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-

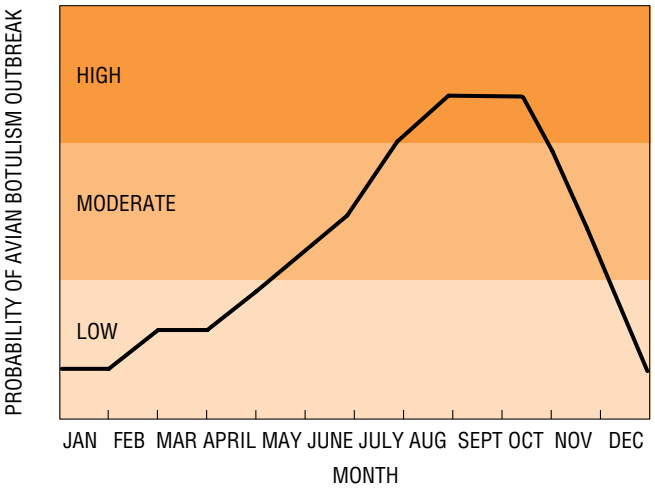

Figure 38.1 *Relative seasonal probability of type C botulism in North American water birds.*

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.*

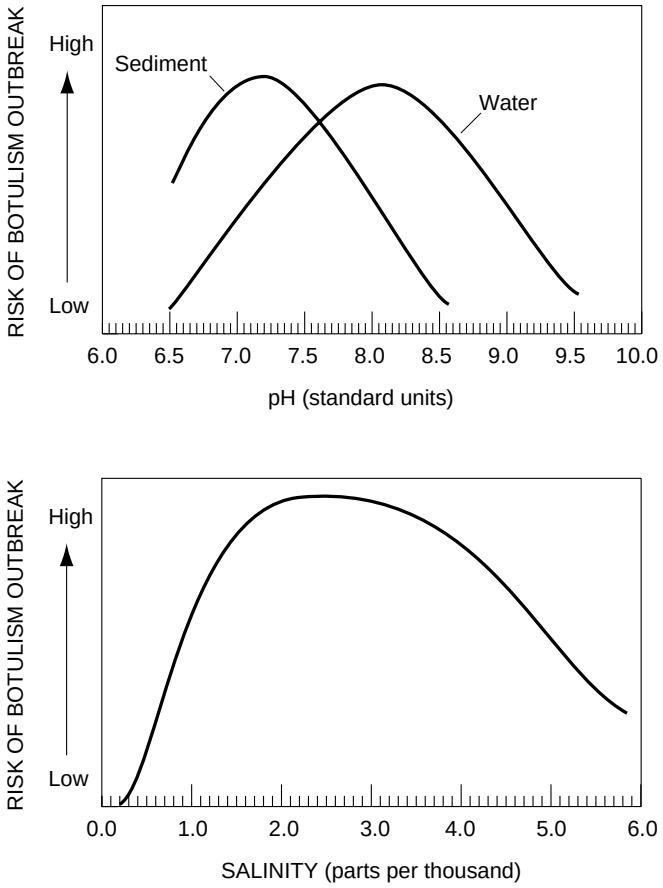

