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Links between type E botulism outbreaks, lake levels, and surface water temperatures in Lake Michigan, 1963–2008

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ABSTRACT

Relationships between large-scale environmental factors and the incidence of type E avian botulism outbreaks in Lake Michigan were examined from 1963 to 2008. Avian botulism outbreaks most frequently occurred in years with low mean annual water levels, and lake levels were significantly lower in outbreak years than in non-outbreak years. Mean surface water temperatures in northern Lake Michigan during the period when type E outbreaks tend to occur (July through September) were significantly higher in outbreak years than in non-outbreak years. Trends in fish populations did not strongly correlate with botulism outbreaks, although botulism outbreaks in the 1960s coincided with high alewife abundance, and recent botulism outbreaks coincided with rapidly increasing round goby abundance. Botulism outbreaks occurred cyclically, and the frequency of outbreaks did not increase over the period of record. Climate change scenarios for the Great Lakes predict lower water levels and warmer water temperatures. As a consequence, the frequency and magnitude of type E botulism outbreaks in the Great Lakes may increase.

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Introduction

Botulism is caused by ingestion of neurotoxin, produced by the anaerobic spore-forming bacterium *Clostridium botulinum*, that impacts neuromuscular function and that may result in paralysis and death of the intoxicated host. Botulinum neurotoxin (BoNT) is the most toxic substance known (Arnon et al., 2001). *C. botulinum* is classified into seven serotypes (A–G) based on BoNT characteristics (Rocke and Bollinger, 2007). Of the seven serotypes, BoNT types C and E are commonly involved in wildlife mortality events (Rocke and Bollinger, 2007). Botulinum neurotoxin type E has caused periodic mortality of fish and fish-eating birds in the Great Lakes since at least the 1960s (Brand et al., 1983; Riley et al., 2008), and types C and E avian botulism may be the most significant cause of wild bird mortality worldwide, sometimes resulting in the death of over one million birds per year (Friend et al., 2001; Newman et al., 2007; Rocke and Bollinger, 2007).

Type E botulism outbreaks have become relatively common in the Great Lakes region since the early 2000s, particularly in lakes Huron, Erie, Ontario, and Michigan. In 2002 and 2007, type E botulism outbreaks in the Great Lakes caused estimated annual mortality of over 18,000 and 17,000 birds, respectively (U.S. Geological Survey National Wildlife Health Center, unpublished data). Large numbers of dead birds observed in northern Lake Michigan in 2007, especially at Sleeping Bear Dunes National Lakeshore where federally endangered piping plovers were affected, have caused great concern among the Great Lakes scientific community and the general public. Botulism-related wild bird mortality has been recently reported in other stressed ecosystems such as the Baltic Sea (Neimanis et al., 2007) and may reflect undesirable changes in aquatic ecosystem health or function (Newman et al., 2007; Riley et al., 2008).

C. botulinum spores commonly occur in soil, surface waters, aquatic sediments, and the tissues of fish and other animals (Jensen and Allen, 1960; Bott et al., 1968; Sayler et al., 1976; Huss, 1980; Reed and Rocke, 1992; Hielm et al., 1998), but BoNT is only produced when the bacterium encounters anoxic conditions within specific pH and temperature ranges (Brand et al., 1983). The environmental factors required for BoNTs to accumulate to levels sufficient to cause fish and bird mortality are not yet fully understood (Rocke and Samuel, 1999; Rocke and Bollinger, 2007). Decaying organic matter, particularly plants or animal carcasses, may enhance conditions for *C. botulinum* spore germination and BoNT production (Reed and Rocke, 1992; Barras and Kadlec, 2000). Additionally, abundant populations of invertebrates and

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their predators may also contribute to the mobilization of BoNTs and subsequent accumulation in the food chain (Jensen and Allen, 1960; Rocke et al., 1999; Kadlec, 2002; Pérez-Fuentetaja et al., 2006).

