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# AVIAN BOTULISM—ANOTHER PERSPECTIVE

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ABSTRACT: Waterfowl botulism is unique among intoxications because toxin produced within its victims leads to secondary poisoning of other birds. Because of this phenomenon, the epizootiology of the carcass-maggot cycle of botulism resembles that of an infectious disease and the reproductive rate (R) of the disease could be defined as the average number of secondary intoxications attributable to a single carcass introduced into a marsh. I propose that toxin production and botulism occur commonly at a low level in many marshes and that factors which influence R determine when the disease expands into a large epizootic. A model that incorporates the number of carcasses occurring in a marsh, the probability of a carcass containing spores, the probability of a carcass persisting until toxin-bearing maggots emerge, and the contact rate between live birds and toxin, may be useful for predicting the extent of secondary poisoning, for identifying questions for research, and as a theoretical basis for management.
*Key words:* *Clostridium botulinum*, waterfowl, epizootiology, model, research, management.

## INTRODUCTION

Botulism causes mortality of wild waterfowl in many areas of the world. The etiology has been known for many years (Kalmbach and Gunderson, 1934) and the microbiology of the disease is well understood. Botulism in waterfowl is caused by ingestion of a potent neurotoxin ($C_1$) produced by the α strain of *Clostridium botulinum* type C. Toxigenicity in type C depends on infection of the bacterium by specific bacteriophages that carry the neurotoxin gene (Eklund et al., 1987). The organism grows vegetatively under anaerobic conditions and the optimal temperature for growth is comparatively high (30 to 37 C) (Cato et al., 1986). Spores of *C. botulinum* type C are common in marsh soil and can persist there for years (Smith et al., 1982). It appears that decaying vegetation is poor substrate for vegetative growth of the organism and toxigenesis, while decaying animal material is highly suitable (Bell et al., 1955). Vertebrate carcasses are particularly important (Smith and Turner, 1987), as they provide a large amount of substrate, a self-contained anaerobic microenvironment, and the high temperatures optimal for growth and toxin production (Wobeser and Galmut, 1984). Animals in a marsh environment ingest spores frequently; for example, >50% of healthy sentinel mallards (*Anas platyrhynchos*) in a marsh with a history of botulism had *C. botulinum* spores in their liver or intestine (Reed and Rocke, 1992). When such an animal dies for any reason, there is a putrefaction, invasion of tissues by *C. botulinum* from the gut, and toxin production (Notermans et al., 1980; Smith and Turner, 1987). Larvae (maggots) of sarcophagous flies, and other invertebrates, feeding on vertebrate carcasses may contain large amounts of toxin (Duncan and Jensen, 1976; Hubalek and Halouzka, 1991), as well as bacterial cells and spores. Birds that ingest such maggots may die of intoxication and their carcasses then become substrate for generation of further toxin and maggots (Hunter et al., 1970). Despite the information available, management of this disease still consists largely of collecting carcasses during epizootics. Viewing known data from a variety of perspectives and assembling information into simple models often help to identify knowledge gaps and hypotheses that can be tested. This paper is presented for that purpose.

## BOTULISM AS A CONTAGIOUS DISEASE

Botulism in waterfowl is distinct from any other intoxication in birds, and even from botulism in most other species, because the production of toxin within its

