REVIEW

DOI: 10.7589/2015-05-117

Journal of Wildlife Diseases, 52(3), 2016, pp. 441–458

MORTALITY OF SELECTED AVIAN ORDERS SUBMITTED TO A WILDLIFE DIAGNOSTIC LABORATORY (SOUTHEASTERN COOPERATIVE WILDLIFE DISEASE STUDY, USA): A 36-YEAR RETROSPECTIVE ANALYSIS

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ABSTRACT: To determine the relative importance of mortality factors for birds and to assess for patterns in avian mortality over time, we retrospectively examined data of birds submitted to the Southeastern Cooperative Wildlife Disease Study (SCWDS; http://vet.uga.edu/scwds), US, from 1976 to 2012. During this period, SCWDS, a wildlife diagnostic laboratory, received 2,583 wild bird specimens, from the taxonomic orders Apodiformes, Caprimulgiformes, Cuculiformes, Passeriformes, and Piciformes, originating from 22 states. Data from 2,001 of these birds were analyzed using log-linear models to explore correlations between causes of mortality, taxonomic family, demography, geographic location, and seasonality. Toxicosis was the major cause of mortality, followed by trauma, bacterial infection, physiologic stress, viral infection, and other (mortality causes with low sample numbers and etiologies inconsistent with established categories). Birds submitted during fall and winter had a higher frequency of parasitic infections, trauma, and toxicoses, whereas birds submitted during the spring and summer were more likely to die of an infectious disease, physiologic stress, or trauma. We noted a decrease in toxicoses concurrent with an increase in bacterial infections and trauma diagnoses after the mid-1990s. Toxicosis was the most commonly diagnosed cause of death among adult birds; the majority of juveniles died from physiologic stress, trauma, or viral infections. Infectious agents were diagnosed more often within the families Cardinalidae and Fringilidae, whereas noninfectious etiologies were the primary diagnoses in the Bombycillidae, Parulidae, Sturnidae, Turdidae, and Icteridae. There are important inherent limitations in the examination of data from diagnostic labs, as submission of cases varies in timing, frequency, location, and species and is often influenced by several factors, including media coverage of high-profile mortality events. Notwithstanding, our data provide a rare opportunity to examine long-term, regional, and temporal patterns in causes of avian mortality, and they allow for the analysis of novel and rare mortality factors.

Key words: Data set, diagnosis, disease, infection, mortality, wild bird.

INTRODUCTION

Wild birds provide valuable ecosystem services such as seed dispersal, pollination, and insect control, and they serve as prey to a variety of natural predators. In addition, birds are valued as part of the expanding wildlife recreation economy that generates millions of

dollars annually through bird watching, photography, hunting, and feeding, all of which contribute to an increasing need to understand the causes of avian morbidity and mortality. Urbanization and other landscape changes apply pressure on wild birds to live at the interfaces with domestic animals and humans. This provides potential opportunities for zoonotic disease transmission to humans, and exposure to potentially higher rates of noninfectious, and often anthropogenic, mortality factors for birds. Previous morbidity and mortality investigations of wild birds have revealed numerous infectious (Hall and Saito 2008; Hoque et al. 2012) and noninfectious causes of morbidity and mortality (Fleischi et al. 2004; Martins et al. 2010) that may have population-level implications for birds in North America. Examples include salmonellosis (Hall and Saito 2008), mycoplasmal conjunctivitis (Dhondt et al. 2005), West Nile virus (WNV; genus *Flavivirus*) infection, trauma, and pesticides (Fleischi et al. 2004).

Anthropogenic factors can be a significant additive force contributing to declining avian populations. An estimated 500 million to 1 billion birds die annually due to anthropogenic factors, including collisions with artificial structures (e.g., buildings, wind turbines, windows, vehicles, and power lines), electrocution, predation by feral or domestic animals, contamination from pesticides and oil spills, fishing by-catch, and enhancement of pathogen transmission in manipulated environments (e.g., bird feeding stations) (Banks 1979; Erikson et al. 2005). Recently, species have suffered from highly visible mortality events (e.g., icterids in Arkansas; Southeastern Cooperative Wildlife Disease Study [SCWDS] 2011) that generated attention and concern from the general public. Yet, similar events may remain undetected or underreported.

The majority of studies analyzing mortality data have had small sample sizes or focused on a specific taxonomic group, mortality cause, or season. In addition, mortality causes and frequency estimates based on diagnostic reports require careful interpretation because of inherent bias resulting from 1) the geographic location of the laboratory, which may influence case submission; 2) disproportionate attention given to some syndromes or species over others based on natural resource agency priorities; 3) disproportionate submission of cases based on public perceptions, which can change over time; 4) difficulty of accurately diagnosing cases due to a lack of suitable specimens or available expertise; and 5) underdiagnosing certain conditions due to a lack of specific tests for some mortality causes (Bernardino et al. 2012). Regardless, patterns of mortality from diagnostic laboratory studies remain an important source of information and often guide and encourage further research (Nettles et al. 2002; Fleischi et al. 2004; Gottdenker et al. 2008; Stauber et al. 2010; Hoque et al. 2012; Nemeth et al. 2014).

Herein, we 1) summarize causes of avian mortalities across taxonomic orders diagnosed at the SCWDS from 1976 to 2012; 2) investigate temporal patterns in the most common diagnoses; and 3) determine whether age, sex, temporal, or spatial factors were associated with mortalities. Because of the very large number of avian species discussed, we do not provide scientific names in the text. They are all listed along with their standard common names (Clements et al. 2015) in Supplementary Material Table S1.

MATERIALS AND METHODS

Study population

We reviewed all avian submissions to the SCWDS diagnostic service located at the University of Georgia, Athens, Georgia, USA, from 1 September 1976 to 31 December 2012. The SCWDS primarily receives cases from 22 states that have contracts to conduct mortality investigations. Throughout the study, several states became members of SCWDS, but in general, states submitting cases were concentrated in the southeastern US (SCWDS 2016). Case submissions included single birds (with no additional recognized, associated mortalities); small groups of birds of one or more species; or larger events involving hundreds or rarely thousands of birds, usually of multiple species, as reported to SCWDS by field biologists and other stakeholders. Data from diagnostic cases have inherent biases due to the increased likelihood that biologists or the public observe, collect, and submit carcasses that are highly visible or

