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13.2.4. Avian Botulism: Geographic Expansion of a Historic Disease

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Synonyms

Limberneck, western duck sickness, duck disease, alkali poisoning

Cause

Avian botulism is a paralytic, often fatal disease of birds resulting from ingestion of toxin produced by the bacterium *Clostridium botulinum*. Waterfowl die-offs from the botulism are usually caused by type C toxin; sporadic die-offs among fish-eating birds, such as common loons (*Gavia immer*) and gulls, have been caused by type E toxin.

Not enough is known about avian botulism to precisely identify the factors leading to an outbreak. When an outbreak does occur, it is usually perpetuated by a well-understood bird-maggot cycle (Figure 1).

Clostridium botulinum persists in wetlands in a spore form that is resistant to heat and drying and in some instances remains viable for years. Toxin production occurs during multiplication of the vegetative form of the bacteria following spore germination. The vegetative form requires dead organic matter and a complete absence of oxygen to grow and produce toxin. Optimum growth of the bacteria occurs at about 25° C (77° F). Toxin production is optimized within a pH range of 5.7 to 6.2 and depends on the protein content of the medium in which the bacteria are growing. All kinds of animal protein are suitable for toxin production. Especially potent toxin is produced in bird, mammal, and a variety of invertebrate carcasses. This entire process is further complicated by a poorly understood but important role of bacteriophages—viruses that infect bacteria. Recent findings show that bacteriophages determine if toxin will be produced during *C. botulinum* growth and multiplication stages.

Important environmental factors that contribute to initiation of avian botulism outbreaks include water depth, water level fluctuations, and water quality; the presence of vertebrate and invertebrate carcasses; rotting vegetation; and high ambient temperatures.

Shallow water permits rapid warming of the submerged marsh soil during periods of high ambient temperatures. Toxin is produced when these soils contain both the spores of *C. botulinum* and suitable organic nutrients for spore germination and reproduction of bacterial cells. Fluctuating water levels that produce “feather edge” shorelines contribute to avian botulism outbreaks when terrestrial and aquatic invertebrates die as land areas are flooded and the underwater areas subsequently become dry when the water recedes. Fertilization of a marsh with sewage or run-off from agricultural activities can stimulate plant or invertebrate animal population growth for short periods, but results in plant and vertebrate die-offs once this stimulus subsides. The resulting mass of nutrients is then

Adapted from: Friend, M., editor. 1987. Field guide to wildlife diseases. U.S. Fish Wildl. Serv., *Resour. Publ.* 167. 225 pp.

