

Lead from spent ammunition: a source of exposure and poisoning in bald eagles

LUIS CRUZ-MARTINEZ, Department of Ecosystem and Public Health, Faculty of Veterinary Medicine, University of Calgary, 3280 Hospital Drive NW, Calgary, AB T2N4Z6, Canada luis.cruz-martinez@ucalgary.ca

PATRICK T. REDIG, The Raptor Center, College of Veterinary Medicine, University of Minnesota, 1920 Fitch Avenue, Saint Paul, MN 55108, USA

JOHN DEEN, Veterinary Population Medicine, College of Veterinary Medicine, University of Minnesota, 385A AnSci/VetMed, 1988 Fitch Avenue, Saint Paul, MN 55108, USA

Abstract: Ongoing occurrence of elevated levels of lead in bald eagles (*Haliaeetus leucocephalus*) following the ban on lead shot for waterfowl hunting led us to hypothesize that spent lead from ammunition, which is present in field residues of white-tailed deer (*Odocoileus virginianus*), represented a source of lead exposure in eagles. We conducted a case-control study using data from 1,277 bald eagles admitted for rehabilitation from January 1996 through December 2009. A multivariate logistic regression model was used to predict the odds of elevated lead levels using admission date in relation to deer hunting season, recovery location in relation to deer hunting zones, and age as predictors. We also assessed mean liver copper concentrations from eagles with elevated lead levels and from eagles with background lead levels, because most high velocity rifle bullets that are used for deer hunting are jacketed in copper. We found 334 bald eagles with elevated lead levels out of 1,277 bald eagles we examined. We detected significantly increased odds for elevated lead levels based on season (late fall and early winter), deer hunting rifle zone, and age (adult birds). The mean liver copper concentration was higher ($P = 0.02$) in eagles with elevated lead levels. These combined results supported our hypothesis that eagles are acquiring lead from hunter-shot deer. Further research is needed to determine whether this exposure to lead is having a population-level impact.

Key words: ammunition, bald eagles, copper, human–wildlife conflicts, lead, poisoning, white-tailed deer

HISTORICALLY, SECONDARY LEAD POISONING in bald eagles (*Haliaeetus leucocephalus*) was thought to occur incidentally as a result of ingestion of spent lead shot from hunters that crippled or poisoned waterfowl (Pattee and Hennes 1983). Researchers reported heavily hunted wetland areas as locations of high risk of lead poisoning for waterfowl and for migrant bald eagles, which were an endangered species at that time (Feierabend and Myers 1984). Experimental studies have established that bald eagles are susceptible to lead toxicity. An average of 82.5 mg of lead (eroded from lead shot) was sufficient to cause morbidity and mortality in bald eagles (Pattee et al. 1981), along with alterations in serum aminolevulinic acid dehydratase, hematocrit, hemoglobin concentration and various serum chemistry parameters (Hoffman et al. 1981).

This problematic association of waterfowl and eagles led ultimately to a legislatively mandated 5-year period for phasing out lead shot for waterfowl hunting, concluding in

a nationwide ban in 1991 on the use of lead shot on federal land in the United States (U.S. Department of the Interior 1986). Many states, including Minnesota, banned the use of lead shot on state-owned lands also. Subsequently, as a result of growing concerns for health of subsistence hunters from ingestion of lead shot, Canada banned lead shot for waterfowl hunting in 1999 (Scheuhammer 2009).

These legislative restrictions resulted in the effective reduction of lead poisoning for waterfowl in the United States (Rattner et al. 2009) and Canada (Scheuhammer 2009). However, researchers reported that the prevalence of lead poisoning in eagles admitted for rehabilitation did not change with the lead shot restrictions (Kramer and Redig 1997). In addition, these authors described a yearly seasonal trend in which most of the lead poisoning cases occurred coincidentally with deer hunting season. This temporal association suggested that deer offal left in the field after their evisceration by hunters were potential

sources of lead exposure for bald eagles (Janssen et al. 1986, Kramer and Redig 1997).

Deer hunting season in the Upper Midwest (Minnesota, Wisconsin, and Iowa) generally starts the first Saturday of November and lasts for about 3 weeks to a month. In Minnesota, approximately a half-million hunters harvest between 200,000 and 250,000 deer per year (Grund et al. 2010), with an estimated 10% of the harvested deer being fatally wounded but not retrieved by hunters (L. Cornicelli, Minnesota Department of Natural Resources, personal communication). Two legal deer hunting zones in Minnesota restrict the types of firearms permitted in rifle and shotgun zones. In the former, high-power centerfire rifles are allowed, whereas, in near-urban deer-hunting regions, only shotguns and muzzleloader rifles are permitted (Grund et al. 2010).

