HABITAT AND MANAGEMENT

TROUBLE IN THE SWAMP: AN ESSAY ON AVIAN BOTULISM

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The marshlands of the western Canadian prairie are spectacular in the fall. On a bright October day, blue sky, blue water and the blended autumn hues of bulrush and prairie grass weave a splendid tapestry of gold and sapphire that shimmers with an ever-changing pattern of wild birds that swarm and disperse in ceaseless motion. The air is vibrant with the annual fall burst of abundance that inspires thanksgiving and foreshadows southward migrations and the frozen peace of winter.

Whitewater Lake, a large marsh and lake in southwestern Manitoba, is such a place. Yet some who stood on its shore in October 1996 were more discouraged than inspired by the autumn spectacle, their spirits wounded by recent and bitter experience. For this scene of plenty was, only a few short weeks before, the killing field for more than 170,000 wild birds. Few people were troubled by this striking paradox of nature because few were there to see it, and fewer still would have known what drama and devastation the scene subsumed, should they have happened by. The bodies of the dead lay buried in limed pits, the fruit of some 4,000 hours of labour, and there was nothing left to be seen. For Larry Bidlake of the Manitoba Department of Natural Resources, however, devastation dominated the view over Whitewater Lake. He had spent a whole season here, July, August and September, coordinating teams of wildlife agents in a futile battle with nature to stop the killing. There was discouragement also among senior managers of his department, and of others in the Canadian Wildlife Service and at Ducks Unlimited Canada, who had to pay the bills for this battle they had not expected to fight and which they had lost — hundreds of thousands of dollars stolen from their plans and dreams for that year and the next. Another 100,000 birds would die at Whitewater Lake in 1997 and the disease would occur again in 1998 and 1999.

These people were not alone in their uneasy contemplations of nature's harshness. In southern Saskatchewan, 140,000 bird carcasses lay strewn across Old Wives Lake that same October, and 1,000,000 would die there in 1997. In southeastern Alberta, Pakowki Lake was the scene of similar devastations: over 100,000 birds in 1994, over 200,000 in 1995. What killed these birds is no mystery. They died of botulism, a well-known form of food poisoning. South of the border, the same disease had also held deadly sway in many smaller outbreaks. From the quarterly reports of the National Wildlife Health Center (U.S. Geological Survey), Madison, Wisconsin: July to October 1994, 10,000 ducks and coots in the Klamath Basin of California, 17,000 at Lake Venturia in North Dakota, 30,000 on the Great Salt Lake wetlands of Utah; July to October 1995, 10,000 ducks and coots in Lower Klamath, 16,600 ducks, cranes and shorebirds at Horsehead Lake in North Dakota; July to October 1996, 14,000 pelicans and egrets on the Salton Sea in California, 15,000 ducks, coots and shorebirds at refuges in North Dakota. Annual total body counts of 74,000, 53,000 and 49,000 in each of these years tell only part of the story. Multiply these figures by three or by six to include the bodies of ducks and other large birds missed in counting. Multiply again by 2 or by 10 or by 100, no one knows the right number, to include the outbreaks neither seen nor counted. And then consider the small birds that are much harder to find and count, the hatchlings of the year and the sandpipers and other small denizens of the shore. If one duck found represents three or six ducks dead, how many dead birds does the corpse of one poisoned sandpiper represent? Surely, a great many.

Persons knowledgeable and concerned about wild birds contemplate such statistics with discomfort. One such person is Terry Neraasen, a biologist at the headquarters of Ducks Unlimited Canada, near Winnipeg. His job is to conserve prairie wetlands and the richness of animals and plants that they contain, to reverse, perhaps, the past 100 years of steady destruction of such places that has occurred throughout the continent. For Terry, botulism is a problem. Death of water birds due to botulism appears to be on the rise. More than 80 percent of such mortality that has ever been recorded in prairie Canada has occurred in the current decade. The number of ducks that died of botulism at Whitewater Lake in 1996 exceeded by some tens of thousands the total number taken by hunters in Manitoba the preceding year. The number that died at Pakowki Lake in 1995 exceeded the total of ducks estimated to have hatched and fledged on all wetlands secured and managed for waterbirds in prairie Alberta in that same year. More birds died from botulism in 1996 in Canada's three prairie provinces than are thought to have died from all of the oil spilled into Prince William Sound, Alaska, by the Exxon Valdez; three times that many died in 1997.

