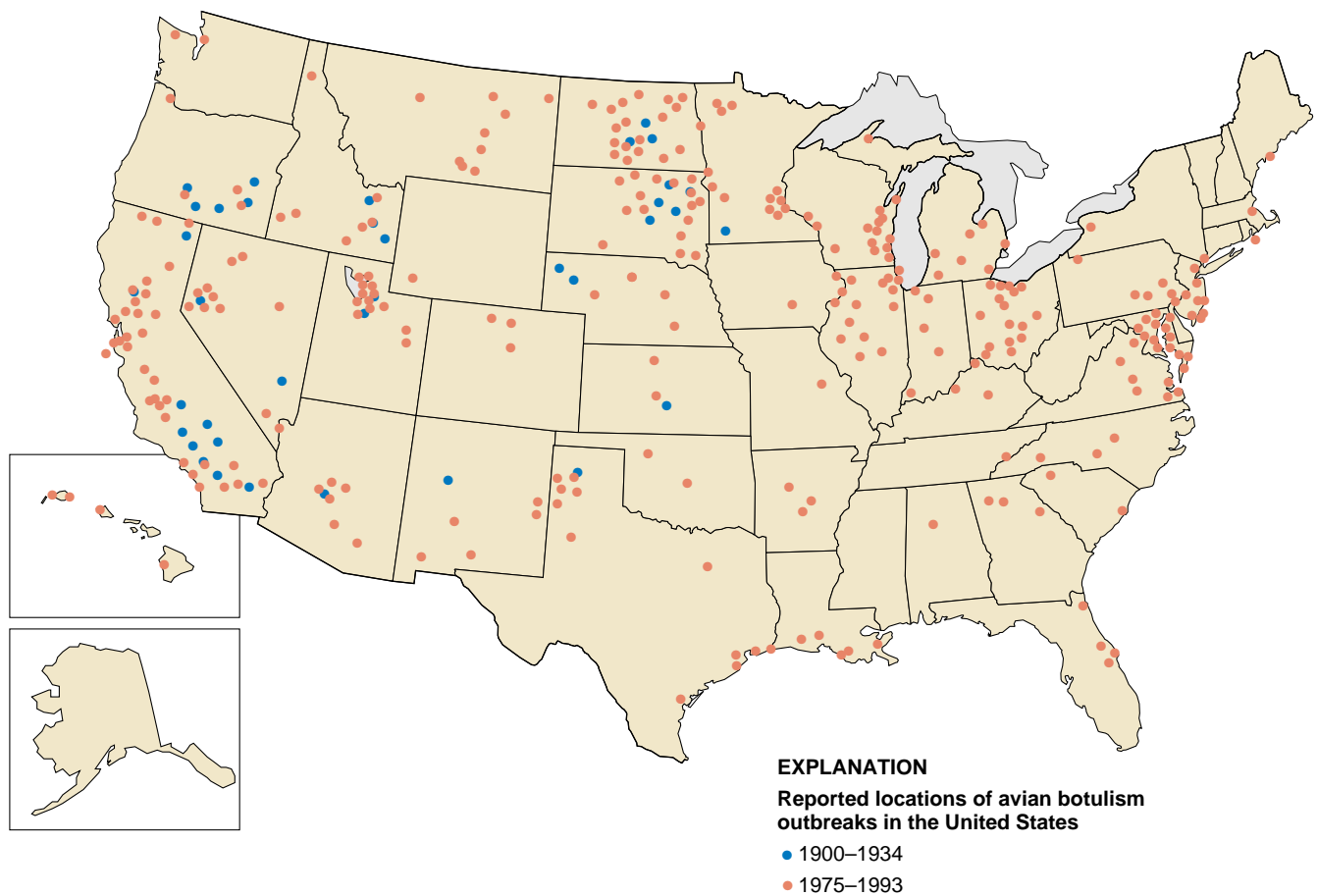
Healthy birds, sick, and recently dead birds will commonly be found together during a botulism outbreak, along with carcasses in various stages of postmortem decay. Often, species representing two, three, or even more orders of birds suffer losses simultaneously.



**Figure 38.7** Type C botulism outbreaks in wild birds.

Avian botulism affects the peripheral nerves and results in paralysis of voluntary muscles. Inability to sustain flight is seen early in botulism, but this sign is not useful for distinguishing botulism-intoxicated birds from those affected by other diseases. Because ducks suffering from botulism cannot fly and their legs become paralyzed, they often propel themselves across the water and mud flats with their wings (Fig. 38.11) (see also Fig. 1.2 in Chapter 1, Recording and Submitting Specimen Data). This sequence of signs contrasts with that of lead-poisoned birds, which retain their ability to walk and run although flight becomes difficult (see Chapter 43).

Paralysis of the inner eyelid or nictitating membrane (Fig. 38.12) and neck muscles follow, resulting in inability to hold the head erect (Fig. 38.13). These are the two most easily recognized signs of avian botulism. When birds reach this stage, they often drown before they might otherwise die from the respiratory failure caused by botulinum toxin.