Lead-based ammunition is widely used for game hunting because of its ballistic qualities, cost effectiveness, and capacity for producing efficient, humane kills (Oltrogge 2009). However, as a consequence of the softness of the metal and the muzzle velocities of modern ammunition, the degree of bullet fragmentation and dispersal of fragments around the wound channel and adjacent tissues are far greater than previously thought (Hunt et al. 2006, Dobrowolska and Melosik 2008, Grund et al. 2010). Thus, lead contamination occurs in both carcasses and viscera of animals shot with lead-based ammunition, particularly with bullets designed to expand rapidly upon impact.

Pure lead bullets have a tendency to foul the riflings in the gun barrel because the muzzle velocity of high-powered rifle ammunition can exceed 914 m/second. This problem has been overcome by manufacturing bullets with a copper jacket around the lead core. We reasoned that eagles that have fed on carcass residues from deer killed with high-powered rifle bullets would ingest copper fragments in addition to lead; detection of this would link the source of lead in eagles to ammunition.

The objective of this study was to investigate whether elevated lead levels in bald eagles were related to ingestion of lead tainted deer remains. Our hypothesis was that spent lead from ammunition, present in the carcasses and viscera of white-tailed deer (*Odocoileus virginianus*), represented an important source

of lead exposure for bald eagles in the Upper Midwest.

Materials and methods

Bald eagle study population

Our study group consisted of injured or sick free-ranging bald eagles admitted to The Raptor Center (TRC) of the University of Minnesota from January 1996 to December 2009. The individuals were brought to TRC from diverse sources, such as conservation officers, animal control staff, The Humane Society volunteers, state highway patrol, and the general public.

Data collection and analyses

The data for this study were obtained from each eagle's medical records. Epidemiologic data were entered upon admission into an electronic database (Access 2003, Microsoft Office 2003, Microsoft Corporation, Redmond, Wash.), and included admission date (day, month, and year), recovery location (county and state), age (nestling, hatch year, second year, after second year, and adult), and sex.

Lead concentrations in blood (antemortem) and the presence or absence of metal foreign bodies within the gastrointestinal tract were measured at admission. Additionally, we measured lead in soft tissues at postmortem in 89 eagles. Blood lead concentration was measured by graphite furnace atomic absorption spectrophotometry (Varian, Model 300+ atomic absorption spectrophotometer, Varian Inc., Australia) performed at the University of Minnesota Veterinary Diagnostic laboratory (UMNVDL) and by an electrochemical method (ESA LeadCare blood lead testing system, ESA Inc., Chelmsford, Mass.) performed in-house. Soft tissue lead concentrations and other metal concentrations (i.e., copper, arsenic, selenium) were measured in liver and kidney by an inductively coupled plasma mass spectrophotometer at UMNVDL. Finally, the presence or absence of radio-dense foreign bodies in the gastrointestinal tract was determined by whole body radiographs (ventro-dorsal and lateral views).

Epidemiological data were categorized for analysis as follows. Individuals were grouped as recovered from either the rifle or shotgun zone (based on the legal firearm deer hunting zones prescribed by the departments of natural

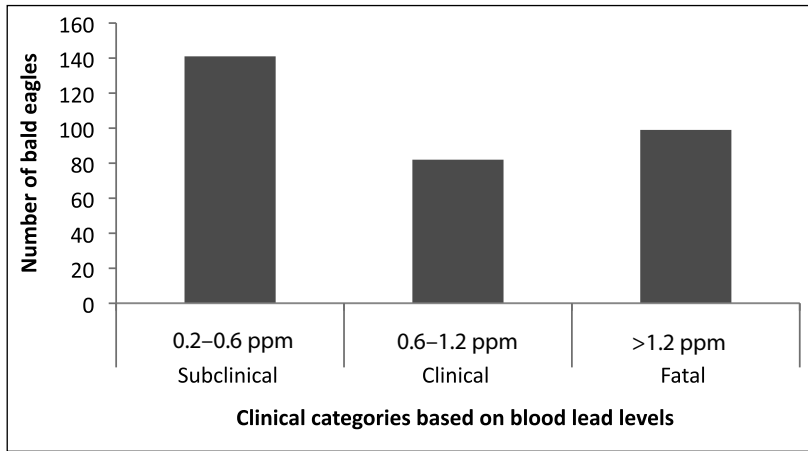


Figure 1. While it is difficult to determine if lead exposure was the primary cause of morbidity and mortality for the eagles in the subclinical category, blood lead levels of ≥ 0.6 ppm are reported to be the primary cause of morbidity and mortality for bald eagles. The scope of this paper, however, was not to determine primary and secondary morbidity and mortality causes related to lead exposure, but to provide evidence of a linkage between spent lead from ammunition used for deer hunting and elevated lead levels in bald eagles.