Invasive species, particularly dreissenid mussels and round gobies, have been hypothesized to play a role in recent type E botulism outbreaks in Great Lakes fish and birds (Getchell and Bowser, 2006). For example, dreissenid mussels may enhance the growth of benthic algae (Hecky et al., 2004; Ozersky et al., 2009) and associated invertebrates (Lowe and Carter, 2004). Subsequent death and decomposition of these organisms may then help create conditions required for *C. botulinum* spore germination and BoNT production. The invasive round goby, which consumes both native invertebrates and dreissenid mussels, may also facilitate toxin transfer to other piscivorous fish and bird species. Although this hypothesized pathway of BoNT transfer has been circulated in the scientific literature (*e.g.*, Getchell and Bowser, 2006) and the media, such food-chain linkages have not been confirmed.

Attempts to detect *C. botulinum* vegetative cells or BoNT in food web components in Lakes Erie, Ontario, and Michigan have had limited success (Getchell et al., 2006; Pérez-Fuentetaja et al., 2006) due to analytical difficulties associated with BoNT detection, spatial and temporal inconsistencies in *C. botulinum* vegetative cell growth and BoNT production, or a combination of these and other factors. Getchell and Bowser (2006) noted that microhabitat conditions in dreissenid mussel beds may affect the timing and extent of BoNT production, and Pérez-Fuentetaja et al. (2006) concluded that a very intensive sampling approach would be necessary to detect *C. botulinum* vegetative cells or BoNT for large-scale studies. Thus, understanding the mechanism(s) of BoNT mobilization through Great Lakes food chains will likely require additional laboratory tools and a better understanding of food web dynamics.

Large-scale factors such as weather and climate could also affect the frequency and severity of botulism outbreaks (Rocke and Bollinger, 2007). In inland wetlands, the risk for type C avian botulism outbreaks may increase at higher water temperatures (Rocke and Samuel, 1999) and may also be associated with water-level fluctuations (Galvin et al., 1985; Barras and Kadlec, 2000). Botulism outbreaks were related to summer air temperatures and precipitation–evaporation balance in several English waterways, prompting Smith (1979) to suggest that existing climate datasets may help to understand and predict disease outbreaks. Here we explore the relationship of such large-scale environmental factors to the incidence of type E botulism outbreaks in Lake Michigan from 1963 to 2008. Specifically, we consider the relationship of type E botulism outbreaks in Lake Michigan to water levels, surface water temperatures, and fish population dynamics.

Methods

Data acquisition

Confirmed type E botulism avian mortality events for Lake Michigan were compiled for the period 1963–2008 from the U.S. Geological Survey (USGS) – National Wildlife Health Center (NWHC) database and cross-referenced with data from the Michigan Department of Natural Resources Wildlife Disease Laboratory. Cases for which only type C botulism was detected were excluded from this analysis. Spatial and temporal differences in monitoring intensity likely bias these mortality estimates. For example, for two years during the period of record, 1965 and 1966, substantial outbreaks were noted in historical records (e.g., Rocke and Bollinger, 2007), but avian mortality was not quantified. Regardless, these data represent the most comprehensive attempt to quantify long-term type E avian botulism mortality in Lake Michigan to date and are likely conservative estimates.

Lake level data for the Lake Michigan–Huron system were provided by the Detroit District Office of the U.S. Army Corps of Engineers for the period 1963–2008. Data were collected hourly from six gauging stations around Lakes Michigan and Huron, operated in the U.S. by the National Oceanographic and Atmospheric Administration (NOAA) and in Canada by the Canadian Hydrographic Service. Daily system-wide mean lake levels were calculated from the hourly data, and mean monthly and mean annual lake levels were calculated from those values.

