LEAD, MERCURY, SELENIUM, AND OTHER TRACE ELEMENTS IN TISSUES OF GOLDEN EAGLES FROM SOUTHWESTERN MONTANA, USA

Alan R. Harmata,[1,3] **and Marco Restani**[2]

[1] Ecology Department, Montana State University, Bozeman, Montana 59717, USA
[2] Department of Biological Sciences, St. Cloud State University, St. Cloud, Minnesota 56301, USA
[3] Corresponding author (email: ubijt@montana.edu)

ABSTRACT: Lead-based rifle bullets, used in game hunting and recreational shooting, fragment when striking bone and soft tissues. Lead fragments may be ingested by birds scavenging offal piles or nonretrieved carcasses and therefore pose a poisoning risk. We captured and sampled 74 Golden Eagles (*Aquila chrysaetos*) in southwestern Montana, USA, from 2008 to 2010 to evaluate levels of lead, mercury, selenium, and 13 other trace elements in blood and feathers. Lead was detected in blood of most (97%, $n=70$) eagles; mean blood level was 0.26 parts per million (ppm). Most eagles (65%) had background levels (<0.2 ppm), 29% had elevated levels (0.2–0.5 ppm), 13% had chronic levels (0.51–1.0 ppm), and 3% had acute levels (>1.0 ppm) in blood. Lead in blood decreased from winter to spring. Resident eagles had higher lead levels than eagles of unknown residency. Mercury was detected in few eagles, whereas selenium was detected in all, but at a low level (0.36 ppm). Other chemical elements in blood were at low or biologically appropriate levels. Lead in feathers ($n=29$) was correlated with blood lead ($P=0.010$), as was mercury in blood and feathers ($n=48$; $P=0.003$). Concentrations of lead and mercury in feathers were higher in adults than in juveniles and immatures ($P<0.016$) and both elements tended to increase with age. Selenium in feathers ($n=48$) appeared stable across plumage classes. Although detection rates of lead in blood of eagles captured in spring increased from 1985–1993 to 2008–2010, mean levels decreased ($P<0.023$) between periods, as did proportions of eagles exhibiting above background levels (>0.2 ppm; $P<0.02$).

Key words: Golden Eagle, lead, mercury, Montana, selenium, trace elements.

INTRODUCTION

Lead-based rifle bullets, used for harvesting game and recreational shooting of varmints, fragment when striking bone and soft tissues. These lead fragments, which can number in the hundreds per carcass, may be ingested by birds scavenging offal piles (Hunt et al., 2006) or nonretrieved carcasses (Knopper et al., 2006; Pauli and Buskirk, 2007). The most highly publicized example of such lead poisoning concerned deaths of reintroduced California Condors (*Gymnogyps californianus*; Church et al., 2006). These deaths led California to prohibit use of leaded bullets within the Condor's range (Kelly et al., 2011) and Arizona to encourage use of nonleaded bullets (Walters et al., 2010). Researchers hypothesized that lead-poisoned Golden Eagles (*Aquila chrysaetos*) may also have ingested bullet fragments while scavenging, because most were admitted to rehabilitation centers during or shortly after big game hunting seasons (Kramer and Redig, 1997; Wayland et al., 2003; Stauber et al., 2010). In fact, lead levels in Golden Eagles decreased after a ban on the use of lead ammunition within the range of Condors in southern California was imposed (Kelly et al., 2011).

During winters 2005–2007, six of 10 of Golden Eagles submitted to the Montana Raptor Conservation Center (MRCC), a raptor rehabilitation center in Bozeman, Montana, USA, exhibited blood concentrations of lead in excess of that considered "invariably fatal" (≥1.2 parts per million [ppm] wet wt.; Kramer and Redig, 1997). All were recovered between December and March and most recovered within 30 km of the Upper Missouri River watershed in southwestern Montana. Number and morbidity of eagles submitted during this period was unusual, and MRCC staff suspected an emerging problem with lead in the local environment. To

