ECOTOXICOLOGY

Demographic implications of lead poisoning for eagles across North America

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Lead poisoning occurs worldwide in populations of predatory birds, but exposure rates and population impacts are known only from regional studies. We evaluated the lead exposure of 1210 bald and golden eagles from 38 US states across North America, including 620 live eagles. We detected unexpectedly high frequencies of lead poisoning of eagles, both chronic (46 to 47% of bald and golden eagles, as measured in bone) and acute (27 to 33% of bald eagles and 7 to 35% of golden eagles, as measured in liver, blood, and feathers). Frequency of lead poisoning was influenced by age and, for bald eagles, by region and season. Continent-wide demographic modeling suggests that poisoning at this level suppresses population growth rates for bald eagles by 3.8% (95% confidence interval: 2.5%, 5.4%) and for golden eagles by 0.8% (0.7%, 0.9%). Lead poisoning is an underappreciated but important constraint on continent-wide populations of these iconic protected species.

Lead, the most abundant nonessential heavy metal in Earth's crust, is also one of the most common environmental toxicants released by human activity (*1*, *2*). Although clinically relevant exposure to anthropogenically released lead has been documented for multiple wildlife taxa (*2*), the population-wide demographic effects of this exposure are, for nearly all species, completely unknown. Bald eagles (*Haliaeetus leucocephalus*) and golden eagles (*Aquila chrysaetos*) are iconic apex predators widely distributed across North America (*3*, *4*). Both species have been the subject of large-scale conservation actions epitomized by efforts within the US and globally (*3*, *4*). Despite these efforts, there is evidence of widespread and localized hotspots of acute lead exposure for both species (*5*–*7*). However, there is no understanding of large-scale spatial and temporal patterns of lead exposure, nor of the demographic consequences of lead-induced mortality for these species (*8*).

We quantified the lead exposure of 1210 bald and golden eagles sampled over the annual cycle and across North America from 2010 to 2018 (Fig. 1A). We used multiple lines of evidence from blood of live eagles (n = 237 bald, 383 golden) and from bone, liver, and feathers of dead eagles (n = 343 bald, 270 golden, of which 21 bald and 2 golden were sampled both ante- and postmortem) to test hypotheses about (i) the spatial, temporal, and demographic extent of lead exposure across the continent, and (ii) the degree to which lead exposure influences the trajectory of populations of these two species in North America.

Chronic poisoning suggests repeated exposure to lead over the long term and, in vertebrate species, can be measured in bone (*9*). Inductively coupled plasma mass spectrometry indicated that of 448 dead birds, 47% of bald eagles and 46% of golden eagles had bone lead concentrations above thresholds for chronic poisoning (i.e., above thresholds used by veterinary pathologists as indicative of a "clinical poisoning"; threshold >10 μg/g for femur, n = 226 bald, 222 golden; Fig. 1B and table S1) (*10*).

We detected age-related variation in the frequency of chronic poisoning as indicated by femur lead concentrations of both bald and golden eagles, but regional differences only for bald eagles (Fig. 2, fig. S1, and tables S1, S5, and S6). For both species, adults were more frequently chronically poisoned than subadults (bald, P = 0.02; golden, P < 0.01) and juveniles (bald, P < 0.01; golden, P < 0.01). Bald eagles in the Central Flyway exhibited higher rates of chronic lead poisoning than did those in the Atlantic (P < 0.01) and Pacific Flyways (P < 0.01).

Acute lead poisoning suggests a short-term high-exposure event and is best measured in blood, liver, or feather tissue [i.e., poisoning defined as above a threshold of >40 μg/dl wet weight for blood, >20 μg/g dry weight for liver, >2.1 μg/g dry weight for feathers (*9*–*11*)]. Of 620 live birds, 28% of bald eagles and 9% of golden eagles had blood lead concentrations indicative of acute poisoning (n = 237 bald, 383 golden; Fig. 1C and table S2). Similarly, 27% of dead bald eagles and 7% of dead golden eagles had liver lead concentrations indicative of acute poisoning (n = 271 bald, 163 golden; Fig. 1D and table S3). Feather lead concentrations can be used to identify acute poisoning events during the time period of feather growth (*11*). Lead profiles for feathers with ≥4 weeks of growth revealed that 35% of dead golden eagles (one feather sampled from each of n = 23 birds) and 33% of dead bald eagles (one feather sampled from each of n = 3 birds) experienced at least one acute lead poisoning event during the growth of that individual feather (Fig. 1E and table S4).

We detected age-related, seasonal, and regional differences in frequency of acute poisoning of bald eagles but not golden eagles (Fig. 2, figs. S1 and S2, and tables S2, S3, S5, and S6). Liver lead concentrations suggested that adult bald eagles were more frequently poisoned than were juveniles (P = 0.03). Likewise, blood lead concentrations indicated that acute poisoning of bald eagles was less common in summer than in fall (P = 0.02) or winter (P < 0.01). Blood lead concentrations also showed that bald eagles in the Central Flyway exhibited a higher rate of lead poisoning than did those in the Atlantic (P = 0.03) and Mississippi Flyways (P = 0.01).

Veterinary pathologists use thresholds of lead concentrations in the liver of dead birds, along with other postmortem findings, to

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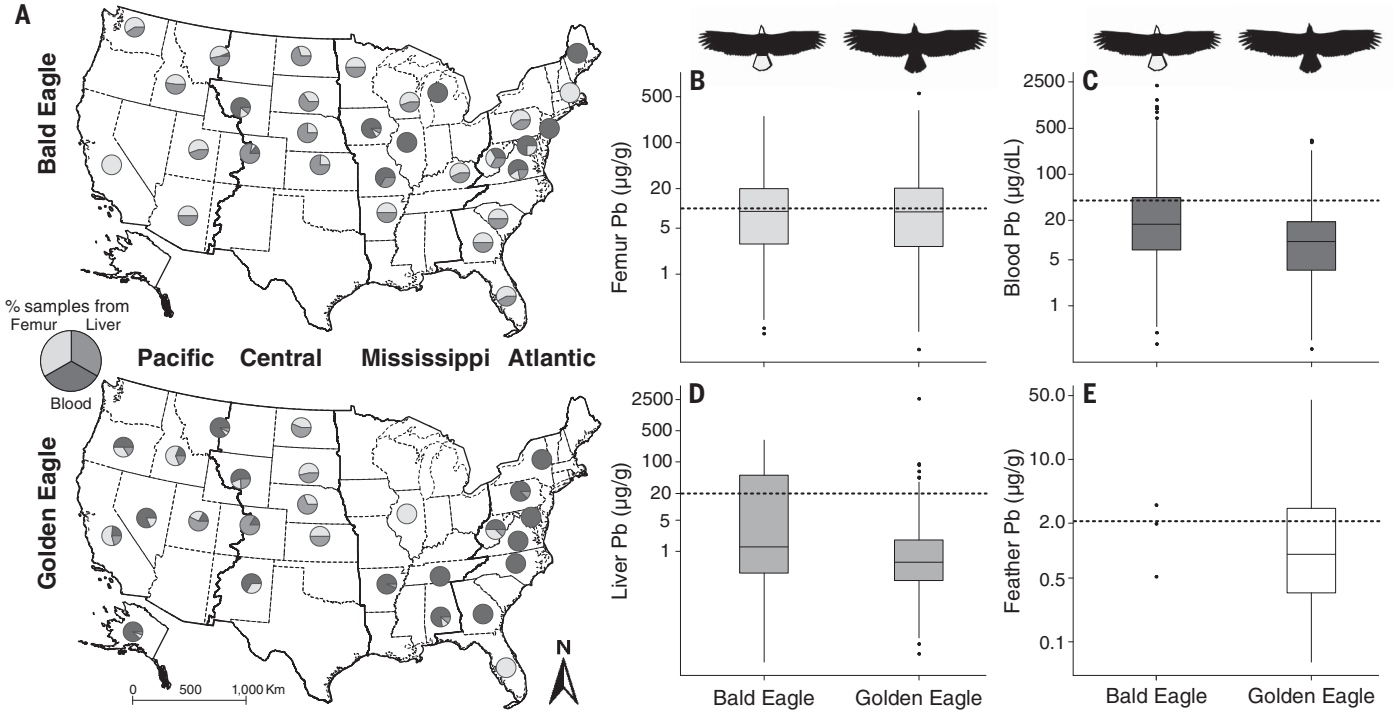

Fig. 1. Origins and lead concentrations of eagles used to interpret demographic effects of lead poisoning. (**A**) Collection locations (by state and US Fish & Wildlife Service–designated flyway) for eagle blood (bald, 237; golden, 383) taken from live birds, and eagle liver (bald, 271; golden, 163) and femur (bald, 226; golden, 222) from dead birds. (**B** to **D**) Censored boxplots (*16*) of lead concentrations in femur (dry weight) (B), blood (wet weight) (C), and liver (dry weight) (D), all shown on a log scale. (**E**) Peak feather (dry weight) lead concentration measured across ≥4 weeks of growth. Feather samples were collected from birds in six US states (see supplementary materials for details). Dotted horizontal lines on boxplots represent thresholds designating clinical poisoning (*9*–*11*, *17*).

determine cause of death (*9*). Measurements of blood lead concentrations from live birds are generally considered a good indicator of recent acute-exposure events, but because the birds are released back into the wild with unknown survival outcomes, there is no empirically defined blood lead concentration threshold associated with death (*6*, *9*). Our analyses suggest that liver lead concentrations above the thresholds used to define severe clinical poisoning occur in 4.9% of dead golden eagles and 25.8% of dead bald eagles. (If liver lead concentrations are above that threshold, then lead poisoning is generally determined to be the cause of death; this threshold is substantially higher and more conservative than the clinical poisoning threshold described above.) Hypothetical matrix population models built for both species suggest that if liver lead concentrations above that conservative threshold always result in death, then the continent-wide population growth rates of these species are being suppressed, for bald eagles by 3.8% (95% confidence interval: 2.5%, 5.4%) and for golden eagles by 0.8% (0.7%, 0.9%; tables S7 and S8), with probable long-term impacts to the population (Fig. 3). If only 75% of birds with liver lead concentrations above that threshold die, then there is a smaller but still demographically relevant suppression of population growth rates (fig. S3).