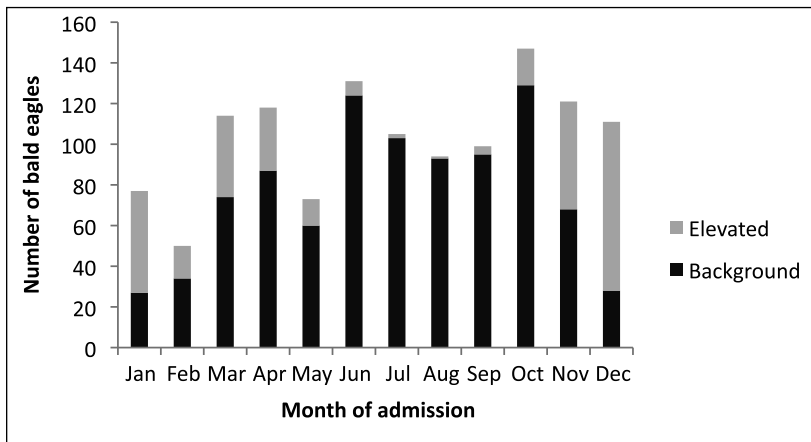


Figure 2. Elevated lead levels are seen at a significantly higher rate in eagles admitted during winter and early spring months (November to April).

resources in each state). Age was categorized into 3 groups: hatch year (<1 year); immature (2 to 5 years); or adults (>5 years), based on plumage characteristics (Bortolotti 1984). Lead data were used to classify the cases as elevated or background. Cases were classified as having elevated lead levels when blood lead concentration was ≥ 0.2 ppm (20 $\mu\text{g}/\text{dl}$), or liver lead concentration was ≥ 2 ppm (200 $\mu\text{g}/\text{dl}$) wet weight. When blood lead concentration was < 0.2 ppm or liver lead concentration was < 2 ppm wet weight, individuals were classified as having background levels of lead. The rationale for assigning an eagle with elevated or background lead levels was based on lead

exposure categories reported by Kramer and Redig (1997), clinical data on lead exposure in bald eagles (Redig and Arent 2008, Redig and Cruz-Martinez 2009), and data on lead concentration in tissues (Franson 1996, Friend and Franson 1999).

Descriptive statistics were used initially to assess the completeness of the data and to further characterize our interpretation of the admission data. We used 1-way analysis of variance (ANOVA) to determine if month and year of admission, recovery location (hunting zones and state) and age, significantly affected the outcome (elevated versus background). If significant ($P < 0.05$), Tukey’s test was used

Table 1. Distribution of bald eagles with elevated and background lead levels admitted to The Raptor Center of the University of Minnesota by state and deer hunting firearm zone from January 1996 to December 2009.

State	Elevated (<i>n</i>)	Background (<i>n</i>)
Minnesota	177	460
Wisconsin	103	328
Iowa	17	45
Other*	25	92
Firearm zone		
Rifle	168	456
Shotgun	99	244
Unknown	50	229
Total	317	929

*Michigan, Illinois, North Dakota, South Dakota, Ohio, Missouri, Nebraska, Indiana, Montana, California, New York, Kentucky, Washington

Table 2. Age distribution, based on plumage characteristics, of bald eagles with elevated and background levels of lead admitted to The Raptor Center of the University of Minnesota from January 1996 to December 2009.

Age groups	Elevated (<i>n</i>)	Background (<i>n</i>)
Adults	249	462
Immature	45	170
Hatch year	28	255
Unknown	2	36
Total	324	923

to determine which groups (levels) differed. A multivariate logistic regression model was then built using the significant effect variables to predict the odds for an eagle of having elevated lead levels. For instance, for month of admission, the groups used were October, November, December, January, February, March, April, and May; for recovery location (firearm zones), the group "rifle" was used, and for age, the group "adults" was used. For year of admission, this model was used to estimate the increase in risk per year for an eagle of having elevated lead levels. Finally, we conducted a 1-tailed Student's *t*-test to compare the liver copper concentrations between eagles with elevated or background lead levels. The above statistical tests were conducted using Statistix 8.0 (Analytical Software, Tallahassee, Fla.).

Table 3. Results from the logistic regression model to predict odds ratios for elevated lead levels in bald eagles admitted to The Raptor Center of the University of Minnesota from January 1996 to December 2009.

Predictor variable	Odds ratio	95% CI (lower limit)	95% CI (upper limit)
October	3.28	1.64	6.55
November	19.14	10.37	35.30
December	81.22	41.61	158.54
January	37.01	18.61	73.58
February	12.35	5.61	27.19
March	9.81	5.23	18.40
April	6.99	3.68	13.28
May	4.07	1.86	8.87
Year	1.05	1.02	1.08
Rifle zone	1.68	1.22	2.32
Adults	2.77	1.94	3.96

Results

Bald eagle study population

From 1996 to 2009, 1,277 bald eagles were admitted to TRC. Of these, 1,247 individuals were admitted live, and 30 were dead on arrival (DOA). Of the total sample size, 334 (26%) individuals were identified as elevated lead level cases; 322 of these were admitted live and 12 were DOA. The blood lead concentration data of eagles with elevated levels were categorized as described by Kramer and Redig (1997; Figure 1). Bald eagles were admitted in every month (Figure 2) and from various geographical regions (Table 1). Adults (>5 years) were the predominant age group admitted (Table 2) with elevated lead levels. We found a bimodal distribution of the cases in which the higher number of eagles with elevated lead levels (58%) was observed during the first 13 weeks after the onset of deer season (Figure 3).