address this concern, we initiated a study in 2008 to investigate lead contamination in blood and feathers of wild Golden Eagles from southwestern Montana. We also screened eagle blood and feathers for mercury because, like lead, it is a nonessential element, is toxic, and has documented effects on avian health and reproduction (reviews by Eisler, 1987; Boening, 2000). Although mercury is of primary concern in aquatic environments (e.g., Scheuhammer and Graham, 1999), little is known of mercury contamination in predators associated with terrestrial habitats and mammalian prey. We also evaluated levels of selenium because of its detoxification properties for mercury (Yoneda and Suzuki, 1997; Berry and Ralston, 2008). Thirteen other trace elements biologically essential, beneficial, or toxic that are not normally reported in raptor contaminant studies were also analyzed (see Rodrigues et al., 2010).

METHODS

We captured Golden Eagles in southwestern Montana from January 2008 to April 2010. Eagles were captured in three areas: 1) the Upper Missouri River watershed within 11 km of the Madison River at its inlet to Ennis Lake to its confluence with the Missouri River, a distance of approximately 67 km (centered 45°39′3″N, 111°31′40″W); 2) within 3 km of the Gallatin River from the mouth of Gallatin Canyon to its confluence with the Missouri River, a distance of approximately 35 km (centered 45°39′49″N, 111°12′9″W); and 3) within a 10-km radius of the town of Ringling, Meagher County, Montana (45°16′18″N, 110°48′41″W).

We captured eagles with a remote-controlled Coda net launcher (Coda Enterprises, Mesa, Arizona, USA). The launcher fired 7.3×7.3-m polyester net with 10.2×10.2 cm mesh. Road-killed ungulate carcasses, mostly white-tailed deer (*Odocoileus virginianus*) and domestic bovine calf carcasses were used as bait.

We also used padded leghold traps (Lockhart method; Miner, 1975) during spring, with Richardson's ground squirrels (*Urocitellus richardsonii*) and white-tailed jackrabbits (*Lepus townsendii*) as bait. Occasionally, we used captive adult female and male Golden Eagles as lures to facilitate the timely visitation of trap sites.

Eagles captured between 1 January and 28 February each year were considered wintering and those captured after 1 March were considered vernal migrants. Eleven eagles captured as part of a wind power study were included in contaminant analysis and all were radio-tagged with ventrally tail-mounted, 65-g, 6-mo–life transmitters and wing notched for visual identification. Marked eagles observed after 15 April were considered resident.

We determined sex based on size (Harmata and Montopoli, 2013) and age based on tail feather coloration of eagles (Bloom and Clark, 2001). Age classes were juvenile, immature (formative and basic II plumage), subadult (basic III plumage), and adult. Eagles that possessed no more than one rectrix with subadult characteristics (white base) were classified as adult because breeding eagles occasionally display at least one subadult rectrix (Steenhof et al., 1983).

We collected 1 cc of whole blood from the brachial vein and 250–500 mg of feathers from the lower breast or abdomen of most captured eagles. Feathers were clipped within 2 cm of the skin with surgical scissors and deposited in plastic sandwich bags. All feathers were fully developed when collected. Blood and feather samples were refrigerated and shipped for analysis at the end of each year.

Analyses of eagle blood and feathers were conducted at Michigan State University, Veterinary Medical Center, Diagnostic Center for Population and Animal Health, Clinical Pathology Laboratory, East Lansing, Michigan, USA. Metals were analyzed by inductively coupled plasma mass spectrometry (ICPMS; Agilent 7500ce ICP-MS,

TABLE 1.   Concentrations (parts per million [ppm]) of lead (Pb), mercury (Hg), and selenium (Se) in blood and feathers of Golden Eagles (*Aquila chrysaetos*) captured (*n*=69) and recovered (*n*=5) in southwestern Montana, USA, 2008–2010.

Element	Tissue	*n*	% Detected[a]	$\bar{x}$ (SE)	GM[b]	Median	Range
Pb	Blood	70	96	0.26 (0.03)	0.16	0.16	0–1.36
	Feathers	29[c]	100	0.68 (0.09)	0.52	0.51	0.07–2.00
Hg	Blood	70	21	0.03 (0.08)	—	0.0	0–0.50
	Feathers	48	83	0.31 (0.36)	0.17	0.18	0–1.50
Se	Blood	70	100	0.36 (0.01)	0.35	0.33	0.20–0.74
	Feathers	48	100	0.89 (0.47)	0.80	0.73	0.28–2.90

[a] ≥5 parts per billion.