Acute poisoning of both species was generally higher in winter months, when bald and golden eagles commonly scavenge (*3*–*5*). Elevated lead concentrations in predatory and scavenging birds are usually caused by primary lead poisoning, most frequently direct ingestion of lead fragments from ammunition (*2*, *12*, *13*). Use of lead in ammunition during hunting seasons corresponds directly, both spatially and temporally, with the feeding ecology of facultative scavengers such as bald and golden eagles (*5*, *14*), a problem that has been studied extensively (*5*, *14*, *15*). Our data show a continent-wide temporal correspondence between acute lead poisoning of eagles and the use of lead ammunition.

Our large-scale data set hints at drivers of spatial and subcontinental trends in the frequency of lead poisoning of eagles that would be impossible to detect in local studies. For example, the high frequency of acute lead poisoning we detected for bald eagles in the Central Flyway could be influenced in part by differential timing of sampling (i.e., if more samples were taken in winter in that flyway than in other flyways). However, such an argument would not hold for the similar spatial patterns in chronic poisoning. Therefore, a more plausible explanation for these two patterns together lies in the potential for unexplained differential scavenging rates of bald eagles in the different flyways.

The age-related patterns we found in lead poisoning in the bones of bald and golden eagles reflect the accumulation of lead in scavenging birds as they age. Metallic lead is ingested, corroded by digestive acidity, incorporated into the bloodstream, absorbed by soft-tissue organs such as liver, and ultimately stored in the skeletal system (*6*, *9*). Thus, the age-related patterns we document show that across North America, eagles are repeatedly exposed to lead that builds up in their bodies as they age, creating an underappreciated demographic constraint for North American eagles.

Of the two eagle species, acute poisoning was more common for bald eagles. Although we did not test hypotheses to explain this, our data suggest that despite the rapidly increasing numbers of this species, their continent-wide populations are still vulnerable to negative demographic consequences associated with lead poisoning.

Demographic modeling of these populations implicates lead poisoning in suppression of growth rates of 0.8 to 3.8% per year, with

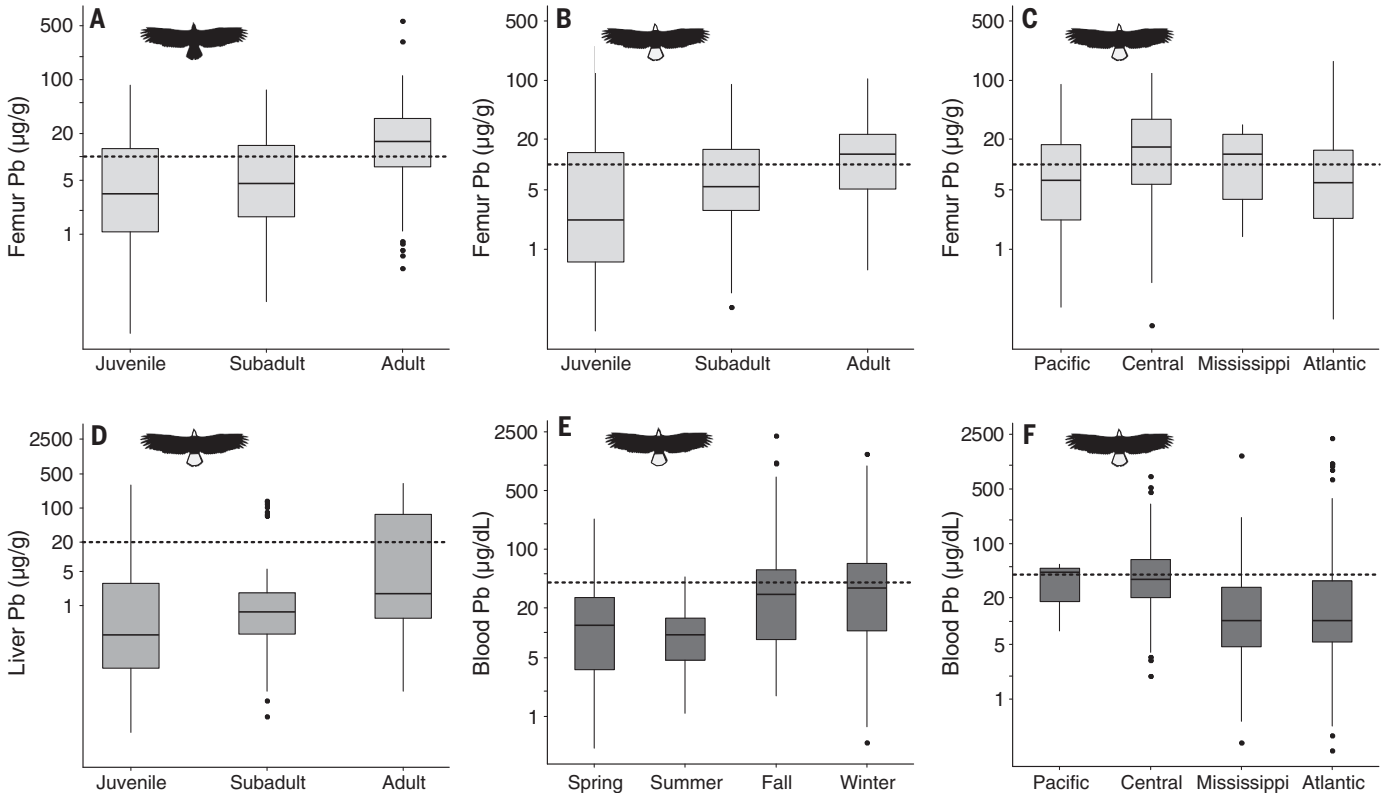

Fig. 2. Lead concentrations in femur, liver, and blood of bald and golden eagles, grouped by age, flyway, and season. (**A**) Censored boxplots of lead concentrations in golden eagle femur (dry weight), sorted by age. (**B** and **C**) Same as (A) for bald eagle femur lead concentrations, sorted by age (B) and by flyway (C). (**D** to **F**) Bald eagle lead concentrations in liver (dry weight) sorted by age (D), in blood (wet weight) sorted by season (E), and in blood, sorted by flyway (F). Boxplots are presented on a log scale; sample sizes are in tables S1 to S3. Dotted horizontal lines on boxplots represent thresholds for clinical poisoning (*9*, *10*, *17*).

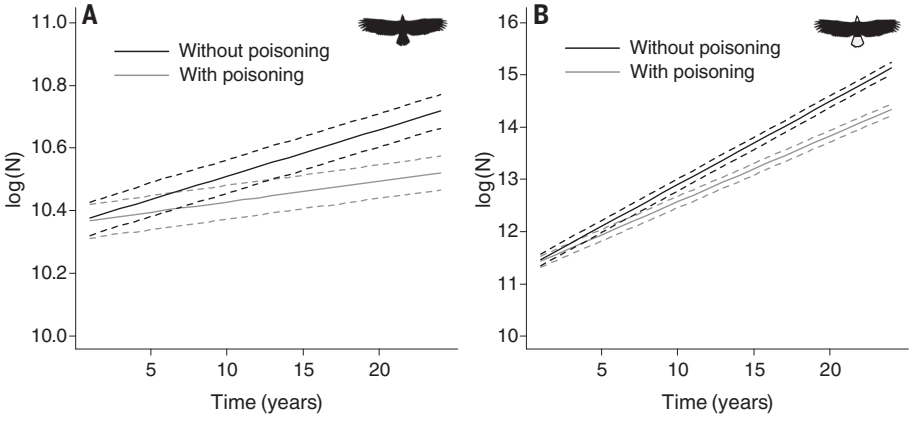

Fig. 3. Deterministic projections for populations of golden and bald eagles with and without effects to growth rates of lead poisoning. (**A**) Hypothetical matrix model projections for populations of golden eagles in scenarios without lead poisoning (upper black line) and with lead poisoning (lower gray line) at levels documented in this study. Solid lines are median estimates; dotted lines are 95% confidence intervals. (**B**) Same as (A) for bald eagles. The model assumes 100% mortality of individuals with liver lead concentrations above the threshold for severe clinical poisoning [33 µg/g dry weight (*15*)]. To isolate the effect of lead-caused mortality on eagle populations, these plots incorporate variation in lambda but no stochastic variation in population size.

consequences over the long term for populations of both species. Such a finding highlights the spatial and temporal extents to which lead poisoning affects populations of bald and golden eagles across North America. Our data identify directions for future conservation action supporting populations of these iconic species.

