The Journal of Wildlife Management 84(3):553–561; 2020; DOI: 10.1002/jwmg.21822

Research Article

Sources of Mortality in Bald Eagles in Michigan, 1986–2017

KENDALL L. SIMON,[1] *U.S. Fish and Wildlife Service, Ecological Services Field Office, 4 East Jimmie Leeds Road, Suite 4, Galloway, NJ 08205, USA*

DAVID A. BEST, *U.S. Fish and Wildlife Service—retired, Ecological Services Field Office, 2651 Coolidge Road, Suite 101, East Lansing, MI 48823, USA*

JAMES G. SIKARSKIE, *Department of Small Animal Clinical Sciences—retired, College of Veterinary Medicine, Michigan State University, 736 Wilson Road, East Lansing, MI 48824, USA*

H. TYLER PITTMAN, *University of Florida/Institute of Food and Agricultural Sciences Marion County Extension, 2232 NE Jacksonville Road, Ocala, FL 34470, USA*

WILLIAM W. BOWERMAN, *University of Maryland, Department of Environmental Science and Technology, 1426 Animal Sciences Building, College Park, MD 20742, USA*

THOMAS M. COOLEY, *Michigan Department of Natural Resources, Wildlife Disease Laboratory, 4125 Beaumont Rd. Room 250, Lansing, MI 48910-8106, USA*

SCOTT STOLZ, *Ramboll, 2260 E. Saginaw Street, East Lansing, MI 48823, USA*

ABSTRACT As bald eagle populations recover, defining major sources of mortality provides managers important information to develop management plans and mitigation efforts. We obtained data from necropsies on 1,490 dead bald eagles (*Haliaeetus leucocephalus*) collected in Michigan, USA, conducted from 1986 to 2017 to determine causes of death (COD). Trauma and poisoning were the most common primary COD categories, followed by disease. Within trauma and poisoning, vehicular trauma ($n = 532$) and lead poisoning ($n = 176$) were the leading COD subcategories, respectively. Females comprised a greater number of carcasses for most COD diagnoses. The proportion of trauma and poisoning CODs significantly increased in the last few years of the study in comparison to a select few years at the beginning. Trauma CODs were greater in autumn months during whitetail deer (*Odocoileus virginianus*) breeding and hunting seasons and in February, when aquatic foraging is unavailable and eagles are likely forced to scavenge along roadsides. Poisoning CODs were greatest in late winter and early spring months, when deer carcasses containing lead ammunition, which are preserved by the cold weather, also become a supplemental food source. The major infectious disease CODs, West Nile virus and botulism (*Clostridium botulinum* type E), were more prevalent during summer months. We recommend moving road-killed carcasses, especially white-tailed deer, from the main thoroughfare to the back of the right-of-way, and the transition from lead ammunition and fishing tackle to non-toxic alternatives to decrease these main anthropogenic sources of mortality for bald eagles, and other scavenger species.

KEY WORDS bald eagle, *Haliaeetus leucocephalus*, lead poisoning, Michigan, mortality, trauma.

The bald eagle (*Haliaeetus leucocephalus*) is a large fish-eating bird of prey with an extensive breeding and wintering range located mostly in the contiguous United States, southern Canada, and coastal areas of northern Canada and Alaska, USA (Buehler 2000). Populations of bald eagles declined in the mid-twentieth century mostly because of human persecution and the release of organochlorine pesticides and polychlorinated biphenyls. After the Endangered Species Act of 1973, and the ban of numerous organochlorine compounds by the Environmental Protection Agency in the 1970s, bald eagle populations recovered to their historical levels in some areas (Buehler 2000). Because gunshot, trapping, and organochlorine pesticides have decreased as sources of bald eagle mortality, other anthropogenic sources (e.g., vehicular trauma) are now the greatest discernible threats to bald eagles (Harris and Sleeman 2007). Researchers summarizing the cause of death for 2,980 bald eagles between 1975 and 2013 reported that most bald eagles died because of poisoning (25.6%), followed by trauma (22.9%), and electrocution (12.5%). And of the eagles that died because of poisoning in that study, 63.5% of those cases were due to lead toxicosis (Russell and Franson 2014).

Lead toxicosis is a well-known source of anthropogenic mortality for bald eagles (Hunt et al. 2006, Stauber et al. 2010, Pagel et al. 2012, Kelly et al. 2014, Warner et al. 2014). Historically, lead toxicosis in bald eagles was a direct result of the ingestion of lead shot from dead or wounded waterfowl, and was a major factor leading to the ban of lead shot for waterfowl hunting in 1991 (Kendall et al. 1996, Friend et al. 2009). Recently, researchers have linked lead

Received: 31 May 2019; Accepted: 25 November 2019

---

[1] *E-mail: kendall_simon@fws.gov*

toxicosis in bald eagles to ingested lead fragments embedded in tissues or offal from lost or discarded upland and large game animals (Hunt et al. 2006, Pagel et al. 2012, Kelly et al. 2014, Warner et al. 2014). Amplifying this exposure source is the high incidence of lead bullet fissuring upon impact, resulting in fragments of irregular shapes and greater surface areas that dissolve more easily in stomach acids, increasing metal retention and ultimately the magnitude of exposure (Scheuhammer and Templeton 1998, Fisher et al. 2006, Hunt et al. 2009, Warner et al. 2014). In addition, the high density and small particle size of fragments increase the probability of ingestion by bald eagles and scavengers (Haig et al. 2014).

Although typically a fish-eating bird, the bald eagle is an adaptable forager and will hunt or scavenge a variety of other prey including mammals, birds, and reptiles (Buehler 2000). Predatory and scavenging birds, like bald eagles, often rely on roadside carrion as a main prey base, making them particularly vulnerable to trauma-based mortality caused by vehicular collision (Wood et al. 1990, Kelly et al. 2014, Russell and Franson 2014). A change in the causes of mortality has occurred through legislation protecting bald eagles from certain sources (Russell and Franson 2014). As bald eagle populations recover, estimates of increasing sources of mortality, such as trauma, or emerging sources such as disease, will become important population vital rates when trying to assess the turnover and the vulnerabilities of different age groups or sexes within populations (Newton 1979). Defining those changes in major sources of mortality provides managers important information to develop management plans and mitigation efforts.

Our goals of this study were to determine the major sources of mortality within the bald eagle population in Michigan, USA, and to identify any spatial and temporal trends in these sources of mortality, especially those that might be mitigated through management efforts. We investigated life-history traits, in relation to human-influenced factors, that predispose bald eagles to the major sources of mortality.