[b] Geometric mean; not calculated if <50% detection rate.

[c] Element tested in feathers only in 2009.

Santa Clara, California, USA; Goulle et al., 2005; Wahlen et al., 2005). Analytical methods followed those of Harmata (2011). Reportable quantization limits for metals were as follows: 1 part per billion (ppb) for lead, selenium, cobalt, iron, manganese, molybdenum, and zinc; 5 ppb for mercury, arsenic, cadmium, copper, and thallium; and 25 ppb for antimony, chromium, nickel, and vanadium.

We have presented arithmetic means ($\bar{x}$) and medians and proportions of contaminant levels for comparisons within and among groups. Nonparametric statistical tests with nontransformed data were used to compare elemental tissue concentrations among, between, or within groups. Mann-Whitney *U*-tests were used to test for differences between groups. Spearman rank order correlation tests were used to test relationships among categories. Contingency tables, χ^2 test of independence, and paired-sample proportion tests were used to test differences in frequencies or proportions. If generalized tests (Kruskal-Wallis analysis of variance, χ^2) detected differences among groups, multiple two-sample post-hoc tests (Mann-Whitney *U*, paired-sample proportion) were performed with Bonferroni adjustments for an appropriate *P* value (i.e., 0.05/*n* tests) to detect differences. All statistical tests were performed and graphics produced in various modules of STATISTICA ver. 6.1 (StatSoft, Inc., 2003). Although geometric means

(GMs) may be biased low (Parkhurst, 1998), we have presented these estimates to allow comparisons with other contaminant studies.

We statistically reanalyzed blood level results for lead, mercury, and selenium in Golden Eagles captured from 1985 to 1993 in west-central Montana (Harmata and Restani, 1995) to compare them with our 2008–2010 results. Only eagles captured between 1 March and 15 April during the present study were compared with those captured by Harmata and Restani (1995). Detections of elements below 0.1 ppm (limit for 1985–1993 analyses) in 2008–2010 were considered nondetections for comparison with 1985–1993 data. Chemical analysis methods differed between periods. Samples obtained between 1985 and 1993 were analyzed by graphite furnace atomic absorption spectroscopy (GFAAS) and those obtained between 2008 and 2010 were analyzed by ICPMS. Although Zhang et al. (1997) found ICPMS results tend to be 10% lower than GFAAS results in the analysis of lead in human blood, Bedrosian et al. (2009) found GFAAS and ICPMS results "directly comparable" for Golden Eagles. Further, Rose et al. (2001) found neither GFAAS nor ICPMS was more accurate than the other for determining concentrations of lead and mercury, whereas Forrer et al. (1998) found equivalency in accuracy and precision for both

methods for determining selenium in human and animal serum. Thus, results from both periods were considered comparable.

We categorized lead concentrations in blood as background (<0.2 ppm), elevated (0.2–0.5 ppm), chronic (0.51–1.0 ppm), and acute (>1.0 ppm). We avoided use of category names based on clinical veterinary experiences because we sampled free-flying Golden Eagles that were not injured and/or overtly expressing signs of lead poisoning, but retained ascending exposure level concentration ranges comparable to those found in other studies (Kramer and Redig, 1997; Stauber et al., 2010). Exposure levels for other trace elements in blood that potentially may affect health and reproduction have been established for some mammal and bird species (e.g., Burgess et al., 2005; Burger and Gochfeld, 2009) but not Golden Eagles.

RESULTS

We obtained blood and feather samples from 70 Golden Eagles (69 captures and one fresh [<1 hr] roadkill). Feather samples were also obtained within 2 days of death from four eagles killed by electrocution. Males and females were sampled equally ($n=37$). Most eagles sampled were juveniles ($n=22$), followed by adults ($n=20$), immatures ($n=17$), and subadults ($n=15$). Residency status for most eagles was unknown, but 69% of eagles were sampled in winter and 31% in spring. Transmitter detections and wing notch observations of flying eagles after 15 April indicated that at least four adults, two juveniles, and one subadult were year-round residents. All were captured during winter.