victims leads to secondary poisoning. It could be considered a contagious disease, if contagion is defined as "the communication of disease from body to body by contact direct or mediate" (Simpson and Weiner, 1989) and also as a vector-borne disease because of the role of maggots in transmission. The similarities to an infectious disease provide a perspective from which to consider the epizootiology of botulism. Research on avian botulism has concentrated on defining the environmental conditions that favor growth and toxin production by the organism, while the population biology of the disease has received little attention. I believe that bacterial growth and toxin production occur frequently in botulism-prone marshes, because spores are abundant and the carcass microenvironment is largely independent of environmental factors (Wobeser and Galmut, 1984; Wobeser et al., 1987). Further, I propose that the unique phenomenon of secondary poisoning is the major factor that determines when a large epizootic will occur. The reproductive rate (R) of an infectious disease has been defined as the "average number of secondary infections attributable to a single infectious case introduced into a fully susceptible population" (Fine et al., 1982). I would define R for botulism as the average number of secondary intoxications attributable to a single carcass introduced into a marsh. In infectious diseases, when $R = 1$, on average only a single secondary case results from each infectious case and the infection is barely able to maintain itself. If $R < 1$, the disease cannot persist, while if $R > 1$, the disease incidence will increase. There likely is a similar relationship between the size of R and the epizootiology of botulism in a population.

Animals that have *C. botulinum* type C spores in their tissues die continuously of many causes in wetlands. Toxin production occurs in some of these, for example, toxin formed in the carcasses of 41% of maggot-infested mallards that died or were killed in a botulism-prone marsh (Reed and Rocke, 1992). Since a bird carcass may contain thousands of maggots, and even one maggot may be sufficient to poison a duck (Hubalek and Halouzka, 1991), R is potentially very large. However, large epizootics of botulism occur only sporadically on most wetlands. At Eyebrow Lake, Saskatchewan, (50°55′N, 106°08′W), a botulism-prone marsh that I have visited frequently during the past 20 years, individual birds with botulism can be found during most summers if a careful search is made, but epizootics have been separated by 2 to 6 yr intervals with no major mortality. I suspect that in most years at Eyebrow Lake, and probably many other marshes, vertebrates die continuously, toxin is formed in some of their carcasses, and cases of botulism occur; but $R \leq 1$, so that the disease flickers or smolders in a limited manner. I would describe botulism at Eyebrow Lake as enzootic (occurring at a regular, predictable or expected rate) with periodic epizootics (occurring with a frequency substantially greater that expected), the latter probably occurring when $R > 1$.

Factors that could influence R include: (1) the proportion of carcasses that contain spores, since those that do not contain spores represent no risk; (2) the rate at which carcasses are removed by scavengers, since carcasses consumed before maggots are available represent no risk; and (3) the probability of birds contacting and consuming maggots from carcasses. The R for botulism is equivalent to $M_2/M_1$ where $M_2$ = number of animals dying of secondary poisoning originating from $M_1$, and $M_1$ = number of animals dying in a marsh for any reason during a particular period. Further, $M_2 = M_1(P_s)(P_m)(\beta)$ with:

$P_s$ = proportion of carcasses that contain spores of toxigenic *C. botulinum*.

$P_m$ = proportion of carcasses that become infested with maggots and persist until toxin-laden maggots emerge.

$\beta$ = an intoxication coefficient that consists of two components; the first (C) represents the frequency of contact between live birds and toxic material, and the second ($P_i$) is the proportion of such contacts that result in intoxication, that is, the proportion of birds that ingest sufficient toxic maggots to cause death. The intoxication coefficient, $\beta$, is analogous to the transmission coefficient (Anderson, 1982) used for infectious diseases.

If every carcass in a marsh contained spores, became infested with maggots, and persisted until maggots were available, $P_s$ and $P_m$ could be ignored. However, these are variable and may be very important. If, for example, 40% of the animals that died in a marsh contained spores ($P_s = 0.4$) but 90% of carcasses were removed by scavengers before toxic maggots develop ($P_m = 0.1$), only 4% of animals that die would produce toxin-laden maggots that might result in secondary poisoning.

The proportion of carcasses that contain spores ($P_s$) probably is related directly to the number of spores in the marsh soil, as well as to the food habits of the animals present. Differences in $P_s$, as a result of differences in spore density in soil, might be sufficient to explain why some marshes have botulism and others do not. If maggots transmit spores as well as toxin, birds dying of botulism would be more likely to become toxic than animals dying from other reasons; thus, $P_s$ may be greater for birds dying of botulism than for birds dying after collision with an overhead wire in the same marsh.