REFERENCES AND NOTES

1. J. Liang, J. Mao, *Environ. Sci. Pollut. Res. Int.* **22**, 7129–7138 (2015).
2. D. Avery, in *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans* (Peregrine Fund, 2009; www.peregrinefund.org/Lead_conference/PDF/0115 Avery.pdf.
3. D. A. Buehler, Bald Eagle (*Haliaeetus leucocephalus*) (Birds of the World, 2020); https://birdsoftheworld.org/bow/species/baleag/cur/introduction.
4. T. E. Katzner *et al*., Golden Eagle (*Aquila chrysaetos*) (Birds of the World, 2020); https://birdsoftheworld.org/bow/species/goleag/cur/introduction.
5. V. A. Slabe *et al*., *Environ. Toxicol. Chem.* **39**, 882–892 (2020).
6. J. C. Franson, R. E. Russell, *Ecotoxicology* **23**, 1722–1731 (2014).
7. A. R. Harmata, M. Restani, *J. Wildl. Dis.* **49**, 114–124 (2013).
8. W. G. Hunt *et al*., *PLOS ONE* **12**, e0172232 (2017).
9. J. C. Franson, D. Pain, in *Environmental Contaminants in Biota* (CRC Press, 2011), pp. 563–593.
10. P. A. Martin, D. Campbell, K. Hughes, T. McDaniel, *Sci. Total Environ.* **391**, 96–103 (2008).
11. M. E. Finkelstein *et al*., *Environ. Sci. Technol.* **44**, 2639–2647 (2010).
12. N. H. Golden, S. E. Warner, M. J. Coffey, *Rev. Environ. Contam. Toxicol.* **237**, 123–191 (2016).
13. L. K. Manning *et al*., *Vet. Pathol.* **56**, 289–299 (2019).
14. M. Nadjafzadeh, H. Hofer, O. Krone, *J. Wildl. Manage.* **77**, 48–57 (2013).
15. L. Cruz-Martinez, P. T. Redig, J. Deen, *Hum. Wildl. Interact.* **6**, 94–104 (2012).
16. D. Helsel, *Statistics for Censored Environmental Data Using Minitab and R* (Wiley, 2011).
17. J. A. Fallon, P. Redig, T. A. Miller, M. Lanzone, T. Katzner, *Wildl. Soc. Bull.* **41**, 205–211 (2017).

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SUPPLEMENTARY MATERIALS

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Materials and Methods
Tables S1 to S8
Figs. S1 to S3
References (*18*–*36*)

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A persistent lead problem

Although it occurs naturally, lead levels increased during the industrial revolution and have posed serious problems for humans and animals. Since the mid-1900s, efforts were made to limit anthropogenic sources of lead in the environment, and these were largely considered successful. Despite this headway, anthropogenic lead remains an underappreciated threat to wildlife. Slabe *et al*. looked at lead levels in samples collected from bald and golden eagles across the United States. They found that almost half of all animals sampled had chronic, toxic levels of lead. Demographic modeling suggested that these levels are high enough to suppress population growth in both species. — SNV

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Supplementary Materials for

Demographic implications of lead poisoning for eagles across North America

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The PDF file includes:

Materials and Methods

Study area and focal species

Bald eagles are endemic to and common throughout most of North America (*3*). Golden eagles are not as widely distributed in North America, but they have a circumpolar distribution at northern latitudes (*4*). Breeding populations of both species in Canada and Alaska are often medium- or long-distance southerly migrants (*3, 18, 19*). Bald eagles in the southern U.S.A. (New Mexico, Florida) may migrate north in summer (*3*). There are no known breeding territories of golden eagles in the U.S.A. east of the Mississippi River. Golden eagles that winter in the eastern U.S.A. are from a small population that breeds in Quebec, Labrador, or Ontario (*19-22*). Both eagle species are facultative scavengers and will utilize carrion as a food source year-round, but particularly during the winter months when live prey is less abundant (*3, 4, 5*).

We expected seasonal variation in lead exposure of eagles (*5, 13, 14*). Golden eagles of all ages are thought to be occasionally exposed to lead during summer months when consuming lead shot prey such as ground squirrels and more frequently exposed in the winter when feeding on carrion (*4*). Similarly, bald eagles of all ages are exposed to lead by consuming carrion during the winter months but also occasionally during the breeding season by consuming fish and waterfowl containing lead fishing equipment or gunshot (*3*). We also expected variation in lead exposure among age classes of eagles since previous studies have shown adults of both species are more likely to exhibit higher lead concentrations then younger age classes (*5, 7*). Furthermore, there are substantial differences in land cover, climate, and human hunting behavior among regions of North America. However, shooting of wildlife is widespread and prior localized studies have suggested that lead exposure of eagles occurs in multiple regions of the continent (*5, 7, 13*). For these reasons we felt it was appropriate to test for differences in lead exposure among flyways, but our expectation was that lead exposure should be similar regardless of region.

Data collection

We collected blood samples from 237 live bald and 383 live golden eagles, and liver, femur, and feather samples from 322 dead bald and 268 dead golden eagles during all astronomical seasons of the year and from states in each of the four major flyways throughout the continental U.S.A. (Tables S1- S3; flyways designated by U.S. Fish and Wildlife Service) (*23*). Additionally, we collected blood from 21 bald and 2 golden eagles upon admission to rehabilitation facilities and then we collected liver and femur tissue from these birds after their subsequent death. Whenever possible, birds were aged as adult, subadult, or juvenile (*24, 25*) and date of sample collection was recorded; in some cases it was not possible to collect this information because molt data were insufficient for aging or because eagle carcasses were found months after death.

These different types of samples provide diverse information on lead exposure of eagles. Blood samples are typically used to understand recent lead exposure of living birds (*17*). However, relationships between blood lead concentrations and health outcomes vary extensively (*9*). Liver lead concentrations are most commonly used by pathologists to diagnose cause of death during necropsy. This index is considered more robust because high concentrations of liver lead are often associated with other clinical signs of poisoning at the time of necropsy (gross and microscopic lesions, green staining around the vent, contracted talons, etc., *9*). Femur lead concentration is indicative of chronic exposure and is the least useful in interpreting recent exposure. However, bone lead concentrations are considered useful for identifying spatial patterns of lead exposure within populations (*9, 10*). Finally, feather lead concentrations can be informative about exposure events at specific times. Such data are particularly useful when birds

can be handled regularly and timing of feather growth is known (*11*). Together, these multiple lines of evidence of lead exposure can provide stronger inference than would be possible with data from any one sample type alone.

Samples were conducted under scientific collecting and bird banding permits issued by the appropriate state and federal authorities and, in the case of live animal collections, with Institutional Animal Care and Use Committee Protocols. All samples were initially received, archived and prepared for analysis at the U.S. Geological Survey in Boise, ID, under U.S. Migratory Bird Scientific Collecting Permit #MB41892B or its predecessors, and Idaho Department of Fish and Game Wildlife Collection/Banding/Possession permit #110728.

Blood samples were collected from living wild free-flying birds upon capture or within 48 hours of admission to licensed rehabilitation facilities (n = 237 bald and 383 golden). We captured free-flying birds with noose traps, rocket nets, net launchers and bow nets (*26*). Once in hand, we harvested blood samples from the brachial vein using 25 or 26 gauge needles and 3 mL syringes to draw 1 – 3 mL of blood. We immediately transferred the blood to low lead BD Vacutainers (BD Vacutainer®) and stored the samples in a conventional freezer.

Liver, feather, and femur samples were collected from dead birds by personnel at licensed rehabilitation facilities or sampled by the authors. Locations of blood, femur, and liver samples are shown in Fig. 1; feathers were collected from birds found dead in the U.S. states of California (golden = 15), Colorado (golden = 1), Florida (bald = 3), Idaho (golden = 1), Utah (golden = 5) and Wyoming (golden = 1). Bird carcasses were recovered in the field by permitted university, private, state, or federal field biologists or law enforcement agents (n = 343 bald, 270 golden). Typical known causes of death of these birds included conspecific fighting, emaciation, unknown trauma, toxicant poisoning (rodenticides and lead), gunshot, electrocution, or collision with wind turbines, power lines, or vehicles. In the case of gunshot birds (n = 11 femur, 14 liver), we did not collect samples from tissue that was obviously damaged by bullet shrapnel. For birds admitted at rehabilitation facilities, we adopted National Wildlife Health Center protocols and only included liver samples from birds that died or were euthanized within 72 hours of admission (NWHC unpublished data). Frequency of lead poisoning in our sample of rehabilitated birds was generally similar to or lower than that in our sample of free-flying birds (Table S1, S2, S3) and statistical tests indicated no difference between the two frequencies.

Sample processing and laboratory analysis

We prepared whole blood samples by drawing aliquots of 100-1000 µL from vacutainers and pipetting them in to 1 mL microcentrifuge tubes or 7.4 mL glass vials. When blood was partially clotted, we recorded wet weights of the entire sample, dried the samples overnight in a 95°C oven, and recorded the dry weights prior to digestion (see below). Dry weight results were converted to wet weights by using a known dry:wet weight ratio (*27*).

We collected one liver lobe and one 2.5-cm section from the diaphysis of the femur from each dead bird we sampled. To ensure no cross contamination between individuals, we used separate disposable scalpel blades to collect tissue from each bird. Other instruments were cleaned with a 70% bleach solution after tissue harvest from each bird. Liver and femur samples were immediately placed in a plastic bag and stored in a conventional freezer. For preparation, liver and femur samples were thawed to room temperature and a 1g (± 0.05g) section was weighed wet, dried in a 95°C oven, weighed again, and then sent to the Michigan State Veterinary Diagnostic Laboratory for lead concentration analyses.