Botulism is a disease known since the dawn of human culture. Botulus is the Latin word for sausage, an etymological link to the fact that preserved meats gone bad have been a common source of such food poisoning since antiquity. Botulism occurs when a particular bacterium, now known as *Clostridium botulinum*, grows in foodstuff and produces powerful toxins. The toxins affect nerves and cause an often fatal paralysis.

A canoe trip early on a summer morning through a prairie marsh where botulism is at work is not soon forgotten, either for its natural majesty or its dispassionate cruelty. The water is green with photosynthesis and a-boil with tiny swimming creatures. From the clustered bulrushes, Yellow-headed Blackbirds come and go, sparkling in the newly risen sun like agitated Christmas ornaments, and punctuating the background din of a million voices with their own peculiar eructated song. The smell is rich and salty. A bow-wave of startled and escaping birds precedes the canoe as it threads its way among the reeds. A Northern Harrier tilts suddenly away in its low, kite-like flight across the marsh, and a sated coyote yaps annoyance from the shore. Life is here in breath- taking concentration, but death is here as well.

Death from botulism generally is slow and unpleasant, though perhaps not directly painful. Botulism causes a flaccid paralysis, a progressive and relentless weakening of the muscles. In a bird, it starts in the legs and then the wings. Look carefully and you will see that some of the birds escaping the canoe's intrusion do so slowly, with a peculiar alternating tilt from side to side as reluctant legs are still convinced to swim. When the legs at last refuse, the birds begin to paddle with their wings like small, weak, side-wheeled steamboats. But the wings weaken also, and soon the neck muscles begin to fail. An old and aptly descriptive name for botulism in birds is "limber neck", a moniker earned by the fact that when you pick up such a bird, its neck is limp and its head hangs straight down. When neck paralysis sets in, the head of a bird on water gradually sinks below the surface and it drowns, perhaps a mercifully early end. Many birds drag themselves out on the shore, however, and eventually come to a halt, unable to move. Here they will die fairly soon if their dose of toxin is high and their breathing muscles are quickly paralysed. But wild birds actually are quite resistant to botulism toxins; the dose required to kill a duck would kill 5,000 or more laboratory mice. Thus many birds linger on in a partially paralysed state, unable to get to food or water, alert and aware, and possibly terrified. If not dispatched by some predator, they will die slowly of dehydration.

And so, to left and to right as the canoe advances, the dead and the dying are much in evidence. The bodies of young coots lie in disorder on their nest mounds and tiny grebes attract blow-flies in the weeds. The transparent third eyelids of poisoned birds are paralysed early in the course of the disease. They bulge with eye fluids and sometimes drip clear drops; one must be disciplined not to think of these as tears.

The toxins produced by *Clostridium botulinum* are some of nature's most potent poisons. About half a pound of one such toxin

would be enough to kill all of the five-plus billion human beings alive today. Botulism toxins are always found on the classified lists of agents of high potential in programs of biological warfare. Fortunately for us, the particular botulism toxin that kills wetland birds seldom, if ever, causes human disease. Fortunately as well, these bacteria are very particular about where and when they will grow. Their environment must be warm and alkaline and free of oxygen, like the inside of a spoiled sausage or an improperly sterilized can of beans. But where does a duck or a coot or a Piping Plover find such poisoned foods? According to Dr. Gary Wobeser of the Canadian Cooperative Wildlife Health Centre at the University of Saskatchewan and author of the definitive textbook on waterfowl diseases, the answer is "maggots".