associated with anthropogenic factors. Higher numbers of affected birds often equate to increased visibility, evidenced by several cases described by the submitters as outbreaks in which dozens or hundreds of birds died. In such cases, accurate estimates of the numbers of birds affected were obtained to the extent possible. Biases regarding geography and landscape are also inherent; for example, mortalities are more likely visible in areas 1) that contained member states; 2) with high human density; 3) with regular monitoring by biologists and other wildlife personnel; 4) that house species of interest because of their conservation status, or public health awareness and concern (e.g., American Crows and other corvids used in WNV surveillance); 5) with public access to areas where birds reside; and 6) where predators and scavengers have had limited access to carcasses. Typically, in mortality events consisting of 10 or more birds, a subset of the available carcasses was subjected to diagnostic evaluation. We defined a "mortality event" as any SCWDS submission involving one or more birds where each bird involved was considered a "case." Submissions included whole carcasses and field-collected tissues. Each submission included a brief summary of the mortality event as described by the submitters (e.g., state wildlife biologists and veterinarians). Summaries generally included location, species, number of animals affected, clinical symptoms if moribund birds were present, and time frame of observed morbidity and mortality. Diagnostic results from some taxonomic groups (e.g., waterfowl, raptors, columbids, and galliforms) are not included in our analysis because they have been previously published (Quist et al. 1995; Fischer et al. 2006; Gerhold et al. 2007). Submitters often noted details about human activities known to occur in the area. For most birds, sex was determined by plumage characteristics and morphometrics or during postmortem examination. Age classes included adult (for those with definitive plumage) and juvenile (hatchlings, nestlings, fledglings, hatch-year, and after-hatch-year).

Diagnostic evaluation and laboratory tests

Diagnostic evaluations of wildlife (carcasses and biological samples) performed at SCWDS included postmortem evaluations and ancillary testing (e.g., bacteriology, virology, and toxicology) as necessary to determine cause of death. Tissues from major organ systems were fixed in 10% neutral-buffered formalin, routinely processed and paraffin-embedded, sectioned at 4 μm, mounted on glass slides, and stained with hematoxylin and eosin. In some cases, impression smears of fresh tissues were stained with Wright's stain to visualize microbes (e.g., trichomonads). Ancillary tests were performed at various diagnostic laboratories, such as at SCWDS, the Athens Diagnostic Laboratory (University of Georgia), Michigan State University, or the California Animal Health and Food Safety Laboratory (University of California–Davis), among others. These tests included bacterial and fungal culture, virus isolation, PCR, fluorescent antibody test, special histochemical stains, immunohistochemistry (e.g., for virus-specific antigens) and toxicologic tests (e.g., cholinesterase assays and mass spectroscopy). Organophosphate (OP) and carbamate (CB) toxicities were determined by measuring cholinesterase inhibition in the central nervous system or by quantifying pesticide concentration in blood, liver, brain, or gastrointestinal contents. Pooled or single tissues (e.g., liver, kidney, heart, and brain) or proventricular, ventricular, or intestinal contents were screened for toxins by mass spectroscopy or gas chromatography. Additional toxicologic assays included enzyme activity (Glaser 2001), reactivation analyses (Kiffer and Minard 1986), and spectrophotometry (Fitzgerald et al. 1990). Ethanol toxicosis was diagnosed based on postmortem findings, absence of evidence of pesticides in tissues, and background information. Although considered valuable, incidental parasitologic findings were not quantitatively assessed due to lack of a consistent parasitologic workup.

Quantitative data analysis

We used log-linear models in R version 3.0.0 (R Development Core Team 2013) to understand whether mortality causes differed among seasons, years, bird taxonomic family, or an individual's age or sex. Seasons were defined as winter (December–February), spring (March–May), summer (June–August), and fall (September–November). Years were grouped into four decades. To determine whether there were any differences in the number of mortalities caused by infectious and noninfectious categories following the enactment of the Food Quality Protection Act (FQPA), we used log-linear models to analyze whether mortality causes differed between the period before 1996, when the FQPA was passed, and after 1996. Causes of mortality were separated into nine broad categories: bacteria, viruses, fungi, ectoparasites, endoparasites, physiologic stress (e.g., emaciation, starvation, malnourishment, temperature stress, and postmigratory stress), toxicosis, trauma, and "other" (mortality causes with low sample numbers and etiologies inconsistent with established categories). Incidental findings (e.g., parasite and bacterial infections) and additional findings not considered the primary causes of death (e.g., secondary bacterial and fungal infections) are included. Spatial data were not quantitatively analyzed because collection was not uniform across the landscape.

RESULTS

Study population

From 1976 to 2012, 2,583 bird carcasses (from 813 mortality events) were submitted to SCWDS from 22 states. Five taxonomic orders were represented, including 30 families and 101 species (see Supplementary Material Table S1). From these, 2,001 complete diagnostic evaluations were conducted resulting in 2,105 primary mortality causes for analysis (1,897 birds with one mortality cause, 102 with two causes, and two with three causes; see Supplementary Material Table S2). Submitted birds included 1,040 males, 613 females, and 641 birds of undetermined sex, with 1,558 adults, 290 juveniles, and 446 birds of undetermined age. Birds for which mortality causes were determined (i.e., included in the analyses) included the following: 554 females, 917 males, and 539 of unknown sex, with 1,376 adults, 256 juveniles, and 378 individuals of undetermined age.

Mortality categories

Toxicosis was the most common diagnosis amongst wild bird carcasses submitted during the study (n=1,021; prevalence=0.49, 95% confidence interval [CI]: 0.46–0.51). Organophosphate toxicosis was the most frequently determined cause of death (n=511). The most common OPs implicated were famphur (n=204), diazinon (n=115), chlorpyrifos (n=55), and dursban and methamidophos (n=24 each) (see Supplementary Material Table S2). The concentration of OPs in pooled samples (e.g., kidney, liver, heart, and brain) ranged from >0.075 to 4,220 ppm, and all concentrations within this range were interpreted as a toxicosis based on diagnostic background. Lower concentrations (e.g., 0.075 ppm) were considered as toxicosis after considering that the mortality event comprised all of the known risk factors (e.g., bird family, location, and numbers of birds affected) and the status of the diagnostic sample. The second most commonly diagnosed cause of mortality was trauma, resulting in 463 mortalities (prevalence=0.22, 95% CI: 0.20–0.24). These were usually caused by collisions with motor vehicles or buildings. Bacterial infections were the third leading cause (n=346; prevalence=0.16, 95% CI: 0.15–0.18), followed by stress (n=82; prevalence=0.04, 95% CI: 0.03–0.05) and viral infections (n=51; prevalence=0.02, 95% CI: 0.019–0.03). All other mortality categories combined encompassed <50 cases and included the following: ectoparasites (n=48; prevalence=0.02, 95% CI: 0.017–0.03), endoparasites (n=41; prevalence=0.02, 95% CI: 0.01–0.03), other miscellaneous causes (n=36; prevalence=0.015, 95% CI: 0.01–0.02), and fungal infections (n=17; prevalence=0.009, 95% CI: 0.005–0.014). Collectively, noninfectious causes were responsible for 1,583 bird deaths (prevalence=0.75, 95% CI: 0.73–0.77) and 519 mortalities were due to infectious agents (prevalence=0.25, 95% CI: 0.23–0.27).

