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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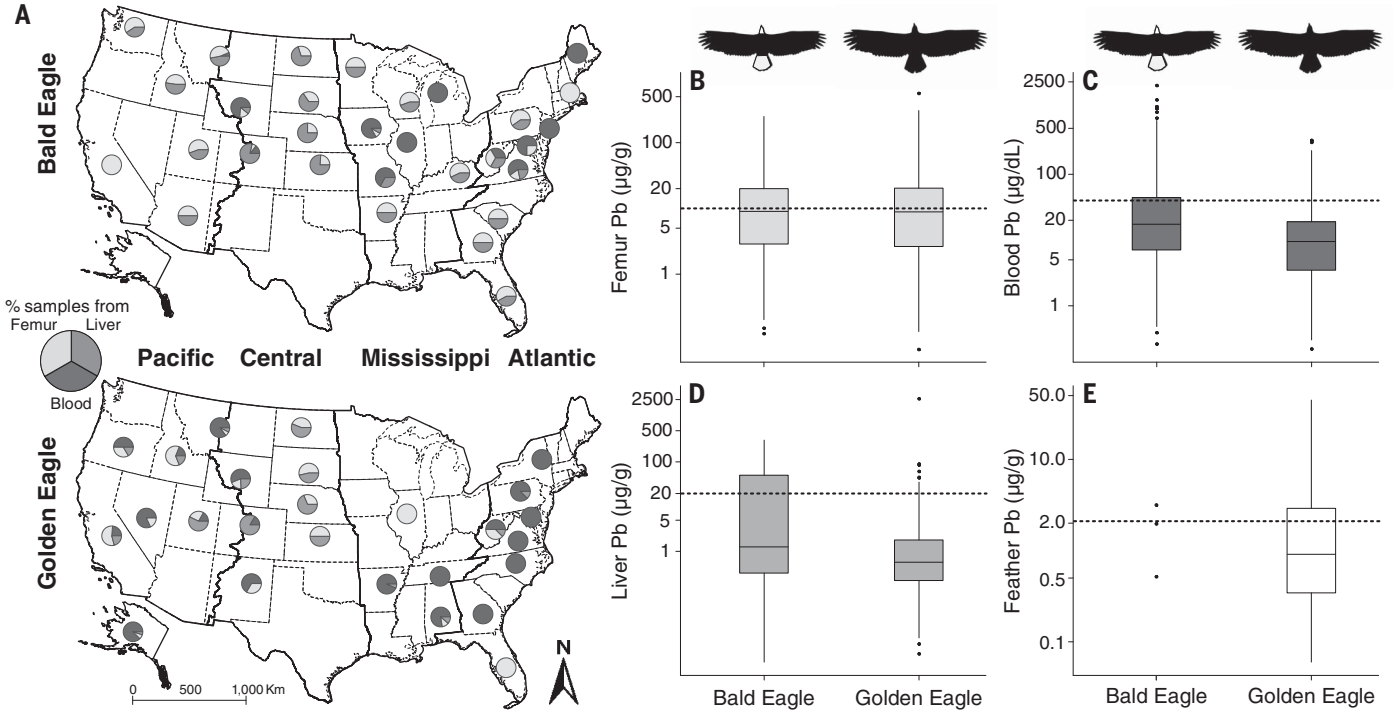

Fig. 1. Origins and lead concentrations of eagles used to interpret demographic effects of lead poisoning. (**A**) Collection locations (by state and US Fish & Wildlife Service–designated flyway) for eagle blood (bald, 237; golden, 383) taken from live birds, and eagle liver (bald, 271; golden, 163) 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