STUDY AREA

Data used for the analysis of this study was based on carcasses of bald eagles collected throughout the state of Michigan. Located in the upper midwestern portion of the United States, Michigan is surrounded by 4 of the 5 Great Lakes: Lake Michigan, Lake Superior, Lake Huron, and Lake Erie. Michigan's geomorphology is classified as Central Lowland plains, with elevations ranging from 175 m to 396 m in the Lower Peninsula and 176 m to 256 m in the Upper Peninsula. The Lower Peninsula of Michigan is characterized by low-relief, hilly landscapes that are a product of past glaciation. Large portions of the southern Lower Peninsula are farms and cropland, transitioning into forests of maple (*Acer* spp.)-beech (*Fagus* spp.)-birch (*Betula* spp.), aspen (*Populus* spp.)-birch, white (*Pinus strobus*)-red pine (*Pinus resinosa*), and oak (*Quercus* spp.)-hickory (*Carya* spp.) cover types in the northern Lower Peninsula. The Upper Peninsula of Michigan is characterized by a landscape of flat to gently rolling plains with exposed bedrock knobs. Land cover is predominantly forested, consisting of is aspen-birch, maple-beech-birch, white-red-jack (*Pinus banksiana*), and spruce (*Picea* spp.)-fir (*Abies* spp.). Michigan experiences a 4-season continental climatic regime, with lake-effect snow influence along the Great Lakes. Winters are moderately long with continual ground snow cover in the northern regions, and warm summers (McNab et al. 2007).

METHODS

The Michigan bald eagle population monitoring program (MBEPMP) began in cooperation with the Continental Bald Eagle Project of the National Audubon Society in 1961 (Postupalsky 1985). The program monitored and assessed bald eagle populations through annual aerial surveys, and documented the population recovery from a low of 52 occupied breeding areas in 1961 to approximately 835 in 2017. The United States Fish and Wildlife Service (USFWS), through the National Wildlife Health Center as part of the MBEPMP, attempted to determine the cause of death for all bald eagle carcasses collected across the continental United States (USFWS 1983). Upon cessation of this program in 1981, the Michigan Department of Natural Resources (MDNR), through its Wildlife Disease Laboratory, began conducting necropsies on all carcasses collected in Michigan. During this same period, MDNR in cooperation with the Veterinary Clinical Center at Michigan State University, also collected, treated, and if possible released grounded bald eagles.

The bald eagle recovery program, implemented by the MDNR, determined a cause of death (COD) for every eagle carcass collected in the state of Michigan from 1986 to 2017 using a generalized necropsy examination. The necropsy results determined a primary COD category and a COD subcategory for each eagle. We divided diagnoses into 8 primary COD categories: disease, electrocution, emaciation, gunshot or trap, other, poisoning, trauma, and no diagnoses. Causes of death designated as other included a variety of diagnosis but mainly drowning and deformity. Subcategories are reported for only 2 primary CODs, trauma and poisoning. The COD subcategories for trauma are airplane, predator aggression, fishing gear, golf ball, intraspecific aggression, powerline collision, tower collision, tree accident, undetermined, vehicular collision, and wind turbine collision. We diagnosed a COD as a tree accident when the eagle had been trapped in either a crack of a tree, or under a blown down nest, branch, or tree trunk. The COD subcategories for poisoning are lead toxicosis, barbiturates (pentobarbital and phenytoin), and organochlorines. We also tallied months in which eagles were submitted for necropsy for those cases where the sex of the bird could be determined.

We diagnosed trauma CODs based on the presence of bone fractures and hemorrhaging in eagles. Histories provided with the eagle also gave insight into the specific trauma event (e.g., found along roadside or under a powerline). We sampled and tested livers from every carcass for lead levels. We considered lead levels ≥5 ppm wet weight to be significant and indicative of lead poisoning. This value is conservative based on previous literature values reporting lead poisoning in eagles (Franson and Russell 2014, Warner et al. 2014). We tested carcasses collected in late summer or early fall for botulism (*Clostridium botulinum* type C and E) using a mouse bioassay described by Quortrup and Sudheimer (1943). Only carcasses that had been collected within a few weeks of the mortality event, and did not show signs of decomposition, were sampled for botulism to protect against false positive results due to postmortem production of toxins. We tested all eagles collected from landfills for pentobarbital and phenytoin poisoning, or when the histories provided suggested a poisoning event. We considered any detection of pentobarbital or phenytoin in an eagle to be considered the cause of death. When discussing study results, we considered the winter months to be December–February, the spring months to be March–May, the summer months to be June–August, and the fall months to be September–November.

Analyses

We performed generalized logit multinomial regressions to determine the proportion of eagles by month and sex for the 4 most prevalent COD categories (Table 1): disease, other, poisoning, and trauma. We summed proportions of the counts to 1 across the 4 general COD categories for each month and sex. We ran all analyses in WinBUGS (Lunn et al. 2000) and plotted results using the R package ggplot2 (Wickham 2016). We performed multinomial logistic regressions using counts of carcasses. We modeled multinomial responses (y_{isk}) in month (i), for each sex (s), and COD (k), where $L(p_{isk} \mid n_{is}, y_{isk}) = C\prod_{k=1}^{k} p_{isk}^{y_{isk}}$. In this case, L is the log likelihood, C is the multinomial coefficient, n_{is} is the total number of carcasses from month i, in sex s, and p_{isk} is the proportion of carcasses in COD k. The multinomial distribution function is formulated in WinBUGS as $y_{is,1:k} \sim \mathrm{dmulti}(p_{is,1:k}, n_{is})$, where $p_{isk} = \exp(n_{isk})/\Sigma \exp(n_{is,1:k})$ and n_{isk} is the log odds of being COD k, month i, and sex s for $k > 1$.

We also compared yearly variations of eagle carcasses submitted for each COD during the 32 years using a binomial random effects model for true mortality probabilities. The WinBUGS binomial distribution function was $r_i \sim \mathrm{dbin}(p_i, n_i)$; the proportion of carcasses in year i, for a specific COD r_i, is a binary response variable with a true mortality probability (p_i), in relation to the total mortality (n_i). Because mortality rates are not entirely independent for each year, we used a random effects model for true mortality probabilities, where we specified non-informative priors for the population mean (logit) probability of mortality (μ), and precision (τ).

Table 1. Causes of death for bald eagles collected in Michigan, USA, 1986–2017. Percentages indicate the percent of all mortalities attributed to the cause of death by sex and overall. Total counts include diagnoses where sex was not specified or could not be determined.