The Michigan State Veterinary Diagnostic Laboratory digested blood, liver and femur

samples in nitric acid, diluted the samples with Millipore water prior to determining lead concentrations with an Agilent 7900 inductively coupled mass spectrometer (ICP-MS) (Agilent Technologies, Santa Clara, CA) fitted with a Cetac Auto Sampler (Cetac, Omaha, NE) and Micro Mist Nebulizer (Agilent). The ICP-MS was calibrated for lead concentration determinations with 1000 ug/mL stock solutions (Alfa Aesar, Ward Hill, MA) in accordance with standard operating procedures published by the manufacturer (Agilent Technologies 2008). Standard reference materials were used for matrix matched quality control of lead concentrations and initial instrument calibrations. Initial instrument calibrations were performed with standard reference materials in triplicate at a limit of detection of 0.5 mg/dL ± 2.9% (relative standard deviation) and a 95% confidence interval of 2.4 to 3.7%. Run-specific calibrations were similar and within the goals for precision listed by the manufacturer. Data on liver and femur lead concentrations are reported on a dry weight basis (dry weight) in micrograms per gram (µg/g) and blood lead concentrations are reported on a wet weight basis (wet weight) in micrograms per deciliter (µg/dL).

Feather samples were stored dry, at room temperature, in a paper envelope and sent to the University of California – Santa Cruz for lead concentration analyses. Feathers were processed and analyzed according to published methods (*11*) using established trace metal clean techniques under HEPA filtered air laboratory conditions (*28*). Briefly, ~2cm individual sections of a feather vane >14 cm long (between 3-6 sections analyzed per feather, depending on the size of the feather) were treated as separate samples and each feather section was rinsed sequentially with HPLC grade methanol, ultrapure water, 1% HNO3, and rinsed a final time with ultrapure water. Samples were then dried, weighed, and digested overnight in 2 mL sub-boiling concentrated HNO_3 in closed Teflon vials, evaporated to dryness, and reconstituted in 1% HNO_3 for analysis. Sample lead concentrations were determined by ICP-MS, Element XR high-resolution or X-Series II quadrupole), measuring masses of ^{206}Pb, ^{207}Pb, and ^{208}Pb with ^{205}Tl added as an internal standard. We then identified the highest lead concentration in the analyzed segments of a single feather and designated this the "peak" lead concentration in that feather.

Statistical Analyses

Many of the lead concentrations recorded were below variable analytical limits of quantitation and the data were not normally distributed (Table S1, S2, S3). Thus, we used the Kaplan–Meier method for nonparametric data to calculate summary statistics based on a cumulative probability distribution using a survival curve (package 'NADA' in program R; *26, 29, 30*). Summary statistics, including the median, mean, standard deviation and 95% confidence limits are computed based on modeled survival probabilities rather than a predefined distribution (16).For each eagle species, and for femur, liver and blood samples, we used the R function 'glm' to build logistic regression models to compare proportions of individuals for which measured lead concentrations were above and below established thresholds for clinical lead poisoning (40 µg/dL wet weight for blood, 20 µg/g dry weight for liver, 2.1 µg/g dry weight for feather and 10 µg/g dry weight for femur; *9, 10, 17, 30*). An acute lead poisoning threshold in feathers was based on the blood lead threshold of >40 µg/dL and estimated using a feather:blood ratio of 1 µg/g feather lead:19 µg/dL blood lead (*27*); as such, >2.1 µg/g feather lead represents >40 µg/dL blood lead. In each of these statistical models, a binary variable describing whether the sample was above or below the threshold for clinical poisoning was the response and the predictors were either the migratory flyway, age group, disposition, or astronomical season of the year. To reduce the probability of Type 2 error, we do not report statistical significance for comparisons when the sample size of one of the groups was < 10.

We used R package 'emmeans' to obtain *post hoc* pairwise comparisons among the migratory flyways, among age groups, among seasons, and by disposition of the bird at time of sampling (free-flying or admitted to a rehabilitation facility; *31*). There were very few birds from which we were able to collect all three tissue types (i.e., we did not collect bone or liver from a live bird, although sometimes a bird whose blood was sampled later turned up dead). Likewise, we sometimes could not ascertain age or season of death (i.e., some birds were found long after death and thus only a desiccated carcass or bone tissue could be collected and we had limited information on age or season of death). Because our metadata records were incomplete in this way, we could not build a single model to evaluate all predictors simultaneously in the context of all others. In theory there could be interactions among lead concentrations in different tissues that influence fatality risk and our analyses do not consider those types of effects. Model estimates for blood of bald eagles and liver of both species, all collected during the summer months, were not well estimated because, during these time periods, we had few measurements with lead concentrations above poisoning thresholds (Table S5, S6)

Demographic Models

We used species-specific demographic parameter estimates in stage-structured matrix models to interpret demography of eagles with and without lead mortality (*32, 33*). For both eagle species we used a 4-stage Leslie matrix model, with one first year age class, two subadult classes, and one adult stage. In each case adults were the only breeders. Prior studies show that golden eagle survival differs among year classes until the adult stage at year four, but that for bald eagles survival only differs substantially between the first and subsequent years (*32, 33*). For this reason we included age-specific survival rates for each age class in the golden eagle model, but for bald eagles we only used first-year and a pooled after-first-year survival rate. We used the survival rates reported from recent comprehensive North American studies for both species (for bald eagles, *32*; for golden eagles, *33*). To estimate fecundity parameters for both species, we used data from a meta-analysis that included samples from across North America (*32* and references to specific studies therein).

Our objective with the demographic models was to compare population growth rates (λ) with lead mortality, and without lead mortality, using simulations. We discounted any possible positive effects of lead on survival based on abundant literature that shows only negative effects of lead on avian survival (*6, 34, 35*). Consequently, we set our demographic analyses up to estimate the magnitude of the positive changes expected in the absence of lead mortality, as opposed to testing whether the effect of removing lead was >0. For the purposes of this modeling, we ignore potential sublethal effects of lead exposure and we made the simplifying assumption that lead poisoning only affected survival, not fecundity, despite evidence from other taxa that lead also may reduce production of offspring (*36*). To focus on the effect of lead on population growth rate, our models only consider uncertainty in that rate parameter, and they do not consider density dependence or uncertainty in population size.

We incorporated uncertainty in the baseline demographic rates for all parameters by specifying distributions that accounted for environmental and sampling variance (i.e., beta distributions for survival, lognormal distributions for fecundity), using parameter values reported in the source literature. We drew 10,000 random samples for each age-specific survival rate and for fecundity for each species from distributions representing baseline conditions and we populated 10,000 baseline Leslie matrices with these samples. We extracted the dominate eigenvalue from each matrix as our measure of λ, and we calculated summary statistics for each

set of 10,000 λ values (median, lower 0.025 and upper 0.975 quantiles of λ; we used the latter two as 95% confidence limits). Model selection for both species suggested that a constant survival rate model fit the data better than models that allowed for temporal variation.

We then took the random baseline samples for age- and species-specific survival, and we removed the age and species-specific rate of lead mortality to create an identical sample but without mortality from lead poisoning. To estimate rates of lead poisoning and lead-caused mortality, we used the field and laboratory data we report in this study. We based our inference about lead poisoning on numbers of birds whose liver lead concentrations were higher than the published threshold for severe clinical lead poisoning (33 µg/g dry weight; *9).* Using the "severe clinical" poisoning threshold is reasonable since previous study confirmed that lead poisoning was the cause of death of 94% of eagles whose liver lead concentrations were over the published threshold for the substantially lower "clinical" lead poisoning threshold (20 µg/g dry weight; *9*). However, there is great uncertainty about the relationship between the state and exposure history of the individual, the timing of the lead exposure, the measurement taken at the time of sampling and the ultimate survival of the bird. Likewise, although blood lead concentrations sample the living portion of the population, the connection between blood lead concentrations and survival outcomes is poorly established (*17*). Thus, given the well-studied connection between liver lead concentrations and accompanying post mortem findings (*9*), we chose to use liver for these models. We compared baseline model outputs to two different lead poisoning scenarios. First, we modeled scenarios in which death occurs for 100% of the birds with liver lead concentrations over the severe clinical poisoning threshold. Because there is a chance that not all birds die once lead concentrations reach the severe clinical poisoning threshold, we also modeled populations under scenarios where 75% of birds die (Table S7, S8, Fig. S3). We chose this 75% threshold because it is substantially lower than the 94% (as reported above in (*6*), but still reflective of the seriousness with which pathologists consider this level of poisoning. This lower threshold thus likely reflects a scenario that is extremely conservative with regard to lead poisoning outcomes.

Liver lead concentrations provide inference into the proportion of dead birds killed by lead poisoning. As such, in our models that excluded lead mortality, we reduced baseline mortality rates by age-specific proportions of birds whose liver lead concentrations were above the severe clinical poisoning threshold (i.e., we assumed that birds would not die, or die less frequently, from lead poisoning). For example, for first-year bald eagles in our 100% mortality model, we reduced the baseline mortality rate of 14.1% by the proportion of first year birds we evaluated that a pathologist would say were killed by lead poisoning (i.e. 12.8% of birds had severe clinical lead poisoning), resulting in a subsequent baseline mortality rate for first year birds of 12.3%. We repeated this for both age classes of bald eagles and for all four ages of golden eagles. We retained all other forms of mortality in our modified survival rates, so our modified survival distributions reflected survival with all forms of mortality except lead poisoning.

We repeated the matrix simulations with the modified survival rates (1 – the revised mortality rate), and collected a pooled sample of 10,000 species-specific λ values in the absence of lead mortality. We computed the same summary statistics for this sample. We assessed the change in λ attributable to lead removal by subtracting the set of 10,000 baseline λ values from the set of λ values without lead mortality, and we computed summary statistics for the set of 10,000 differences. To explore the long-term consequences of lead removal, we projected current United States population size estimates for each eagle species (*32, 33*) using baseline λ and "no-lead" λ. For this assessment, we multiplied the median of the population size estimates for each

species (N_t) by the respective sets of λ distributions to obtain an estimate of N_{t+1}. We repeated this exercise for ~25 years (i.e., $N_{t \text{ to } t+24}$), essentially two generations of each species (*32*), to show how populations could grow in the absence of lead versus with no change in lead in the environment. We used this short time period since density dependence is unlikely to be influential within two generations. Because we did not consider how density dependence, other environmental factors, or uncertainty in N might affect the outcome, this modeling represents an illustrative exercise rather than a prediction.