Bird taxonomy and mortality causes

For birds in the order Passeriformes (n=1,927; 92% of all mortalities), there was a strong association between avian family and whether the cause of death was infectious or noninfectious (likelihood ratio χ^2=742.92, Pearson=782.19, df=9, P<0.05). Infectious causes of mortality were more common than noninfectious causes the families Cardinalidae (n=83; 87% of cases were Northern Cardinals) and Fringillidae (n=268; 95% of cases were Pine Siskins, House Finches, and American Goldfinches), whereas rates of noninfectious causes of mortality were significantly higher than rates of infectious causes among the Bombycillidae (n=134; 100% of cases were Cedar Waxwings), Icteridae (n=747; 97% of cases were the Brown-headed Cowbirds, Common Grackles, Boat-tailed Grackles, and Red-winged Blackbirds), Parulidae (n=94; 23 species, no predominant species), Sturnidae (n=102; 100% of cases were European Starlings), and Turdidae (n=157; 76% of cases were American Robins) (Fig. 1a). In contrast, the number of bird deaths attributed to infectious versus noninfectious causes was not statistically different for birds in the Corvidae (n=117; 68% of cases were American Crows), Emberizidae (n=88; 10 species,

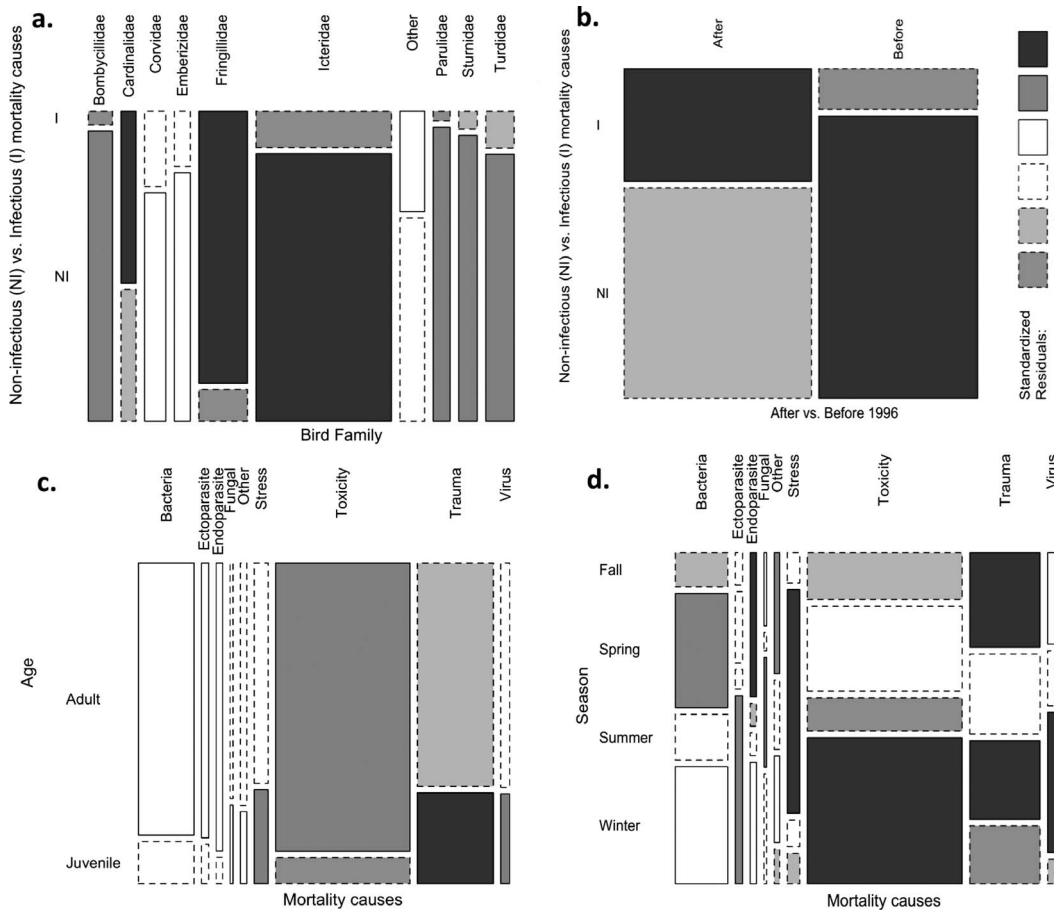

FIGURE 1. Mosaic plots showing the relationships between the causes of mortality among birds from the USA submitted to the Southeastern Cooperative Wildlife Disease Study, 1976–2012 and several factors. Each mosaic plot (a–d) represents an overall statistically significant relationship between the two variables on the *x*- and *y*-axes, as determined using log-linear models. The log-linear model determines the overall relationship between the two variables, whereas the mosaic plot provides a visual of which observations or categories within the two variables are specifically driving the statistical difference. These "strengths of associations" are indicated in the mosaic plots as follows: black boxes correspond to a 0.01% significance; gray boxes correspond to a 5% significance; and white boxes correspond to <5%, or a nonsignificant difference. A solid line around the box indicates an observed frequency that is higher than what is expected by chance, whereas a dotted line indicates a lower than expected frequency. Panels a and b group the causes of mortality into two categories, infectious and noninfectious causes; panels c and d show the causes of mortality as the nine broad categories, as described in the text. Causes of mortality showed strong relationships with bird family (a), years pre– and post–Food Quality Protection Act legislation (b), age (c), and season (d). The first box in each column represents the first category on the *y*-axis; the second box corresponds to the second category on the *y*-axis and so on when there are more than two categories on the *y*-axis. The width of each column indicates the proportion of individuals from that category out of the total number of individuals analyzed.

none dominant), and the grouping of families classified as "other" (n=137; 15 families, with 1–45 records per family) (Fig. 1a). Common Grackles (n=6) were the only species infected with the bacterium *Hafnia alvei*.