	Male		Female		Total	
Cause of death	Number	%	Number	%	Number	%
Disease	29	7.8	55	7.5	92	6.2
Electrocution	13	3.5	31	4.2	62	4.2
Emaciation	23	6.2	35	4.8	75	5.0
Gunshot or trap	13	3.5	32	4.3	65	4.4
Other	13	3.5	31	4.2	51	3.4
Poisoning	70	18.8	96	13.0	185	12.4
Trauma	199	53.5	435	59.0	770	51.7
No diagnosis	12	3.2	22	3.0	190	12.7
Total	372		737		1,490	

We also used a binomial random effects model to separately calculate probabilities of total, trauma, and poisoning true mortality in comparison to the total bald eagle population by each county in which carcasses had been reported to be collected. We averaged total, trauma, and poisoning mortalities using data from 2013–2017. We calculated the 5-year average total population for each county for the same years, with the following equation:

$$N_{5-yr\ Average\ Total\ Population} = \frac{N_{Average\ Occ.Terr\,(2013-2017)} \times 2}{p\,(age \geq 4)},$$

where $N_{Occ.Terr}$ is the number of occupied nesting territories for each county, and $p(age \geq 4)$ is the stable-age distribution proportion of the population ≥4 years old (USFWS 2016). We formulated the WinBUGS binomial distribution function as $r_i \sim \mathrm{dbin}(p_i, n_i)$; the proportion of carcasses in county i, for a specific COD r_i, is a binary response variable with a true 5-year average p_i, in relation to the 5-year average n_i. Because mortality rates are not entirely independent for each county, we again used a random effects model for true mortality probabilities. We then plotted true mortality probabilities and categorized them by quintiles for each county in ArcMap 10.6 (Esri, Redlands, CA, USA).

RESULTS

The bald eagle recovery program implemented by MDNR collected 1,490 bald eagles from 1986 to 2017. As expected in a recovering population, the number of eagle carcasses collected for necropsy increased proportionally with the number of occupied breeding areas (Fig. 1). The greatest number of eagles necropsied was in 2016, when 112 eagles were collected and evaluated for CODs. Females comprised the greatest number of carcasses in which sex could be determined ($n = 737$), almost double that of males ($n = 372$; Table 1). Trauma was the COD with the greatest number of definitive diagnoses (51.7%), followed by poisoning (12.4%), disease (6.2%), and emaciation (5.0%). Females again comprised the greatest number of trauma carcasses ($n = 435$), double that of males ($n = 199$; Table 2). Within the trauma category, vehicular collisions were the most common definitive COD subcategory (69%), followed by powerline collisions (5.5%), and tree accidents (2.2%).

Lead toxicosis was the most common COD subcategory diagnosis within the poisoning COD (95.1%), followed by barbiturates (4.3%) and organochlorines (0.5%; Table 3). The difference between the number of female carcasses ($n = 96$) in comparison to males ($n = 70$) diagnosed with a poisoning COD was less than the difference between females and males

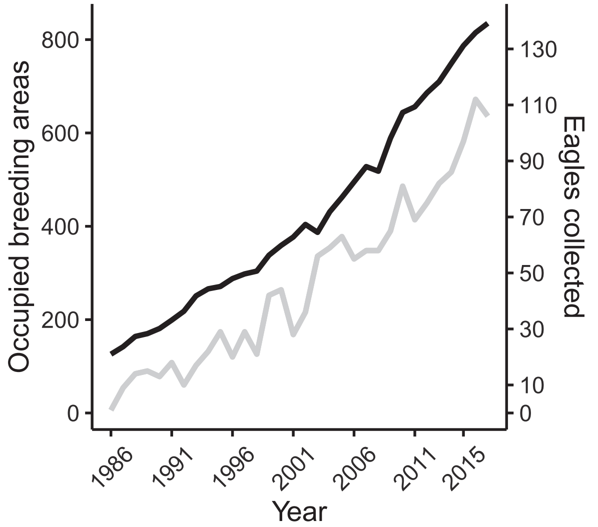

Figure 1. Number of occupied bald eagle breeding areas (in black), and eagle carcasses collected for necropsy (in gray) in Michigan, USA, 1986–2017.

for primary CODs and trauma CODs. Disease was the COD with the third greatest number of definitive diagnoses, with the 3 most common infectious diseases being West Nile virus (*n* = 17), aspergillosis (*n* = 8), and botulism (*n* = 8).

Sex and Month Proportions

The months with the greatest proportion of trauma CODs were February, September, and October for males and females. In contrast, April was the month with the least proportion of trauma CODs, with credible intervals (CrIs) that did not overlap with February, September, and October (Fig. 2). The proportion of bald eagles diagnosed with a poisoning COD increased during the winter months to peak in February, March, and April for both females and males. For poisoning CODs, the lower 95% CrIs for January through April, were greater than the upper 95% CrIs for June through October (Fig. 2). The proportion of bald eagles diagnosed with a disease COD was greatest for the summer months of June, July, and August for both males and females. For disease CODs, the lower 95% CrIs for summer months were greater than the upper 95% CrIs for the winter months of December, January, February, and March (Fig. 2). The monthly proportions in which bald eagles were diagnosed with all other CODs were similar, with the exception of February. The proportion of other CODs were least for both males and females in February, with upper 95% CrIs that were less than the lower 95% CrIs for all months, except March and November (Fig. 2).

Table 2. Summary of trauma subcategory causes of death for bald eagles collected in Michigan, USA, 1986–2017. Percentages indicate the percent of all mortalities attributed to the cause of death by sex and overall. Total counts include diagnoses where sex was not specified or could not be determined.

	Male		Female		Total	
Cause of death	Number	%	Number	%	Number	%
Airplane	0	0	4	0.9	5	0.7
Predator aggression	0	0	0	0	2	0.3
Fishing gear	0	0	1	0.2	1	0.1
Golf ball	0	0	1	0.2	1	0.1
Intraspecific aggression	8	4.0	6	1.4	14	1.8
Powerline collision	9	4.5	24	5.5	42	5.5
Tower collision	1	0.5	2	0.5	3	0.4
Tree accident	3	1.5	10	2.3	17	2.2
Undetermined	42	21.1	97	22.3	150	19.5
Vehicular collision	136	68.3	288	66.2	532	69
Wind turbine collision	0	0	2	0.5	3	0.4
Total	199		435		770	

Table 3. Summary of poisoning subcategory causes of death for bald eagles collected in Michigan, USA, 1986–2017. Percentages indicate the percent of all mortalities attributed to the cause of death by sex and overall. Total counts include diagnoses where sex was not specified or could not be determined.

