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Minor differentiation of foraging niche may have a major impact on the incidence of avian botulism in shorebirds

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Abstract

Avian botulism is a fatal disease of birds caused by ingestion of neurotoxins produced by *Clostridium botulinum* type C and is now recognized as the most common cause of death in waterbirds worldwide. Although tens of species have been reported to suffer from avian botulism, it remains unknown which ecological factors primarily determine inter-specific variation in the incidence of this disease. We hypothesized that an exposure of birds to botulin may largely depend on their foraging niche, as the toxin is available mostly at the sediment surface, especially during the carcass-maggot stage of botulism epizootics. To test this hypothesis we used capture-recapture methods to estimate mortality of two shorebird species differing in bill morphology and foraging niche, wood sandpiper *Tringa glareola* (short bill, surface-feeding) and common snipe *Gallinago gallinago* (long bill, deep probing), during a major avian type C botulism outbreak in central Poland. All the reported cases of shorebird mortality were attributed to botulism and we found large differences in daily survival rates of both species (0.87 and 0.99 in wood sandpipers and common snipe, respectively). Even assuming much shorter stopover duration of wood sandpipers, survival rate over the entire stopover period was estimated at 0.57 in the wood sandpiper and at 0.90 in the common snipe. To our knowledge, this is the first non-circumstantial evidence that relatively minor differentiation of foraging niche may have a major impact on the incidence of avian botulism in birds. Our data might also suggest that, on the evolutionary time scale, avian type C botulism may constitute a strong selective pressure acting on foraging niches of shorebirds, and possibly other waterbirds.

Zusammenfassung

Botulismus ist bei Vögeln eine tödliche Krankheit, die durch die Aufnahme von Neurotoxinen, die von *Clostridium botulinum* Typ C produziert werden, hervorgerufen wird und mittlerweile als die weltweit häufigste Todesursache bei Wasservögeln erkannt worden ist. Wenn auch bei Dutzenden von Vogelarten Botulismus gefunden wurde, ist unbekannt, welche ökologischen Faktoren hauptsächlich die interspezifischen Unterschiede in den Fallzahlen bestimmen. Wir nahmen an, dass der Kontakt zum Butulinumtoxin weitgehend von der Nahrungsnische abhängen sollte, weil das Toxin meist auf der Oberfläche des Sediments vorhanden ist, insbesondere während der Maden-Kadaver-Phase von Botulismus-Seuchen. Um diese Hypothese zu testen, nutzten wir während eines größeren Botulismusausbruchs in Mittelpolen Fang-Wiederfang Methoden zur Bestimmung der Mortalität von zwei Limikolenarten, die sich hinsichtlich Schnabellänge und Nahrungsnische unterscheiden: Bruchwasserläufer *Tringa glareola* (kurzer Schnabel, Nahrungssuche auf dem Sediment) und Bekassine *Gallinago gallinago* (langer Schnabel, stochert tief im Untergrund). Alle registrierten Sterbefälle der Limikolen wurden auf Botulismus zurückgeführt, und wir fanden große Unterschiede für die täglichen Sterberaten von Bruchwasserläufer (0,87) und Bekassine (0,99). Selbst wenn man eine deutlich kürzere Aufenthaltsdauer für den Bruchwasserläufer annimmt, war seine Überlebensrate für die gesamte Aufenthaltsdauer viel geringer

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(0,57) als bei der Bekassine (0,90). Nach unserer Kenntnis ist dies der erste direkte Hinweis darauf, dass ein verhältnismäßig kleiner Unterschied in der Nahrungsnische größeren Einfluss auf die Häufigkeit von Botulismusfällen bei Vögeln haben könnte. Unsere Ergebnisse könnten auch nahelegen, dass in evolutionären Zeiträumen Botulismus Typ C einen starken Selektionsdruck auf die Nahrungsnischen von Watvögeln und möglicherweise anderen Wasservögeln ausübt.

Keywords: Capture-recapture; *Clostridium botulinum*; Foraging niche; Shorebirds; Survival rate; Type C botulism

Introduction

Avian botulism is a paralytic and often fatal disease of birds caused by ingestion of neurotoxins that are produced by *Clostridium botulinum* type C (Rocke and Bollinger 2007). Sporadic avian mortality has also been caused by type E toxin, although it has been reported almost exclusively for fish-eating waterfowl (Brand, Schmitt, Duncan, & Cooley 1988; Hannett, Stone, Davis, & Wroblewski 2011). Under aerobic and other adverse conditions, *C. botulinum* forms dormant spores, which are harmless until they germinate into vegetative cells and begin multiplying. After germination, bacterial cells may start to produce neurotoxins which are encoded by specific bacteriophages that infect and replicate specifically within *Clostridium* bacteria (Eklund, Poysky, Reed, & Smith 1971). Optimal environmental conditions that promote germination of botulinum spores are relatively poorly recognized, but botulism outbreaks usually develop in shallow waters that are rich in decaying organic matter (Rocke and Samuel 1999).