Lead, mercury, and selenium

No differences were found among years for detection rate and levels of lead, mercury, and selenium in blood of Golden Eagles ($P>0.06$). Lead was detected in blood of most eagles tested, mercury in
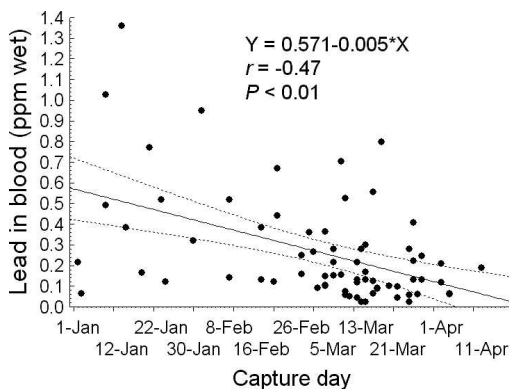

FIGURE 1. Lead concentrations (parts per million [ppm] wet wt.) in blood of Golden Eagles (*Aquila chrysaetos*) by capture day (2008–2010 combined), in southwestern Montana. Independent variable (X) in regression is number of days from 31 December.

very few, and selenium in all (Table 1). Mercury and selenium in blood were positively correlated (Spearman's $R=0.25$, $P=0.037$). No relationship existed between plumage class or sex and lead, mercury, or selenium levels in blood ($P>0.077$). Mean blood lead (Table 1) was above background, and 29% of eagles sampled exhibited elevated levels (0.2–0.5 ppm) and 13% exhibited chronic levels (>0.5–1.0 ppm). Although two eagles (3%), a resident male and resident female captured in winter, had lead in blood categorized as acute (>1.0 ppm), both appeared in excellent condition and showed no signs of debilitation or disease. Lead in blood of resident eagles ($n=7$; $\bar{x}=0.66$ ppm, SE=0.18, range 0.13–1.36 ppm) was higher than in those of unknown residency ($n=63$, $\bar{x}=0.23$ ppm, SE=0.03, range 0–0.80 ppm) ($Z=2.57$, $P=0.01$). Lead in blood tended to decrease from winter to spring both years (Fig. 1).

Only one Golden Eagle, an adult male, had a mercury level (0.50 ppm) in blood above that considered background in piscivorous birds (0.4 ppm; Eisler, 1987; Burgess et al., 2005). Only one eagle had a selenium blood level below background, but no eagles had levels above those considered toxic in birds (0.2 and 2.0 ppm, respectively; Eisler, 1985; Burgess et al., 2005).
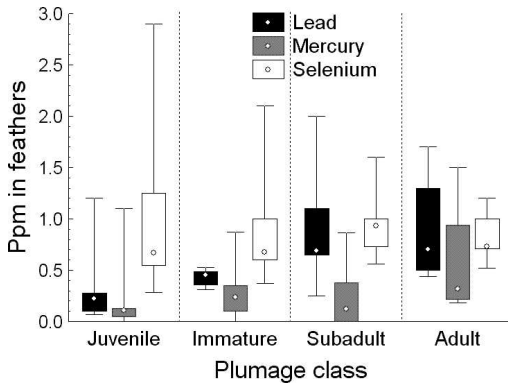
FIGURE 2. Median (point), minimum-maximum (whiskers), and quartile (central 50%) distribution (box) of lead, mercury, and selenium concentrations (parts per million [ppm] wet wt.) in feathers of Golden Eagles (*Aquila chrysaetos*) captured in southwestern Montana, USA, 2008–2010.

Lead in feathers was correlated with lead in blood (Spearman's $R=0.51$, $P=0.010$), and mercury levels in blood and feathers were also correlated (Spearman's $R=0.44$, $P=0.003$). Concentrations in feathers were higher in adults than juveniles and immatures for lead ($H_{3,29}=10.364$, $P<0.016$) and mercury ($H_{3,48}=11.685$, $P<0.008$), and both tended to increase with plumage class (i.e., age), whereas selenium levels appeared stable across plumage classes (Fig. 2). No differences were detected in lead, mercury, or selenium levels in feathers between unknown and known residents ($P>0.213$). Selenium in blood was not correlated with lead, mercury, or selenium in feathers ($P>0.109$).