	Male		Female		Total	
Cause of death	Number	%	Number	%	Number	%
Lead	68	97.1	90	93.8	176	95.1
Barbiturates	2	2.9	6	6.2	8	4.3
Organochlorines	0	0	0	0	1	0.5
Total	70		96		185	

Yearly True Mortality Probabilities

Yearly probability estimates of bald eagles diagnosed with trauma CODs exhibited a general increase from 1986 to 2017. The majority of yearly probability estimates for trauma were not significantly different throughout time, until the 2 most recent years in the time period evaluated. The lower 95% CrIs for the years of 2016 and 2017 did not overlap the upper 95% CrIs for the first 7 years of the time period, from 1986 to 1992 (Fig. 3).

Yearly probability estimates of bald eagles diagnosed with poisoning CODs were not significant, with nonoverlapping CrIs, with the exception of 2010 and 2015. Lower 95% CrIs for 2010 and 2015 did not overlap with the majority of upper 95% CrIs from 1986 to 2003 (Fig. 3).

Yearly probability estimates of bald eagles diagnosed with gunshot or trap CODs exhibited a general decrease from 1986 to 2017. The majority of probability estimates were not significantly different throughout time, until the 2 most recent years. The upper 95% CrIs for 2016 and 2017 were below the lower 95% CrIs for the majority of the first 13 years of the time period, from 1986 to 1998 (Fig. 3).

Similar to gunshot or trap CODs, probability estimates of bald eagles diagnosed with emaciation CODs also exhibited a general decrease over time. The majority of yearly probability estimates are not significantly different throughout time until the 3 most recent years. The upper 95% CrIs for the years of 2015, 2016, and 2017 were below the lower 95% CrIs for the majority of the first 14 years of the time period, 1986 to 1994 (Fig. 3).

The majority of yearly probability estimates of bald eagles with no COD diagnosis were not significantly different throughout time, although they generally trend downward (Fig. 3). The majority of yearly probability estimates of bald eagles with disease CODs were not significantly different throughout time, although they trend slightly upward

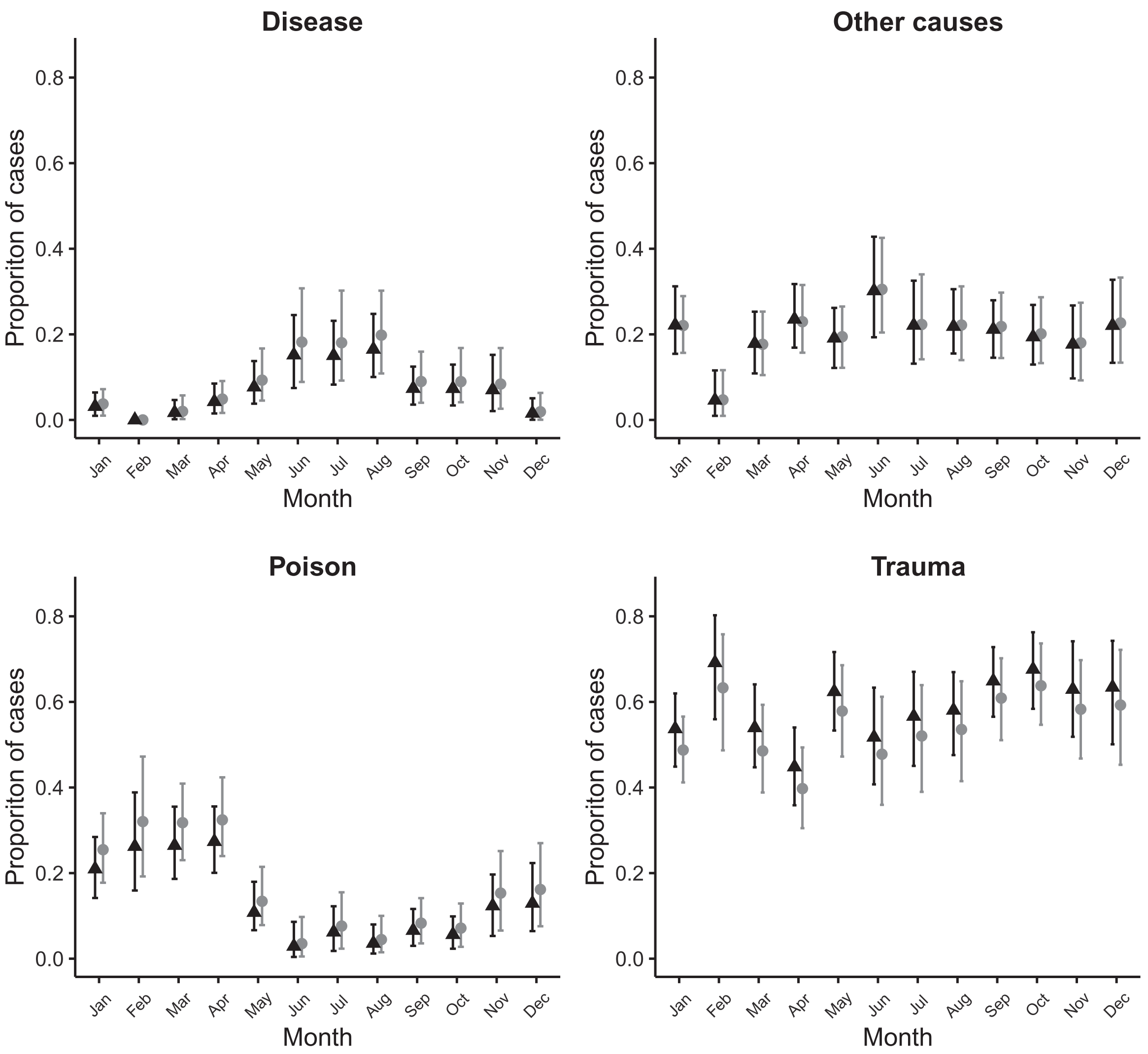

Figure 2. Proportion of female (black triangle) and male (grey circle) bald eagle carcasses with a primary cause of death diagnosed as disease, other causes, poisoning, or trauma collected in Michigan, USA, 1986 and 2017. We calculated proportions by month and sex. Bars represent 95% credible intervals.

(Fig. 3). Yearly probability estimates for electrocution and all other CODs were not significantly different throughout time, and did not indicate general increasing or decreasing trends.