In order for a botulism outbreak to occur the toxin must become available for birds. In the early stages of epizootics the toxin is presumably transferred from the substrate to the birds through zooplankton and benthic epifauna. Invertebrates that feed on decaying matter remain unaffected by the toxin and may act as toxin reservoirs (Duncan and Jensen 1976; Rocke and Bollinger 2007). An alternative route to a massive botulism outbreak is through toxicoinfection, resulting from ingestion of *C. botulinum* spores, followed by multiplication of bacteria within the gastrointestinal tract, with subsequent toxigenesis and absorption of toxins (Critchley 1991; Trampel, Smith, & Rocke 2005). Thus, the incidence and numbers of *C. botulinum* spores and vegetative cells, as well as the concentration of botulinum neurotoxin in the environment could contribute significantly to the onset of a large outbreak. As soon as the first botulism-related casualties occur, an outbreak often becomes self-perpetuating, as foraging waterbirds tend to accidentally ingest toxic fly larvae that feed on the carcasses of infected birds. This, so called, carcass-maggot cycle rapidly accelerates the spread of the disease, causing massive avian botulism outbreaks (Espelund and Klaveness 2014). In fact, outbreaks with losses of up to 50,000 birds are relatively common and botulism epizootics with more than a million deaths have been reported (Rocke and Bollinger 2007). In consequence, avian botulism has been recognized as the most common cause of death in waterbirds worldwide.

Most shorebirds (Charadrii) typically use shallow water and mudflat habitats to replenish fuel reserves during migration and, thus, are potentially highly exposed to avian botulism. However, this group of birds shows extraordinary divergence of micro-habitat use and foraging niche, mostly attributable to high inter-specific variation in bill morphology (Nebel, Jackson, & Elner 2005). One of the primary determinants of foraging behavior in shorebirds is bill length, as species/individuals with shorter bills are expected to peck epifaunal prey or probe near the sediment surface, while species/individuals with longer bills probe deeper into the sediments and feed mainly on infaunal invertebrates. We hypothesized that such differentiation of foraging niche may have an impact on the incidence of avian botulism, as botulinum toxins are available mostly at the sediment surface, especially during the carcass-maggot stage of botulism epizootics. To test this hypothesis we used capture-recapture methods to estimate survival rate of two shorebird species differing in bill morphology, wood sandpiper *Tringa glareola* and common snipe *Gallinago gallinago*, during a major avian type C botulism outbreak. Wood sandpiper has a relatively short bill (interquartile range: 27.0–28.8 mm, Fig. 1) and it

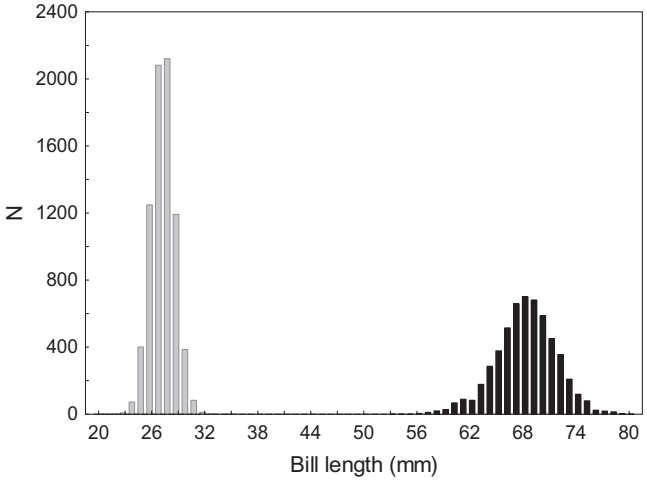

Fig. 1. Divergence in bill length among wood sandpiper (grey bars, $n = 7609$ individuals) and common snipe (black bars, $n = 5569$ individuals). Data collected during autumn migration period between 1989 and 2013 at Jeziorsko reservoir, central Poland.

frequently catches free-living and mobile prey from the sediment surface (Krupa et al. 2009). Although common snipe is only slightly larger, its bill is over two times longer (interquartile range: 66.5–70.7 mm, Fig. 1) and adapted specifically for deep probing. As a result, we expected wood sandpipers to be more exposed to botulinum neurotoxins and, thus, to show elevated rate of botulism-related mortality.