Historical comparisons

Detection rates of lead and selenium in blood of Golden Eagles captured during this study were higher than rates for those captured in west-central Montana in 1985–1993 (two-sided proportion test, $P=0.018$), but mercury was detected at a similar rate ($P=0.42$) (Table 2). Lead levels were higher in 1985–1993 than in 2008–2010 (Mann-Whitney $Z=2.26$, $P<0.023$), but mercury and selenium levels were not different (Mann-Whitney $Z<0.55$, $P>0.582$) between sampling periods (Table 2). The proportion of eagles captured in spring exhibiting lead levels below background (<0.2 ppm) increased from the late 20th century (44%, $n=87$) to the early 21st century (67%, $n=48$) (two-sided proportion test, $P<0.02$). The proportion of eagles exhibiting elevated lead levels was lower in 2008–2010 than 1985–1993 (two-sided proportion test, $P=0.043$), but small sample size in chronic and acute categories prevented adequate statistical evaluation despite an apparent decline (Fig. 3).

Other blood contaminants

Other chemical elements in blood of Golden Eagles were at low or biologically appropriate concentrations (Table 3). No captured eagle exhibited signs of toxicity or teratogenic or mutagenic effects. We found no differences in blood concentrations of other chemical elements among

TABLE 2. Concentrations (parts per million [ppm]) of lead (Pb), mercury (Hg), and selenium (Se) in blood of Golden Eagles (*Aquila chrysaetos*) captured in Montana, USA, during spring (1 March–15 April) 1985–1993 (analysis by graphite furnace atomic absorption spectroscopy) and 2008–2010 (analysis by inductively coupled plasma mass spectrometry).

Element	Years	n	% Detected[a]	$\bar{x}$ (SE)	GM[b]	Median	Range
Pb	1985–1993	87	85	0.28 (0.03)	0.20	0.20	0–1.30
	2008–2010	48	100	0.19 (0.02)	0.14	0.13	0.04–0.80
Hg	1985–1993	69	25	0.12 (0.04)	—	0	0–2.30
	2008–2010	48	23	0.03 (0.01)	—	0	0–0.50
Se	1985–1993	78	87	0.43 (0.07)	0.28	0.30	0–5.00
	2008–2010	48	100	0.36 (0.01)	0.35	0.34	0.20–0.74

[a] ≥ 0.1 ppm.

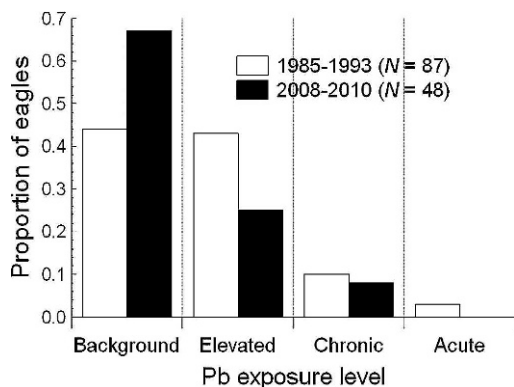[b] Geometric mean; not calculated if <50% detection rate.

FIGURE 3. Proportion of Golden Eagles (*Aquila chrysaetos*) exhibiting background (<0.2 parts per million [ppm]), elevated (0.2–0.5 ppm), chronic (0.51–1.0 ppm), and acute (>1.0 ppm) levels of lead (Pb) in blood that were captured and sampled during spring (1 March–15 April) in southwestern Montana, USA, in 1985–1993 (Pb analysis by graphite furnace atomic absorption spectroscopy) and 2008–2010 (analysis by inductively coupled plasma mass spectrometry). Proportions of eagles exhibiting background and elevated levels were different between periods ($P<0.05$).

plumage classes, between sexes, or among years ($P>0.05$).