Bird demographics and mortality causes

Mortality in juvenile birds was more often attributed to physiologic stress, trauma, and viral infection compared to the other causal agents, whereas adults died more frequently

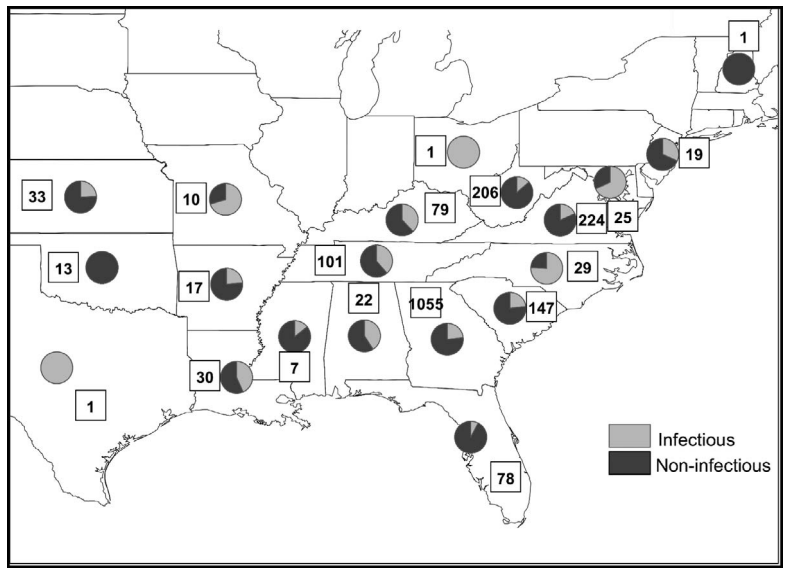

FIGURE 2. Geographic distribution within the USA of infectious and noninfectious mortality causes of wild birds submitted to the Southeastern Cooperative Wildlife Disease Study, 1976–2012.

from toxicosis (likelihood ratio χ^2=102.81, Pearson=105.89, df=8, $P<0.05$) (Fig. 1c). Frequencies of mortality were not significantly different among categories for male versus female birds (likelihood ratio χ^2=6.38, Pearson=6.46, df=8, P=0.60).

Temporal, seasonal, and geographic trends in mortality

There was a significant difference in the numbers of infectious versus noninfectious case submissions before and after 1996 (likelihood ratio χ^2=145.47, Pearson=138.70, df=1, $P<0.05$), with more wild birds dying from noninfectious mortality causes before 1996 (Fig. 1b). The numbers of cases varied greatly within decades from the late 1970s through 2012, with peaks in case submissions occurring every 7–10 yr. There was a significant association between the season in which bird carcasses were found (i.e., submitted) and the mortality category (likelihood ratio χ^2 =347.74, Pearson=359.49, df=8, $P<0.05$). For example, during fall, frequencies of mortality were comparatively low for bacteria and toxicologic etiologies and higher for endoparasitic infections, trauma, and other causes. In contrast, deaths during spring were more frequently caused by bacteria and stress and less commonly by endoparasites. During summer, mortalities were more often caused by fungal and viral infections and trauma than toxicoses. Finally, during winter, mortality rates from ectoparasites and toxicoses were higher than those caused by fungi, other, trauma, and viral infections (Fig. 1d). Throughout the study, the southeastern US was overrepresented in submission origins compared to other areas. Noninfectious mortalities were more often diagnosed in the Southeast than in other regions (Fig. 2).

DISCUSSION

The most common causes of mortality for avian case submissions in this study were of noninfectious origin, specifically toxicoses (most commonly OPs) and trauma, followed by bacterial infections, physiologic stress, and viral infections. Sampling was opportunistic and not designed to represent specific avian taxa, habitat types, or geographic locations. Location bias includes greater numbers of birds submitted to SCWDS from Georgia (where SCWDS is located) and nearby SCWDS charter member states. More recent-

ly, SCWDS has included samples from more distant states, again affecting geographic patterns of diagnoses. Although results should be interpreted with an understanding of the limitations of cases submitted to a diagnostic center, this type of surveillance is an invaluable method for discovering patterns of mortality that often lead to subsequent research. For example, isolated outbreak events were noticed in wild ungulates in Canada during early 1970s, and individuals submitted to a wildlife pathology laboratory for necropsy were diagnosed with epizootic necrobacillosis. This finding led to epizootiologic studies that determined the risk factors that were associated with the morbidity and mortality events (Wobeser et al. 1975).

Toxicoses

Organophosphate compounds were the most common causes of toxicosis diagnosed during the present study. More than 100 bird species have been documented with mortality due to OP and CB toxicoses (Glaser 2001). The impact of human-made toxins in general on wild birds is also evident in several European countries, in which bird diversity reportedly decreased following the application of pesticides to fields (Geiger et al. 2010). Although all birds are susceptible to pesticide exposure, species-specific natural history, behavior, and physiology affect the likelihood of exposure and the lethal dose threshold. In general, altricial and gregarious species (e.g., Common Grackle, American Crow, and American Robin and other turdids) are at increased risk for OP toxicosis (Glaser 2001), consistent with results from the present study, in which more than half of the individuals affected by OPs were icterids (primarily the Common Grackle). This observation coincides with previous reports in which seedeaters, jays, crows, and blackbirds accounted for more than 45% of individual birds diagnosed with toxicoses in general (Augspurger et al. 1996; Glaser 2001). In OP toxicosis, death is typically due to pesticide-induced anorexia (Grue 1982) or paralysis, and subsequent respiratory failure from the inhibition of acetylcholinesterase activity, which can be accelerated with elevated ambient temperatures, decreased availability of resources, and physiologic stressors (Glaser 2001). Accidental (e.g., insecticides) and intentional (Avitrol® or 4-aminopyridine [Avitrol Corporation, Tulsa, Oklahoma, USA] and Starlicide® or 3-chloro-*p*-toluidine hydrochloride [Ralston-Purina, St. Louis, Missouri, USA]) poisoning of wild birds and other vertebrates with insecticides can result in localized die-offs. However, our results suggest that in some regions, exposure to agricultural pesticides can account for much higher numbers of mortalities than these intentional or avian-targeted compounds. Most OPs are commonly used for pest control in agricultural, residential, recreational, and livestock settings (Glaser 2001). Many pesticides have nontarget consequences, and previous studies in wild birds have determined high lethality rates with exposure to certain CBs (carbofuran) and OPs (famphur and diazinon) (Augspurger et al. 1996; Fleischi et al. 2004). In this study, avicides (Avitrol and Starlicide) only affected target species (i.e., Common Grackle, Red-winged Blackbird, and European Starling).

