Wildlife Health Australia

Botulism in Australian wild birds

Fact sheet

Introductory statement

Botulism is a paralytic disease caused by ingestion of a toxin produced by the bacterium *Clostridium botulinum*. Avian botulism can occur in any bird species, but is most frequently seen in ducks, geese, swans, ibis, egrets and pelicans. It occurs worldwide and is commonly reported in wild birds in Australia. The majority of birds that develop botulism will die if not treated. Toxin production by *C. botulinum* only occurs under specific environmental conditions. Most outbreaks of botulism occur in the summer and autumn and often occur repeatedly in the same environment. Large numbers of birds can die during outbreaks and this can contribute to local and regional population declines.

Aetiology

Order: *Clostridiales* Family: *Clostridiaceae* Genus: *Clostridium*

Clostridium botulinum is a Gram-positive, rod-shaped bacterium that produces environmentally resistant spores when faced with unfavourable conditions. It grows in environments where there is little or no oxygen. *Clostridium botulinum* isolates can produce any one of seven distinct types of neurotoxins (A-G). Most produce only one neurotoxin, but some isolates will produce multiple toxins. *Clostridium botulinum* isolates are also categorized into groups I-IV by their ability to use protein substrates for energy. Toxin production is generally group specific and Group III strains are associated with animal disease. Ingestion of the toxin causes the paralytic disease known as botulism. Waterfowl mortalities due to botulism are usually caused by type C toxin (or type C/D mosaic), although type E has caused sporadic mortalities among fish-eating birds (such as gulls) mainly around the Great Lakes in North America (Rocke and Bollinger 2007; Le Maréchal et al. 2016).

Natural hosts

Clostridium botulinum is a soil bacterium that is particularly common in wetland environments. There is also some evidence that it may grow in the digestive system of some species of birds (Vidal et al. 2011). *Clostridium botulinum* also grows well in decomposing carcasses. Toxins can accumulate in invertebrate hosts,

such as the maggots that feed on these carcasses. Spores produced by the bacterium persist in the environment for years (Rocke 2006).

World distribution

Outbreaks of avian botulism have been reported since the beginning of the 20th Century in North America, South America, Europe, Africa, Asia and Australia. Type C botulism has been reported in over 260 bird species (39 families) in at least 28 countries. Outbreaks have killed hundreds of thousands of birds in North America (Le Maréchal et al. 2016).

Occurrences in Australia

Outbreaks of avian botulism type C are known to have occurred in Australia since 1934 and occur regularly in many areas of the country (Pullar 1934; Grubb 1964; McKenzie et al. 1982; Woodall 1982; Galvin et al. 1985). Numbers of birds that die in these outbreaks are generally less than 100, but the death of up to 1,500 birds (predominately grey teal [*Anas gibberifrons gracilis*]) was recorded in one outbreak in Victoria (Galvin et al. 1985).

Sixty-eight events of suspected or confirmed avian botulism were recorded in Australia between 2006 and 2012. In ten of these events, multiple species of birds from several bird orders were affected, however Anseriformes (ducks, geese and swans) were involved at the majority of events. Most of the reported cases occurred from November to April, with fewer cases in the cooler months (Grillo et al. 2013).

Ten wild bird mortality events were attributed to suspected or confirmed cases of botulism from October to December 2012 (Grillo et al. 2013). Botulism was confirmed by enzyme-linked immunosorbent assay (ELISA) in one pelican (*Pelecanus* sp.) and five ducks found dead in a lake near Port Headland, WA. In the other nine events, a presumptive diagnosis of botulism was made based on clinical signs, environmental conditions and a lack of lesions upon necropsy. Five of the events reported were from Victoria, involving mostly Australian white ibis (*Threskiornis molucca*), but in some instances wild ducks and pelicans were also affected. Two events each were also recorded in NSW and Qld with only wild ducks affected.

Between January and March 2011, a number of suspected cases of botulism were reported in waterbirds across Australia. In NSW, reports were received from areas near Tweed Heads, Newcastle, Albury and Wagga Wagga. Reports were also received near Brisbane in Qld, Alice Springs in the NT, and Melbourne, Shepparton, and Bendigo in Vic. Anseriformes were involved in all events, with multiple species affected including magpie geese (*Anseranas semipalmata*), Australasian grey teal, Pacific black ducks (*Anas superciliosa*), Muscovy ducks (*Cairina moschata*), masked lapwings (*Vanellus miles*), and black swans (*Cygnus atratus*) (Cox-Witton et al. 2011).