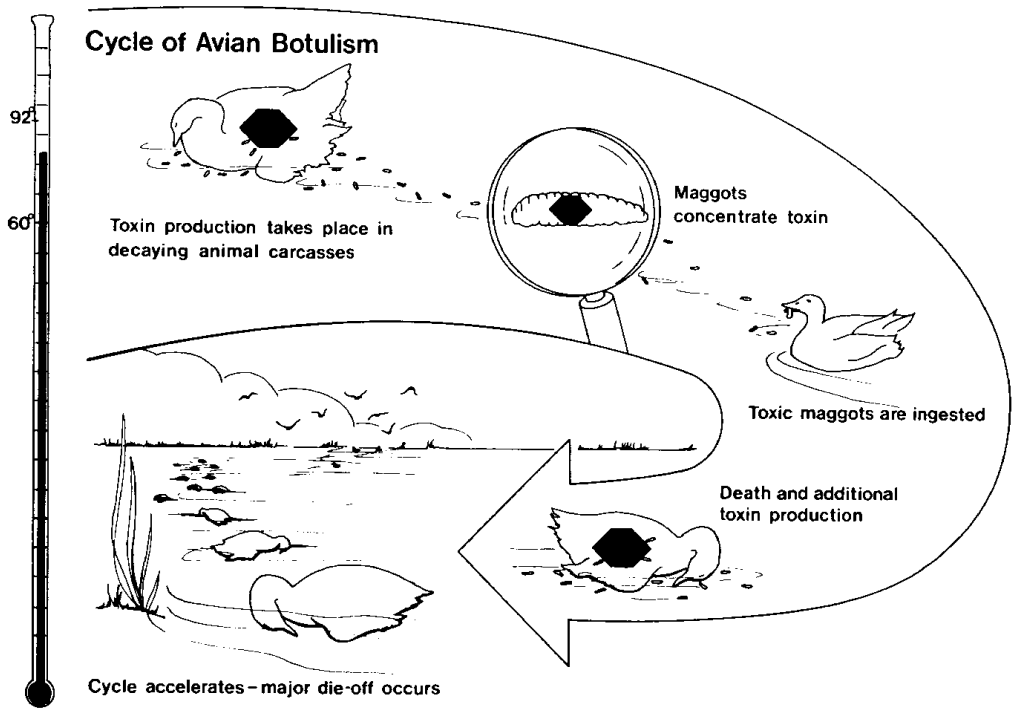

Figure 1. Avian botulism cycle.

available for growth of *C. botulinum* and toxin production. Dense vegetation can entrap and thus kill fish, amphibians, or invertebrates, and masses of rotting marsh plants can reduce oxygen levels to the point that aquatic animal life is killed. Both of these conditions provide large amounts of growth material for toxin production. The presence of vertebrate carcasses and high ambient temperatures are also conducive to the buildup of fly populations involved in the bird-maggot cycle for avian botulism transmission.

Species Affected

Many species of birds and some mammals are affected by type C botulism. In the wild, waterfowl and shorebirds are most often affected (Figure 2). Vultures are known to be highly resistant to type C toxin.

Losses vary a great deal from year to year at site-specific locations and from species to species. A few hundred birds may die in 1 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 have been relatively common (Table 1).

Figure 2. Frequency of botulism in major groups of wild birds.

Type of bird	Type C	Type E
Waterfowl	●●●●	●
Loons	○	●●●
Herons	●●	○
Shorebirds	●●●●	○
Gulls	●●●	●●●
Raptors	●	○
Upland game birds	●	○
Songbirds	●	○

●●●● Common; die-offs occur almost yearly
●●● Frequent
●● Occasional
● Infrequent
○ Not reported

Table 1. *Major waterfowl botulism outbreaks.*

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 (near Billings)	1978	50,000
Montana (near Billings)	1979	100,000
Great Salt Lake, Utah	1980	110,000

Table 2. *Initial outbreaks by location of type C avian botulism in wild waterfowl.*

Location	Year	Location	Year
The Americas		Europe	
United States	1910	Sweden	1963
Canada	1913	Denmark	1967
Uruguay	1921	England	1969
Mexico	1976	Netherlands	1970
Argentina	1979	East Germany	1971
Brazil	1982	West Germany	1971
Australia-Asia		Italy	1973
Australia	1934	Spain	1973
New Zealand	1972	Norway	1975
Japan	1973	Scotland	1977
Africa		Czechoslovakia	1981
Union of South Africa	1956	Wales	1983

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 to occur in many other countries. Most of these reports are recent, usually within the past 20 years (Table 2). Most type C outbreaks within the United States occur west of the Mississippi River; however, outbreaks have occurred from coast-to-coast and border-to-border (Figure 3). Type E outbreaks in birds are much less frequent and within the conterminous United States have been confined to the Great Lakes region.

