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How do recent changes in Lake Erie affect birds? Part three: type-E botulism

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The health of Lake Erie reached a low point in the 1960s and 1970s, and then improved greatly by the 1980s (Makarewicz and Bertram 1991). Now, Lake Erie is suffering from harmful algal blooms, botulism, invasive species, climate change and other issues. Why is this? What has brought about so many new issues? What does it all mean for birds? This review article is the last of a series of three articles in *Ontario Birds*. The articles provide an overview of some of the current environmental and ecological issues for Lake Erie, with emphasis on the implications for the numerous bird species that depend on the lake for nesting and migration. There are dozens of worthy issues to profile. We chose to begin, in part one, with invasive *Phragmites* (Tozer and Beck 2018); in part two, we tackled invasive Zebra Mussels and Quagga Mussels (*Dreissena polymorpha* and *D. rostriformis bugensis*, respectively; Tozer and Beck 2019); and here, in part three, we take on the impacts of type-E botulism. In addition to a review of each issue, the articles present new analysis of relevant citizen science data and suggest actions that we, as birders, can take to help alleviate the issues.

Botulism is a disease that infects humans as well as wildlife (Critchley 1991). It is caused by neurotoxins produced by the bacterium *Clostridium botulinum* (Desta *et al.* 2016). There are several types or strains of *C. botulinum* (types A through H), each surviving best under different conditions (Hannett *et al.* 2011). Botulism rarely infects humans because thorough cooking, good hygiene and other routine measures prevent the acquisition of the toxins (Shapiro *et al.* 1998). A few of the strains infect wild birds: type-C causes die-offs of waterfowl throughout the world, particularly in western North America (Wobeser *et al.* 1987) and type-E causes die-offs in the Great Lakes (Wijesinghe *et al.* 2015). *C. botulinum* is a native species that occurs in soils and sediments throughout the Great Lakes (Graikoski *et al.* 1968). It survives for long periods, sometimes for many decades, as harmless dormant spores, but once favourable growing conditions are encountered it starts growing and produces botulinum neurotoxin

Figure 1. Tens of thousands of individuals belonging to dozens of different waterbird species, such as this Common Loon, sometimes die during late summer and autumn on Lake Erie and the other Great Lakes due to outbreaks of type-E botulism. *Photo: Bird Studies Canada*

(Long and Trauscher 2006). The neurotoxin is one of the most potent toxins known (Singh 2000) and although many aspects of the disease have been well-studied for nearly two centuries (Cherington 2004), the function that the toxin serves for the bacteria, if there is one, remains unclear (Simpson 1986). Alternatively, the toxin may be a by-product of a complicated evolutionary history involving lateral transfer of toxin-producing genes to the bacteria, perhaps from a virus, with no obvious subsequent benefit to the bacteria (Poulain and Popoff 2019). As such, survival is just as high and distribution in the environment is just as extensive in toxic and non-toxic forms of certain strains of *C. botulinum* (Poulain and Popoff 2019). Therefore, the current type-E botulism issue for waterbirds (i.e., any aquatic bird species) in Lake Erie and the rest of the Great Lakes might ultimately be attributable to a mere chance event deep in *C. botulinum's* evolutionary history!

The problem for waterbirds, or any other vertebrate such as fish, is that botulinum toxin interferes with transmission of nerve impulses intended to stimulate peripheral or voluntary muscles (Desta *et al.* 2016). Through a series of different steps, the toxin prevents the neurotransmitter acetylcholine from traveling out from nerve endings, effectively killing nerve impulses so they never reach intended muscles (Gundersen 1980). The result is flaccid paralysis, and waterbirds with even very small doses of the toxin are unable to keep their eyes open, raise their wings, move their feet, or hold up their heads, hence the common name "limberneck disease" (Cherington 2004). In the end, infected waterbirds typically die a slow death due to starvation, drowning, depredation, or other complications (Figure 1).