DISCUSSION

Lead

The initial impetus for this study was to determine how lead in blood of debilitated Golden Eagles submitted to the MRCC raptor rehabilitation center during winter reflected profiles of contamination in the local, free-flying Golden Eagle population. The proportion of eagles in the highest exposure category in Montana from 2008 to 2010 was much lower than that reported from the Midwest (Kramer and Redig, 1997), Canadian prairie provinces (Wayland and Bollinger, 1999; Wayland et al., 2003), and Pacific Northwest (Stauber et al., 2010). Mean lead level in blood of Golden Eagles captured in southwestern Montana from 2008 to 2010 also was below that found during 1985–1993 (Harmata and Restani, 1995) but similar to levels from 1985–1986 in California (Pattee et al., 1990). Moreover, the proportion

of eagles in the background exposure category in Montana increased over time, with a reciprocal decrease in proportions in all higher exposure categories (elevated, chronic, acute). Our results from Montana may be more representative of lead levels within the general Golden Eagle population because we sampled free-flying individuals rather than those found debilitated, moribund, or dead, as was the case in the studies cited above and those treated by MRCC.

Profiles and timing of contamination in blood of captured eagles suggested that at least some contamination occurred in southwestern Montana. Two resident adults captured in winter were the only eagles sampled that exhibited acute levels of lead in blood, likely from lead fragments or projectiles ingested while feeding on unrecovered ungulate carcasses or discarded offal associated with hunting (e.g., Hunt et al., 2009).

Declining residues of lead in blood of Golden Eagles from winter to spring may indicate 1) a temporal difference in exposure or 2) a spatial difference in exposure manifested through population turnover during migration. Eagles captured from January through February may have recently arrived from northern areas with higher lead contamination or may have been residents exposed to a locally contaminated food base during autumn hunting seasons. Eagles captured in March may have arrived from southern areas with lower lead contamination or may have been residents that purged lead from their systems after exposure during or prior to spring. Reduction in lead in blood of vernal migrant Golden Eagles since the last century is encouraging and may be a function of bans of lead shot and programs promoting a voluntary switch from lead to copper ammunition for big game.

Feathers are important bioindicators of environmental contamination (Burger, 1994), and deposition of metals in developing feathers is important in excretion of

TABLE 3.   Concentrations (parts per million [ppm] wet wt.) of essential, beneficial, and toxic elements analyzed in blood and feathers of Golden Eagles (*Aquila chrysaetos*) captured (*n*=69) or recovered (*n*=5) in southwestern Montana, USA, 2008–2010. Beryllium[a] and thallium[a] were also tested but not detected in either tissue of any Golden Eagle. ND = none detected.

Element[b]	Tissue	*n*	% Detected	$\bar{x}$ (SE)	GM[c]	Median	Range
Sb[a]	Blood	44	ND				
	Feathers	25	8	0.10 (0.01)		0.01	0.08–0.11
As[a]	Blood	70	ND				
	Feathers	25	84	0.01 (0.01)	0.08	0.09	0.20–0.21
Cd[a]	Blood	70	ND				
	Feathers	25	8	0.09 (0.02)		0.09	0.10–0.02
Cr[d]	Blood	43	58	0.19 (0.03)	0.07	0.14	0.67–0.26
	Feathers	29	100	0.91 (0.04)	0.89	0.86	0.59–1.60
Co[d]	Blood	25	4			0.08[e]	
	Feathers	29	59	0.09 (0.01)	0.05	0.08	0.08–0.15
Cu[f]	Blood	25	100	0.19 (0.01)	0.18	0.20	0.25–0.28
	Feathers	29	100	8.40 (0.24)	8.23	8.40	6.00–11.00
Fe[f]	Blood	25	100	358 (4.42)	358	360	300–390
	Feathers	29	100	57 (8.12)	42.9	41.0	10.0–200.0
Mn[f]	Blood	25	100	0.125 (0.01)	0.11	0.10	0.30–0.23
	Feathers	29	100	5.30 (0.62)	4.62	5.30	1.50–13.0
Mo[f]	Blood	25	ND				
	Feathers	29	3[e]			0.07	
Ni[f]	Blood	44	18	0.08 (0.01)		0.07	0.03–0.15
	Feathers	29	100	0.33 (0.04)	0.27	0.24	0.09–1.10
V[a]	Blood	25	ND				
	Feathers	29	100	0.12	0.08	0.08	0.03–0.54
Zn[f]	Blood	25	100	5.65 (0.23)	5.55	5.40	4.00–7.90
	Feathers	29	100	119.2 (4.69)	116.5	120	64.0–180

[a] Toxic (element that forms poisonous soluble compounds and has no biologic role).