Water temperature data were acquired from the NOAA National Data Buoy Center (http://www.ndbc.noaa.gov/; accessed 4 February 2009). Hourly surface water temperatures for the northern Lake Michigan station (identifier 45002) were downloaded for the station's entire period of record (September 1979 through November 2008). The northern Lake Michigan station is located halfway between North Manitou and Washington Islands, which is the part of the lake where avian botulism outbreaks have been most commonly reported. Monthly median water temperatures were estimated for use in this analysis, and annual means and July–September means were calculated from those values.

Long-term fish population data were acquired from the U.S. Geological Survey Great Lakes Science Center for 1973–2008 based on annual bottom-trawl surveys conducted at seven Lake Michigan sites (see Bunnell et al., 2006). Data were compiled for fish species that consistently made up more than 1% of the total catch, including alewife *Alosa pseudoharengus*, bloater *Coregonus hoyi*, deepwater sculpin *Myoxocephalus thompsonii*, ninespine stickleback *Pungitius pungitius*, rainbow smelt *Osmerus mordax*, round goby *Neogobius melanostomus*, slimy sculpin *Cottus cognatus*, and yellow perch *Perca flavescens*. For analysis, fish species were categorized as benthic (i.e., deepwater sculpin, ninespine stickleback, round goby, and slimy sculpin) or pelagic (i.e., alewife, bloater, rainbow smelt, and yellow perch). Aggregate variables (i.e., total fish abundance and invasive (alewife, rainbow smelt, and round goby) fish abundance) were also calculated. Species-specific and aggregate abundance values were reported as the estimated mean number of individuals per hectare for each year.

Data analysis

Avian mortality, lake levels, surface water temperatures, and fish populations were examined using individual and overlapping annual time series graphs to identify potential correlations. Outbreak and non-outbreak years were defined based on the presence or absence of type E botulism cases in the study area. Despite the lack of quantitative avian mortality data, based on published reports (*e.g.*, Rocke and Bollinger, 2007), 1965 and 1966 were identified as outbreak years. To compare lake levels and surface water temperatures in outbreak and non-outbreak years, two-sample t-tests were performed using mean annual lake levels and mean July–September surface water temperatures (the months for which the long-term temperature mean was within the range of optimal *Cladophora* growth (Higgins et al., 2008)). Spearman correlations were used to evaluate the strength of relationships between bird mortality, population levels of individual fish species, and aggregate fish variables; correlations for which rho (ρ) was greater than 0.250 were noted. A combined analysis of all factors using logistic regression was not performed because the period of record for temperature and fish abundance data did not cover the entire period of record for avian mortality data.

The five-year moving average proportion of years with botulism outbreaks and the five-year mean estimated avian botulism mortality for Lake Michigan were calculated for the period of record to identify trends in the frequency of botulism outbreaks. A five-year period was chosen because the five-year means tracked the mortality data closely while filtering out extreme values. Statistical evaluation was not applied in support of this analysis because the data were insufficient to support complex models representing the cyclical nature of the data.

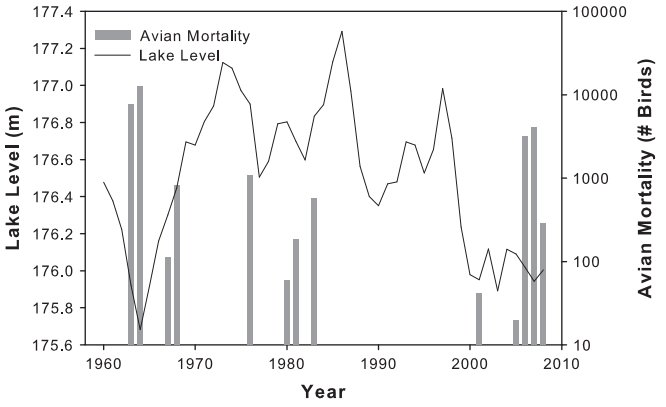

Fig. 1. Estimated Lake Michigan avian botulism mortality (1963–2008) and Lake Michigan water levels (1960–2008). Note that 1965 and 1966 were years with documented botulism outbreaks but undetermined mortality estimates.