The occurrence and magnitude of waterbird mortalities due to type-E botulism is highly variable over space and time (Lafrancois *et al.* 2011). Die-offs of waterbirds were first recorded in the Great Lakes in the early 1960s, continued sporadically during the 1970s and 1980s and were absent during most of the 1990s (Brand *et al.* 1983, 1988; Cabrera 2014). Die-offs of waterbirds due to type-E botulism have occurred annually in Lake Huron since 1998, Lake Erie since 1999 and Lake Ontario since 2002 (Cabrera 2014). The die-offs involve a variety of different species including loons, grebes, herons, cormorants, ducks, coots, shorebirds, eagles, gulls, terns and crows (Carpentier 2000, Canadian Cooperative Wildlife Health Centre 2008, Chipault *et al.* 2015). In some years, only a small number of birds are found dead, whereas in 2002, one of the worst years on record, over 20,000 individuals washed up dead on beaches and shorelines throughout the Great Lakes (Cabrera 2014). The number of dead individuals recorded is, of course, only an unknown fraction of the total number killed, given that carcasses sink before they wash up on shore or carcasses wash up in locations where they go unrecorded. Recent research using radio transmitters implanted in floating Common Loon carcasses released throughout Lake Michigan aimed to uncover how wind, waves and water currents influence the trajectory of floating carcasses, which will help identify botulism hotspots and may help better understand how many carcasses go undetected (Kenow *et al.* 2016).

What happened starting in the late 1990s and early 2000s to cause annual die-offs of waterbirds in Lake Erie and most of the other Great Lakes? The quick answer is: we don't really know. Evidence is building to suggest that recurring type-E botulism is the result of multiple, complicated, interacting pathways of infection involving nutrient runoff, invasive species, algal blooms, climate change and perhaps additional factors yet to be discovered. The basics go like this: *C. botulinum* spores become activated and produce toxin when they encounter warm water with abundant nutrients and no oxygen (Espelund and Klaveness 2014). We know from long-term monitoring that water temperature (Mason *et al.* 2016), extent of deoxygenated water or hypoxia (Zhou *et al.* 2013) and soluble reactive phosphorus (Daloğlu *et al.* 2012) have all increased in recent decades in Lake Erie and some of the other

Great Lakes. The result: increasingly good conditions for production of botulism neurotoxin.

Now add a dash of invasive species and a pinch of algae. *C. botulinum* spores become very active and produce large amounts of neurotoxin within hot, oxygen-depleted, nutrient-rich mats of rotting algae of the genus *Cladophora* (mostly *C. glomerata*, hereafter "*Cladophora*"), which piles up in shallow water and along shorelines in late summer (Byappanahalli and Whitman 2009, Chun *et al.* 2013, 2015). Submerged aquatic vegetation dominated by *Cladophora* has increased in extent in all of the Great Lakes in recent decades as shown by remote sensing satellite data (Brooks *et al.* 2015). The increase in *Cladophora* is in part due to increases in water clarity and associated light penetration brought about by the super-efficient filter feeding of non-native invasive Zebra Mussels and Quagga Mussels, which remove light-blocking phytoplankton and other particles from the water column (Auer *et al.* 2010). The increase in *Cladophora* is also due to increases in hard substrate for *Cladophora* to attach to in the form of the mussels' shells, since *Cladophora* only grows on hard surfaces and not on soft mud or sand (Higgins *et al.* 2008). The mussels also concentrate nutrients for *Cladophora* by filtering nutrients out of the water column and eliminating them in their feces (Hecky *et al.* 2004, Dayton *et al.* 2014). The enhancement of algal growth by the mussels is made even stronger by recent increases in nutrient runoff into Lake Erie and the other Great Lakes due to increases in surface application of fertilizer on agricultural fields during the non-growing season, which is more likely to wash downstream into the lakes during increasingly frequent storms, themselves a consequence of climate change (Smith *et al.* 2015). Indeed, the overall enhancing effect of the mussels on algal growth has been shown convincingly through experimental manipulation using pre-constructed "colonies" of live and dead mussels either with or without artificial addition of nutrients (Francoeur *et al.* 2017). In short, bring in non-native invasive mussels, clear the water column, increase the light, increase the nutrients, bring on the *Cladophora* and produce lots of botulism neurotoxin in dead mats of algae at the end of the summer. As further evidence of these relationships, multiple studies show correlations at various scales between type-E botulism outbreaks in waterbirds and warm water, low water (shallow water tends to be warmer) and *Cladophora* (Wijesinghe *et al.* 2015, Princé *et al.* 2017).