Figure 38.3 *Relationship of pH and salinity to avian botulism outbreaks.*

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

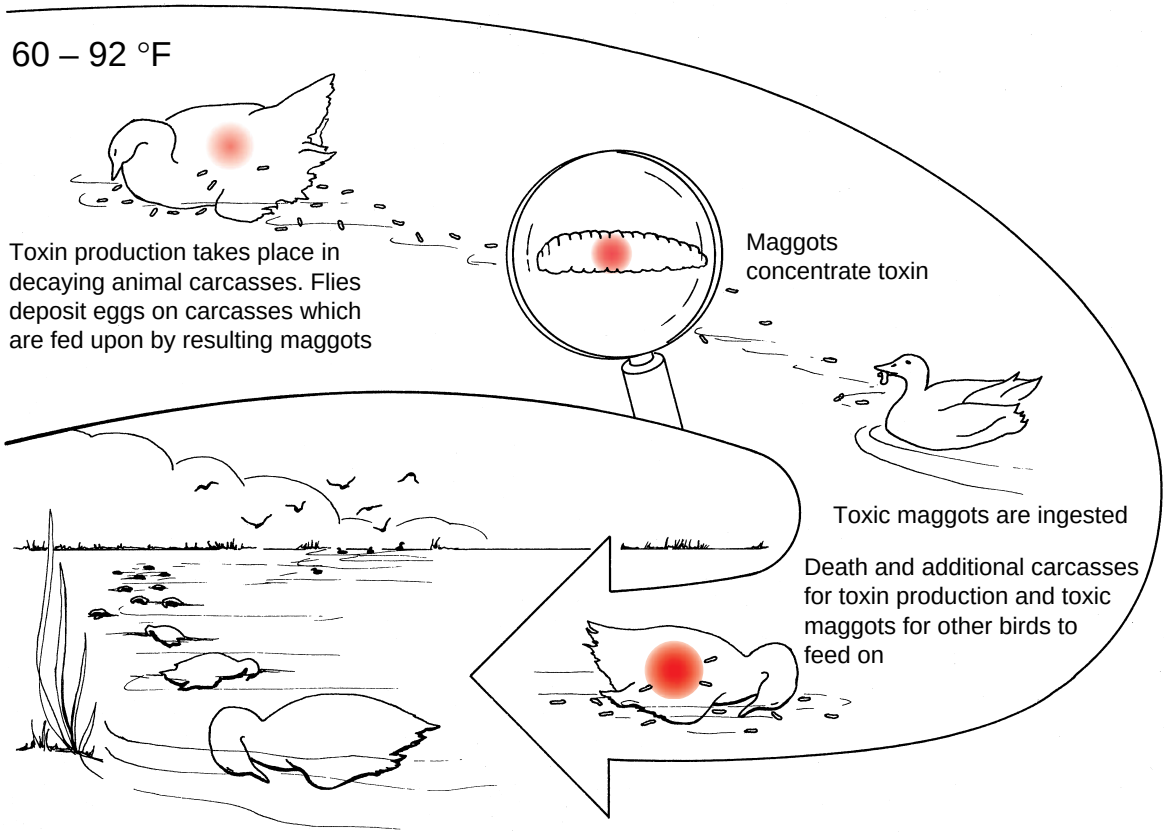

Figure 38.4 *Carcass-maggot cycle of avian botulism.*

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.