Cyanide led to 33 mortality cases in our study and mostly involved passerines such as warblers, sparrows, and thrushes; this case number was consistent with that of a past study (Friend and Franson 2001). One mortality event involved carcasses of several species recovered from a tailing impoundment contaminated with waste from a gold mining operation; such operations are known to use cyanide in gold extraction from low-grade ore (Gönen et al. 2004).

Lead poisoning has been documented in North American waterfowl since 1874, with estimated annual losses of up to 2.4 million birds (Friend and Franson 2001). Although the use of lead shot for hunting waterfowl was banned in the US in 1991, lead-related mortalities have continued among wild birds (Friend et al. 2009). Lead toxicosis can occur in nontarget species (e.g., raptors, scavenging birds, and diving waterfowl) after consumption of spent lead shot, projectiles, and fishing sinkers (Stauber et al. 2010). However, lead

toxicosis has also been documented in songbirds (e.g., Parulidae) in periurban areas (Martins et al. 2010) and can occur via ingestion of contaminated invertebrates (e.g., earthworms) (Nelson-Beyer et al. 2013). In our study, lead toxicosis was diagnosed in Yellow-rumped Warblers, a Blue Jay, and a Solitary Vireo collected from a law-enforcement training center (Lewis et al. 2001). Although estimating the potential population-level effects for the target species in this study is not feasible, it is important to continue to recognize lead as a potential source of mortality in all wild vertebrates.

Strychnine is used in agriculture as a rodenticide and repellent that can cause muscle tetany, seizures, and convulsions in birds or mammals within hours of consumption (Wobeser and Blakley 1987; Friend and Franson 2001). It is usually available as an odorless, white crystalline powder applied to milo, a form that can be fatal when ingested by granivorous birds (Hegdal and Gatz 1976). Icterids were the most common species diagnosed with strychnine toxicosis in our study, mostly during late fall. This may be related icterids often being the numerically dominant family foraging in agricultural fields, where they tend to feed in large groups (Glaser 2001). Although dieldrin (a chlorinated hydrocarbon insecticide) and DDT are banned in the US (Environmental Protection Agency [EPA] 2014a), some birds had DDT residues in tissues. DDT has been linked to reproductive disorders in other avian taxa, especially in tertiary consumers such as Osprey (due to bioaccumulation and biomagnification; Lundholm 1997). Studies evaluating the effects in other avian taxa should be considered due to the long-term availability of these compounds in the environment.

Reports of ethanol intoxication in wild birds often involve Cedar Waxwings in the US, and Common Blackbirds (*Turdus merula*) and Redwings (*Turdus iliacus*) in the UK (Fitzgerald et al. 1990; Duff et al. 2012). In our study, the majority of diagnoses of ethanol toxicoses involved Cedar Waxwings submitted during late winter and early spring. Ethanol derives from fermented fruits and sap from plants; the timing coincides when nomadic flocks eat recently thawed, overwintered berries in which yeast ferments glucose into pyruvate and subsequently into ethanol (Fitzgerald et al. 1990). Diagnosing ethanol toxicoses is challenging due to lack of a specific diagnostic test and a poor understanding of ethanol metabolism in wild birds. A presumed diagnosis is made based on history, signalment, evidence of acute trauma, and ruling out additional potential predisposing or primary causes of death. Although ethanol poisoning can be a primary cause of death, trauma is often the proximate cause due to collisions with buildings or other objects after sublethal intoxication (Fitzgerald et al. 1990). Cedar Waxwings may be more susceptible because they are frugivorous and tend to overfeed when foraging in flocks. Indeed, these birds were often diagnosed as small groups of individuals found within the same area.

Future amendments or revisions to the application of agricultural crop products should address any wild bird mortalities resulting from the consumption of the toxic compounds mentioned above. Revision of the current policies regulating the marketing of avicides could aid in reducing their impacts on nontarget wild bird species.

Trauma

Trauma has been considered an important cause of death in birds for decades (Austin 1931). Trauma accounted for mortality in about 20% of all cases in our study, and it affected all age classes and sexes similarly. The leading traumatic cause of death was in-flight collisions (e.g., windows or motor vehicles; see Supplementary Material Fig. S1a), which most often involved short- (e.g., Cedar Waxwing) and long-distance (e.g., Mourning Warbler) migrants. These mortalities are a continuous conservation concern for migrating birds (Gottdenker et al. 2008; Longcore et al. 2013), and numerous additional reports have documented extensive passerine mortalities associated with artificial structures (Erikson et al. 2005). To the extent possible, flight patterns and habitat use should be considered

when routing power lines, roads, airstrips, wind turbines, and other structures. Adverse weather, artificial lighting, and tower height are also important contributors to avian traumatic collisions (Longcore et al. 2013). Predation, often evidenced by skin punctures, lacerations, and hemorrhages, accounted for 1% of trauma diagnoses. This percentage is likely an underestimate of deaths due to predation, as predators commonly leave little evidence of their prey. Shooting of nongame bird species became illegal in the US with the enactment of the Migratory Bird Act Treaty of 1918. Illegal shooting of wild birds accounted for nearly 7% of traumatic deaths in this study, affecting corvids, fringillids, icterids, turdids, and picids. Half of these cases were American Crows, for which hunting is seasonally regulated in most states; however, these crows were shot during closed seasons. The remaining cases were in common songbird species. Depredation permits are available for some songbird species (Banks 1979); however, there was no indication that these deaths were legally permitted.

Physiologic stress as a cause of mortality

The most common factors associated with the diagnosis of physiologic stress were postmigratory and weather related. Avian migratory routes often span habitat that is unsuitable for feeding and cover, thereby requiring adequate fat stores to provide energy for long-distance flights to breeding or wintering grounds (Klaasen 1996). Shortly after migration, birds with depleted fat stores are more vulnerable to environmental stresses. Physiologic stress is still loosely defined and more often considered a diagnosis of exclusion, made based on signalment, history, and an absence of other etiologies that might otherwise explain emaciation (see Supplementary Material Fig. S1b) or poor condition. Physiologic stressors may be seen in chicks due to nest abandonment, populations from poor-quality habitats with inadequate resources, after recent or ongoing migratory activity, and from extreme temperatures or other adverse weather.