How do botulism bacteria or its neurotoxin get from rotting mats of algae or other sources into waterbirds? One way is through invertebrates such as fly maggots (Diptera larva) that pick up the bacteria or toxin while feeding on or within rotting carcases or algae washed up along the lakeshore. Notably, invertebrates such as aquatic insects and mussels are unaffected by the neurotoxin (Pérez-Fuentetaja *et al.* 2011). Shorebirds feed on the tainted invertebrates (Figure 2) and gulls and other scavengers feed on the infected carcases, and in turn can become infected. These pathways are likely the most common in the summer and early fall (Canadian Cooperative Wildlife Health Centre 2008). The presence of botulism bacteria

Figure 2. Some waterbirds, such as this Spotted Sandpiper, become infected with type-E botulism bacteria or its toxin while feeding on or within tainted algae washed up along lakeshores.
Photo: Leslie Abram

or its toxin is also found at high levels in the tissues of living and dead Zebra Mussels and Quagga Mussels, as well as in various other aquatic invertebrates, such as larval midges (Chironomidae) and worms (Oligochaeta) (Pérez-Fuentetaja *et al.* 2006, 2011). The chironomids, oligochetes and other non-mussel invertebrates likely acquire the bacteria or its toxin through direct ingestion or while eating detritus or sediment, particularly near rotting algae, whereas the mussels likely acquire the toxin by filtering it out of the water column or filtering out smaller invertebrates that have already acquired it (Getchell and Bowser 2006, Pérez-Fuentetaja *et al.* 2006). Certain waterfowl, such as the Long-tailed Duck (*Clangula hyemalis*), Common Goldeneye (*Bucephala clangula*) and Bufflehead (*B. albeola*) feed on the tainted mussels and the toxin-laden aquatic invertebrates (Schummer *et al.* 2008a,b). Fish feed on the infected mussels and other infected invertebrates and due to biomagnification acquire high concentrations of the bacteria or toxin (Bott *et al.* 1966). The tainted fish are in turn fed on by a variety of different waterbirds (Essian *et al.* 2016). In an ironic final twist, another non-native invasive species, the Round Goby (*Neogobius melanostomus*), specializes in feeding on non-native invasive mussels and is itself especially common in the stomachs of botulism-killed waterbirds, particularly cormorants and loons (Hebert *et al.* 2014, Essian *et al.* 2016, King *et al.* 2017). Furthermore, botulism-infected fish that are partially paralyzed are attractive, easy pickings for fish-eating waterbirds (Yule *et al.* 2006). The pathways leading up through aquatic invertebrates, mussels and fish to waterbirds are likely the most common in autumn (Canadian Cooperative Wildlife Health Centre 2008). There are probably other sources and production pathways of botulism yet to be discovered or better described, such as water currents that bring spores up from the sediment so they are available to enter

the food chain and additional complicating factors involving competition between *C. botulinum* and other microorganisms that keep botulism bacteria at low numbers (Pérez-Fuentetaja *et al.* 2006). The bottom line is that recurring outbreaks of type-E botulism on the Great Lakes over the past two decades appear to be the result of multiple, complicated, interacting ecological and environmental changes ultimately brought about by non-native invasive Zebra Mussels and Quagga Mussels, nutrient loading and climate change.