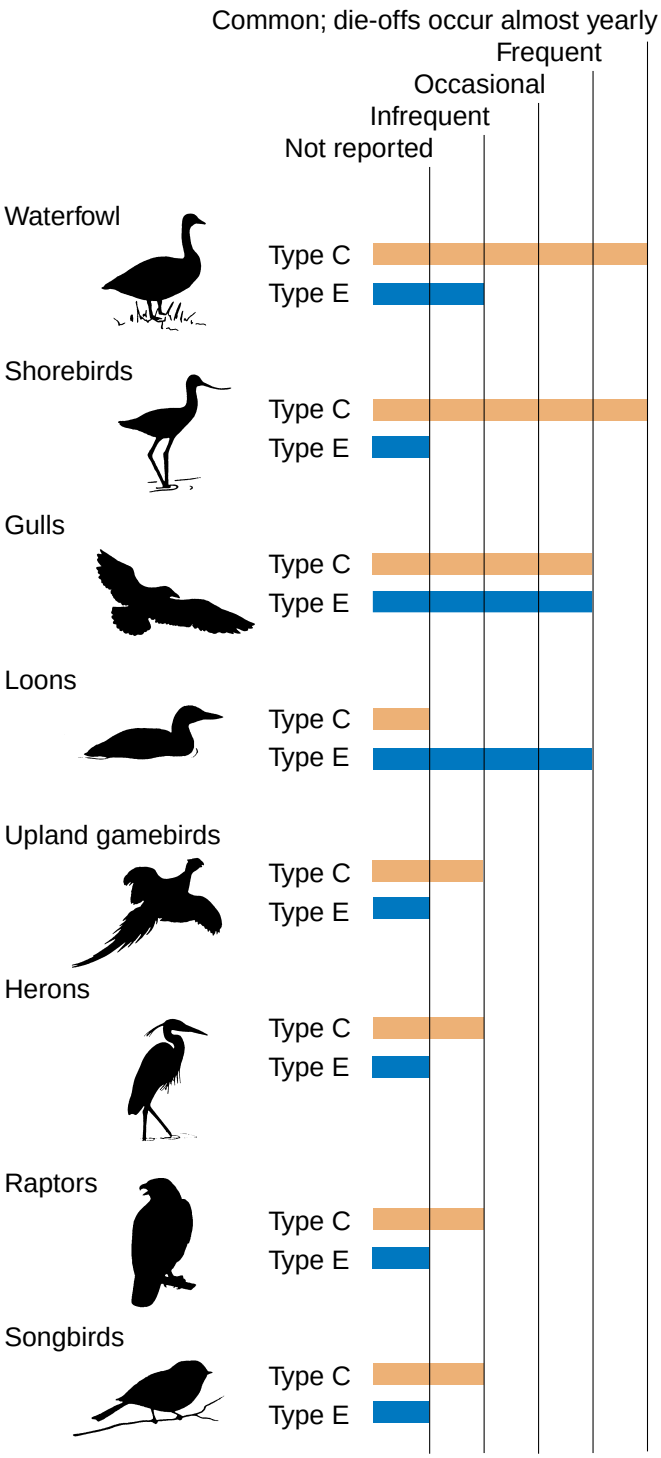

Figure 38.5 *Frequency of botulism in major groups of wild birds.*

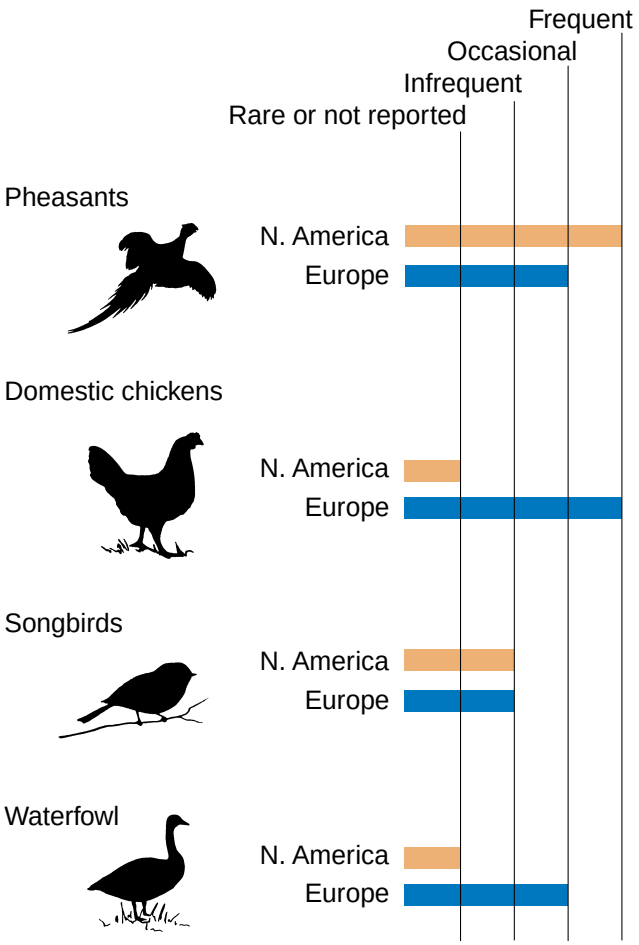

Figure 38.6 *Frequency of botulism in captive birds.*

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	100,000–300,000
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.