Bacterial diseases

Avian salmonellosis was the major mortality factor for fringillids (some with *Trichomonas* sp. coinfections; see Supplementary Material Fig. S1c); cardinalids and passerids; and to a lesser extent, icterids. The prevalence of *Salmonella enterica* subsp. *enterica* serovar Typhimurium in this study was 11% (nearly 200 birds), which is relatively high compared to previous studies in free-ranging birds (5.4%) (Hall and Saito 2008). Although all avian species are likely susceptible to salmonellosis, it was exclusively diagnosed in passerines in our study (see Supplementary Material Fig. S1c). Salmonellosis is most commonly diagnosed in passerines (Pennycott et al. 1998; Hall and Saito 2008; Hernandez et al. 2012) and generally involves species that congregate at bird feeders where the pathogen is spread via the fecal-oral route (Friend and Franson 2001). Because birds may be asymptomatic carriers, salmonellosis is best diagnosed based on epidemiologic patterns in conjunction with clinical signs and postmortem examination (Kirkwood 2008). Consistent with our study, the southeastern US has experienced a recent increase in numbers of avian salmonellosis cases (Hall and Saito 2008). This disease is an important threat to the health of wild birds and has led to adverse population-level effects in some species in the UK (Lawson et al. 2014). Avian salmonellosis has been documented throughout the year (Hall and Saito 2008); however, in this and previous studies, the timing of outbreaks was often associated with the seasonal use of bird feeders (i.e., January–April; Hernandez et al. 2012).

Numerous *Mycoplasma* spp. can cause disease in birds; however, *Mycoplasma gallisepticum* has likely had the greatest impact on wild bird populations in the US (Ley et al. 1998), primarily affecting members of the Fringillidae (Mikaelian et al. 2001). Mycoplasmal conjunctivitis emerged as an important disease in eastern House Finches in 1994, initially affecting birds in the mid-Atlantic US, but subsequently expanded its range and caused seasonal epizootics and population

declines in eastern House Finches (Altizer et al. 2004; Ley et al. 2010). Mycoplasmal conjunctivitis was one of the most common bacterial infections in our study. We observed a gradual decline in mycoplasmal conjunctivitis diagnoses since 1994. This could suggest flock immunity (Bonneaud et al. 2012), decreased virulence (Grodio et al. 2012), dampened transmission due to a decline in finch population numbers (Hochachka and Dhondt 2000), or reporter fatigue. Similar to *Salmonella* spp. transmission, aggregation of birds at feeders likely facilitates transmission of *Mycoplasma* spp. through increased contact with infected hosts or substrates (Friend and Franson 2001), which is consistent with the high case numbers detected during colder months. Apodids have also been diagnosed with *M. gallisepticum*–induced sinusitis and tracheitis (Murakami et al. 2002). A European Starling was infected with *Mycoplasma sturni*, a pathogen that was previously reported in corvids and mimids with conjunctivitis (Ley et al. 1998) and in asymptomatic corvids and turdids (Wellehan et al. 2001).

Infections with bacteria other than those mentioned above were relatively rare and included *Clostridium perfringens*, *Escherichia coli*, *H. alvei*, and *Staphylococcus aureus*. *Clostridium perfringens* was isolated from four corvids, although enterotoxemia was not confirmed. *Clostridium perfringens* is typically linked to die-offs in waterfowl, domestic ducks, and poultry (Friend and Franson 2001). Dietary changes can lead to disruption of commensal intestinal microflora, thereby allowing *C. perfringens* to thrive and produce an enterotoxin (Friend and Franson 2001). In our study, *C. perfringens*–positive corvids were collected in January and therefore may have experienced a recent seasonal dietary change due to seasonal decreases in available resources. Two of these corvids were diagnosed with a reovirus infection and were concurrently culture positive for intestinal *C. perfringens* and *E. coli*. Collibacilosis (*E. coli* infection) was diagnosed in a small number of fringillids, corvids, and mimids, usually between April and September. One House Finch (fringillid) had ulcerative dermatitis associated with *S. aureus* infection. *Hafnia alvei* is an enterobacterium that is commonly isolated from environmental sources (e.g., water and soil) (Janda and Abbott 1998). Information regarding *H. alvei* infection in birds is limited to fecal prevalence studies and detections in subclinically infected poultry (Rogers 2006). In our study, six Common Grackles (icterids) collected during winter in South Carolina died from infection with *H. alvei*, which has similar pathogenesis in birds as *E. coli* (Albert et al. 1992). To our knowledge, this is the first report of mortality caused by *H. alvei* in wild birds.

Viral diseases

About one-third of all viral infections, mainly involving avian poxvirus (genus *Avipoxvirus*; see Supplementary Material Fig. S1d) and WNV, were diagnosed in corvids. However, avian pox was also diagnosed in passerids and mimids. Avian poxvirus infection has been documented in at least 278 bird species of 70 families and 20 orders, and it has caused epornitics in passerines (primarily House Sparrows) at bird feeders (McFerran et al. 1976; Van Riper and Forrester 2007). Poxvirus infections in fringillids, mimids, passerids, and emberizids were infrequently diagnosed in our study. Although infections may occur year-round (Friend and Franson 2001), most birds diagnosed with poxvirus infection in our study were collected during summer. Environmental conditions (e.g., humidity and temperature) and mosquito activity may prolong virus survival in the environment and contribute to increased mechanical transmission (Friend and Franson 2001).

West Nile virus has caused significant mortality in numerous North American bird species (LaDeau et al. 2007). Birds are the primary amplifying host of this mosquito-borne virus, and although most avian infections are likely subclinical, corvids and some other birds are highly susceptible to fatal infections (Komar et al. 2003). In our study, corvids were diagnosed with WNV and poxvirus at an approximately equal frequency, although viral infections did not account for

the majority of mortalities in corvids (11%). West Nile virus was also diagnosed in birds in the families Fringillidae, Passeridae, Mimidae, and Emberizidae. In some areas of the US, American Crow populations have experienced significant declines due to WNV (LaDeau et al. 2007), raising concern about endangered corvids, such as the Hawaiian Crow (*Corvus hawaiiensis*) and the Florida Scrub-jay (*Aphelocoma coerulescens*).

A small number of house finches were positive for eastern equine encephalitis virus (EEEV; family *Togaviridae*) in our study. Songbirds in eastern North America have a relatively high EEEV antibody prevalence, indicating exposure, compared to other bird groups. These birds are also thought to play a major role in enzootic transmission cycles; however, most infections are asymptomatic (Friend and Franson 2001).

Reovirus (family *Reoviridae*) prevalence was low in our study and was detected as a primary pathogen in a small number of corvids coinfected with *C. perfringens*. Reovirus-associated clinical disease has been described for psittacines, ducks, and pigeons (McFerran et al. 1976; Graham 1987). In Belgium and Finland, a reovirus-like virus was isolated during an epornitic involving Hooded Crows (*Corvus corone*; Mast et al. 2006; Huhtamo et al. 2007). In North America, reoviral die-offs in woodcocks were first described in the 1980s and were frequently associated with enteritis and concurrent bacterial infections (Friend and Franson 2001).