**Figure 38.8** Locations of avian botulism outbreaks in the United States.



Photo by James Runnigen

**Figure 38.9** Typical scene of avian botulism. Dead birds are often found along the shore in parallel rows that represent receding water levels.



Photo by Milton Friend

**Figure 38.10** Botulism-affected birds tend to congregate along vegetated peninsulas and islands. Both dead and sick birds are evident in the photograph.



Photo by Milton Friend

**Figure 38.11** Botulism-intoxicated birds that have lost the power of flight and use of their legs often attempt escape by propelling themselves across water or land using their wings.



Photo by Milton Friend

**Figure 38.12** Paralysis of the inner eyelid is a common sign in botulism-intoxicated birds.



Photo by Milton Friend

**Figure 38.13** Paralysis of the neck muscles in botulism-intoxicated birds results in inability to hold the head erect (limberneck). Death by drowning often results.

## Gross Lesions

There are no characteristic or diagnostic gross lesions in waterfowl dying of either type C or type E botulism. Often, affected birds die by drowning, and lesions associated with drowning may be present.

## Diagnosis

The most widely used test for avian botulism is the mouse-protection test, although an enzyme-linked immunosorbent assay (ELISA) for type C toxin has been developed recently. For the mouse test, blood is collected from a sick or freshly dead bird and the serum fraction is then inoculated into two groups of laboratory mice, one group of which has been given type-specific antitoxin. The mice receiving antitoxin will survive, and those that receive no antitoxin will become sick with characteristic signs or die if botulism toxin is present in the serum sample. The ELISA is an *in vitro* test that detects inactive as well as biologically active toxin.

A presumptive diagnosis is often based on a combination of signs observed in sick birds and the absence of obvious lesions of disease when the internal organs and tissues of sick and dead birds are examined. However, this initial diagnosis must be confirmed by the mouse-protection or ELISA test to separate avian botulism from algal poisoning, castor-bean poisoning, and other toxic processes that cause similar signs of disease. Avian botulism should be suspected when maggots are found as part of the ingesta of gizzard contents of dead birds (Fig. 38.14), however, such findings are rare. After a bird ingests toxin, it takes several hours to days before the bird develops signs of the disease and dies. By this time, most food items ingested at the time of intoxication have been eliminated.



Photo by Milton Friend

**Figure 38.14** Waterfowl and other birds readily feed on maggots as exemplified by their presence in the gizzard of this duck that died from avian botulism.

## Prevention and Control

Prevention of avian botulism outbreaks in waterbirds will depend on a thorough understanding of the interactions between the agent, the host, and the environment. Because botulism spores are so ubiquitous in wetlands and are resilient, attempts to reduce or eliminate the agent are not currently feasible, but some actions can be taken to mitigate environmental conditions that increase the likelihood of outbreaks.

### Management of Environment

Attempts should be made to reduce organic inputs into wetlands or to eliminate factors that introduce large amounts of decaying matter. For example, in areas that are managed primarily for migratory waterfowl (ducks, geese, swans), reflooding land that has been dry for a long time is not recommended during the summer. Similarly, avoid sharp water drawdowns in the summer because they could result in fish-kills and die-offs of aquatic invertebrates whose carcasses could then become substrates for *C. botulinum* growth. In areas managed primarily for shorebirds, water drawdowns provide essential habitat; thus, botulism control must focus on cleaning up any vertebrate carcasses that may result from drawdowns.

Prompt removal and proper disposal of vertebrate carcasses by burial or burning, especially during outbreaks, are highly effective for removing substrates for toxin production. The importance of prompt and thorough carcass removal and proper disposal cannot be overemphasized. Several thousand toxic maggots can be produced from a single waterfowl carcass (Fig. 38.15). Consumption of as few as two to four of these toxic maggots can kill a duck, thereby perpetuating the botulism cycle. It is not uncommon to find three or



Photo by Milton Friend

**Figure 38.15** Several thousand toxic maggots can be produced from a single waterfowl carcass.

four freshly dead birds within a few feet of a maggot-laden carcass. Failure to carry out adequate carcass removal and disposal programs can cause a rapid build-up of highly toxic decaying matter and toxin-laden maggots, thereby accelerating losses in waterbirds, as well as seeding the environment with more botulism spores as the carcasses decompose.

Many botulism outbreaks occur on the same wetlands year after year and within a wetland there may be localized “hot spots.” Also, outbreaks often follow a fairly consistent and predictable timeframe. These conditions have direct management implications that should be applied towards minimizing losses. Specific actions that should be taken include accurately documenting conditions and dates of outbreaks in problem areas, planning for and implementing intensified surveillance and carcass pickup and disposal, and modifying habitat to reduce the potential for botulism losses or deny bird use of major problem areas during the botulism “season” or both.

### **Management actions for minimizing losses from avian botulism**

Document environmental conditions, specific impoundments or areas of outbreaks, and dates of occurrence and cessation.

Plan for and implement intensive surveillance and vertebrate carcass pickup and disposal starting 10–15 days before the earliest documented cases until 10–15 days after the end of the botulism “season.”

Where possible, monitor and modify environmental conditions to prevent the pH and salinity of wetlands from reaching or being maintained within high hazard levels.