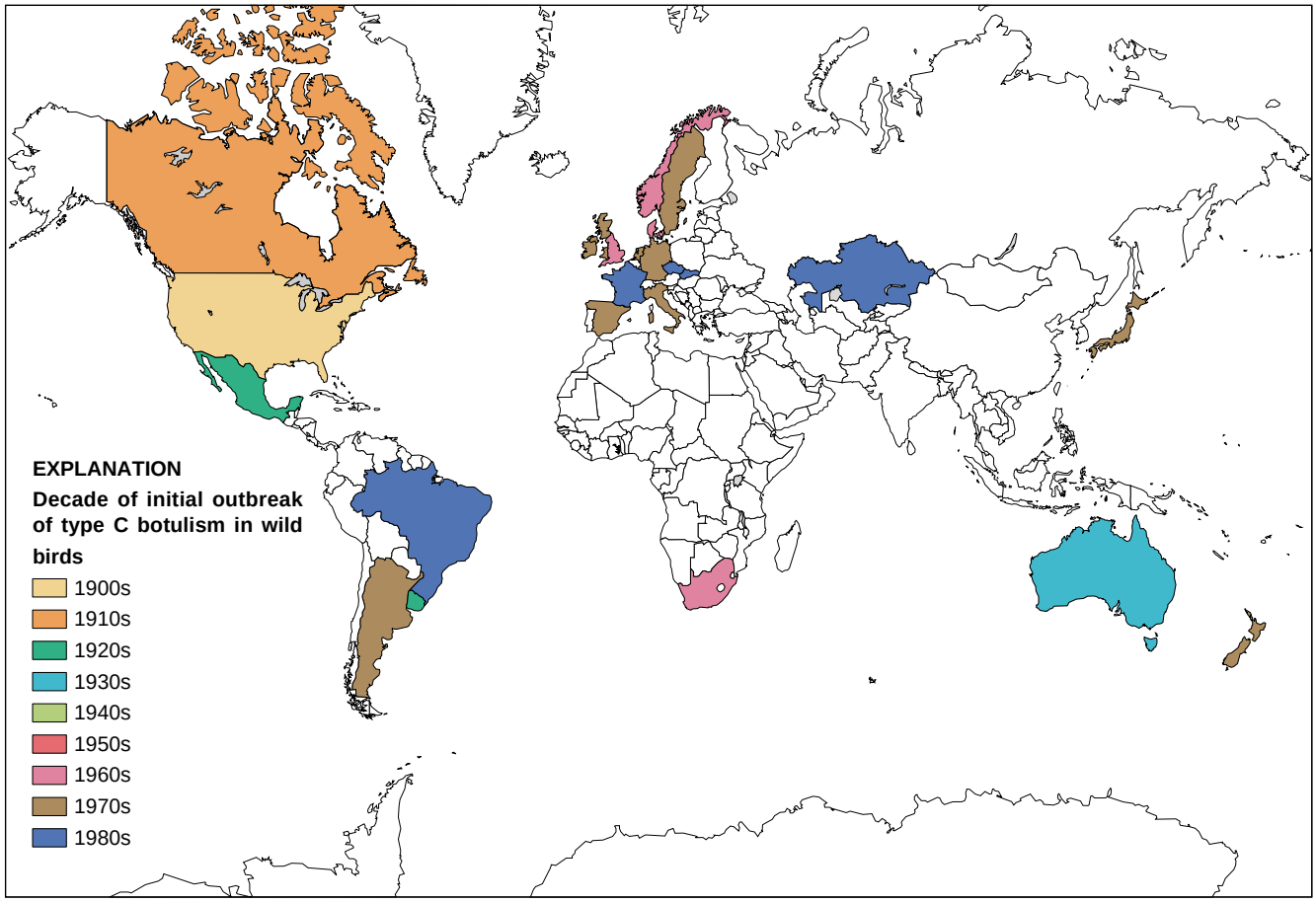

Figure 38.7 *Type C botulism outbreaks in wild birds.*

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.