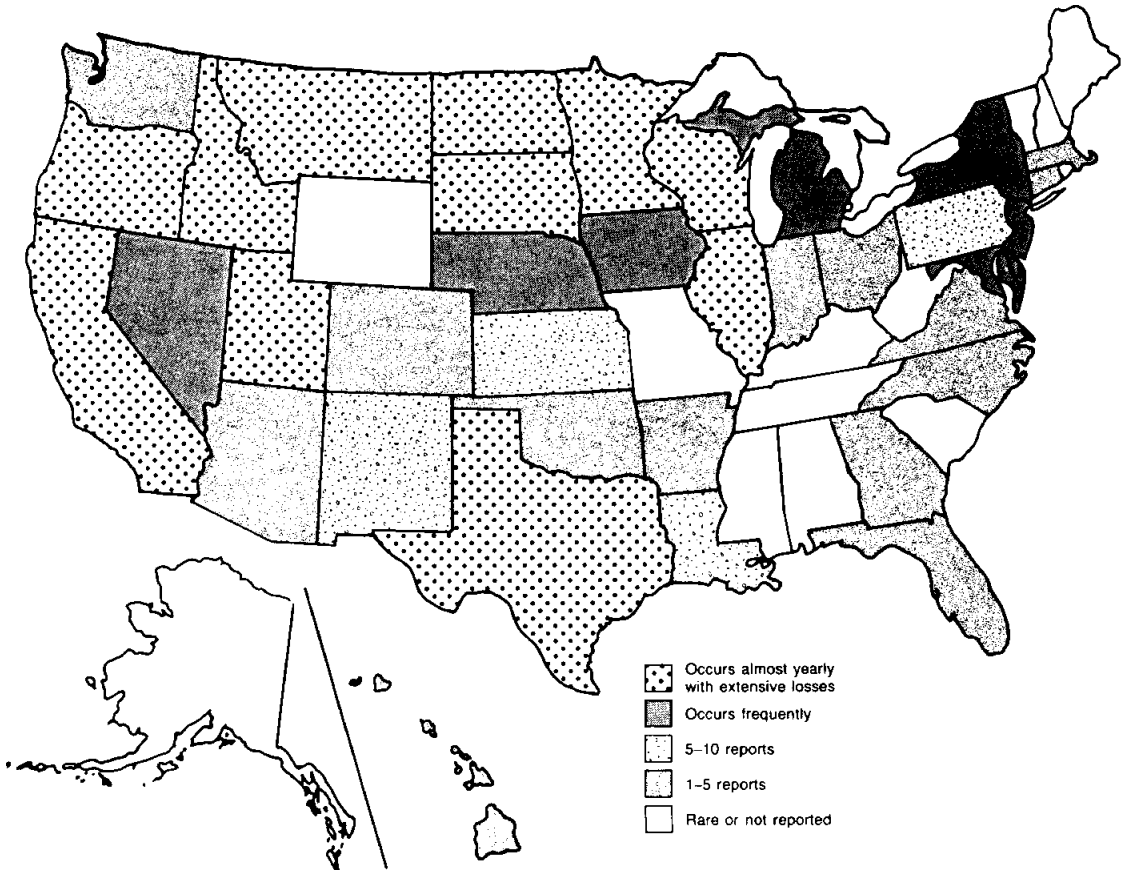

Figure 3. Frequency of type C botulism in waterfowl.

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 portions 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 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 vegetation bordering the water or the original water's edge. 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.

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

Avian botulism affects the peripheral nerves and results in paralysis of voluntary muscles. Inability to sustain flight is seen early in botulism. Once the power of flight is lost and paralysis of leg muscles has occurred, ducks suffering from botulism often propel themselves across the water and mud flats with their wings. This sequence of signs contrasts with that of lead-poisoned birds, which retain their ability to walk and run even though flight becomes difficult.

Paralysis of the inner eyelid or nictitating membrane (Figure 4) and neck muscles follows, resulting in inability to hold the head erect (Figure 5). These are the two most easily recognizable signs of avian botulism. Once birds reach this stage, death from drowning often occurs before the bird might otherwise die from the respiratory failure caused by botulinum toxin.

Avian botulism often occurs in the seasons when waterfowl are flightless because of wing molt. Care then must be taken to separate birds in molt from those with early stages of intoxication because the behavior of these birds may be similar. Molting birds are difficult to catch and birds that cannot be captured with a reasonable effort should not be pursued further. If these birds are suffering from botulism, they can be easily captured when they become unable to dive to escape pursuit. Birds at this level of intoxication still have a high probability for survival if proper treatment is administered.

Figure 4. Paralysis of the inner eyelid is a common sign in botulinum-intoxicated birds.

Figure 5. Paralysis of the neck muscles in bitulinum-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 type C or type E botulism.

Diagnosis

The most reliable test for avian botulism is the mouse protection 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 or die with characteristic signs if botulinum toxin is present in the serum sample.