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Table S1. Statistics describing central tendencies (median, mean, standard deviation, and confidence intervals) of femur lead concentrations (µg/g dry weight) of (a) bald eagles and (b) golden eagles, by age, flyway, and disposition. Samples were collected during the period from 2010-2018. "Poisoned" is a count of the number of birds for which lead concentrations were above a clinical threshold for lead poisoning as described in the Materials and Methods. LCL = lower confidence limit, UCL = upper confidence limit.

(a) Bald Eagle

Age	**n**	**Poisoned**	**Median**	**Mean**	**SD**	**95% LCL**	**95% UCL**
Adult	137	77	13.27	17.91	18.43	14.82	21.00
Juvenile	50	16	2.11	14.52	41.92	2.90	26.15
Subadult	39	13	5.54	13.22	20.35	6.84	19.61
Flyway							
Atlantic	89	31	6.13	11.88	21.40	7.43	16.32
Central	63	42	16.37	26.90	36.10	17.98	35.81
Mississippi	16	9	11.43	13.47	9.75	8.70	18.25
Pacific	58	24	5.95	12.55	17.07	8.16	16.94
Disposition							
Free-flying	171	84	9.55	17.98	28.13	13.77	22.20
Rehabilitation	55	22	6.42	11.27	15.22	7.24	15.29

(b) Golden Eagle

Age	**n**	**Poisoned**	**Median**	**Mean**	**SD**	**95% LCL**	**95% UCL**
Adult	89	59	15.93	34.07	69.95	19.54	48.60
Juvenile	39	13	3.29	10.84	16.91	5.54	16.15
Subadult	94	30	4.27	10.99	15.03	7.95	14.02
Flyway							
Atlantic	10	6	14.88	21.85	24.33	6.77	36.93
Central	63	37	13.83	26.49	72.26	8.64	44.33
Pacific	149	59	5.67	17.45	32.70	12.20	22.70
Disposition							
Free flying	177	86	9.22	20.43	47.15	13.49	27.38
Rehabilitation	45	16	5.10	19.36	47.60	5.45	33.27

Table S2 Statistics describing central tendencies (median, mean, standard deviation, and confidence intervals) of blood lead concentrations (µg/dL) of (a) bald eagles and (b) golden eagles from North America, by age, season, flyway, and disposition. Samples were collected during the period from 2010-2018. "Poisoned" is a count of the number of birds for which lead concentrations were above a clinical threshold for lead poisoning as described in the Materials and Methods. Summary statistics, including the median, mean, standard deviation and 95% confidence limits are computed based on modeled survival probabilities rather than a defined distribution (16). LCL = lower confidence limit, UCL = upper confidence limit.

(a) Bald Eagle

Age	**n**	**Poisoned**	**Median**	**Mean**	**SD**	**95% LCL**	**95% UCL**
Adult	145	44	19.04	92.78	264.79	49.68	135.87
Juvenile	39	7	11.18	36.19	79.40	11.27	61.11
Subadult	53	17	23.04	62.72	160.01	19.64	105.80
Season							
Fall	61	20	28.90	122.77	347.87	35.48	210.07
Spring	39	5	12.24	25.15	47.83	10.14	40.16
Summer	48	2	9.27	11.41	10.47	8.45	14.37
Winter	89	41	34.20	102.97	211.26	59.08	146.86
Flyway							
Atlantic	123	22	10.31	82.78	271.46	34.81	130.75
Central	83	39	34.20	70.60	113.38	46.21	94.99
Mississippi	28	5	10.13	72.78	253.17	-21.00	166.55
Pacific	3	2	43.03	34.73	24.12	7.43	62.02
Disposition							
Free-flying	128	45	25.20	52.15	95.43	35.62	68.68
Rehabilitation	109	23	10.88	105.65	311.07	47.25	164.04

(b) Golden Eagle

Age	**n**	**Poisoned**	**Median**	**Mean**	**SD**	**95% LCL**	**95% UCL**
Adult	116	14	11.99	22.78	40.84	15.35	30.21
Juvenile	60	1	2.03	6.21	10.08	3.66	8.76
Subadult	207	19	10.22	18.91	36.52	13.94	23.89
Season							
Fall	97	3	4.39	8.95	12.38	6.49	11.41
Spring	99	9	10.99	17.14	21.92	12.82	21.46
Summer	10	0	6.02	8.59	9.48	2.72	14.46
Winter	177	22	12.00	24.16	48.00	17.08	31.23
Flyway							
Atlantic	57	8	14.94	26.63	45.08	14.93	38.33
Central	112	12	8.79	20.82	43.24	12.81	28.83
Pacific	214	14	8.30	14.35	26.96	10.74	17.96
Disposition							
Free-flying	354	31	9.70	17.17	30.95	13.94	20.39
Rehabilitation	29	3	8.17	29.24	71.20	3.33	55.16

Table S3. Statistics describing central tendencies (median, mean, standard deviation, and confidence intervals) of liver lead concentrations (µg/g dry weight) of (a) bald eagles and (b) golden eagles from North America, by age, season, flyway, and disposition. Samples were collected during the period from 2010-2018. "Poisoned" is a count of the number of birds for which lead concentrations were above a clinical threshold for lead poisoning as described in the Materials and Methods. Summary statistics, including the median, mean, standard deviation and 95% confidence limits are computed based on modeled survival probabilities rather than a defined distribution (16). LCL = lower confidence limit, UCL = upper confidence limit.

(a) Bald Eagle

Age	**n**	**Poisoned**	**Median**	**Mean**	**SD**	**95% LCL**	**95% UCL**
Adult	179	61	1.73	44.83	73.67	34.03	55.62
Juvenile	47	6	0.25	17.73	51.73	2.94	32.52
Subadult	45	7	0.72	16.65	38.75	5.33	27.97
Season							
Fall	45	10	1.40	33.52	65.45	14.40	52.64
Spring	71	21	1.28	45.09	82.81	25.83	64.35
Summer	35	0	0.33	0.88	1.25	0.46	1.29
Winter	120	43	1.96	40.55	63.59	29.17	51.93
Flyway							
Atlantic	53	10	1.29	18.70	40.26	7.86	29.54
Central	131	42	1.82	46.05	77.77	32.73	59.37
Mississippi	39	11	0.74	39.94	73.46	16.89	63.00
Pacific	48	11	1.08	21.37	42.69	9.29	33.45
Disposition							
Free flying	218	62	1.22	37.98	69.81	28.71	47.25
Rehabilitation	53	12	1.55	25.03	51.25	11.23	38.83

(b) Golden Eagle

Age	**n**	**Poisoned**	**Median**	**Mean**	**SD**	**95% LCL**	**95% UCL**
Adult	74	8	1.15	7.16	17.77	3.11	11.20
Juvenile	39	1	0.46	1.62	4.32	0.27	2.98
Subadult	50	3	0.37	54.41	369.8	-48.10	156.93
Season							
Fall	28	1	0.85	3.22	8.31	0.14	6.30
Spring	37	4	0.52	6.30	17.76	0.57	12.02
Summer	32	0	0.38	0.53	0.43	0.38	0.68
Winter	66	7	0.75	45.05	321.5	-32.52	122.63
Flyway							
Atlantic	3	1	3.05	13.08	17.89	-7.16	33.32
Central	83	6	0.75	5.52	16.10	2.06	8.98
Pacific	77	5	0.48	36.57	297.7	-29.93	103.08
Disposition							
Free flying	136	7	0.57	22.94	224.3	-14.76	60.63
Rehabilitation	27	5	0.75	7.18	13.57	2.06	12.30

Table S4. Statistics describing central tendencies (median, mean, standard deviation, and confidence intervals) of peak feather lead concentrations (µg/g) of bald eagles and golden eagles identified across the ≥ 4 weeks of feather growth. Samples were collected during the period from 2010-2018. "Poisoned" is a count of the number of birds for which peak lead concentrations were above a clinical threshold for lead poisoning, estimated as described in the Materials and Methods. Summary statistics, including the median, mean, standard deviation and 95% confidence limits are computed based on modeled survival probabilities rather than a defined distribution (16). LCL = lower confidence limit, UCL = upper confidence limit.

Species	**n**	**Poisoned**	**Median**	**Mean**	**SD**	**95% LCL**	**95% UCL**
Bald Eagle	3	1	1.97	1.89	1.33	0.39	3.39
Golden Eagle	23	8	0.91	5.26	11.00	0.77	9.76

Table S5. For bald eagles, beta coefficients (β), standard errors (SE), Z-scores (Z) and P values (P) for the predictor variables in the logistic regression models describing frequency of clinical poisoning (see methods for thresholds used in this determination) for blood, liver, and femur lead concentrations. We did not test for seasonal differences in frequency of chronic (femur) lead poisoning. Model estimates for a summer effect on lead concentrations in liver are not well estimated because few measurements of lead concentrations were above poisoning thresholds.