Here the story of botulism in water birds gets complicated and stretches our understanding. The maggots, at least, we can explain. Scattered in the fertile ooze of wetlands are the spores of *Clostridium botulinum*; inert, dormant, thick-walled forms of the bacterium that appear to last forever. Animals that feed in the marsh, whether tadpoles or wigeon, ingest these spores along with the mud and water that accompanies whatever food they are seeking. This normal situation produces no disease; the tissues of the living are inhospitable to the growth of this bacterium. However, should such an animal die for some reason — from some accident or disease, for example — the bacterium suddenly finds itself in fertile ground. The tissues of the dead in summer are warm and rich, and the spores of the bacterium come to life, multiply and produce toxins. As the carcass begins to swarm with bacteria on the inside, another set of necrophiles is at work on the outside. Ubiquitous blow-flies have laid masses of eggs on the hapless victim even before it has died. Maggots hatched from these eggs bore into the nutritious putrefaction of the carcass, converting dead flesh to new life with wonderful efficiency. Among the nutrients, the maggots also encounter the botulism toxins. The toxins do the maggots no harm, but they are absorbed, turning each maggot into a tiny pill of poison. When the carcass has been reduced to bone and feathers and no nutrients remain, the maggots abandon ship, swarming overboard in all directions to seek suitable places to pupate and reach adulthood.

These maggots are the agents that carry the poison of botulism from bacterium to bird. Nauseating though this may seem to us, birds love maggots. Out in the marsh, birds that pass by a carcass where *Clostridium botulinum* is at work are quick to snap up any maggots that show themselves. Eating as few as four or five of these toxin-laden maggots will kill a duck; just one will kill a duckling or a Piping Plover. But, of course, that duck or plover also has been feeding in the marsh. It, too, has dormant spores of the bacterium in its body. When it dies from eating the poisonous maggots, it becomes another carcass in which the bacterium can grow and elaborate toxins that are then packaged into more maggots and presented as food to other birds. And so, by the mathematics of compound interest, one dead bird may be magnified to 100,000 dead birds.

At some point in every major outbreak of botulism in birds, this cycle of death, bacterial growth and poisonous maggots becomes the dominant process that perpetuates the disease and multiplies its victims. But the earlier events in botulism outbreaks are more uncertain. What really starts these outbreaks? Why do they occur on some wetlands more often or more severely than on some others? Why do some occurrences of botulism result in truckloads of dead birds and months of dying, while others are self-limiting? It seems that botulism behaves rather like a forest fire. A spark of some kind ignites the tinder, but a multitude of factors such as wind, rain, the age of the forest and the thickness of the undergrowth, determine whether the fire will just fizzle out where it started, smolder for a while and erupt later on, or progress immediately to a raging conflagration. For avian botulism, neither the "spark" nor the limiting conditions that determine the magnitude of the "fire" are fully understood.

Some scientists think that the critical events that contribute to the occurrence of botulism take place in the ooze. Dr. Tonie Rocke of the National Wildlife Health Center thinks this could be the case. Not much is known about the natural history of *Clostridium botulinum* in its native mud, but the little we

do know is intriguing. For example, the bacterium, by itself, cannot produce what we call "botulism" toxins; it does not have the genes for these poisons. Rather, the genes are carried to the bacterium by a virus that infects it and ultimately kills it. Thus, only *Clostridium botulinum* infected with this lethal virus can produce toxin. The evolutionary good sense of this arrangement, or its practical implications for understanding avian botulism, are not apparent, yet an understanding of these microbial mysteries could provide important clues.

Another view is that the events that start and perpetuate a botulism outbreak are not so mysterious as they are multiple and interrelated in complicated ways. The existence of the carcass-maggot cycle implies that any animal dying for any reason anywhere in a marsh could start an outbreak, if conditions are suitable. It is more likely to be these conditions, rather than the "spark", that determine the nature of the "fire". But what are the critical conditions? Gary Wobeser thinks there is a series of such critical factors, each with a certain probability of occurrence: a probability that a carcass will contain Clostridium botulinum, that toxin will be produced, that toxin-laden maggots will emerge, that the maggots will be found and consumed by other birds. These probabilities are affected by factors such as the density of bacterial spores in the local mud, the number of predators and scavengers, like coyotes and crows, that can remove carcasses before they produce poisonous maggots, and the number of birds present on the marsh. For Larry Bidlake and Terry Neraasen, understanding of botulism outbreaks is an urgent, practical concern. These people, and hundreds like them across North America, are expected to "do something" when botulism strikes. Not really knowing what to do is no defence; inaction is not permitted. But what can they do? At the moment, they do what is possible and what seems to make sense: they collect and destroy the bodies of the dead and dying to try to break the carcass-maggot cycle.