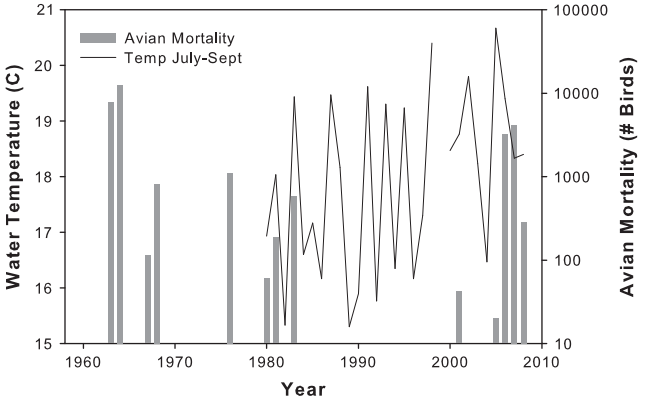

Fig. 3. Estimated Lake Michigan avian botulism mortality (1963–2008) and mean July through September surface water temperatures (1979–2008) in northern Lake Michigan. Note that 1965 and 1966 were years with documented botulism outbreaks but undetermined mortality estimates.

Results

Lake Michigan avian botulism outbreaks were reported in the 1960s, late 1970s, early 1980s, and 2000s, but not in the 1990s. The four largest documented outbreaks occurred in 1963 (7725 birds), 1964 (12,650 birds), 2006 (3187 birds), and 2007 (4158 birds) (Fig. 1). The highest number of outbreak years per decade occurred in the 1960s and the 2000s, with mortality events reported annually in Lake Michigan since 2005 (including 2009, National Park Service, unpublished data).

Mean annual water levels varied substantially in Lake Michigan from 1963–2008, with particularly low water periods occurring in the 1960s and 2000s (Fig. 1). Botulism outbreaks tended to occur in years with low mean annual water levels (Fig. 1). The late 1980s to early 1990s represented the only low water period for which avian botulism outbreaks were not reported. Mean annual lake levels were significantly lower in outbreak years (mean = 176.2 m) than in non-outbreak years (mean = 176.6 m; $t = 3.37$, $p = 0.002$; Fig. 2). Outbreak years occurred only when Lake Michigan mean annual water levels were below 176.8 m, and the largest avian mortality events (i.e., 1963–1964 and 2006–2007) were recorded when mean annual water levels were less than 176.1 m.

Mean annual and mean July–September surface water temperatures in northern Lake Michigan showed an increasing trend over the last three decades (Fig. 3), as also noted by Dobiesz and Lester (2009). Mean July–September water temperatures were significantly higher in outbreak years (18.7 °C) than in non-outbreak years (17.5 °C) ($t = 2.290$, $p = 0.034$; Fig. 4). Outbreak years tended to occur when mean July–September surface water temperatures were greater than 17 °C (ranging from 16.9 °C to 20.7 °C). During the large outbreaks of 2006 and 2007, mean July–September surface water temperatures exceeded 18 °C.

Fish population trends from 1973–2008 varied widely among species and had few consistent relationships to botulism outbreaks. Benthic fish abundance was positively correlated with avian mortality over the period of record ($\rho = 0.341$), as was the abundance of slimy sculpin ($\rho = 0.271$) and round goby ($\rho = 0.540$). The most recent outbreaks, from 2000 to 2008, coincided with rapidly increasing abundance of round gobies in Lake Michigan (D. Bunnell, USGS Great Lakes Science Center, personal communication).

No clear trends in the frequency or magnitude of avian botulism mortality events were apparent for the period of record. The five-year moving average of the frequency of occurrence of botulism outbreaks showed a cyclical pattern but did not increase over the period of record. A high frequency of occurrence of outbreaks was documented in the 1960s and the 2000s (Fig. 5). Five-year mean avian mortality also did not show a clear pattern over time, but was greatest during the 1960s.