		β	SE	Z	P
Blood	(Intercept)	-1.81	0.70	-2.59	0.01
	Flyway - Central	1.89	0.68	2.78	0.01
	Flyway - Mississippi	-0.07	0.72	-0.10	0.92
	Flyway - Pacific	2.38	1.47	1.62	0.11
	Age - Juvenile	-0.77	0.52	-1.48	0.14
	Age - Subadult	-0.44	0.42	-1.06	0.29
	Season - Spring	-0.66	0.58	-1.13	0.26
	Season - Summer	-2.28	0.79	-2.88	< 0.01
	Season - Winter	0.68	0.38	1.80	0.07
	Rehabilitation - True	0.79	0.65	1.23	0.22
Liver	(Intercept)	-1.78	0.74	-2.39	0.02
	Flyway - Central	0.92	0.65	1.41	0.16
	Flyway - Mississippi	0.44	0.69	0.64	0.52
	Flyway - Pacific	0.41	0.74	0.55	0.58
	Age - Juvenile	-1.06	0.49	-2.17	0.03
	Age - Subadult	-0.76	0.46	-1.65	0.10
	Season - Spring	0.48	0.45	1.05	0.29
	Season - Summer	-16.91	1077.72	-0.02	0.99
	Season - Winter	0.76	0.43	1.79	0.07
	Rehabilitation - True	0.44	0.62	0.72	0.47
Femur	(Intercept)	-0.53	0.37	-1.45	0.15
	Flyway - Central	1.73	0.46	3.77	< 0.01
	Flyway - Mississippi	0.88	0.62	1.43	0.15
	Flyway - Pacific	0.55	0.44	1.25	0.21
	Age - Juvenile	-1.19	0.38	-3.16	< 0.01
	Age - Subadult	-0.90	0.40	-2.25	0.02
	Rehabilitation - True	0.52	0.44	1.17	0.24

Table S6. For golden eagles, beta coefficients (β), standard errors (SE), Z-scores (Z) and P values (P) for the predictor variables in the logistic regression models describing frequency of clinical poisoning (see methods for thresholds used in this determination) for blood, liver, and femur lead concentrations. We did not test for seasonal differences in frequency of chronic (femur) lead poisoning. Numbers of livers and femurs sampled at rehabilitation facilities were so small that, for this species, we did not test for differences in frequency of lead poisoning between rehabilitated and wild-caught birds. Model estimates for a summer effect on lead concentrations in liver and blood are not well estimated because few measurements of lead concentrations were above poisoning thresholds.

		β	SE	Z	P
Blood	(Intercept)	-2.54	0.86	-2.97	< 0.01
	Flyway - Central	0.02	0.51	0.03	0.97
	Flyway - Pacific	-0.41	0.50	-0.82	0.41
	Age - Juvenile	-1.34	1.15	-1.16	0.25
	Age - Subadult	-0.25	0.39	-0.65	0.51
	Season - Spring	0.60	0.74	0.81	0.42
	Season - Summer	-14.37	1230.76	-0.01	0.99
	Season - Winter	0.93	0.70	1.31	0.19
	Rehabilitation - True	0.05	0.67	0.07	0.94
Liver	(Intercept)	-1.81	1.41	-1.29	0.20
	Flyway - Central	-1.50	1.34	-1.12	0.26
	Flyway - Pacific	-1.52	1.39	-1.10	0.27
	Age - Juvenile	-0.92	1.12	-0.82	0.41
	Age - Subadult	-0.22	0.79	-0.28	0.78
	Season - Spring	1.37	1.21	1.14	0.26
	Season - Summer	-15.80	1776.02	-0.01	0.99
	Season - Winter	1.24	1.14	1.08	0.28
Femur	(Intercept)	0.65	0.64	1.01	0.31
	Flyway - Central	0.24	0.70	0.35	0.73
	Flyway - Pacific	-0.21	0.67	-0.31	0.75
	Age - Juvenile	-1.37	0.41	-3.32	< 0.01
	Age - Subadult	-1.23	0.34	-3.61	< 0.01

Table S7. For bald eagles, output of simulation modeling of continent-wide populations, showing population growth rates (λ), population size (N), and age structures (proportions (p) of individuals in each of four age classes). Three scenarios are shown, (a) baseline (present-day) conditions; (b) simulations showing increase in population growth rate in a scenario in which mortality of lead poisoned birds does not occur, and mortality rate of birds with liver lead concentrations above a clinically accepted threshold for poisoning is 100% (i.e., 100% of poisoning fatalities do not occur); and (c) simulations showing increase in population growth rate in a scenario in which mortality of lead poisoned birds does not occur, and mortality rate of birds with liver lead concentrations above a clinically accepted threshold for poisoning is 75% (i.e., 25% of poisoning fatalities do not occur. Note that although the models were different, model outputs in (b) and (c) are similar. Shown for each parameter are means, standard deviations (SD) and the 2.5%, 50% and 97.5% quantiles.

(a) Baseline conditions for bald eagle populations

Parameter	**Mean**	**SD**	**2.5%**	**50%**	**97.5%**
λ	1.14	0.06	1.03	1.14	1.28
N	82,457	14,016	58,405	81,201	112,661
p juvenile	0.21	0.04	0.13	0.21	0.30
p 2nd year	0.16	0.02	0.11	0.16	0.20
p 3rd year	0.13	0.01	0.10	0.13	0.14
p >3rd Year	0.50	0.08	0.35	0.50	0.66

(b) Without deaths from lead poisoning, assumes 100% of poisoned birds would have died

Parameter	**Mean**	**SD**	**2.5%**	**50%**	**97.5%**
λ	1.18	0.06	1.07	1.17	1.
N	85,586	14,569	60,601	84,322	117,372
p juvenile	0.21	0.04	0.13	0.21	0.30
p 2nd year	0.16	0.02	0.11	0.16	0.20
p 3rd year	0.12	0.01	0.10	0.13	0.14
p >3rd Year	0.50	0.08	0.35	0.50	0.66

(c) Without deaths from lead poisoning, assumes 75% of poisoned birds would have died

Parameter	**Mean**	**SD**	**2.5%**	**50%**	**97.5%**
λ	1.17	0.06	1.06	1.16	1.31
N	84,525	14,445	59,983	83,407	116,122
p juvenile	0.21	0.04	0.13	0.21	0.30
p 2nd year	0.16	0.02	0.11	0.16	0.20
p 3rd year	0.12	0.01	0.10	0.13	0.14
p >3rd Year	0.50	0.08	0.35	0.50	0.66

Table S8. For golden eagles, output of simulation modeling of continent-wide populations, showing population growth rates (λ), population size (N), and age structures (proportions (p) of individuals in each of four age classes). Three scenarios are shown, (a) baseline (present-day) conditions; (b) simulations showing increase in population growth rate in a scenario in which mortality of lead poisoned birds does not occur, and mortality rate of birds with liver lead concentrations above a clinically accepted threshold for poisoning is 100% (i.e., 100% of poisoning events do not occur); and (c) simulations showing increase in population growth rate in a scenario in which mortality of lead poisoned birds does not occur, and mortality rate of birds with liver lead concentrations above a clinically accepted threshold for poisoning is 75% (i.e., 25% of poisoning fatalities do not occur. Note that although the models were different, model outputs in (b) and (c) are similar. Shown for each parameter are means, standard deviations (SD) and the 2.5%, 50% and 97.5% quantiles.

(a) Baseline conditions for golden eagle populations

Parameter	**Mean**	**SD**	**2.5%**	**50%**	**97.5%**
λ	1.01	0.03	0.96	1.01	1.07
N	31,783	3,735	25,123	31,554	39,635
p juvenile	0.15	0.03	0.10	0.15	0.22
p 2nd year	0.10	0.02	0.07	0.10	0.14
p 3rd year	0.09	0.01	0.06	0.09	0.11
p >3rd Year	0.66	0.06	0.53	0.66	0.76

(b) Without deaths from lead poisoning, assumes 100% of poisoned birds would have died

Parameter	**Mean**	**SD**	**2.5%**	**50%**	**97.5%**
λ	1.02	0.03	0.97	1.01	1.08
N	31,938	3,704	25,344	31,712	39,679
p juvenile	0.15	0.03	0.10	0.15	0.22
p 2nd year	0.11	0.02	0.07	0.10	0.14
p 3rd year	0.09	0.01	0.06	0.09	0.11
p >3rd Year	0.65	0.06	0.53	0.66	0.76

(c) Without deaths from lead poisoning, assumes 75% of poisoned birds would have died

Parameter	**Mean**	**SD**	**2.5%**	**50%**	**97.5%**
λ	1.02	0.03	0.97	1.01	1.07
N	31,928	3,761	25,258	31,707	39,916
p juvenile	0.15	0.03	0.10	0.15	0.22
p 2nd year	0.11	0.02	0.07	0.10	0.14
p 3rd year	0.09	0.01	0.06	0.09	0.11
p >3rd Year	0.65	0.06	0.54	0.66	0.76