The ANOVA test showed the following predictors to be positively associated with outcome: month of admission ($P = 0.01$), year of admission ($P = 0.009$), firearm zone ($P = 0.002$) and age ($P = 0.0005$); the state where the eagle was recovered was not a significant predictor of elevated lead levels ($P = 0.33$). Significant predictors were grouped as follows. For month of admission, the 3 different groups (ranked

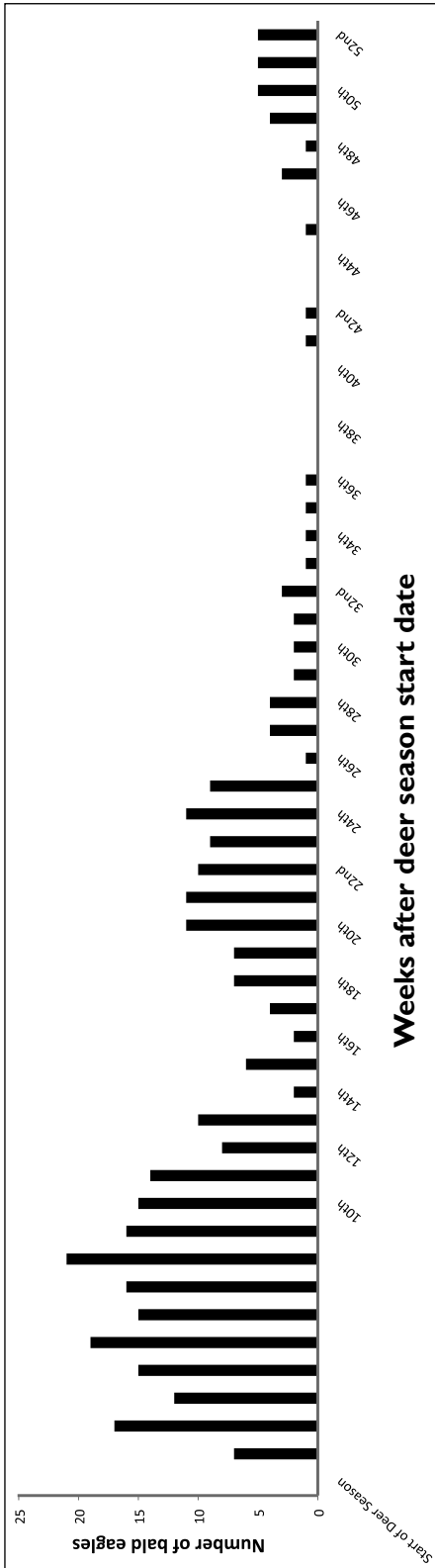


Figure 3. The greater number of cases was found immediately after the start of deer season, beginning on week 2 and peaking at week 8. The decline that followed this first seasonal peak was probably due to a combination of heavy snow cover and onset of cold temperatures that forced eagles to move away from open land toward river valleys, decreasing the chances of being found and brought in for rehabilitation. The second wave of cases occurred when warmer temperatures started melting the snow cover, exposing cold-preserved deer carcasses and viscera.

in descending order) were: December and January versus November; February and April versus October and May. For year of admission, 3 different groups (ranked in descending order) were identified: 2009 versus 1997 to 2008 versus 1996. For hunting zones, 2 different groups were identified: rifle zone and shotgun zone versus unknown zone. For age, 3 different groups (ranked in descending order) were identified: adults, immature, and hatch year.

The highest odds for bald eagles having elevated lead levels occurred during the winter months in eagles recovered from the rifle zone and in adult individuals (Table 3). In addition, the model showed that the odds of elevated lead level cases increased 5% on average per year during the study period.

Copper concentration in the liver was available for 89 individuals. Of these, 63 eagles had elevated lead levels, and 26 (30%) eagles had background lead levels (Table 3). The results from the 1-tailed t-test showed that the mean copper concentration from eagles with elevated lead levels ($\bar{x} = 8.93$, $SE = 1.64$) ppm wet weight was higher ($P = 0.02$) than that of background lead level eagles ($\bar{x} = 5.19$, $SE = 0.77$) ppm wet weight (Figure 4).

Of 322 live eagles with elevated lead levels, only 34 (11%) individuals had radiographically visible metal objects in their stomachs; 10 eagles had shotgun pellets in their stomachs, and the remaining 24 eagles had metallic shrapnel in their stomachs (Figure 5).