The county analysis of true mortality indicated that the probability of a mortality event was generally greater in the Lower Peninsula for both total CODs (Fig. 4A) and trauma CODs (Fig. 4B), given the fewer number of breeding pairs in a given county. With the exception of a few counties, true population mortality for poisoning CODs was generally greater in the Northern Lower Peninsula and Upper Peninsula (Fig. 4C).

DISCUSSION

Bald eagles are highly dependent on non-aquatic food sources, such as carrion (Stauber et al. 2010, Warner et al. 2014). The growing population of bald eagles in Michigan has also led to rising numbers of eagles feeding on carrion, and the subsequently proportional increase in trauma CODs. This is likely due to the high availability of deer carcasses and carrion along roadways following deer-vehicle collisions (Sudharsan et al. 2006). We observed this functional response spatially; the probability of true mortality, in relation to population by county, was greater in densely populated counties containing urban roads and municipalities (Fig. 4). We also observed this response temporally towards the end of the study period, in which the yearly probabilities of true mortality were significantly greater for 2016 and 2017 in comparison to the first 7 years (1986–1992). In Michigan, 50,949 deer-vehicle collisions were reported in 2017 alone, with the majority of these collisions occurring in October and November (Michigan Department of State Police 2017). The greater number of deer-vehicle collisions during these months is likely the result of increased deer movement related to breeding behaviors and fall hunting seasons (Etter et al. 2002, Sudharsan et al. 2006). Consequently, the greatest proportions of bald eagle CODs were attributed to vehicular trauma during fall.

The second highest incidence of vehicular collision mortality occurred in February. This may also be a functional response resulting from changes in the availability of aquatic

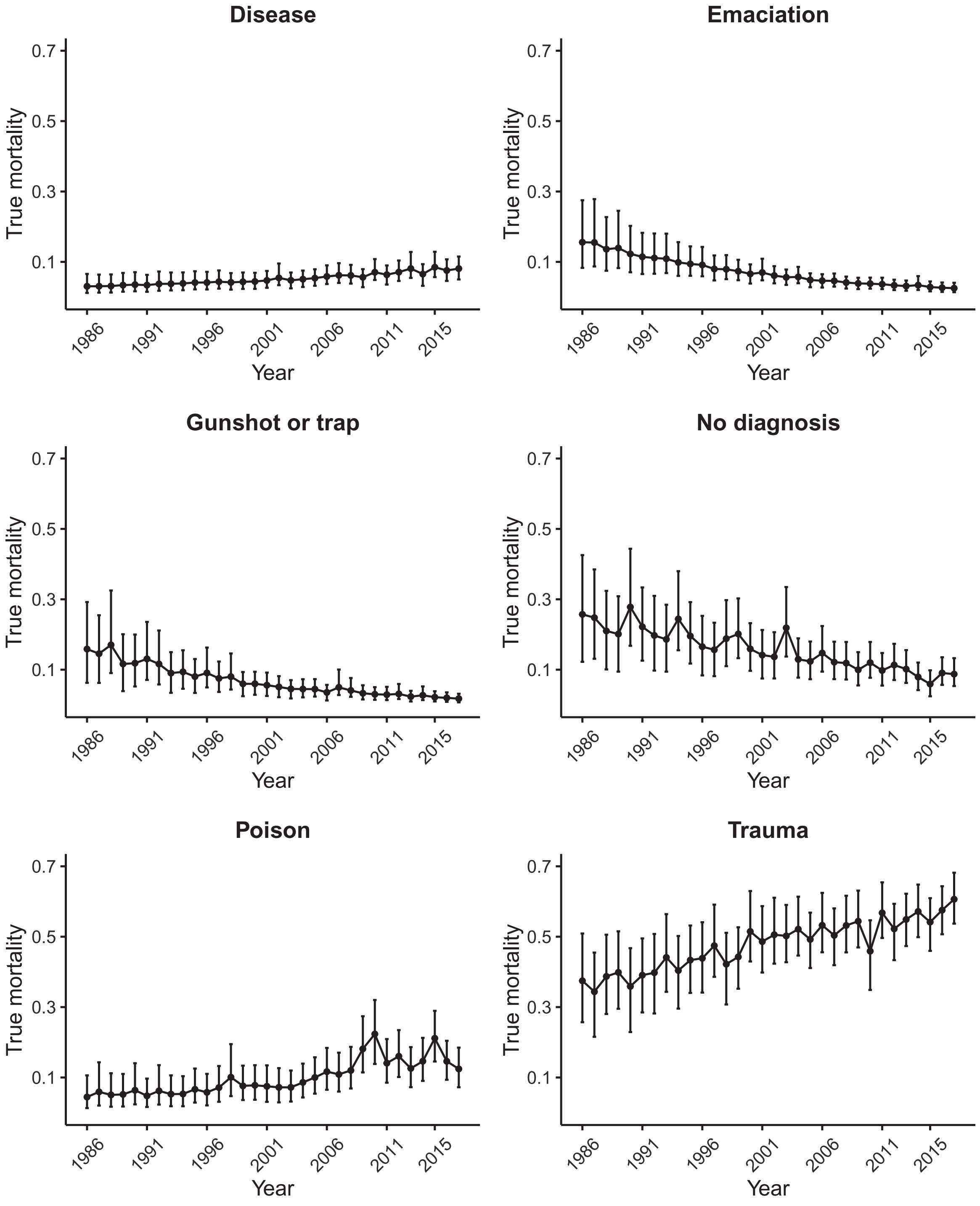

Figure 3. True mortality probabilities of bald eagle carcasses with a primary cause of death diagnosed as disease, emaciation, gunshot or trap, no diagnosis, poisoning, or trauma collected in Michigan, USA, between 1986 and 2017. We calculated probabilities by year. Bars represent 95% credible intervals.

food sources to terrestrial food sources during winter, when many lakes and rivers are frozen, as similarly proposed by Grubb and Lopez (2000), Stauber et al. (2010), Nadjafzadeh et al. (2013), and Warner et al. (2014). Females were responsible for the majority of vehicular collision mortality events (288) in comparison to males (136). We hypothesize that the greater body size of female eagles results in less maneuverability and a longer take off time in the event of oncoming traffic. The female majority in vehicular trauma CODs may also be due to dominant females out-competing smaller males for carrion when food sources are limited (Franson and Russell 2014). The high number of vehicular trauma mortalities may be exacerbated when roadside conditions prevent escape routes from oncoming traffic and limit flight paths to traffic lanes.