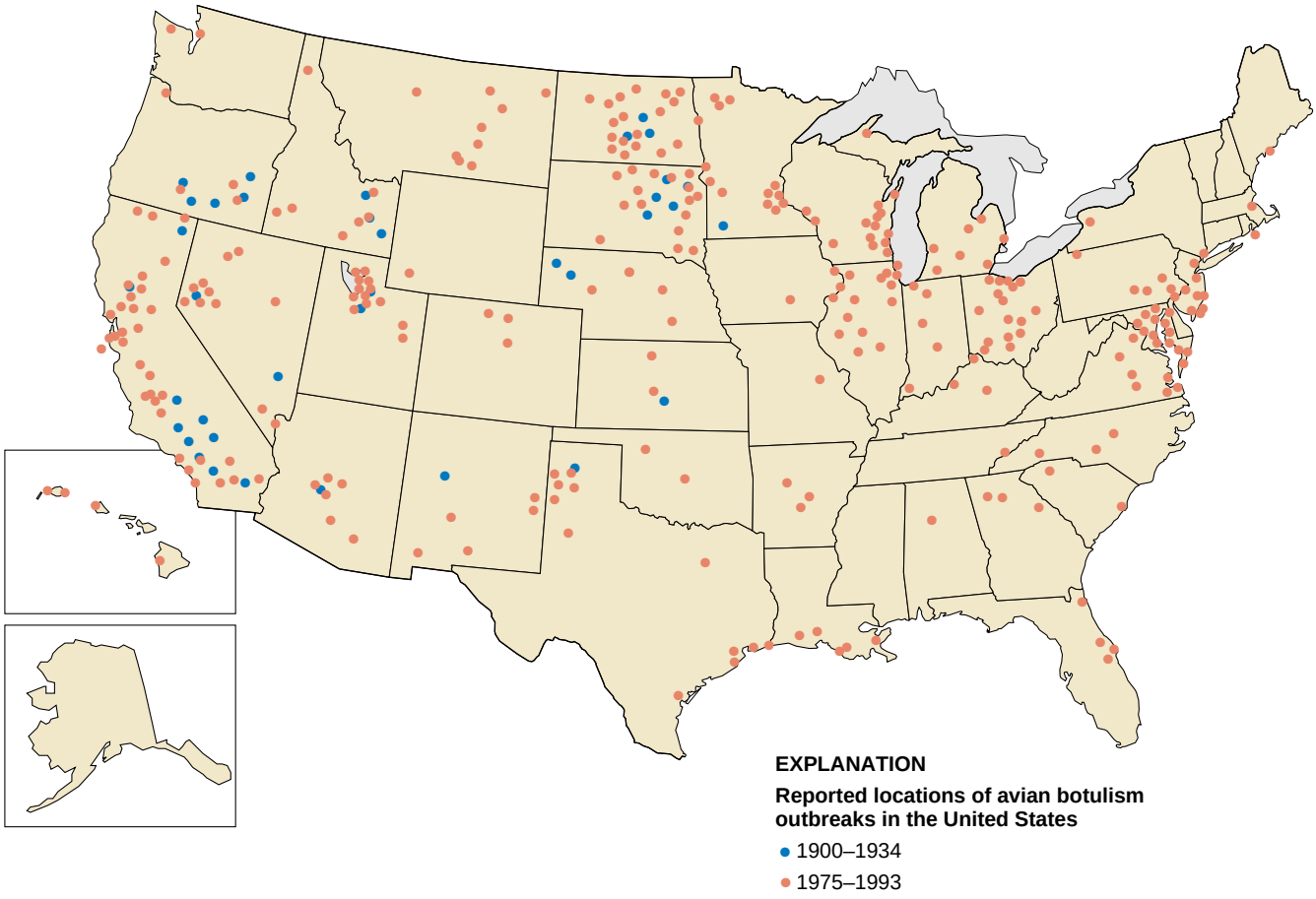

Figure 38.8 *Locations of avian botulism outbreaks in the United States.*

Photo by James Runningen

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, castorbean 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.

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

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[1] clinical signs	Severe[1] 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

[1] 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

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