Epidemiology

Outbreaks of avian botulism type C (or C/D) can occur in both sporadic, unpredictable patterns and highly predictable patterns (occurring at the same location and time year after year). Clostridial spores are widespread in many aquatic environments (Soos and Wobeser 2006). The factors that predispose to botulism outbreaks are not fully understood but suitable growth conditions include anaerobic conditions, the presence of a source of protein, high temperatures (25-42 C) and moisture (Le Maréchal et al. 2016). Most outbreaks occur in the summer or autumn and increased water temperature is considered to be a risk factor. Other

factors that have been associated with avian botulism outbreaks include increased organic material in the sediment, low or fluctuating water levels, high water pH (7.5-9.0), redox potential (increased risk if less than 100) and increased salinity (> 2 ppt) (Rocke and Samuel 1999).

Dabbling and filter-feeding waterfowl, shorebirds that feed near the surface waterbodies, and sediment- or fish-eating birds are considered higher risk of developing botulism than other waterfowl (Le Maréchal et al. 2016).

Increased bird mortalities (as the result of other factors) may also trigger outbreaks (Soos and Wobeser 2006). Carcasses act as an environment where *C. botulinum* can grow and produce toxins. Maggots feeding on the carcasses concentrate toxin and poison other birds when they are eaten, resulting in an escalating spiral of mortality. Species of birds that feed on maggots are at greatest risk for intoxication, but intoxication can also occur in predatory birds and mammals (Le Maréchal et al. 2016).

Outbreaks of avian botulism type E have primarily been described in the Great Lakes of North America. The vast majority of species affected are birds that eat fish (loons, ducks, gulls and terns). It is hypothesized that these birds consume fish that are sick as the result of their consumption of invertebrates containing this toxin (Hannett et al. 2011). It has also been postulated that an introduced species of fish, the round gobi (*Neogobius melanostomus*) may play an important role in these outbreaks (Ruffing 2004).

Clinical signs

Botulism causes flaccid paralysis; clinical signs are indicative, but not specific for this disease. Frequently, the first indication of an outbreak of avian botulism is a sudden increase in the number of dead birds on the edge of a water body (Rocke and Friend 1999). Birds exhibit signs of progressive weakness and paralysis of skeletal muscles, with the first obvious sign typically the loss of ability to fly. Birds may lose the ability to use their legs but may still be able to shuffle forward using their wings. In advanced cases, they can no longer hold up their head, hence the name, 'limberneck,' and the third eyelid protrudes across the eye. Birds may have a change in voice or completely lose the ability to vocalise. Death is a result of drowning, asphyxiation due to paralysis of respiratory muscles, or starvation and dehydration. Affected birds are also more likely to be subjected to predation (Rose 2005; Raymundo et al. 2012).

Diagnosis and pathology

Diagnosing botulism can be challenging as there are no associated gross or microscopic lesions (Rose 2005). A presumptive diagnosis of avian botulism is based on the geographic location (whether previous outbreaks have occurred in the area), the time of the year, the species of the birds affected, clinical signs and absence of significant lesions at necropsy. The finding of maggots in the digestive tract may also be suggestive of botulism intoxication (Le Maréchal et al. 2016). Detection of the toxin can be difficult however, finding toxin in serum, gut, organs or maggots from dead birds further supports a diagnosis of botulism (Rocke and Bollinger 2007). *Clostridium botulinum* can be carried in the gut of healthy birds without clinical signs, but there is limited evidence that types C, D and C/D mosaic are part of the normal avian gut flora (Le Maréchal et al. 2016; Palmer 2018). The presence of *C. botulinum* in the gut alone is not diagnostic for the disease and a definitive diagnosis requires the demonstration of toxin in serum or tissue and exclusion of other differential diagnoses. See also WHA Fact Sheet "Diagnosing Botulism in Birds in Australia".

Differential diagnosis

A list of differential diagnoses for avian botulism will depend on the geographic location of the outbreak. Other toxins including organophosphates, organochlorides, mercury, algal toxins, castor bean and lead can cause neurological signs that include weakness and paralysis. These intoxications could be difficult to differentiate from avian botulism as they may not be associated with gross or microscopic lesions. Viral diseases including avian influenza, exotic Newcastle disease, duck plague, West Nile virus, and avian bornavirus (Canada goose genotype) can also cause significant mortality events and some birds may exhibit neurological signs. Fowl cholera (*Pasteurella multocida*) causes large waterfowl die offs but is more likely to occur in the winter. Nutritional deficiencies e.g. calcium, thiamine, vitamin A, vitamin E and selenium all have the potential to cause neurologic diseases in birds, but are not likely to do so in multiple birds, especially adult birds, over a short stretch of time (Rose 2005; Sonne et al. 2012).