Discussion

The frequency of occurrence of type E avian botulism outbreaks in Lake Michigan from 1963–2008 was related to lower water levels and higher summer surface water temperatures. Previous studies have linked type C avian botulism outbreaks to water-level fluctuations in

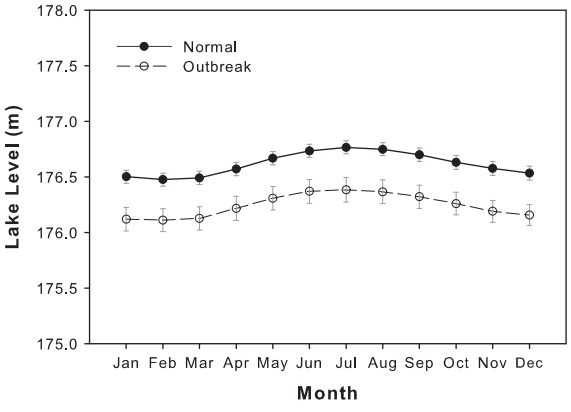

Fig. 2. Mean annual Lake Michigan water levels by month in botulism outbreak and non-outbreak years (1963–2008). Error bars denote one standard error of the mean.

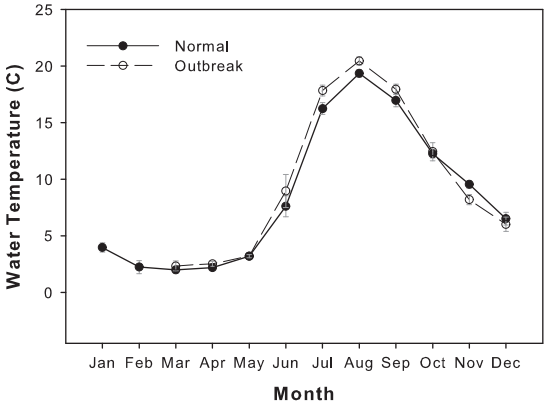

Fig. 4. Mean northern Lake Michigan surface temperatures by month in botulism outbreak and non-outbreak years (1979–2008). Error bars denote one standard error of the mean.

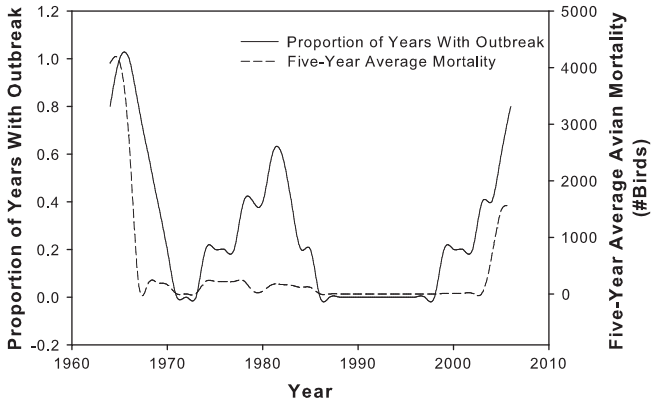

Fig. 5. Five-year moving average frequency of occurrence of botulism outbreaks (solid line) and estimated bird mortality (dashed line) in Lake Michigan (1963–2008).