Materials and methods

The fieldwork was conducted at the Jeziorsko reservoir (51°40′N, 18°40′E), central Poland, in 2011, when a major avian type C botulism outbreak took place (for details please see Włodarczyk et al. 2014). The first botulism-related bird mortalities were recorded at the reservoir in mid-May and the epizootic peaked in July-August. At the end of August the number of affected birds started to decrease, and the outbreak abated gradually in mid-September. Using the data from regular counts (plots and transects), the total number of affected waterbirds at the reservoir was estimated at ∼5500 individuals. Mallard *Anas platyrhynchos* and Eurasian coot *Fulica atra* dominated among intoxicated birds (42.5% of all affected individuals). Ten species of shorebirds were also recorded to suffer from intoxication (Włodarczyk et al. 2014). The presence of botulinum toxin type C in the internal organs (liver, spleen, kidneys) of sick birds was confirmed with mouse lethality assay, following Solomon and Lilly (2001). Strains isolated from the samples were also tested for the presence of gene (*nhnt*) specific for *C. botulinum* by real-time PCR and for the presence of genes specific for *C. botulinum* type C and D (*bontC*, *bontD*) by conventional PCR (Grenda & Kwiatek 2009). Comparison of obtained PCR product sequence with GeneBank database revealed 99.7% identity with the neurotoxin gene of *C. botulinum* type C isolated from birds (for details of laboratory analyses please see Włodarczyk et al. 2014).

We started to capture shorebirds during the peak of the botulism epizootic (12 August) and continued trapping for the next 40 days (until 20 September, the final stage of the epizootic). In total, we captured 188 wood sandpipers and 158 common snipes. All captured sandpipers and most snipes (88%) were first-year birds. Each captured individual was marked and released at the reservoir as soon as the standard ringing procedures and data collection were completed.

In order to estimate botulism-related mortality of shorebirds, sick individuals and carcasses were counted at a 10 ha (0.1 km^2) mudflat plot, where birds were captured. The entire plot was checked twice a day and all carcasses were removed from the plot on each occasion. All recorded cases of shorebird mortality were attributed to botulism. Individuals recognized as affected by botulinum toxin showed typical intoxication symptoms, including limb paresis, flaccid neck paralysis, frequent ventroflexion of the neck, or a complete paralysis. None of the collected carcasses showed any signs of predation. In general, predation-related mortality of shorebirds at the site is negligible due to the absence of medium-sized and large falcons which are considered the major predators of migrating shorebirds (Ydenberg et al. 2004; Van Den Hout, Spaans, & Piersma 2008).

For the analysis we used the Burnham capture-recapture model for both live and dead encounters (Burnham and Anderson 2002). The model estimates the following parameters: survival probability (S), recapture probability (p), recovery probability (r) and fidelity rate (F). In all the models we assumed that recovery probability r did not differ between species, as sandpiper and snipe carcasses were collected at the same plot with the same methodology and, technically, localization of carcasses at an open mudflat was highly effective (we collected ∼100% of all carcasses at each occasion). Thus, r was either constrained as constant or time-dependent (41 encounter occasions = days). All other parameters: (1) survival rate S, (2) recapture probability p, and (3) fidelity rate F, were tested for inter-specific variation, time dependency, and an interaction between them, resulting in 128 models fitted to the data. Goodness-of-fit was tested on a fully parameterized model with a bootstrap routine. We conducted 100 simulations and compared deviance of the fitted model with randomly generated deviance values. In order to estimate the variance inflation factor (ĉ) we divided the deviance estimate for the actual data by the average deviation of the simulated data. Using this method, ĉ was estimated at 1.34, indicating that the model fitted the data reasonably well. All fitted models were ranked using Akaike's Information Criterion corrected for small sample size and adjusted for overdispersion ($QAIC_C$). The models were also compared with Akaike weights that are interpreted as the weights of evidence in favour of a given model against all other hypothesized models. Relative importance of each explanatory variable was evaluated by summing Akaike weights across all models in which the variable occurred (Burnham and Anderson 2002). Likelihood-ratio (LR) test was used to compare selected pairs of general and reduced (nested) models. We also used model averaging to calculate a weighted average of parameters estimates based on Akaike weights. This approach produces robust parameter estimates by reducing model selection bias and accounting for model selection uncertainty (Johnson and Omland 2004). All calculations were conducted in program MARK (White and Burnham 1999). We reported all estimated parameters as mean ± SE.