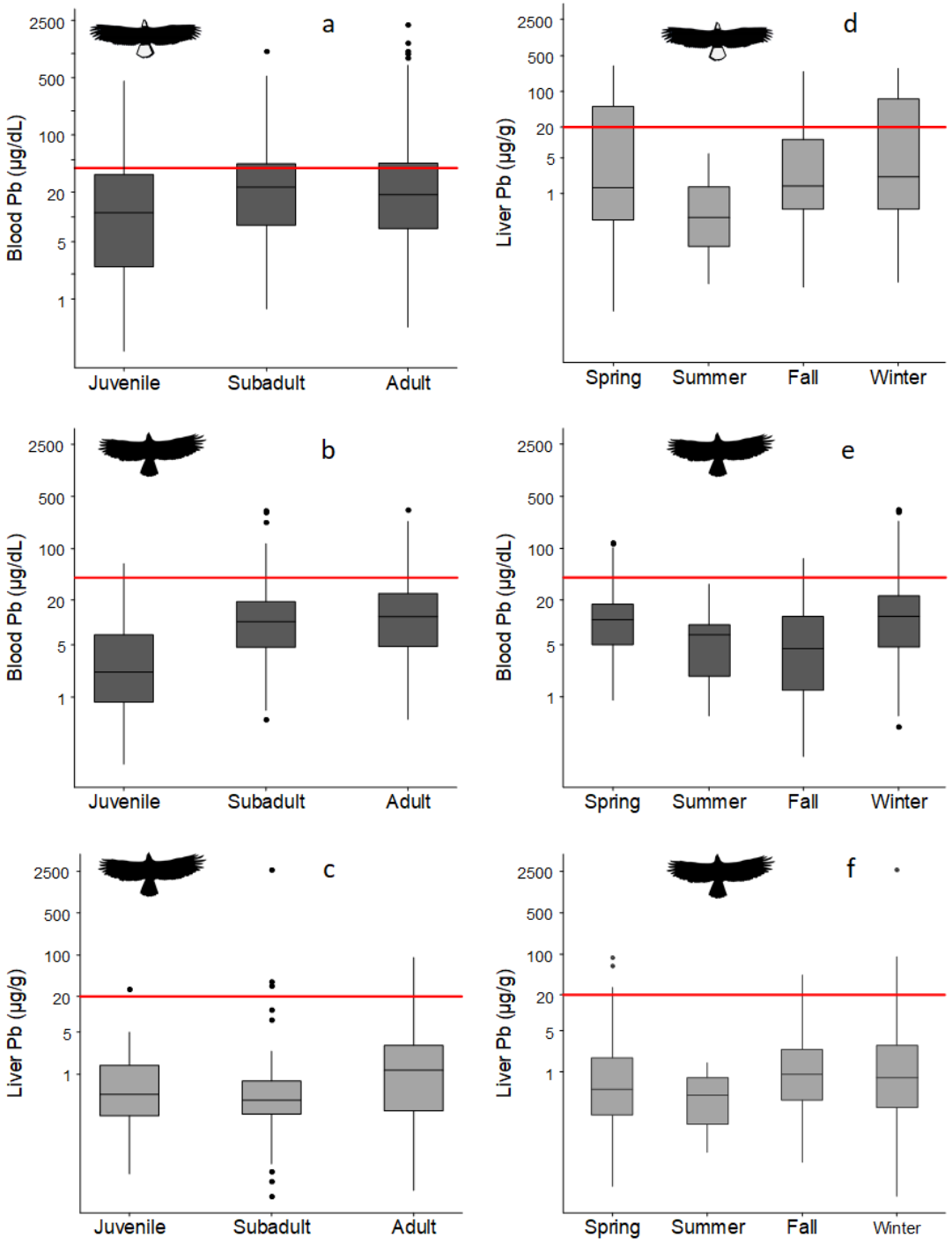

Fig. S1. Lead concentrations in liver and blood of bald and golden eagles, organized by age and season. Censored boxplots (*16*) of lead concentrations for (a) bald eagle blood (wet weight), organized by age, golden eagle (b) blood and (c) liver (dry weight) by age, and (d) bald eagle liver, and golden eagle (e) blood and (f) liver, all by season. Boxplots are presented on a log scale. Sample sizes are in Tables S2 and S3. Red horizontal lines on boxplots represent thresholds for clinical poisoning (*9, 10, 17*).

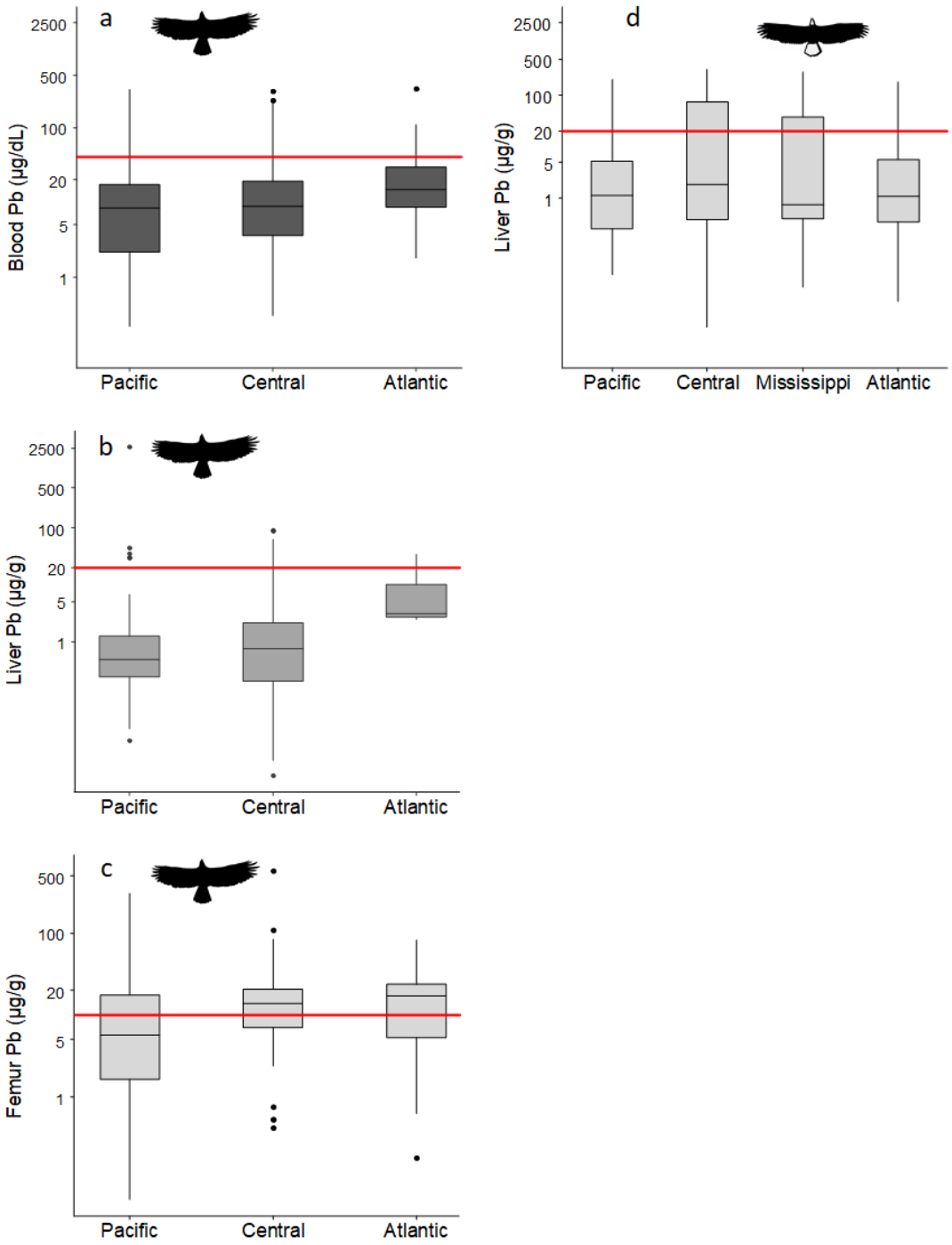

Fig. S2. Lead concentrations in blood, liver, and femur of bald and golden eagles, organized by flyway. Censored boxplots (*16*) of lead concentrations organized by flyway for (a) blood (wet weight), (b) liver (dry weight), and (c) femur (dry weight) of golden eagles and (d) of liver of bald eagles. Boxplots are presented on a log scale. Sample sizes are in Tables S1, S2 and S3. Red horizontal lines on boxplots represent thresholds for clinical poisoning (*9, 10, 17*).