Poisoning was the second highest definitive cause of bald eagle mortality, with lead toxicosis being the greatest COD subcategory. Our results agree with previous studies

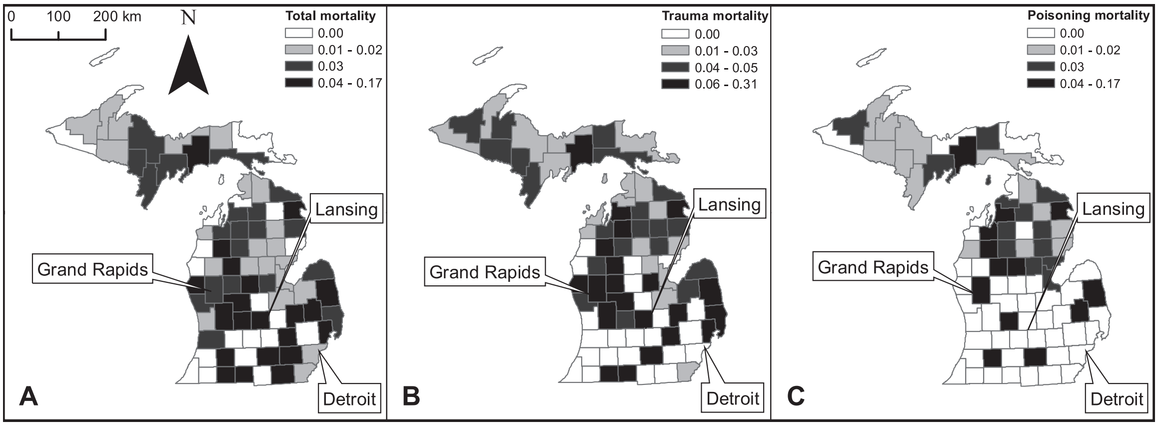

Figure 4. True mortality probabilities, in relation to bald eagle population, of bald eagle carcasses for all causes of death (A), and causes of death diagnosed as trauma (B) and poisoning (C) collected in Michigan, USA, 1986–2017. We calculated probabilities by county using a 5-year average (2013–2017) of mortality and population data.

concluding that the majority of lead-poisoned bald eagles were found between the months of January and April, when carcasses preserved by the cold weather, and containing lead ammunition, become a secondary or supplemental food source (Pattee et al. 1990). White-tailed eagles (*Haliaeetus albicilla*) in Germany shift to more terrestrial prey, especially lost or discarded upland game, in winter because frozen waterbodies limit aquatic food sources (Nadjafzadeh et al. 2013). We suspect this may be a reason for the greater observed frequency of lead poisoning before ice-out in March and April, when deer that were wounded during hunting season, and subsequently stressed by decreased food availability and heavy snows die to then become exposed carcasses during early spring snow-melt (Neumann 2009). Our spatial analyses also support this conclusion; probabilities of true poisoning mortality, in relation to total population by county, are more prevalent in northern counties where greater hunting activity is expected (Fig. 4C). Furthermore, Pattee et al. (1990) suggested that the continued observation of lead poisoning in the months following the fall hunting season could be due to a refractory period between ingestion and observed adverse toxicological effects. For example, death of bald eagles following ingestion of lead shot takes anywhere from 10 days to 133 days (Pattee et al. 1981).

The general increase in the proportion of poisoned eagles suggests that as the bald eagle population has increased, so has the prevalence of eagles foraging on terrestrial prey sources, and subsequent lead poisoning events from these sources. Greater frequencies of raptors foraging on non-preferred food sources as breeding densities increase has been suggested by Krüger and Lindström (2001) and Ferrer et al. (2006), who reported that inexperienced or non-dominant birds were forced to settle in lower quality habitat, and thus were more likely to forage on the non-preferred food sources in that habitat. In accordance, eagles occupying lower-quality habitat (habitat lacking access to open water or located in close proximity to humans) and winter migrant eagles may be more likely to forage on available upland game rather than fish. The general increasing proportion of poisoned eagles may also be the result of a greater likelihood of a poisoned eagle being detected if the lower-quality eagle habitat is closer in proximity to humans.

Monitoring mortalities due to lead poisoning is imperative. Spent ammunition in game animal remains is also detrimental to a variety of scavengers including corvids and terrestrial carnivores (Nadjafzadeh et al. 2013). Although lead ammunition is no longer used in the hunting of waterfowl, it continues to be used in Michigan for hunting upland small game and white-tailed deer despite the availability of copper or copper-zinc alloy bullets as a cost effective and equally effective alternative to lead-based bullets (Trinogga et al. 2013). In addition to not posing a toxic threat if ingested (Thomas 2013), copper bullets are less likely to fragment upon impact, reducing the likelihood of ingestion and increasing the ease of regurgitation.

Fishing tackle is also a source of lead exposure when eagles consume fish or other birds that have ingested lead sinkers (Scheuhammer et al. 2003, Lewin et al. 2006, Haig et al. 2014). The greater frequency of lead poisonings observed during spring may correlate when lost fish, with the line and sinker still attached, that have died during winter become available during ice-out in Michigan.

Greater proportions during summer of disease CODs, the third-most prevalent bald eagle COD, coincides with the prevalence of infectious disease vectors in Michigan. West Nile virus is transmitted to eagles by biting mosquitos (Nemeth et al. 2006), with unsurprisingly greater frequency during summer while mosquitoes are active in Michigan. We observed a proportionally greater number of West Nile cases than a previous eagle mortality study in which only 6 mortality cases were due to the West Nile virus, out of 2,519 bald eagle carcasses from 4 migratory bird flyways in the United States (Russell and Franson 2014).

Natural botulism outbreaks are also more frequent in warmer conditions, occurring cyclically (annually from Jul through Nov) during years of low mean annual water and lake levels in Michigan and warmer mean surface water temperatures (Lafrancois et al. 2011). Fish, particularly the invasive round goby (*Neogobius melanostomas*), walleye

(*Stizostedion vitreum*), and yellow perch (*Perca flavescens*), have been reported as possible significant vectors to various fish-eating birds (Yule et al. 2006). The incidence of type E botulism in bald eagles will likely continue to increase with rising lake temperatures and lower water levels according to Great Lakes climate change predictions (Lafrancois et al. 2011). The slight increasing trend of yearly probability estimates for disease may be a combination of increasing bald eagle populations, and rising lake temperatures.