Results

In total, we collected 69 carcasses of wood sandpipers (19 marked, 50 unmarked) and 17 carcasses of common snipe (2 marked, 15 unmarked) at the selected study plot. At the same time, 4.8% of wood sandpipers and 9.5% of common snipe were recaptured with no clinical signs of botulism. Capture-recapture analysis gave strong evidence for differences in botulism-related survival rate between the species, as indicated by the four most parsimonious models (Table 1).

Table 1. Overview of model selection for botulism-related survival of wood sandpiper and common snipe.

Model	$QAIC_C$	$\Delta QAIC_C$	$QAIC_C$ weight	Np	Deviance
(1) *S*(species) *p*(.) *r*(.) *F*(.)	436.17	0.00	0.51	5	221.5
(2) *S*(species) *p*(.) *r*(.) *F*(species)	438.07	1.90	0.20	6	221.3
(3) *S*(species) *p*(species) *r*(.) *F*(.)	438.12	1.95	0.19	6	221.4
(4) *S*(species) *p*(species) *r*(.) *F*(species)	439.45	3.28	0.10	7	220.6
(5) *S*(.) *p*(.) *r*(.) *F*(species)	445.04	8.87	0.01	5	230.4
(6) *S*(.) *p*(species) *r*(.) *F*(species)	446.86	10.69	0.00	6	230.1
(7) *S*(.) *p*(species) *r*(.) *F*(.)	449.18	13.01	0.00	5	234.5
(8) *S*(.) *p*(.) *r*(.) *F*(.)	449.34	13.17	0.00	4	236.7
(9) *S*(t) *p*(.) *r*(.) *F*(species)	461.62	25.45	0.00	45	153.4
(10) *S*(species) *p*(.) *r*(t) *F*(.)	462.50	26.33	0.00	45	153.3

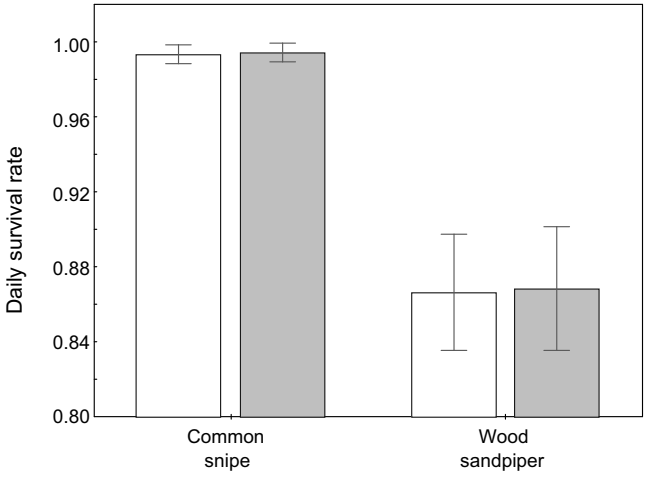

Fig. 2. Daily survival rate (±SE) of common snipe and wood sandpiper during a major avian type C botulism outbreak estimated with the most parsimonious model (white bars) and model averaging (grey bars).

The best-ranked model assuming no inter-specific variation in survival rate poorly fitted the data ($\Delta QAIC_C = 8.87$, Model 5). Comparison of nested models (Model 1 vs. Model 8) with the LR test indicated that the likelihood of the more complex model (inter-specific variation in survival) was significantly greater than the likelihood of a model assuming no inter-specific variation in survival ($\chi^2 = 15.23$, df = 1, $p < 0.001$). Strong relative importance of inter-specific variation in explaining survival rates of both species was supported with high total Akaike weight ($w_i = 0.99$). The best model estimated daily survival at 0.995 ± 0.005 in the common snipe and 0.867 ± 0.031 in the wood sandpiper. Model averaging gave highly consistent estimations of daily survival rate: 0.994 ± 0.005 in the common snipe and 0.869 ± 0.033 in the wood sandpiper (Fig. 2). There was no evidence for inter-specific differences in recapture probability (0.011 ± 0.004, Model 1). The most parsimonious model also indicated no differences in fidelity rate (F) between both species (0.930 ± 0.030; Model 1), while the second best model estimated fidelity rate at 0.75 ± 0.07 in wood sandpiper and at 0.94 ± 0.03 in common snipe. The latter estimates translated into stopover durations $1/(1 - F)$ of 4.0 days in the wood sandpiper and 16.7 days in the common snipe.