Discussion

The present case-based study sought to determine whether epidemiological data would support the hypothesis that a possible source of lead exposure for bald eagles was lead-based ammunition used for deer hunting.

Recovery rates in relation to deer hunting season

We identified a seasonal pattern of elevated levels of lead in eagles starting in October. Upon onset and during hunting season (November), the odds of eagles with elevated levels of lead increased considerably and reached the highest values during the following 2 months (December and January). Then, the percentage of cases, although still in significant numbers, declined from February to May. This marked

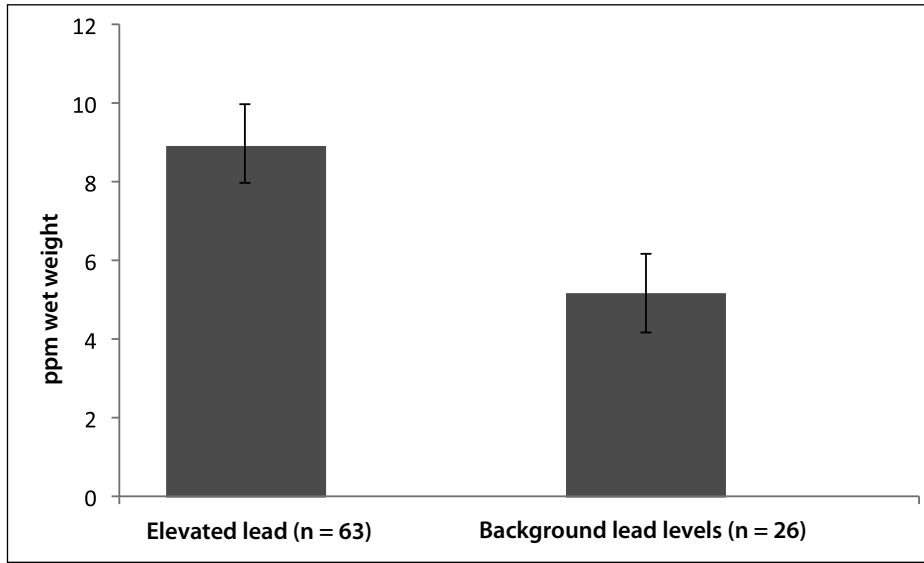


Figure 4. A subset (n = 89) of eagles submitted for histopathology over the course of the study period was evaluated for metal (including lead and copper) analysis in soft tissues. Of these, 63 eagles were categorized as having elevated lead levels, and 26 eagles were categorized as having background lead levels.

seasonal pattern of eagles with elevated lead levels has been reported since the 1980s (Kramer and Redig 1997). In addition, Strom et al. (2009) observed the same seasonal pattern for bald eagle fatalities attributed to lead poisoning in Wisconsin. Few cases of eagles with elevated lead levels were identified during summer and early fall, seasons when angling is a popular activity and lead sinkers and jigs are widely used. These objects are a significant source of exposure for waterbirds, particularly loons (*Gavia immer*; Sidor et al. 2003, Pokras et al. 2009). However, no evidence of ingestion of such objects was observed for bald eagles or osprey (*Pandion haliaeetus*), as per TRC data, even though fish represents a main food source during this time.

An association between lead exposure (and poisoning) in raptors and hunting seasons has been consistently reported in the scientific literature for bald eagles (Kramer and Redig 1997, Strom et al. 2009, Stauber et al. 2010), golden eagles (*Aquila chrysaetos*; Wayland and Bollinger 1999), California condors (*Gymnogyps californianus*; Church et al. 2006, Cade 2007), turkey vultures (*Cathartes aura*; Kelly and Johnson 2011), and common ravens (*Corvus corax*; Craighead and Bedrosian 2008) in the United States; for white-tailed sea eagles (*Haliaeetus albicilla*; Krone et al. 2009) in Germany; and for

Steller's sea eagles (*Aquila crysaetos*) and white tailed sea eagles (*Haliaeetus pelagicus*) in Japan (Saito 2009). In addition, lead from ammunition has been associated with lead exposure and poisoning for 66 species of terrestrial birds worldwide (Fisher et al. 2006, Pain et al. 2009).

Because stomach contents of eagles seldom contained lead fragments or other residues that would be associated with the source of exposure, we cannot be certain in every case that the source was deer residues. It is possible that other non-retrieved, lead ammunition-killed animals, such as coyotes (*Canis latrans*), and small game also could be a source. However, the temporal relationship of cases of elevated lead levels with the onset of the deer-hunting season creates a strong association with deer as the source of lead in bald eagles.