Barbiturate poisonings observed in our study are likely a result of domestic pets and farm animals euthanized by pentobarbitol or phenytoin solutions, then disposed into a landfill or other area accessible to scavenging eagles. Twenty-nine bald eagles were poisoned after feeding on a cow that had been euthanized in British Columbia, Canada, showing the widespread negative effects from a single exposed carcass (Langelier 1993). Secondary sodium pentobarbital poisoning may weaken eagles, causing primary CODs to be caused by blunt trauma (wandering into traffic or falling from perches), predation, drowning, fatal mobbing attacks by other species, power line collision, or electrocution (Friend and Franson 1999, Krueger and Krueger 2003). Veterinary practitioners and animal shelters may reduce barbiturate poisonings in scavengers by wrapping carcasses euthanized with barbiturate solutions before transportation to landfills.

Although the sample size in this study is sufficiently large, it is not a true representative sample of population mortality because of the opportunistic sampling scheme. Some CODs, such as vehicular trauma, are more frequently collected and reported because of direct human reporting and high visibility of carcasses along roadsides. In contrast, the number of eagles collected because of lead poisoning is certainly under-represented because they likely ingest lead from unretrieved, hunter-shot carcasses and die in secluded locations. Deaths due to infectious diseases and barbiturate solutions may also be under-represented because of the decreased likelihood that affected eagles will be discovered. Lastly, when using proportional data analysis methodology, general decreasing trends in proportion to other CODs, such as emaciation and no diagnosis, may contribute to increases in proportions of other CODs with small sample sizes, such as poisoning and disease. For example, the credible intervals around the proportions of emaciation and no diagnosis CODs have narrowed over time (Fig. 3), demonstrating that although the same pathologist performed the majority of necropsies, technological advances in diagnostics have allowed for more precise COD diagnosis throughout the study period. This may overestimate the number of emaciation and no diagnosis mortalities early in the study period that may have actually been caused by poisoning or disease.

MANAGEMENT IMPLICATIONS

Results from this study can be used by the USFWS to determine area-specific opportunities to secure avoidance, minimization, and compensatory mitigation measures to reduce and offset detrimental effects to eagles. As a strategy to mitigate incidental take due to vehicular collision in the state of Michigan, we recommend the removal of deer carcasses and carrion from the roadway by county road commissions and the Department of Transportation. This action will decrease future risk of collision, and the number of bald eagle fatalities. Incidental take of bald eagles due to ingestion of lead in hunter-shot deer carcasses can also be substantially mitigated through a transition from lead ammunition to copper or copper-zinc alloy ammunition. By implementing the mandatory requirement of removal of carcasses from the roadway and the use of copper ammunition, managers can reduce risk to all scavenging species.

ACKNOWLEDGMENTS

We acknowledge the collaboration of many, including the Department of Natural Resources, National Park Service, United States Forest Service, USFWS employees, and private citizens, for collecting and submitting numerous bald eagle carcasses for necropsy. J. R. Melotti assisted T. M. Cooley in numerable necropsies. The Michigan Department of Environmental Quality and the Michigan Department of Natural Resources provided funding for toxicological testing. Financial support was also provided by the USFWS and the University of Maryland.

LITERATURE CITED

Buehler, D. A. 2000. Bald eagle (*Haliaeetus leucocephalus*). Account 506 *in* A. Poole, editor. The birds of North America online. Cornell Lab of Ornithology, Ithaca, New York, USA. ⟨http://bna.birds.cornell.edu/bna/species/506⟩. Accessed 10 Mar 2013.

Etter, D. R., K. M. Hollis, T. R. Van Deelen, D. R. Ludwig, J. E. Chelsvig, C. L. Anchor, and R. E. Warner. 2002. Survival and movements of white-tailed deer in suburban Chicago, Illinois. Journal of Wildlife Management 66:500–510.

Ferrer, M., I. A. N. Newton, and E. V. A. Casado. 2006. How to test different density-dependent fecundity hypotheses in an increasing or stable population. Journal of Animal Ecology 75:111–117.

Fisher, I. J., D. J. Pain, and V. G. Thomas. 2006. A review of lead poisoning from ammunition sources in terrestrial birds. Biological Conservation 131:421–432.

Franson, J. C., and R. E. Russell. 2014. Lead and eagles: demographic and pathological characteristics of poisoning, and exposure levels associated with other causes of mortality. Ecotoxicology 23:1722–1731.

Friend, M., and J. C. Franson. 1999. Field manual of wildlife diseases. General field procedures and diseases of birds. U.S. Geological Survey, Biological Resources Division, National Wildlife Health Center, Madison, Wisconsin, USA.

Friend, M., J. C. Franson, and W. L. Anderson. 2009. Biological and societal dimensions of lead poisoning in birds in the USA. Pages 34–60 *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt, editors. Ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.

Grubb, T. G., and R. G. Lopez. 2000. Food habits of bald eagles wintering in northern Arizona. Journal of Raptor Research 34:287–292.

Haig, S. M., J. D'Elia, C. Eagles-Smith, J. M. Fair, J. Gervais, G. Herring, J. W. Rivers, and J. H. Schulz. 2014. The persistent problem of lead poisoning in birds from ammunition and fishing tackle. Condor 116:408–428.

Harris, M. C., and J. M. Sleeman. 2007. Morbidity and mortality of bald eagles (*Haliaeetus leucocephalus*) and peregrine falcons (*Falco peregrinus*) admitted to the Wildlife Center of Virginia, 1993–2003. Journal of Zoo and Wildlife Medicine 38:62–66.

Hunt, W. G., W. Burnham, C. N. Parish, K. K. Burnham, B. Mutch, and J. L. Oaks. 2006. Bullet fragments in deer remains: implications for lead exposure in avian scavengers. Wildlife Society Bulletin 34:167–170.

Hunt, W. G., R. T. Watson, J. L. Oaks, C. N. Parish, K. K. Burnham, R. L. Tucker, J. R. Belthoff, and G. Hart. 2009. Lead bullet fragments in venison from rifle-killed deer: potential for human dietary exposure. PLoS ONE 4:e5330.

Kelly, T. R., R. H. Poppenga, L. A. Woods, Y. Z. Hernandez, W. M. Boyce, F. J. Samaniego, S. G. Torres, and C. K. Johnson. 2014. Causes of mortality and unintentional poisoning in predatory and scavenging birds in California. Veterinary Record Open 1:e000028.

Kendall, R. J., T. E. Lacker, C. Bunck, B. Daniel, C. Driver, C. E. Grue, F. Leighton, W. Stansley, P. G. Watanabe, and M. Whitworth. 1996. An ecological risk assessment of lead shot exposure in non-waterfowl avian species: upland game birds and raptors. Environmental Toxicology and Chemistry 15:4–20.