Neither the number of animals dying in a marsh ($M_1$) nor the proportion scavenged is constant and these factors are not independent, since the number of carcasses present may influence scavenging. The proportion of carcasses that persist until toxin-laden maggots emerge ($P_m$) will be influenced by fly activity, the number and type of scavengers present, the amount of alternate food available to scavengers, the ease with which scavengers can find carcasses, and the time interval between death of an animal and the emergence of maggots from its carcass. Increased availability of carrion in an area might attract scavengers and increase the scavenging rate over the long term but sudden death of a large number of vertebrates, such as in a hail storm, fish kill, or cyanobacterial bloom, will overload the system with carrion in the short term. A larger proportion of the carcasses would then persist until toxic maggots were available. This may explain why botulism epizootics often follow such events. Scavenger overloading likely occurs during a botulism epizootic, facilitating its perpetuation. We found that only 2.3% of duck carcasses were removed by scavengers during a botulism epizootic at Eyebrow Lake (Cliplef and Wobeser, 1993). If this scavenging rate ($P_m = 0.97$) was combined with $P_s = 0.4$, 39% of birds that die would produce toxin-bearing maggots. Weather might have an effect on $P_m$. Cool temperatures retard maggot infestation of carcasses (Wobeser et al., 1987), so that in cool weather there is a longer time period during which carcasses may be removed by scavengers. This may be one reason why epizootics wane during cool weather. Lack of fly activity because of cold weather during early spring epizootics (Wobeser et al., 1983) also may explain why these do not persist. Any factor that reduces scavenging, such as predator control on areas used by nesting waterfowl, dense emergent vegetation in which carcasses are hidden from scavengers, or death of vertebrates in areas inaccessible to scavengers, could result in a larger proportion of carcasses persisting until toxic maggots are present. Birds dead and dying of botulism are often concentrated about islands at Eyebrow Lake, and in other marshes (Locke and Friend, 1987). The islands at Eyebrow Lake were constructed to reduce predation on nesting waterfowl; since many predators are also scavengers,

one would predict that scavenging of carcasses is reduced on these islands.

The intoxication coefficient ($\beta$) is complex and any factor that increases contact between live birds and carcasses could increase the likelihood of contagion. Increased density of live birds, carcasses containing maggots, or both, in a marsh should increase C and, hence, $\beta$. Epizootics often occur in late summer or autumn when waterfowl populations are at an annual high, and as water levels are declining. Declines in water depth, particularly on wetlands with very shallow gradually sloping profiles, may result in major reductions of water area, increasing bird and carcass density dramatically. It is unlikely that birds and carcasses are distributed homogeneously or randomly in a marsh. Any factor that brings them together could increase $\beta$. Islands are attractive to birds as resting places and might concentrate birds where toxic maggots are plentiful because of reduced scavenging. Similarly, some birds feed along the water's edge where maggots from carcasses drift to shore and pupate in the mud (G. Wobeser, unpubl.). Food habits of the birds could have a major effect on $P_i$. A large proportion of birds that feed preferentially on invertebrates would increase the probability of secondary poisoning.