Laboratory diagnostic specimens and procedures

Capture enzyme-linked immunoassay (cELISA) and PCR are used to detect botulinum toxin. Although previously considered a gold standard for testing, mouse inoculation is no longer used in Australia.

Serum should be collected from living or recently dead birds for toxin detection via cELISA. Liver, ingesta, blood and other organ samples can be collected for PCR testing. Maggots collected from dead birds can be tested (Soos and Wobeser 2006).

Not all States and Territories in Australia perform testing for botulinum toxin. The cELISA is available at the Biosecurity Sciences Laboratory (BSL) in Brisbane, QLD (www.business.qld.gov.au/industry/agriculture/land-management/health-pests-weeds-diseases/sample-testing/acceptance) and Department of Primary Industries and Regional Development (DPIRD) Western Australia Diagnostic Laboratory Services (DDLS). The PCR test is available at the Department of Primary Industries and Regional Development (DPIRD) Western Australia, Diagnostic Laboratory Services (DDLS). Liver, blood and gut content are appropriate samples. See the WHA Fact Sheet “Diagnosing Botulism in Birds in Australia” for more information.

Treatment

The main treatment for avian botulism is supportive care (Rocke and Friend 1999; Le Maréchal et al. 2016). Recovery takes several weeks and fluid therapy and supplemental feeding is generally required. Botulinum antitoxin may be used, but is not commercially available (Rose 2005).

Prevention and control

Artificial water bodies should be designed so that sufficient aeration and water circulation are provided. Steep sides and deeper water may also keep water temperatures stable and prevent botulinum production from occurring (Rose 2005). It is also very important to prevent or limit the organic inputs entering both natural and artificial water bodies, as this reduces the energy resources available for the bacterium to grow (Rocke and Friend 1999).

It has been shown that by reducing carcass numbers and thus maggot load, mortality may be reduced (Evelsizer et al. 2010). Soos and Wobeser (2006) suggested picking up carcasses that died other causes in waterbird breeding colonies before a botulism outbreak occurred might help prevent an outbreak.

Dispersal of birds has also been used to reduce mortality. Many outbreaks occur on breeding grounds in North America and dispersing birds under these conditions is not an option. However, in Australia, outbreaks do not occur in breeding flocks and so efforts to move birds off the water body that is the cause of the outbreak may be an effective way of reducing mortality.

Surveillance and management

Confirmed and suspected cases of botulism can be reported to your local Department of Primary Industries or regional WHA Wildlife Coordinator (www.wildlifehealthaustralia.com.au). Reports of outbreaks of botulism are captured in Australia's wildlife health information system. Botulism is listed by the OIE (World Animal Health Organisation) as a reportable disease in wildlife (www.oie.int) but is not included in AUSVETPLAN.

Research

There is a need for a comprehensive review of all sites and their characteristics where avian botulism outbreaks have occurred in Australia. This information could be used to predict the conditions that would most likely lead to outbreaks, identify sites where outbreaks occur regularly and help communities design ponds and lakes that are less likely to create an environment that is favourable for these outbreaks to occur. Further work is needed to better understand optimal sampling protocols for ante and post mortem test accuracy.

Statistics

Wildlife disease surveillance in Australia is coordinated by the Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia's States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. Please contact admin@wildlifehealthaustralia.com.au. There are over 220 cases of avian botulism recorded in eWHIS, from every state and territory of Australia.

Wildlife Health Australia (WHA) reviewed avian botulism cases over a six-year period (Grillo et al. 2013):

- There were 68 confirmed or suspected cases of avian botulism in Australia 2006-2012
- Most events occurred between November and April
- Ten events included species from multiple bird orders; 54/68 events involved Anseriformes (e.g. ducks, geese, swans) and 11/68 events involved Ciconiiformes (e.g. ibis, egrets, pelicans).

Human health implications

There are no recorded cases of human poisoning due to Type C botulinum toxin (Rocke 2006).

Conclusion

Botulism occurs commonly in water birds across Australia. It should be considered an import differential diagnosis for any mortality event in water birds, particularly if the event occurs during the summer or autumn. The factors that predispose to botulism outbreaks are complex and incompletely understood.

Further studies into the environmental conditions associated with these outbreaks in Australia may help to understand why they occur and mitigate their impact.

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To provide feedback on this fact sheet

We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. If you can help, please contact us at admin@wildlifehealthaustralia.com.au.

Wildlife Health Australia would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to admin@wildlifehealthaustralia.com.au. We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for the network (or update current sheets). A small amount of funding is available to facilitate this.

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