The increase in risk (5% on average per year) of cases of elevated lead levels suggested that while there was a decrease in the hunting population (Grund et al. 2010), the number of deer viscera left in the field every season (>200,000), along with fatally wounded but non-retrieved deer, was a readily available and abundant food source for eagles. Contributing to the above, the nesting population of bald eagles in Minnesota has steadily increased for the last 30 years with a current population of approximately 2,300



Figure 5. Ventro-dorsal view of whole-body radiographs of 2 bald eagles with elevated lead levels. The rounded, well-defined shape of shotgun pellets found in the ventriculus (arrows, right image) is easily distinguished radiographically from the irregular shape of rifle ammunition fragments (arrows, left image).

pairs (C. Henderson, Minnesota Department of Natural Resources, personal communication). Thus, while the toxic load in the environment may have remained more or less constant, the size of the at-risk population of eagles was steadily increasing, thereby accounting for the incremental increase each year.

Recovery location in relation to deer-hunting zones

Eagles that were recovered from the rifle zones had a higher risk for elevated lead levels as compared to those that were recovered from the shotgun zones. A possible explanation for this is based on the ballistics and kinetics of lead-based bullets. Bullets fired from center-fire rifles can reach velocities twice as fast as projectiles (slugs) fired from shotguns and muzzleloaders (Stroud and Hunt 2009). Combined with the bullet's design, composition, and type (particularly rapid-expansion bullets), this high velocity produces greater numbers of fragments upon impact compared to shotgun slugs (Grund et al. 2010). Lead-contaminated tissues (wound channel and adjacent tissues along with viscera)

often are utilized as food sources by bald eagles and other avian scavengers (Hunt et al. 2006, Pauli and Buskirk 2007, Krone et al. 2009, Saito 2009, Grund et al. 2010).

From our study design, it was not possible to determine if the exact location where the eagle was recovered was also the location where it was exposed to lead. Bald eagle movement patterns are affected, in part, by food availability and weather conditions (Buehler 2000). Also, there is individual variation in the timing from exposure to clinical disease or death (Hoffman et al. 1981, Pattee et al. 1981) and, in some areas, close to the borders along the state of Minnesota, Wisconsin, and Iowa, both types of hunting zones are in close proximity to each other. However, our large sample size, combined with biological plausibility, supports our findings of higher risks of elevated lead levels for birds foraging or inhabiting in the deer hunting rifle zone.

Relationship between eagle age and lead levels

We observed most elevated lead level cases among adult individuals (>5 years) as compared

to the other age groups. This age bias has been reported in bald and golden eagles from the Canadian prairies (Wayland and Bollinger 1999) and in Steller's sea eagles from Japan (Saito 2009) and appears to be related to aggressive behavior of eagles at scavenging sites during winter (Buehler 2000).

While bald eagles feed upon a variety of food items, including fish, birds, and mammals, their diet depends on food availability; during winter months, these birds scavenge opportunistically (Lang et al. 1999, Buehler 2000). Researchers report that carcasses of large ungulates, particularly of white-tailed deer, are common food sources for wintering bald eagles (Swenson et al. 1986, Jennelle et al. 2009). We infer that adult bald eagles would have the first choice at consuming flesh of a carcass and would likely choose easily accessible tissues at bullet wound sites (Saito 2009).

Copper concentration

Lead-core, copper-jacketed bullets are the most commonly used bullets for deer hunting. The copper jacket will separate from the lead core upon impact or while travelling through the body, thereby, leaving copper remains in the carcass or viscera of the shot animal (Hunt et al. 2006, Oltrogge 2009). Our findings of higher copper concentrations in livers from eagles with elevated lead levels suggested that this higher concentration may have occurred due to concurrent ingestion of copper fragments present in the remains of deer killed with lead-core, copper-jacketed bullets.

Presence of metal foreign objects in gastrointestinal tract

Only a small proportion (11%) of bald eagles had radiographic evidence of metallic foreign objects in their gastrointestinal tracts. This low proportion has been previously reported for lead-exposed bald eagles (Kramer and Redig 1997). Birds of prey cast undigested food materials (such as hair and feathers) within approximately 12 to 24 hours after ingestion (Duke 1997). By this mechanism, an eagle might orally eliminate large metal fragments or pellets. Researchers report that white-tailed sea eagles tend to avoid large objects while feeding (Krone et al. 2009); however, smaller fragments (as those produced upon impact by certain high-

velocity rifle lead bullets) can be rapidly eroded in the acid gastric environment of eagles or can be overlooked upon radiographic examination, thereby providing additional explanation of this finding (Kramer and Redig 1997, Hunt et al. 2006).

Conclusions

While many lead-based products and lead residues (from smelters and shooting ranges) are still available, most of these are heavily regulated. However, lead-based products for outdoor activities (i.e., hunting) are widely used and, therefore, a significant amount of lead is deposited in aquatic and terrestrial ecosystems (Rattner et al. 2009). Here, we presented evidence that relates spent lead from ammunition to lead exposure and poisoning in eagles. Further, the spatial-temporal association between eagles with elevated lead levels and deer hunting season and zones, along with the metal analysis in relation to commonly used lead bullets for deer-hunting, supported our hypothesis that exposure to spent lead from ammunition ingested by eagles while scavenging on deer residues, is a major source of lead exposure in the Upper Midwest.