Responding to a botulism outbreak is a major undertaking, especially for under-financed wildlife agencies during the busy summer season. At Pakowki Lake in 1995, clean-up operations were continuous from July 17 to October 18, involved 54 people and required 4,800 person-hours of work; at Whitewater Lake in 1996, it was July 9 to September 18 and 4,000 hours. At the peak of the outbreak, crews were collecting over 3,000 carcasses per day. These outbreak areas are large, shallow lakes of 40 and 26 square miles each. Logistical difficulties are legion. The only effective way to collect waterbird carcasses is by air boat — expensive machines, too few in number and prone to regular breakdown. Boats and trucks need gas at lake-side where none is for sale for miles around. Crews need food and drink, transportation, first aid, lodging, discipline and encouragement. Burial pits require backhoes, landowner permission, and environmental approval. The press must be given information and kept out of the way.

Yet, when all is organized and operating, there still is no clear evidence that these cleanup operations make a difference. The large outbreaks seem to run their courses, the collection of carcasses more an accounting of their magnitude than a check on their progress. The birds die faster than they can be collected; carcasses continue to elude detection and to produce those maggot poison pills. When outbreaks have been detected in their very earliest hours by intensive surveillance, carcass collection has seemed to slow their advance. But wildlife agencies seldom have the funds or personnel to undertake such surveillance on all, or even a few, of the many wetlands which we consign to their care. Thus, many attempts to do battle with botulism seem too little, too late.

Trent Bollinger, a wildlife veterinarian with the Canadian Cooperative Wildlife Health Centre, has witnessed this seemingly hopeless war with botulism but has sought within it not the ready harvest of despair but the seeds of a better understanding and, possibly, of hope itself. He now leads a partnership of wildlife agencies from Canada and the United States that is putting unprecedented amounts of time and resources toward learning all that can be learned from these massive outbreaks. Where do they start and how do they progress? What is happening in the water, in the air and among the birds when disease begins? Does carcass pick-up do good, or harm or neither? Which birds are dying and

what might this mean for bird populations? And, from all of this, can botulism be understood sufficiently that we might, one day, be able to alter its course or reduce its impact? Trent and others will dedicate years of work to these questions before answers will emerge.

If we were to join Larry Bidlake on the shore of Whitewater Lake on a bright summer morning to survey this scene of nature's slaughter, how should we feel about botulism in wild birds? Perhaps we should accept that this is no more than the dark side of nature's normal balance. After all, botulism has been known to occur in water birds for as long as anyone has been around to recognize and record it; an account of this "western duck sickness" was published by the United States Department of Agriculture in 1915. Yet we also must wonder whether we ourselves have had some hand in these occurrences. Have the 70 and 80 and 90 percent of wetlands in North America that we have drained or otherwise destroyed during this century caused birds to congregate on the remaining marshes in high numbers that favour disease? Have we harried away the scavenging predators that once beat the maggots and bacteria to their prey? And whether we ourselves are guilty or blameless, how should we respond to botulism in wild birds? Although population trends are disturbingly downward, there still are some 80 million ducks in North America, 33 million or so just on the western prairies. We still shoot eight to ten million ducks each year. We might accept a strictly mathematical view that a few hundred thousand or a million ducks dying of botulism each year is, in this context of many millions, trivial. The possible poisoning of rare animals like the Piping Plover could give us pause, but the precariousness of their grip on life has other, more important causes.

Thus, a broad and biological view might advise us to ignore botulism and place our concerns for nature elsewhere. But I would venture that society at large rejects this view. Whether it is because we wish to hunt these birds at a later date, to revel in the beauty of nature in the wild places that they animate, or simply to know that they are there and may be glimpsed on rare occasions, spring and fall, from our high-rise window, we do not want these birds to die. Our response is informed less by an understanding of the biology of birds than by a sentiment, both simple and primeval, that we wish our fellow creatures well. It is with a hope that may be at once a grand delusion and our salvation that we turn to Larry Bidlake and his colleagues and say "Do something", and to Gary Wobeser, Trent Bollinger and their colleagues and say "Find a better way".

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"God has given us the earth for our life. It is a great entail. It belongs as much to those who come after us as to us; and we have no right, by anything that we do, or neglect to do, to involve them in unnecessary penalties, or to deprive them of benefits which are theirs by right."

- John Ruskin.