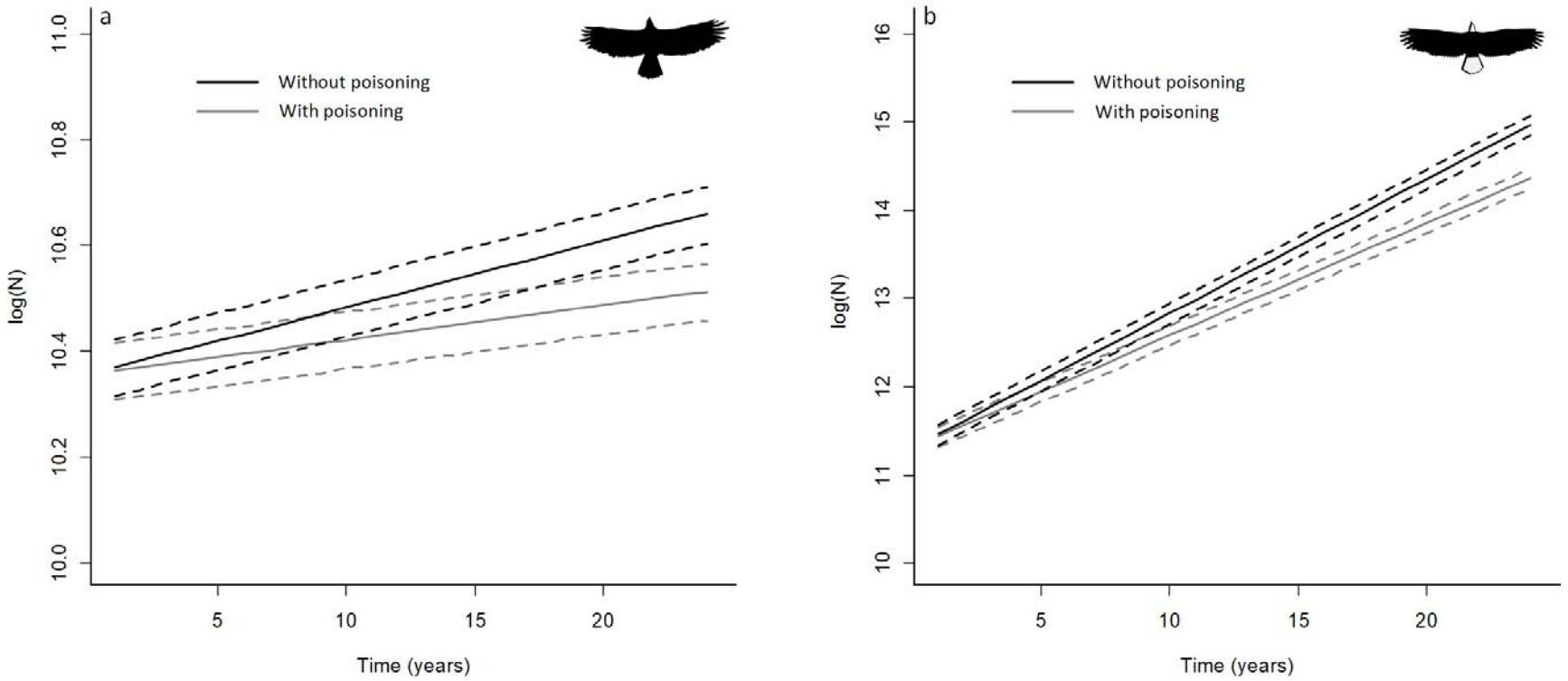

Fig S3. Deterministic projections for populations of golden and bald eagles with and without effects to growth rates of lead poisoning. (A) Hypothetical matrix model projections for populations of golden eagles in scenarios without lead poisoning (upper black line) and with lead poisoning (lower gray line) at levels documented in this study. Solid lines are median estimates and dotted lines are 95% confidence intervals. **(B)** Same as (A) for bald eagles. The model assumes 100% mortality of individuals with liver lead concentrations above the threshold for severe clinical poisoning [33 µg/dL dry weight (15)]. To isolate the effect of lead-caused mortality on eagle populations, these plots incorporate variation in lambda but no stochastic variation in population size.

References and Notes

1. J. Liang, J. Mao, Source analysis of global anthropogenic lead emissions: Their quantities and species. *Environ. Sci. Pollut. Res. Int.* **22**, 7129–7138 (2015).

2. D. Avery, in *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans* (Peregrine Fund, 2009; www.peregrinefund.org/Lead_conference/PDF/0115 Avery.pdf.

3. D. A. Buehler, Bald Eagle (*Haliaeetus leucocephalus*) (Birds of the World, 2020); https://birdsoftheworld.org/bow/species/baleag/cur/introduction.

4. T. E. Katzner *et al*., Golden Eagle (*Aquila chrysaetos*) (Birds of the World, 2020); https://birdsoftheworld.org/bow/species/goleag/cur/introduction.

5. V. A. Slabe, J. T. Anderson, J. Cooper, T. A. Miller, B. Brown, A. Wrona, P. Ortiz, J. Buchweitz, D. McRuer, E. Dominguez-Villegas, S. Behmke, T. Katzner, Feeding ecology drives lead exposure of facultative and obligate avian scavengers in the eastern United States. *Environ. Toxicol. Chem.* **39**, 882–892 (2020).

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

7. A. R. Harmata, M. Restani, Lead, mercury, selenium, and other trace elements in tissues of golden eagles from southwestern Montana, USA. *J. Wildl. Dis.* **49**, 114–124 (2013).

8. W. G. Hunt, J. D. Wiens, P. R. Law, M. R. Fuller, T. L. Hunt, D. E. Driscoll, R. E. Jackman, Quantifying the demographic cost of human-related mortality to a raptor population. *PLOS ONE* **12**, e0172232 (2017).

9. J. C. Franson, D. Pain, in *Environmental Contaminants in Biota* (CRC Press, 2011), pp. 563–593.

10. P. A. Martin, D. Campbell, K. Hughes, T. McDaniel, Lead in the tissues of terrestrial raptors in southern Ontario, Canada, 1995-2001. *Sci. Total Environ.* **391**, 96–103 (2008).

11. M. E. Finkelstein, D. George, S. Scherbinski, R. Gwiazda, M. Johnson, J. Burnett, J. Brandt, S. Lawrey, A. P. Pessier, M. Clark, J. Wynne, J. Grantham, D. R. Smith, Feather lead

concentrations and $^{207}Pb/^{206}Pb$ ratios reveal lead exposure history of California Condors (*Gymnogyps californianus*). *Environ. Sci. Technol.* **44**, 2639–2647 (2010).

12. N. H. Golden, S. E. Warner, M. J. Coffey, A review and assessment of spent lead ammunition and its exposure and effects to scavenging birds in the United States. *Rev. Environ. Contam. Toxicol.* **237**, 123–191 (2016).

13. L. K. Manning, A. Wünschmann, A. G. Armién, M. Willette, K. MacAulay, J. B. Bender, J. P. Buchweitz, P. Redig, Lead intoxication in free-ranging bald eagles (*Haliaeetus leucocephalus*). *Vet. Pathol.* **56**, 289–299 (2019).

14. M. Nadjafzadeh, H. Hofer, O. Krone, The link between feeding ecology and lead poisoning in white-tailed eagles. *J. Wildl. Manage.* **77**, 48–57 (2013).

15. L. Cruz-Martinez, P. T. Redig, J. Deen, Lead from spent ammunition: A source of exposure and poisoning in bald eagles. *Hum. Wildl. Interact.* **6**, 94–104 (2012).

16. D. Helsel, *Statistics for Censored Environmental Data Using Minitab and R* (Wiley, 2011).

17. J. A. Fallon, P. Redig, T. A. Miller, M. Lanzone, T. Katzner, Guidelines for evaluation and treatment of lead poisoning of wild raptors. *Wildl. Soc. Bull.* **41**, 205–211 (2017).

18. B. E. Bedrosian, R. Domenech, A. Shreading, M. M. Hayes, T. L. Booms, C. R. Barger, Migration corridors of adult Golden Eagles originating in northwestern North America. *PLOS ONE* **13**, e0205204 (2018).

19. T. A. Miller, R. P. Brooks, M. J. Lanzone, J. Cooper, K. O'Malley, D. Brandes, A. Duerr, T. E. Katzner, Summer and winter space use and home range characteristics of Golden Eagles (*Aquila chrysaetos*) in eastern North America. *Condor* **119**, 697–719 (2017).

20. T. Katzner *et al*., Status, biology, and conservation priorities for North America's eastern golden eagle (*Aquila chrysaetos*) population. *Auk* **129**, 168–176 (2012).

21. A. J. Dennhardt, A. E. Duerr, D. Brandes, T. E. Katzner, Integrating citizen-science data with movement models to estimate the size of a migratory golden eagle population. *Biol. Conserv.* **184**, 68–78 (2015).

22. A. J. Dennhardt, A. E. Duerr, D. Brandes, T. E. Katzner, Applying citizen-science data and mark–recapture models to estimate numbers of migrant golden eagles in an Important Bird Area in eastern North America. *Condor* **119**, 817–831 (2017).

23. US Fish & Wildlife Service, Migratory Bird Program—Conserving America's Birds; https://www.fws.gov/birds/management/flyways.php.

24. P. Pyle, Molt limits in North American passerines. *N. Am. Bird Bander* **22**, 49–89 (1997).

25. P. H. Bloom, W. S. Clark, Molt and sequence of plumages of golden eagles and a technique for inhand ageing. *N. Am. Bird Bander* **26**, 97–116 (2001).

26. K. L. Bildstein, D. Bird, *Raptor Research and Management Techniques* (Hancock House, ed. 1, 2008).

27. V. A. Slabe, J. T. Anderson, J. Cooper, B. Brown, P. Ortiz, J. Buchweitz, D. McRuer, T. Katzner, Lead in piscivorous raptors during breeding season in the Chesapeake Bay region of Maryland and Virginia, USA. *Environ. Toxicol. Chem.* **38**, 862–871 (2019).

28. A. R. Flegal, D. R. Smith, Current needs for increased accuracy and precision in measurements of low levels of lead in blood. *Environ. Res.* **58**, 125–133 (1992).

29. L. Lee, NADA: Nondetects and Data Analysis for Environmental Data. R package version 1.6-1.1 (2020); https://CRAN.R-project.org/package=NADA.

30. *R: A Language and Environment for Statistical Computing* (R Foundation for Statistical Computing, 2017).

31. R. V. Lenth, emmeans: Estimated Marginal Means, aka Least-Squares Means. R package version 1.5.4 (2021); https://CRAN.R-project.org/package=emmeans.

32. B. Millsap, E. Bjerre, M. Otto, G. Zimmerman, N. Zimpfer, *Bald and Golden Eagles: Population Demographics and Estimation of Sustainable Take in the United States, 2016 Update* (2016).

33. B. Millsap *et al*., Age-specific survival rates, causes of death, and allowable take of golden eagles in the western United States. *Ecol. Appl.* (2021).

34. M. E. Finkelstein, D. F. Doak, D. George, J. Burnett, J. Brandt, M. Church, J. Grantham, D. R. Smith, Lead poisoning and the deceptive recovery of the critically endangered California condor. *Proc. Natl. Acad. Sci. U.S.A.* **109**, 11449–11454 (2012).

35. S. A. Lambertucci, J. A. Donázar, A. D. Huertas, B. Jiménez, M. Sáez, J. A. Sanchez-Zapata, F. Hiraldo, Widening the problem of lead poisoning to a South-American top scavenger: Lead concentrations in feathers of wild Andean condors. *Biol. Conserv.* **144**, 1464–1471 (2011).

36. N. Vallverdú-Coll, F. Mougeot, M. E. Ortiz-Santaliestra, C. Castaño, J. Santiago-Moreno, R. Mateo, Effects of lead exposure on sperm quality and reproductive success in an avian model. *Environ. Sci. Technol.* **50**, 12484–12492 (2016).