Literature cited

- Bortolotti, G. R. 1984. Criteria for determining age and sex of nestling bald eagles. *Journal of Field Ornithology* 55:467–481.
- Buehler, D. A. 2000. Bald eagle (*Haliaeetus leucocephalus*). Pages 1–39 in A. Poole and F. Gill, editors. *The Birds of North America*. Vol. 506. The birds of North America, Philadelphia, Pennsylvania, USA.
- Cade, T. J. 2007. Exposure of California condors to lead from spent ammunition. *Journal of Wildlife Management* 71:2125–2133.
- Church, M. E., R. Gwiazda, R. W. Risebrough, K. Sorenson, C. P. Chamberlain, S. Farry, W. Heinrich, B. A. Rideout, and D. R. Smith. 2006. Ammunition is the principal source of lead accumulated by California condors re-introduced to the wild. *Environmental Science and Technology* 40:6143–6150.
- Craighead, D., and B. Bedrosian. 2008. Blood lead levels of common ravens with access to big-game offal. *Journal of Wildlife Management* 72:240–245.
- Dobrowolska, A., and M. Melosik. 2008. Bullet-de-

- rived lead in tissues of the wild boar (*Sus scrofa*) and red deer (*Cervus elaphus*). *European Journal of Wildlife Research* 54:231–235.
- Duke, G. E. 1997. Gastrointestinal physiology and nutrition in wild birds. Pages 1049–1056 in *Proceedings of the Nutrition Society*. The Nutrition Society, June 24–June 28, 1996, Ulster, Coleraine, UK.
- Feierabend, J. S., and O. Myers. 1984. A national summary of lead poisoning in bald eagles and waterfowl. National Wildlife Federation, Washington, D.C., USA.
- 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. 1996. Interpretation of tissue lead residues in birds other than waterfowl. Page 279 in W. N. Beyer, G. H. Heinz, and A. W. Redmon-Norwood, editors. *Environmental contaminants in wildlife: interpreting tissue concentrations*. Lewis, Boca Raton, Florida, USA.
- Friend, M., and C. Franson. 1999. Lead. Pages 175–189 in M. Friend and C. Franson, editors. *Field manual of wildlife diseases: general field procedures and diseases of birds*. U.S. Department of Interior, U.S. Geological Survey, Washington, D.C., USA.
- Grund, D. M., L. Cornicelli, L. T. Carlson, and E. A. Butler. 2010. Bullet fragmentation and deposition in white-tailed deer and domestic sheep. *Human-Wildlife Interactions* 4:257–265.
- Hoffman, D. J., O. H. Pattee, S. N. Wiemeyer, and B. Mulhern. 1981. Effects of lead shot ingestion on delta-aminolevulinic acid dehydratase activity, hemoglobin concentration, and serum chemistry in bald eagles. *Journal of Wildlife Diseases* 17:423–431.
- 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.
- Janssen, D. L., J. E. Oosterhuis, J. L. Allen, M. P. Anderson, D. G. Kelts, and S. N. Wiemeyer. 1986. Lead poisoning in free-ranging California condors. *Journal of the American Veterinary Medical Association* 155:1052–1056.
- Jennelle, C. S., M. D. Samuel, C. A. Nolden, and E. A. Berkley. 2009. Deer carcass decomposition and potential scavenger exposure to chronic wasting disease. *Journal of Wildlife Management* 73:655–662.
- Kelly, T. R., and C. K. Johnson. 2011. Lead exposure in free-flying turkey vultures is associated with big game hunting in California. *PloS One* 6:e15350.
- Kramer, J. L., and P. T. Redig. 1997. Sixteen years of lead poisoning in eagles, 1980-95: an epizootiologic view. *Journal of Raptor Research* 31:327–332.
- Krone, O., N. Kenntner, A. Trinogga, M. Nadjafzadeh, F. Scholz, J. Sulawa, K. Totschek, P. Schuck-Wersig, and R. Zieschank. 2009. Lead poisoning in white-tailed sea eagles: causes and approaches to solutions in Germany. Pages 289–301 in *Proceedings of the 2008 conference on ingestion of lead from spent ammunition: implications for wildlife and humans*. The Peregrine Fund, Boise, Idaho, USA.
- Lang, A. L., R. A. Andress, and P. A. Martin. 1999. Prey remains in bald eagle, *Haliaeetus leucocephalus*, pellets from a winter roost in the upper St. Lawrence River, 1996 and 1997. *Canadian Field-Naturalist* 113:621–626.
- Oltrogge, V. 2009. Success in developing lead-free, expanding-nose centerfire bullets. Pages 310–317 in *Proceedings of the 2008 conference on ingestion of lead from spent ammunition: implications for wildlife and humans*. The Peregrine Fund, Boise, Idaho, USA.
- Pain, D. J., I. J. Fisher, and V. G. Thomas. 2009. A global update of lead poisoning in terrestrial birds from ammunition sources. Pages 99–118 in *Proceedings of the 2008 conference on ingestion of lead from spent ammunition: implications for wildlife and humans*. The Peregrine Fund, Boise, Idaho, USA.
- Pattee, O. H., and S. K. Hennes. 1983. Bald eagles and waterfowl: the lead shot connection. *Proceedings of the North American Wildlife Conference* 48:230–237.
- 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.
- Pauli, J. N., and S. W. Buskirk. 2007. Recreational shooting of prairie dogs: a portal for lead entering wildlife food chains. *Journal of Wildlife Management* 71:103–108.
- Pokras, M., M. Kneeland, A. Ludi, E. Golden, A. Major, R. Miconi, and R. H. Poppenga. 2009. Lead objects ingested by common loons in New England. *Northeastern Naturalist* 16:177–182.