smaller inland lakes and wetlands (Kalmbach, 1968; Galvin et al., 1985; Barras and Kadlec, 2000), and resource managers have been advised to avoid water draw-downs in these systems to minimize outbreaks (Rocke and Friend, 1999). Causal mechanisms for such water-level-outbreak links, however, have not been clearly demonstrated. Galvin et al (1985) and Barras and Kadlec (2000) speculated that water-level fluctuations cause drying and flooding of sediments, leading to benthic invertebrate mortality, which creates an environment rich in organic matter and conducive to *C. botulinum* spore germination and BoNT production. Similarly, we speculate that declines in Lake Michigan water levels, which are likely most common during or preceding years with low mean water levels, could result in the mass stranding and mortality of aquatic invertebrates, which may then serve as growth substrates for *C. botulinum* (see Rocke and Friend, 1999). Additionally, low water years may impact large areas of Lake Michigan shoreline and nearshore habitat, particularly in northern Lake Michigan where benthic and nearshore habitats are more complex. Deposition of decaying organic matter on these exposed areas (*e.g.*, dead alewife during the 1960s (Bunnell et al., 2006) or sloughed *Cladophora* during the 1960s and 2000s (Higgins et al., 2008)) could result in substantial nutrient and organic matter enrichment. Periodic seiche and storm events may then inundate these enriched shoreline areas, alter water temperature and redox conditions in ways that favor BoNT production (Pérez-Fuentetaja et al., 2006), and facilitate transport of BoNT-enriched substrates into deeper water where they may be consumed by various avian species.

Botulism outbreaks were also linked to higher surface water temperatures in this analysis and in other studies (Rocke and Samuel, 1999; Rocke et al., 1999). July to September surface water temperatures were significantly higher in botulism outbreak years than in non-outbreak years. Rocke et al. (1999) found that the probability of type C botulism outbreaks was associated with increasing water temperature in experimental wetland enclosures, and Rocke and Samuel (1999) reported that the risk for a type C botulism outbreak increased at water temperatures exceeding 20 °C. Higher water temperatures may promote avian botulism outbreaks directly by enhancing *C. botulinum* spore germination and vegetative cell growth or indirectly by exerting effects on benthic invertebrate populations (Rocke and Samuel, 1999).

In this study, outbreaks were most likely to occur when mean July–September surface water temperatures at the northern mid-lake data buoy were greater than 17–18 °C. Temperature data from shallower, more protected nearshore waters may show an even stronger relationship to frequency of outbreak occurrence or outbreak intensity. Water temperatures likely have direct effects on *C. botulinum* growth and BoNT production (*e.g.*, Smith and Turner, 1987). Warmer water temperatures also likely enhance growth and senescence of *Cladophora* (Higgins et al., 2008) and benthic invertebrates (*e.g.*, Plante and Downing, 1989), which in turn would increase organic matter and nutrient availability in shoreline and nearshore habitats, reduce oxygen availability, and indirectly enhance conditions for *C. botulinum* growth and BoNT production. Additionally, higher water temperatures may further enhance conditions for *C. botulinum* growth and BoNT production by decreasing dissolved oxygen availability, as a result of increased benthic respiration (Blumberg and Di Toro, 1990) or reduced oxygen solubility (Wetzel, 2001).

Links between trends in fish populations and botulism outbreaks were examined because fish have been implicated as a likely source of BoNT for piscivorous birds (Rocke et al., 2004; A.M. Yule et al., 2006) and because high prey abundance has been correlated with avian botulism outbreaks elsewhere (Rocke et al., 1999; Nol et al., 2004). In this study, botulism outbreaks were associated with higher abundance of benthic fish species including slimy sculpin and round goby. Further, major outbreaks in the 1960s coincided with large alewife populations (Bunnell et al., 2006), and recent outbreaks coincided with increasing abundance of round gobies in Lake Michigan (Truemper and Lauer, 2005). The fish population data used here did not cover the entire period of record of avian mortality, including the 1960s when alewife dieoffs were common. Moreover, these data were lake-wide mean abundance estimates determined from bottom trawling on soft substrates at fixed sites, a method that may not accurately reflect fish abundance near areas where BoNT occurs and that may not adequately represent fish that prefer rocky substrates, such as round gobies (Ray and Corkum, 2001). Further investigation will be required to fully understand potential linkages between fish abundance and botulism outbreaks.