Control

Management of Environment

Control efforts need to focus on three important factors that contribute to the development and maintenance of avian botulism outbreaks: fluctuating water levels during hot summer months, an abundance of flies, and animal carcasses for toxin production. On areas managed primarily for migratory waterfowl (ducks, geese, swans), reflooding of land that has been dry for a long time is not recommended during summer. Similarly, sharp drawdowns of water should be avoided since they could result in fish-kills and die-offs of aquatic invertebrates whose carcasses could then become centers for the growth of *C. botulinum*. On those areas managed primarily for shorebirds, water drawdowns are essential, and botulism control must focus on a cleanup of any carcasses that may result.

Prompt removal and proper disposal of vertebrate carcasses by burial or burning are highly effective mechanisms for removing the major sources of toxin production and maggot development. 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. Consumption of as few as two to four of these toxic maggots can result in intoxication of a duck, thereby perpetuating the botulism cycle. It is not uncommon to find three or four freshly dead birds within a few feet of a maggot-laden carcass. Failure to carry out adequate carcass removal and disposal programs can result in a rapid buildup of highly toxic materials, and can accelerate losses as well as seed the environment with *C. botulinum* toxin and spores as the carcasses decompose. Toxin formed in these carcasses is quite stable. This preformed toxin can be taken in by invertebrates, remain free in bottom sediments, or become suspended in the water column where it can serve as the source of winter and spring botulism outbreaks when ingested by feeding birds.

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 time sequence. These conditions have direct management implications that should be applied toward 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 habitats to reduce the potential for botulism losses and deny bird use on major problem areas during the botulism “season.” Surveillance and carcass disposal activities should start 10 to 15 days before the earliest documented cases and continue 10 to 15 days after the end of the botulism “season.” Habitat modifications will primarily involve control of water quality and water levels.

Because fish carcasses can also serve as sites for *C. botulinum* growth, they should be promptly removed during fish control programs in marsh environments, or fish control programs should be restricted to the cooler months (non-fly season). Power lines that cross marsh environments have been associated with major botulism outbreaks. Bird carcasses from collisions with power lines have served as initial points for toxin production within the marsh environment. Therefore, if possible, power lines should not be placed across marsh environments used by large concentrations of water birds.

Numerous outbreaks of avian botulism have been associated with sewage and other wastewater discharge into marsh environments. This relation is not presently understood, but has occurred often enough that wetland managers should discourage the discharges of these effluents when substantial waterfowl or shorebird use occurs or is likely to occur on an area during the ensuing 30 days.

Treatment of Sick Birds

Studies at Bear River Refuge, Utah, have clearly demonstrated that a high percentage of botulinum-intoxicated waterfowl can be saved. If the birds are provided with fresh water and shade, or injected with antitoxin, recovery rates of 75–90% and higher can result. In contrast with waterfowl,

very few American coots (*Fulica americana*), shorebirds, gulls, and grebes have survived treatment for botulism. Experience to date with these species indicates that rehabilitation efforts are not worthwhile.

When botulinum-intoxicated birds are treated, the birds should be maintained under conditions that provide unrestricted access to fresh water, maximum provision for shade, an opportunity for birds that recover to fly out of the enclosure when they choose to, and minimum disturbance (including presence of humans). It is also important to remove carcasses daily from enclosures to prevent the buildup of toxic maggots within the treatment area, and to monitor the cause of mortality since one cannot assume botulism is the cause. The weakened condition of botulinum-intoxicated birds can result in the eruption of infectious disease such as avian cholera. Should this occur, it is essential to immediately address the infectious disease problem.

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. The National Wildlife Health Research Center has produced and maintains antitoxin for this purpose. Contact the center's Resource Health Team for assistance.

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 a few human cases of type E botulism in North America as the result of eating improperly smoked or cooked fish or marine products. Although humans are regarded as being fairly resistant to type C botulinum toxin, at least two cases of type C botulism have been reported, although the origins were unidentified. Thorough cooking destroys botulinum toxin in food.

Suggested Reading

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