Avoid water drawdowns for rough fish and vegetation control during warm weather. Collect vertebrate carcasses (fish) and properly dispose of them if drawdowns are necessary during summer and warm fall months.

Construct wetland impoundments in botulism-prone areas in a manner that facilitates rapid and complete drainage thereby encouraging bird movement to alternative impoundments.

Because fish carcasses can also serve as sites for *C. botulinum* growth, they should be promptly removed during fish control programs in marshes, or fish control programs should be restricted to the cooler months of year (the nonfly season). Also, bird collisions with power lines that cross marshes have been the source of major botulism outbreaks because carcasses from these collisions served as initial substrates for toxin production within marshes. Therefore, if possible, power lines should not be placed across marshes used by large concentrations of waterbirds.

Numerous outbreaks of avian botulism have been associated with sewage and other wastewater discharges into marshes. This relationship is not presently understood, but outbreaks have occurred often enough that wetland managers should discourage the discharges of these effluents when many waterfowl or shorebirds are using the area or are likely to use an area during warm weather.

### **Treatment of Sick Birds**

Botulism-intoxicated waterfowl can recover from the disease. If sick birds are provided with freshwater and shade, or injected with antitoxin, recovery rates of 75 to 90 percent and higher can result (Fig. 38.16). In contrast to waterfowl, very few coot, shorebirds, gulls, and grebes survive botulism intoxication, even after treatment. Experience to date with these species indicates that rehabilitation efforts may not be worthwhile.





Because avian botulism most often afflicts waterfowl in the seasons when they are flightless due to wing molt, biologists and rehabilitators must be careful to distinguish between birds in molt and birds with early stages of botulism, because the behavior of these birds may be similar. Molting birds are very difficult to catch, and birds that cannot be captured with a reasonable effort should not be pursued further. Birds that are suffering from botulism can easily be captured when they lose the ability to dive to escape pursuit. Birds at this level of intoxication still have a high probability of surviving if proper treatment is administered.

When botulism-intoxicated birds are treated, they should be maintained under conditions or holding pens that provide free access to freshwater, maximum provision for shade, the opportunity for recovered birds to fly out of the enclosure when they choose to, and minimum disturbance (including the presence of humans). It is also important to remove carcasses daily from holding pens to prevent the buildup of toxic maggots.

Costs associated with capturing and treating sick birds are high. Therefore, the emphasis for dealing with avian botulism should be on prevention and control of this disease rather than on treatment of intoxicated birds. However, antitoxin should be available for use in case endangered species are affected.

### **Human Health Considerations**

Botulism in humans is usually the result of eating improperly home-canned foods and is most often caused by type A or type B botulinum toxin. There have been several human cases of type E botulism in North America from eating improperly smoked or cooked fish or marine products. Type C botulism has not been associated with disease in humans, although several outbreaks have been reported in captive primates. Thorough cooking destroys botulinum toxin in food.

| Duck type  | Amount of antitoxin (cc) | Percentage recovered                 |                                    |
|--|--------------------------|--------------------------------------|------------------------------------|
|  |                          | Moderate <sup>1</sup> clinical signs | Severe <sup>1</sup> clinical signs |
| Pintail  |                          |                                      |                                    |
|   | 0.5                      | 91.9                                 | 69.0                               |
|  | 1.0                      | 93.5                                 | 73.3                               |
| Green-winged teal  |                          |                                      |                                    |
|   | 0.5                      | 93.5                                 | 57.4                               |
|  | 1.0                      | 91.2                                 | 58.1                               |
| Mallard  |                          |                                      |                                    |
|   | 0.5                      | 81.6                                 | 66.3                               |
|  | 1.0                      | 84.9                                 | 67.8                               |
| Shoveler   |                          |                                      |                                    |
|  | 0.5                      | 89.2                                 | 53.1                               |
|  | 1.0                      | 95.1                                 | 57.6                               |

<sup>1</sup> Condition of bird when treated with antitoxin.

**Figure 38.16** Recovery rates of ducks in response to antitoxin therapy.

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## Supplementary Reading

- Eklund, M.W., and Dowell, V.R., eds., 1987, Avian Botulism: An International Perspective: Springfield, Ill., Charles C. Thomas, 405 p.
- Hariharan, H., and Mitchell, W.R., 1977, Type C botulism: the agent, host spectrum and environment: The Veterinary Bulletin, v. 47, p. 95–103.
- Reed, T.M., and Rocke, T.E., 1992, The role of avian carcasses in botulism epizootics: The Wildlife Society Bulletin, v. 20, p. 175–182
- Rocke, T.E., 1993, *Clostridium botulinum*, in Gyles, C.L. and Thoen, C.O., eds., Pathogenesis of bacterial infections in animals (2d ed.): Ames, Iowa, Iowa State University Press, p. 86–96.
- Rocke, T.E., Smith, S.R., and Nashold, S.W., 1998, Preliminary investigation of the *in vitro* test for the diagnosis of avian botulism in wild birds: The Journal of Wildlife Diseases, v. 34 p. 744–751.