Discussion

Our capture-recapture study of two shorebird species, wood sandpiper and common snipe, revealed substantial differences in botulism-related mortality. Daily survival rates during the botulism epizootic were estimated at 0.87 in the wood sandpiper and 0.99 in the common snipe. Even assuming much shorter stopover duration of wood sandpipers (4.0 days vs. 16.7 days in the common snipe), survival rate over the entire stopover period was estimated at 0.57 in the wood sandpiper and at 0.90 in the common snipe. As both species used the same foraging habitat and, thus, were expected to have similar exposure to botulism neurotoxins released into the environment, we suggest that these striking differences in survival were most likely attributable to a differentiation of foraging niche via changes in bill morphology.

The effect of foraging niche on the incidence of botulism in shorebirds has already been suggested based on some circumstantial observational evidence. Surveys of shorebirds affected by botulism in Canada showed a dominance of surface feeding species, indicating that they may be more prone to botulism than probers (reviewed in Adams et al. 2003). For example, two species of dowitchers *Limnodromus*, which are specifically adapted for deep probing, were clearly underrepresented in the mortality surveys during botulism outbreaks (Adams et al. 2003). By contrast, surface-feeding small *Calidris* sandpipers have been reported to be most commonly affected by avian botulism, although the counts were not corrected for relative abundance (Strauman 1996; Peers 1998).

Most avian botulism outbreaks self-perpetuate through the carcass-maggot cycle (Reed and Rocke 1992), as most waterbirds, including shorebirds, find fly larvae palatable (Adams et al. 2003). Several thousand toxic maggots can be produced from a single waterfowl carcass, and consumption of as few

as two maggots may be lethal for a bird, as they concentrate toxin at very high levels (Bell, Sciple, & Hubert 1955; Espelund & Klaveness 2014). Thus, surface-feeding species may become considerably more exposed to the toxin, while probing species that mainly feed on infaunal invertebrates are likely to be much less exposed. We propose that this difference in feeding habits might primarily explain observed differences in botulism-related mortality of wood sandpiper (surface-feeder) and common snipe (prober). Also, botulism neurotoxins are usually released into the environment at relatively low quantities and the toxin concentrations in water are often low (Hubálek and Halouzka 1991), which suggests that variation in the incidence of botulism should be primarily driven by the type of prey consumed (Rocke and Bollinger 2007).

On the other hand, we cannot exclude that the differences in botulism-related mortality between common snipe and wood sandpiper could be, at least to some extent, attributed to different susceptibility of both species to botulinum neurotoxin. In general, the common snipe is about 30% larger than the wood sandpiper (average body mass in 2011: 99.89 ± 0.91 [SE] g vs. 69.03 ± 0.78 [SE] g in common snipe and wood sandpiper, respectively), which means that the minimum lethal dose of the toxin is expected to be slightly larger for the snipe. However, we find this explanation unlikely, as numerous case reports from massive botulism outbreaks indicate that the incidence of botulism is primarily determined by feeding ecology of birds rather than by their mass/size-related susceptibility. In most cases, dabbling ducks *Anatini* and coots have been found to show the highest incidence of botulism (Forrester et al. 1980; Brand, Windingstad, Siegfried, Duncan, & Cook 1988; Shin et al. 2010; Woo et al. 2010; Work, Klavitter, Reynolds, & Blehert 2010), despite the fact that they are several times heavier than the majority of shorebirds, including common snipe and wood sandpiper. Similar situation has been observed at our study site, where mallards (body mass: 750–1450 g) and Eurasian coots (body mass: 600–1000 g) dominated among intoxicated birds (nearly half of all affected individuals) (Włodarczyk et al. 2014). This might be explained by the fact that dabbling ducks are known to readily consume maggots once they are dislodged from the intoxicated carcasses (Duncan and Jensen 1976).

Although avian type C botulism in wild birds was first recorded in North America at the beginning of 20th century, for the last 30 years it has been reported from all the continents except Antarctica (Rocke and Bollinger 2007). Assuming the emerging nature of type C botulism (Dobson and Foufopoulos 2001) and the fatal impact it has on wildlife, we suggest it may become an important regulatory factor in avian populations most susceptible to the disease. Our data might also suggest that, on the evolutionary time scale, avian type C botulism may constitute a strong selective pressure acting on foraging niches of shorebirds, and possibly other waterbirds, but much more empirical evidence is needed to firmly support this hypothesis.

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