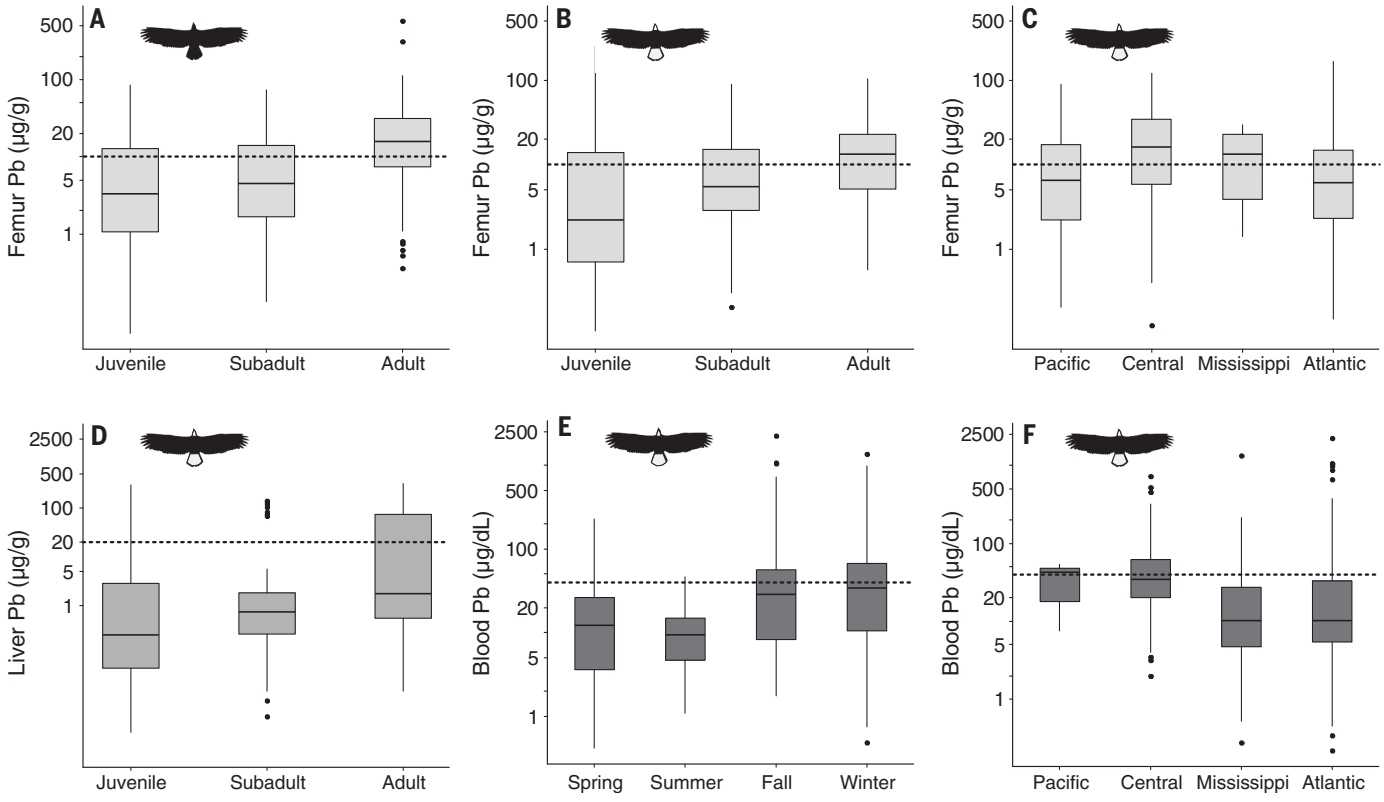

Fig. 2. Lead concentrations in femur, liver, and blood of bald and golden eagles, grouped by age, flyway, and season. (**A**) Censored boxplots of lead concentrations in golden eagle femur (dry weight), sorted by age. (**B** and **C**) Same as (A) for bald eagle femur lead concentrations, sorted by age (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*).

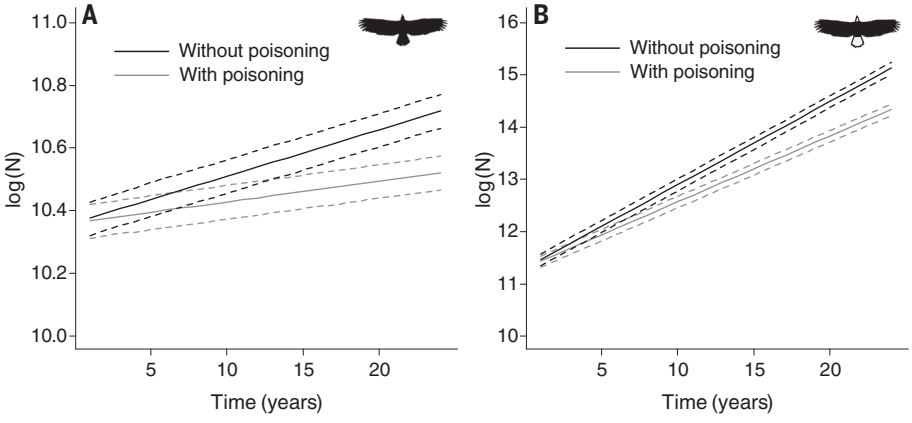

Fig. 3. Deterministic projections for populations of golden and bald eagles with and without effects to growth rates of lead poisoning. (**A**) Hypothetical matrix model projections for populations of golden eagles in scenarios without lead poisoning (upper black line) and with lead poisoning (lower 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