An example may illustrate how differences in R might occur and the effect of such changes. Assume that 10 vertebrates died in a marsh during a period of time ($M_1 = 10$). Of these, four contained spores ($P_s = 0.4$) and eight were consumed by scavengers before maggots emerged ($P_m = 0.2$). Four birds contacted a carcass containing maggots ($C = 4$) but only two ingested sufficient maggots to become poisoned ($P_i = 0.5$), so $\beta = 2$. Applying these values, $M_2 = 10 \times 0.4 \times 0.2 \times 2 = 1.6$ and $R = 1.6/10 = 0.16$. Under such conditions, toxin is formed in some carcasses and birds die of botulism but the amount of secondary poisoning is very limited. However, on an island where scavenging was limited ($P_m = 0.7$) and contact was more frequent ($C = 8$), but other factors remained the same, R would = 1.1. At this site, the disease could persist and perhaps expand slowly. Now let us assume that 500 birds were killed during a hail storm on the marsh. Because so many carcasses were available, scavengers removed only a small proportion ($P_m = 0.9$), and the likelihood of contact increased ($C = 12$), so that $\beta = 6$. Under these conditions, $M_2 = 500 \times 0.4 \times 0.9 \times 6 = 1080$, and $R = 2.2$. While this does not seem a very high reproductive rate, the number of secondary cases of poisoning would more than double with each replication of the cycle and during the third replication >5,000 birds could die of secondary poisoning. If maggots transmit spores, so that $P_s \approx 1$ for the birds that died from botulism, the rate of increase would be more rapid. If $P_m$ and C also increased further, as more carcasses became available, the rate of transmission would accelerate.

## IMPLICATIONS

The concepts that botulism (a) behaves in a manner similar to an infectious disease, and (b) is often enzootic but flares into epizootic proportions when conditions are favorable for secondary poisoning, may be useful in identifying directions for research. A first step would be to determine if, and how commonly, botulism occurs at a low (enzootic) level in marshes of different types. The magnitude of the different components in the model of secondary poisoning proposed above is unknown but some of these can be measured. It may be possible to identify and measure the extent of factors that cause death of vertebrates ($M_1$) in different types of marsh. Both $P_s$ and $P_m$ are measurable and the effect of variables, such as season, weather and habitat conditions, on spore density and on the rate at which carcasses are infested by maggots and removed by scavengers could be determined. Components of $\beta$ may also be measurable.

The model may also provide a theoretical basis for management. The objective

of management would be to reduce R to <1, as for control of an infectious disease (Anderson, 1982). Reducing $M_1$ is currently done on some marshes through measures such as routing overhead transmission lines away from marshes and preventing fish-kills (Locke and Friend, 1987). If other factors that influence mortality of vertebrates can be identified, they might also be manipulated. If $P_s$ is determined primarily by the density of spores in the environment, and spores in soil are very stable, $P_s$ may not be amenable to manipulation. The proportion of carcasses that persist until toxin-laden maggots emerge ($P_m$) is likely to be highly variable and may be amenable to manipulation. Wildlife managers have abundant experience in reducing the impact of predators and scavengers; this may be an occasion to use the knowledge in the reverse direction as a preventive measure for botulism. Carcass collection and disposal in the face of an epizootic is a common and expensive management technique. In essence, it is an attempt to artificially improve scavenging and, hence, to reduce $P_m$ and C when massive numbers of toxin-bearing carcasses are already present. Its efficacy in reducing losses has never been tested. Carcass collection may remain part of the response to epizootics; it might also be used in a preventive manner, particularly after natural events such as hail storms.

Dispersing birds away from the site of epizootics is an obvious management activity to reduce β. If localized foci of intense botulism mortality can be identified, these could be characterized and managed in regard to density of birds, carcasses and scavengers. Construction of ponds that are deep, with steep sides and minimal shoreline, as suggested by Hunter et al. (1970) and Rosen (1971) is not feasible in most circumstances but might reduce the effects of drawdown on bird density, and the area to be searched by scavengers. The effect of features, such as islands, that may concentrate birds and carcasses, and act as nidi of botulism should be examined carefully. In the case of islands, the benefits from increased duckling production as a result of reduced predation might be weighed against disease losses. As the magnitude and degree of variability of the components of the model are determined, sensitivity analysis could be used to determine which factors are most suitable for management.

Friend (1981) categorized management in response to wildlife diseases into four stages. Currently, management of waterfowl botulism is stalled in the intermediate stages characterized by either frustration with the inability to manage the disease, or reaction in a fire-fighting manner to epizootics. This paper is presented in the hope that it may stimulate discussion and facilitate reaching the fourth stage, in which management of botulism is primarily preventive and research is targeted specifically at improving the level of management.

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