Krueger, B., and K. Krueger. 2003. US Fish and Wildlife Service fact sheet: secondary pentobarbital poisoning in wildlife. <http://cpharm.vetmed.vt.edu/USFWS/USFWSFPentobarbFactSheet.pdf>. Accessed 03 Mar 2013.

Krüger, O., and J. Lindström. 2001. Habitat heterogeneity affects population growth in goshawk *Accipiter gentilis*. Journal of Animal Ecology 70:173–181.

Lafrancois, B. M., S. C. Riley, D. S. Blehert, and A. E. Ballmann. 2011. Links between type E botulism outbreaks, lake levels, and surface water temperatures in Lake Michigan, 1963–2008. Journal of Great Lakes Research 37:86–91.

Langelier, K. 1993. Barbiturate poisoning in twenty-nine bald eagles. Pages 231–232 *in* P. T. Redig, editor. Raptor biomedicine. University of Minnesota Press, Minneapolis, USA.

Lewin, W. C., R. Arlinghaus, and T. Mehner. 2006. Documented and potential biological impacts of recreational fishing: insights for management and conservation. Reviews in Fisheries Science 14:305–367.

Lunn, D. J., A. Thomas, N. Best, and D. Spiegelhalter. 2000. WinBUGS—a Bayesian modelling framework: concepts, structure, and extensibility. Statistics and Computing 10:325–337.

McNab, W. H., D. T. Cleland, J. A. Freeouf, J. E. Keys, Jr., G. J. Nowacki, and C. A. Carpenter, compilers. 2007. Description of ecological subregions: sections of the conterminous United States [CD-ROM]. General Technical Report WO-76B. U.S. Department of Agriculture, Forest Service, Washington, D.C., USA.

Michigan Department of State Police. 2017. Michigan Traffic Crash Facts. <http://publications.michigantrafficcrashfacts.org/2017/MTCFVol1.pdf>. Accessed 27 Dec 2017.

Nadjafzadeh, M., H. Hofer, and O. Krone. 2013. The link between feeding ecology and lead poisoning in white-tailed eagles. Journal of Wildlife Management 77:48–57.

Nemeth, N., D. Gould, R. Bowen, and N. Komar. 2006. Natural and experimental west Nile virus in five raptor species. Journal of Wildlife Diseases 42:1–13.

Neumann, K. 2009. Bald eagle lead poisoning in winter. Pages 210–218 *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt, editors. Ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.

Newton, I. 1979. Population ecology of raptors. Buteo Books, Vermillion, South Dakota, USA.

Pagel, J. E., P. B. Sharpe, D. K. Garcelon, A. E. Little, S. K. Taylor, K. R. Faulkner, and C. S. Gorbics. 2012. Exposure of bald eagles to lead on the northern Channel Islands, California. Journal of Raptor Research 46:168–176.

Pattee, O. H., P. H. Bloom, J. M. Scott, and M. R. Smith. 1990. Lead hazards within the range of the California condor. Condor 92:931–937.

Pattee, O. H., S. N. Wiemeyer, B. M. Mulhern, L. Sileo, and J. W. Carpenter. 1981. Experimental lead-shot poisoning in bald eagles. Journal of Wildlife Management 45:806–810.

Postupalsky, S. 1985. The bald eagles' return. Journal of Natural History 87:62–63.

Quortrup, E. R., and R. L. Sudheimer. 1943. Detection of botulinus toxin in the bloodstream of wild ducks. Journal of the American Veterinary Medical Association 102:264–266.

Russell, R. E., and J. C. Franson. 2014. Causes of mortality in eagles submitted to the National Wildlife Health Center 1975–2013. Wildlife Society Bulletin 38:697–704.

Scheuhammer, A. M., S. L. Money, D. A. Kirk, and G. Donaldson. 2003. Lead fishing sinkers and jigs in Canada: review of their use patterns and toxic impacts on wildlife. Canadian Wildlife Service Occasional Paper 108, Ottawa, Canada.

Scheuhammer, A. M., and D. M. Templeton. 1998. Use of stable isotope ratios to distinguish sources of lead exposure in wild birds. Ecotoxicology 7:37–42.

Stauber, E., N. Finch, P. A. Talcott, and J. M. Gay. 2010. Lead poisoning of bald (*Haliaeetus leucocephalus*) and golden (*Aquila chrysaetos*) eagles in the US inland Pacific Northwest region—An 18-year retrospective study: 1991–2008. Journal of Avian Medicine and Surgery 24:279–287.

Sudharsan, K., S. J. Riley, and S. R. Winterstein. 2006. Relationship of autumn hunting season to the frequency of deer-vehicle collisions in Michigan. Journal of Wildlife Management 70:1161–1164.

Thomas, V. G. 2013. Lead-free hunting rifle ammunition: product availability, price, effectiveness, and role in global wildlife conservation. Ambio 42:737–745.

Trinogga, A., G. Fritsch, H. Hofer, and O. Krone. 2013. Are lead-free hunting rifle bullets as effective at killing wildlife as conventional lead bullets? A comparison based on wound size and morphology. Science of the Total Environment 443:226–232.

U.S. Fish and Wildlife Service [USFWS]. 1983. Northern States Bald Eagle Recovery Plan. USFWS, Denver, Colorado, USA.

U.S. Fish and Wildlife Service [USFWS]. 2016. Bald and golden eagles: population demographics and estimation of sustainable take in the United States, 2016 update. Division of Migratory Bird Management, Washington, D.C., USA.

Warner, S. E., E. E. Britton, D. N. Becker, and M. J. Coffey. 2014. Bald eagle lead exposure in the Upper Midwest. Journal of Fish and Wildlife Management 5:208–216.

Wickham, H. 2016. ggplot2: elegant graphics for data analysis. Springer-Verlag, New York, New York, USA.

Wood, P. B., D. A. Buehler, and M. A. Byrd. 1990. Raptor status report–bald eagle. Pages 13–21 *in* G. Pendleton, editor. Proceedings of the Southeast Raptor Management Symposium and Workshop. National Wildlife Federation, Washington, D.C., USA.

Yule, A. M., I. K. Barker, J. W. Austin, and R. D. Moccia. 2006. Toxicity of *Clostridium botulinum* type E neurotoxin to Great Lakes fish: implications for avian botulism. Journal of Wildlife Diseases 42:479–493.

Associate Editor: David Euler.