Several published reports indicate that the frequency of wildlife disease events in aquatic ecosystems has increased in recent decades (Harvell et al., 1999; Ward and Lafferty, 2004; Newman et al., 2007), but a lack of baseline data makes explicit testing of this hypothesis difficult (Lafferty et al., 2004). Recent increases in wild avian mortality from botulism and other causes have been observed in other aquatic systems in the U.S. (Rocke et al., 2004; 2005; Newman et al., 2007) and the Baltic Sea (Neimanis et al., 2007). Although it has been suggested that type E botulism outbreaks have increased in recent years in the Great Lakes (IJC, 2004), these outbreaks have occurred sporadically in Lake Michigan since first documented in the early 1960s. Our dataset indicated that the frequency of outbreaks was high in both the 1960s and 2000s, but there was no clear trend in the frequency of outbreak occurrence over the period of record.

The analyses reported herein have several implications for future botulism research and monitoring efforts in the Great Lakes. Foremost, large-scale factors like lower lake levels and higher summer water temperatures increase the likelihood of botulism outbreaks. Further analyses investigating the relationship of other lake-level metrics (*e.g.*, magnitude or rate of lake-level change) and more specific water temperature metrics (*e.g.*, nearshore water temperature, number of days within *C. botulinum* optimum growth range) as they relate to botulism outbreaks over both annual and historical time scales are warranted.

Although our correlation-based approach identified large-scale relationships, it did not address mechanisms of BoNT production or transfer in the Great Lakes. As demonstrated for type C botulism (Rocke and Samuel, 1999), it is likely that lake levels and water temperatures are among many factors that contribute to type E botulism outbreaks. These factors, however, may only be relevant when they coincide with other environmental conditions (i.e., pH, salinity, and redox potential) conducive to BoNT production. Investigating spatial and temporal variability in chemical factors, particularly at fine spatial scales, in suspected BoNT-production hotspots (*e.g.*, depositional areas for organic matter, sediment–water interfaces, dreissenid mussel beds), and in relation to the onset and end of botulism outbreak periods, will help to further define mechanisms leading to BoNT production.

In addition to physical variables, a food web context conducive to BoNT mobilization must also be present for botulism outbreaks to occur. Botulism outbreaks occurred in the Great Lakes with similar regularity during the 1960s and 2000s, but the 1960s events predated the establishment of dreissenid mussel and round goby populations (key components in the hypothesized type E BoNT mobilization pathway). Exploring the relevance of native invertebrates and fish, and the dominance of key components in Great Lakes food chains (both native and invasive), may help to define the food web context necessary to facilitate BoNT mobilization.

Recent speculation about sites of BoNT production in the Great Lakes has focused on the potential role of deepwater depositional habitats and dreissenid mussel beds (*e.g.*, Getchell and Bowser, 2006). However, efforts to detect BoNT in these habitats have had limited success (Pérez-Fuentetaja et al., 2006; Getchell et al., 2006). As established through this study, the link between water levels and botulism outbreaks in the Great Lakes suggests that shoreline and inshore habitats (i.e., those most affected by lake-level fluctuations) may be more involved in BoNT production than previously thought. Conditions and ecological processes in these areas, particularly those associated with *Cladophora* deposition and decay (Byappanahalli and Whitman, 2009), should be considered in future investigations of BoNT production in Great Lakes environments.

Global climate change scenarios for the Great Lakes predict that warmer temperatures and lower water levels will occur in the future (Magnuson et al., 1997; Angel and Kunkel, 2010; Karl et al., 2009). This analysis indicates that such conditions may cause increased incidence of botulism outbreaks in Lake Michigan with potential negative impacts on fish and bird populations (*e.g.*, Friend et al., 2001). Because the ultimate effects of increased disease pressures on fish and bird populations of the Great Lakes are unknown (Riley et al., 2008), more comprehensive monitoring of botulism-related bird and fish mortalities in the Great Lakes is needed.

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