The botulism issue is complicated and potentially quite big, but is it actually negatively affecting waterbird populations? Long-term bird population monitoring programs are extremely important for answering this type of timely but unforeseen question. As shown above, there are dozens of waterbird species that are potentially affected by type-E botulism on the Great Lakes. Based on observed mortalities, the majority of dead individuals are loons, mergansers, cormorants and gulls (Carpentier 2000, Canadian Cooperative Wildlife Health Centre 2008, Chipault *et al.* 2015). Populations of most of these more-commonly killed species have remained steady or increased over the past two decades in Ontario or the Great Lakes when type-E botulism has been most prevalent. For example, abundance of breeding Common Loons was stable in Ontario during the period (Smith *et al.* 2019); abundance increased for Common Merganser

Figure 3. Type-E botulism may be reducing the reproductive success of the Common Loon (an adult with two chicks is shown here).
Photo: Missy Mandel

(*Mergus merganser*) in southern Ontario and for Red-breasted Merganser (*Mergus serrator*) in northeastern North America (Canadian Wildlife Service Waterfowl Committee 2017); the number of breeding Double-crested Cormorants (*Phalacrocorax auritus*) increased and stabilized throughout the Great Lakes (Weseloh *et al.* 2002, Ridgway *et al.* 2006). Exceptions to that trend are the number of breeding gulls. Ring-billed Gulls (*Larus delawarensis*) and Herring Gulls (*L. argentatus*) declined slightly in the Great Lakes, although this is thought to be due to reduced food availability rather than botulism (Weseloh 2011). By contrast, the Great Black-backed Gull (*L. marinus*) is apparently especially susceptible to type-E botulism and as a result has been extirpated as a breeder from eastern Lake Ontario (Shutt *et al.* 2014) and may soon be eliminated as a breeder from throughout the Great Lakes (Weseloh 2011). Therefore, it seems that the widespread die-offs caused by type-E botulism are not creating long-term population-level impacts—in most cases. However, continued research and monitoring are needed to fully understand population-level impacts.

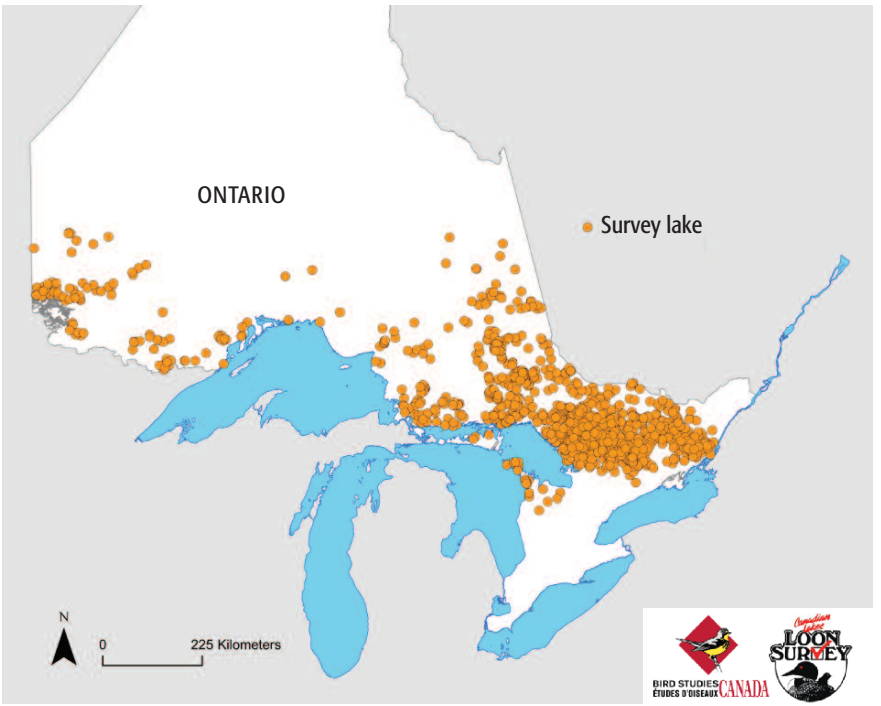
The above examples tell us whether abundance of infected waterbird species is negatively influenced by type-E botulism, but they do not tell us about potential negative effects on reproductive success, which could happen without concurrent changes in population size. For example, we could see stable or nearly stable populations at the same time as declining reproductive success if enough immigrants move in from outside to maintain the population. The Common Loon (Figure 3) seems to be especially vulnerable to type-E botulism given that it figures prominently in counts of botulism-killed carcases that wash up on shore (Carpentier 2000, Canadian Cooperative Wildlife Health Centre 2008, Chipault