- Rattner, B. A., J. C. Franson, S. R. Sheffield, C. I. Goddard, N. J. Leonard, and P. J. Wingate. 2009. Technical review of the sources and implications of lead-based ammunition and fishing tackle to natural resources. Pages 68–70 *in* Proceedings of the 2008 conference on ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.
- Redig, P. T., and L. R. Arent. 2008. Raptor toxicology. *The Veterinary Clinics of North America. Exotic Animal Practice* 11:261–282.
- Redig, P. T., and L. Cruz-Martinez. 2009. Raptors. Pages 209–242 *in* T. N. Tully, G. M. Dorrestein, and A. K. Jones, editors. *Handbook of avian medicine*. Elsevier, New York, New York, USA.
- Saito, K. 2009. Lead poisoning of Steller sea-eagle (*Haliaeetus pelagicus*) and white-tailed eagle (*Haliaeetus albicilla*) caused by the ingestion of lead bullets and slugs, in Hokkaido Japan. Pages 302–309 *in* Proceedings of the 2008 conference on ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.
- Scheuhammer, A. M. 2009. Historical perspective on the hazards of environmental lead from ammunition and fishing weights in Canada. Pages 61–67 *in* Proceedings of the 2008 conference on ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.
- Sidor, I. F., M. A. Pokras, A. R. Major, R. H. Pop-penga, K. M. Taylor, and R. M. Miconi. 2003. Mortality of common loons in New England, 1987 to 2000. *Journal of Wildlife Diseases* 39:306–315.
- 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 U.S. Inland Pacific Northwest region—an 18-year retrospective study: 1991–2008. *Journal of Avian Medicine and Surgery* 24:279–287.
- Strom, S. M., J. A. Langenberg, N. K. Businga, and J. K. Batten. 2009. Lead exposure in Wisconsin birds. Pages 194–201 *in* Proceedings of the 2008 conference on ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.
- Stroud, R. K., and W. G. Hunt. 2009. Gunshot wounds: a source of lead in the environment. Pages 119–125 *in* Proceedings of the 2008 conference on ingestion of lead from spent ammunition: implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA.
- Swenson, J. E., K. L. Alt, and R. L. Eng. 1986. Ecology of bald eagles in the Greater Yellowstone Ecosystem. *Wildlife Monographs* 3–46.
- U. S. Department of Interior. 1986. Final supplemental environmental impact statement: use of lead shot for hunting migratory birds in the United States. Washington, D.C., USA.
- Wayland, M., and T. Bollinger. 1999. Lead exposure and poisoning in bald eagles and golden eagles in the Canadian Prairie provinces. *Environmental Pollution* 104:341–350.
-

LUIS CRUZ-MARTINEZ received his D.V.M. degree from the National University of Costa Rica.



He completed an internship and residency in raptor biomedicine at the Raptor Center of the University of Minnesota while pursuing an M.S. degree at the University of Minnesota. Currently he is a Ph.D. student at the Department of

Ecosystem and Public Health, Faculty of Veterinary Medicine, University of Calgary, Canada, focusing on inhalation toxicology of wild birds.

PATRICK T. REDIG is the founder and director emeritus of the Raptor Center at the University of Minnesota.



Currently, he is a professor of avian medicine and surgery at the College of Veterinary Medicine, University of Minnesota. His research interests include the impacts of lead poisoning on predator and scavenger

populations, surveillance and ecology of ortho and paramyxoviruses, and avian orthopedics.

JOHN DEEN is a professor in epidemiology in the College of Veterinary Medicine



at the University of Minnesota. He obtained his veterinary medicine degree and doctorate in epidemiology from the University of Guelph in Canada and has focused on swine disease and welfare measurement. He is the principal

investigator of the University of Minnesota's contribution to USAID's emerging pandemic threats program in RESPOND that involves a One Health effort at the university's academic health center, which includes the colleges of the nursing, public health, medicine, and veterinary medicine.