The most common fungi detected in the present study were *Aspergillus* spp., which can lead to respiratory infections, especially in immunosuppressed hosts or those exposed to overwhelming environmental loads of *Aspergillus* spp. (Beernaert et al. 2010). Although aspergillosis epornitics are typically documented in the fall and early winter, when moldy postharvest agricultural products are accessible to birds (Friend and Franson 2001), in our study, aspergillosis diagnoses were significantly correlated with birds collected during summer. However, these results must be interpreted with caution, as aspergillosis is often an underlying or subclinical disease that manifests when precipitated by concurrent stress, trauma, or underlying infections. Although coinfections were not detected in birds with aspergillosis in this study, it is important to rule out underlying infections and other potential causes of immunosuppression in these birds.

Parasitism

Protozoal infections were relatively rarely diagnosed in our study, and included *Plasmodium* spp. and coccidia in birds in the family Passeridae, and *Toxoplasma gondii*–associated encephalitis in birds in the Picidae, Icteridae, and Turdidae. Consistent with these findings, wild birds are rarely documented with clinical or subclinical *T. gondii* infection (Dubey 2002), although grackles in Mexico and turdids in Europe have succumbed to fatal toxoplasmosis (Alvarado-Esquivel et al. 2011; Literak et al. 2011). Details of the diagnostic findings for the *T. gondii*–infected Red-bellied Woodpecker in our data set were described previously (Gerhold and Yabsley 2007). Felids are the natural host of *T. gondii*, and isolates from pigeons in Mexico were genetically identical to those of domestic cats (*Felis catus*; Alvarado-Esquivel et al. 2011), suggesting that a shared source of *T. gondii* can cause disease in vulnerable species (Friend and Franson 2001). In this study, avian malaria and coccidiosis diagnoses were limited to House Sparrows. Coccidial infections are rarely reported in wild birds with the exception of cranes and waterfowl, which may develop fatal disseminated infections (Friend and Franson 2001).

Acanthocephalan infections resulted in perforation of the intestine and subsequent coelomitis in two Red-bellied Woodpeckers. One of these infections was attributed to *Mediorhynchus centurorum*, and this bird was coinfected with *Sarcocystis* spp. In the US, picids infected with *M. centurorum* were reported in Louisiana (Nickol 1969) and Florida (Foster et al. 2002). *Mediorhynchus* spp. are the only acanthocephala known to regularly parasitize North American woodpeckers (Nickol 1969), and they can also infest

other bird species, including phasianids and icterids (Marchand and Vassiliades 1982; Richardson and Nickol 2008). The apparent host specificity for woodpeckers by *M. centurorum* may be associated with an insectivorous diet, as the intermediate host for *M. centurorum* is the woodroach (*Parcoblatta pennsylvanica*; Jackson and Nickol 1979). In contrast, icterids were not commonly diagnosed with parasitic infections; two grackles had severe acariasis (*Harpirrhynchus quasimodo*) and another killed by gunshot had a cloacal granuloma induced by the digenetic nematode *Collyriculum faba*. The latter was considered an incidental finding, and infestations by this skin fluke are typically regarded as nonpathogenic in wild birds (Farner and Morgan 1944; Heneberg et al. 2001; Literak et al. 2011). In our study fringillids were coinfected with *Trichomonas* spp. and *Salmonella* spp. Although trichomoniasis outbreaks have been more common in columbids (Sansano-Maestre et al. 2009), the potential adverse effects of this protozoan, in combination with other pathogens, should be considered for other avian taxa.

The most common ectoparasite-associated disease in our study was tick paralysis. Passerines can die from a tick-induced polyneuropathy and paralysis caused by a neurotoxin secreted by the female hard-bodied tick *Ixodes brunneus* while feeding (Friend and Franson 2001). Female *I. brunneus* feed more commonly on passerines that forage in the forest understory, and fringillids were most commonly affected by tick paralysis in this and past studies (Luttrell et al. 1996). In this study, heavy infestations of *Knemidocoptes* spp. and *H. quasimodo* mites were reported in an American Robin and Brown-headed Cowbird, respectively. Infestations by *H. quasimodo* in cowbirds have only recently been reported in the southeastern US (Spalding et al. 2010; Magenwirth et al. 2013). *Knemidocoptes* spp. caused epornitics in the American Robin in the US in the 1990s (Pence et al. 1999) and has also caused mortality in the Evening Grosbeak, House Sparrow, and Cassin's Finch (Carothers et al. 1974).

Seasonal and temporal trends

Avian samples derived from mortality events were submitted more frequently to SCWDS during late fall and early spring than during late spring and summer; however, all seasons had significant associations with different mortality causes. During fall, birds were more frequently diagnosed with endoparasitic infections and trauma. Endoparasitic infections comprised micro-and macroparasites, such as protozoa (primarily *Trichomonas* spp.) and platyhelminths. Summer also was positively correlated with the prevalence of *Aspergillus* infection. Although in general passerines are infrequently diagnosed with *Trichomonas* sp., *T. gallinae* infection is occasionally diagnosed in passerines in late spring, summer, and fall (Friend and Franson 2001), which agrees with our findings.

Ectoparasite infections were more commonly diagnosed in birds collected during fall and winter, consistent with past studies of wild birds in the southeastern US (Luttrell et al. 1996). Trauma diagnoses were made in birds submitted to SCWDS in every month throughout the 36-yr study. However, nearly 20% of mortality cases occurred in October, coinciding with annual fall migration, and they were most commonly attributed to in-flight collisions. The number of case submissions attributed to trauma was significantly lower during summer. One possible explanation is that adult movements may be limited during this time due to breeding, increased local food availability, and rearing of offspring. Seasonal avian mortality due to trauma may be especially common among Neotropical migrants during fall migration (Longcore et al. 2013). Our results are consistent with this phenomenon, with the majority of trauma diagnoses in Neotropical migrants of the family Parulidae during the fall migration across the Atlantic flyway.

Season and toxicosis were strongly correlated. Most submissions diagnosed with toxicosis were submitted to SCWDS from January to March, with infrequent diagnoses during summer. Seasonal detections of toxicosis in birds are often related to pesticide application

to slow-growing crops such as wheat and rye in the southeastern US, which starts early in the year (winter) when food is relatively scarce (US Department of Agriculture 1997).