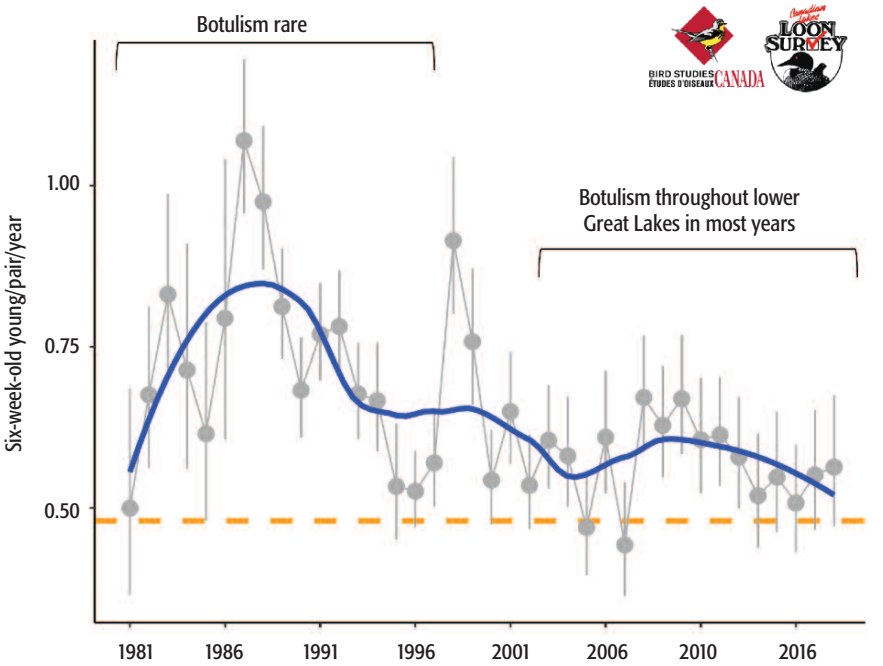
et al. 2015). As such, we used data from Bird Studies Canada's Canadian Lakes Loon Survey (Tozer *et al.* 2013b) to explore the possibility that reproductive success of this hard-hit species might be negatively affected by type-E botulism. Our line of reasoning was as follows: The Common Loon is a long-lived species (up to 20-30 years in the wild) and breeds throughout central and northern Ontario, well to the north of the lower Great Lakes where botulism outbreaks are most common (Evers 2007). Individuals typically spend the first few years of their lives on the ocean and at 3-4 years of age return to the breeding grounds for the first time to acquire a breeding territory (Piper e*t al.* 2015). Based on studies of colour-marked Common Loons, we know that reproductive success is lower for male Common Loons breeding on a territory for the first time; apparently it takes up to two or more years for them to learn, by trial and error, where the best nest sites are located on a territory in order to maximize their reproductive success (Piper *et al.* 2008). Remarkably, nest sites in this species are chosen by males, regardless of previous nesting experience of females, for reasons that remain unclear (Piper *et al.* 2008). We also know from colour-marked individuals that the number of young produced per year for males but not females increases with increasing age up to 15-17 years old (Piper *et al.* 2017). If enough breeding

males die due to botulism on their southward migration through the lower Great Lakes each autumn, then as a result, we would expect a larger proportion of younger, inexperienced, first-time territorial males in the breeding population the following spring and thereafter, with an associated reduction in reproductive success over the years. Thus, if type-E botulism negatively affects the reproductive success of Common Loons, we would expect higher chick production, on average, before the onset of recurring type-E botulism outbreaks in the late 1990s and early 2000s and lower chick production after that time.

We explored these ideas using data from 11,623 Common Loon breeding attempts on 1,317 lakes spread across 38 years in Ontario (Figure 4). Following Tozer *et al.* (2013a), we modeled the number of six-week-old young per territorial pair per year from 1981 to 2018 while controlling statistically for lake size and longitude given that the number of young produced is known to increase with increasing lake size (Alvo 2009, Piper *et al.* 2012) and is higher in the west compared to the east (Tozer *et al.* 2013a). We also included a random effect for each lake because ~50% of the lakes surveyed each year, on average, had reproductive

Figure 4. Lakes with at least one year of data collected by participants in Bird Studies Canada's Canadian Lakes Loon Survey used to determine patterns in Common Loon reproductive success in Ontario between 1981 and 2018 (n = 1,317 lakes).





success data for > 1 pair of loons (Tozer *et al.* 2013a). We focused on six-week-old young because Common Loons of this age have attained nearly adult size and with it a much lower chance of being depredated, making them a reasonable indicator of the number of young actually fledged (Evers 2007). Even though acidity is a strong predictor of loon reproductive success (Alvo *et al.* 1988, Alvo 2009), we were unable to control for differences in pH due to lack of data. We found the number of young per pair per year was highly variable, with no clear difference in reproductive success before and after the onset of recurring type-E botulism in the late 1990s and early 2000s (Figure 5). Nonetheless, it is worth noting that years of extremely good chick production (i.e., at or above 0.75 young

Figure 5. Reproductive success of Common Loons in Ontario between 1981 and 2018. Shown are annual estimates (dots) and associated 95% confidence intervals (vertical gray lines). A loess line of best fit (curved blue line) is included to show overall trajectory. The orange horizontal dashed line denotes an estimate of the minimum number of young per pair per year (0.48) required to prevent population declines (Evers 2007). Years of extremely good chick production (i.e., at or above 0.75 young per pair per year) ceased to occur after 1999 when recurring type-E botulism spread throughout the lower Great Lakes.

Data source: Bird Studies Canada's Canadian Lakes Loon Survey

Botulism-killed Common Loon.
Photo: Bird Studies Canada

per pair per year) ceased to occur after 1999 when recurring type-E botulism started to spread throughout the lower Great Lakes (Figure 5).

Whether this pattern is linked to botulism is unknown. Of course, there are many other factors that influence reproductive success of Common Loons, including acid precipitation, mercury pollution and climate change, any combination of which might also explain the pattern, but the influence of botulism is worth considering. On a happy note, the number of Common Loon chicks produced in most of the years since the early 2000s is apparently high enough to maintain a stable population given the average number of young per pair per year in most years is above 0.48 (Figure 5), which is the best available estimate of the minimum number required to prevent population declines (Evers 2007).

So what does it all mean? We have seen that type-E botulism in waterbirds in Lake Erie and the lower Great Lakes is due to multiple, complicated, interacting ecological and environmental changes ultimately brought about by non-native invasive species, nutrient loading, climate change and probably other factors. Therefore, we cannot stress enough to be extremely careful regarding these issues, especially taking steps to avoid introducing and spreading non-native invasive species (see summary at Ministry of Natural Resources and Forestry 2019), a concluding message we also arrived at in each of our first two articles in this three-part series (Tozer and Beck 2018, 2019). Anything we can do to reduce nutrient loading and climate change in the Great Lakes will also help mitigate the type-E botulism issue for waterbirds (see Environmental Commissioner of Ontario 2018 a,b). In particular, there are actions we can all take as individuals to help mitigate climate change (see Environmental Commissioner of Ontario 2019). In the end, perhaps the most important action we can collectively take as birders to help alleviate botulism and the many other issues currently affecting Lake Erie and the lower Great Lakes is to inform everyone that there is much more at stake than most people typically appreciate. All too often, the negative effects that these issues have on humans receive the most attention by the media and the general public, whereas the negative implications for waterbirds tend to go unnoticed or unaddressed. This is where we, as birders, can help in a big way to achieve a healthier balance.

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