There has been a general decline in the number SCWDS avian case-submissions attributed to toxicosis since the mid-1990s. This trend may, in part, reflect the enactment of the FQPA in 1996, in which the EPA implemented a ban on ~60 pesticides (EPA 2014b). Detections of trauma and infectious diseases in birds show the opposite trend; more cases were diagnosed at SCWDS since 2000. This apparent increase in the numbers of trauma cases may be related to landscape alterations within migratory flyways, including construction of buildings, power towers, and wind turbines. In addition, birds dying of trauma associated with artificial structures may be increasingly visible to the public who may, in turn, be more aware and motivated to report avian mortalities due to recent zoonotic disease surveillance (e.g., WNV and avian influenza). Lastly, increased popularity of bird watching activities and backyard feeding has likely increased the visibility of avian diseases, for which there are increasingly available and improved diagnostic methods.

Demographic factors and causes of mortality

In general, mortality due to toxic compounds was significantly more frequently diagnosed in adults, whereas juvenile birds died more often from physiologic stress, trauma, and viral infections. The former likely represented acute deaths after exposure to contaminated feed, since the majority of birds died from toxicosis during winter, and the OPs and CBs do not bioaccumulate in tissues. Age-association with mortality from pesticide exposure seems to be specific to the compound in question. For example, in cases of bioaccumulation (e.g., when pesticides do not degrade rapidly, such as organochlorides), adults are sublethally affected by impairment of hatching success (Fry 1995; Blus and Henny 1997), especially in raptors. In other taxa, such as galliforms, OP application to croplands has been linked to increased mortality in juveniles, as they are forced to disperse to find arthropods, whereas the adult diet is primarily vegetation based (Blus and Henny 1997). The majority of deaths attributed to physiologic stress were postmigratory or weather related. Mortalities associated with migration have been correlated with in-flight losses (e.g., storms) and unseasonably cold temperatures (Newton 2007). Nestlings lack adequate feathers and adipose tissue for insulation and therefore are more susceptible to cold and blood- or skin-feeding insects, while also undergoing physiologic demands of rapid growth and development (Cluttonbrock et al. 1985). Juveniles are at greater risk of dying of physiologic stress due to food constraints, nest abandonment, or exposure compared to adults, and they may be more susceptible to mortality due to some viruses (Nemeth and Bowen 2007).

Secondary bacterial infections

Twenty-eight birds belonging to families Icteridae (n=20), Fringillidae (n=6), and Hirundinidae and Sturnidae (n=1 each) had secondary bacterial infections, which were not considered the primary cause of death. In these infections, *Staphylococcus* spp. was the most commonly isolated bacteria from the liver and ocular swabs; in the latter case, it was considered an opportunistic pathogen in birds with conjunctivitis. *Escherichia coli* (n=7) was isolated from livers and other tissues of Emberizidae, Hirundinidae, Sturnidae, Turdidae, Mimidae, Fringillidae, and Corvidae and was occasionally isolated along with other bacteria (n=4). *Enterococcus* spp. were exclusively isolated from the intestine of icterids (n=3). *Enterobacter* spp. were isolated from the throat of a Caprimulgidae and from the liver of one Bombycillidae (n=1 each). *Pseudomonas* spp. (including *P. fluorescens*) were isolated from the intestine and liver of Fringillidae and Turdidae (n=1 each).

Incidental parasitologic findings

Four parasites comprised the majority of incidental (not contributing to mortality) parasitologic observations in birds; these

parasites included the protozoa *Sarcocystis* spp.; the filarial nematode *Chandlerella quiscali*; and nematodes *Tetrameres* spp. and *Diplotriaena* spp. Icteridae was the family most commonly infected with these parasites. *Tetrameres* spp. are most often within the proventriculus of birds (Mollhagen 1976) and occasionally within the adjacent esophagus and ventriculus. *Diplotriaena* spp. were identified in air sacs and within the coelomic cavity. *Diplotriaena tricuspis* also infected birds within the family Icteridae, although it was formerly thought to be specific to corvids (Cawthorn and Anderson 1980). *Chandlerella quiscali* can parasitize up to 98% of grackle populations in some areas, and it has also infected other bird species (Granath 1980). *Knemidocoptes* spp. mites were the most commonly observed ectoparasite, most often as incidental findings in the American Robin (see Supplementary Material Table S3). However, severe infestations in the hind limbs were occasionally associated with fatal secondary bacterial infections. Secondary bacterial infections and incidental parasitism is common in wildlife, and parasites are often a normal component of a host's microfauna. The significance of parasites and secondary bacterial infections is judged by first demonstrating the host response to these infections and then weighing it against the overall clinical picture and pathology noted.

Avian mortality comparisons between wildlife diagnostic centers

To determine the broad applicability of the data collected in this study, it is useful to compare our results to the US national database. The US National Wildlife Health Center (NWHC) in Madison, Wisconsin, identifies pathogens and other causes of mortality in wildlife on a national scale, and these data are publicly available. Data from SCWDS are also included in the NWHC database (NWHC 2013). The same broad categories of mortality were important (i.e., infectious, trauma, and toxicities) in both data sets; however, there were variations in the extent to which avian groups were affected by each mortality cause. For example, most deaths at the NWHC were attributed to (in decreasing order of magnitude) infectious disease, toxicosis, and trauma. Both data sets reveal potentially significant negative impacts of infectious diseases (dominated by salmonellosis and WNV), trauma (primarily due to in-flight collisions with artificial structures), and toxicosis (mostly due to OPs, CBs, and Avitrol) on wild bird populations in the US.

Despite the inherent limitations of interpreting data from case submissions to a diagnostic laboratory, data generated from consistent efforts to document mortality causes in wildlife help establish baseline levels of infectious etiologies; offer opportunities to detect novel pathogens and parasites; and provide insights into multifactorial causes of morbidity and mortality in wild animal populations, including trends over time and space. Long-term data sets that encompass a large region are exceedingly rare, but our results illustrate their importance. Trends in avian mortality in our study mirrored the national database and collectively reinforce the urgent need to address current and ongoing anthropogenic impacts on wild birds. Such findings should guide future research, management options, and policies aimed at reducing these impacts.

ACKNOWLEDGMENTS

We thank the many biologists, technicians, the public, and especially SCWDS member states that participated in the collection and submission of the birds in this study. This work was possible due to the expertise of SCWDS diagnostic veterinarians. Special thanks to Cindy O. McElwee and Miryam Astudillo-Hernandez for valuable help in report collection.

SUPPLEMENTARY MATERIAL

Supplementary material for this article is online at http://dx.doi.org/10.7589/2015-05-117.

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Submitted for publication 10 May 2015.
Accepted 19 October 2015.