[b] Sb = antimony; As = arsenic; Cd = cadmium; Cr = chromium; Co = cobalt; Cu = copper; Fe = iron; Mn = manganese; Mo = molybdenum; Ni = nickel; V = vanadium; Zn = zinc.

[c] Geometric mean; not calculated if <50% detection rate.

[d] Considered a biologically beneficial element.

[e] Absolute value (*n*=1).

[f] Considered a biologically essential element.

total body burdens (Furness et al., 1986; Braune and Gaskin, 1987). Unless feather concentrations in Montana Golden Eagles are a result mobilization of lead deposited in bone during previous autumns and mobilized during periods of metabolic stress (breeding), concentrations in feathers provide a profile of seasonal contamination (Finkelstein et al., 2010) and are likely more representative of contamination in the local environment during summer when most feather development occurs (Ellis et al., 2006). We are unaware of any other studies reporting lead in feathers of Golden Eagles. However, Martinez-Lopez et al. (2004) reported mean lead level of 0.72 ppm in Booted Eagle (*Hieraaetus pennatus*) feathers from what they considered an unpolluted environment in Spain. That level is above mean lead feather concentration of Golden Eagles reported here and suggests summer environments of Montana Golden Eagles may be relatively clean.

Exposure to lead may not be exclusively from large-caliber bullet fragments. Lambertucci et al. (2011) found lead isotopic ratios in feathers of most highly contaminated Andean Condors (*Vultur gryphus*) most consistent with .22 rimfire and 20-gauge shotgun ammunition rather than large-caliber rifle ammunition. Recreational

shooting of lagomorphs and varmints (e.g., ground squirrels [*Urocitellus* spp.] and prairie dogs [*Cynomys* spp.]) outside of traditional hunting seasons is common in Montana (Harmata and Restani, 1995) and carcasses are commonly left unrecovered (Knopper et al., 2006; Pauli and Buskirk, 2007). Leaded .22-caliber rimfire and smooth bore ammunition are most often used (legally) for this activity and thus may be equally (or more) culpable as large-caliber ammunition for wide-scale lead contamination of Montana eagles.

Overwhelming evidence indicates lead-containing ammunition is the proximate source of lead in blood and feathers of eagles (Redig et al., 2009) and other scavengers studied (Finkelstein et al., 2010; Lambertucci et al., 2011). Half-life of lead in human blood is approximately 36 days (Todd et al. 1996) and possibly 14 days in Common Ravens (*Corvus corax*; Craighead and Bedrosian, 2008). If Golden Eagle blood chemistry is similar, the high incidence of detection reported here suggests nearly every eagle had been exposed to environmental lead within 1 mo of sampling, with possibly 44% exposed within 2 wk. Unless prior lead contamination predisposes eagles to visit carcass-baited traps, it seems unlikely all had fed on unrecovered animals or offal piles containing lead fragments. Isotopic analysis of feather lead content of otherwise healthy, free-flying eagles may confirm source of lead. Continued monitoring of lead in Golden Eagles along with compliance with lead ammunition bans and use of recommended alternatives may indicate the efficacy of such practices on reducing lead in Golden Eagles.

Mercury and selenium

Absence of mercury in blood of most Golden Eagles and low levels in the few in which it was detected suggested the local terrestrial environment that provided food and water may be relatively free of mercury. Moreover, mercury levels have not changed over nearly three decades.

Geometric mean mercury level in feathers was 0.16 ppm, which was considerably lower than the level found in feathers of Bald Eagles (*Haliaeetus leucocephalus*) captured in the same area during the same time period (>13 ppm, $n=91$; Harmata, 2011). Bowerman et al. (1994) considered mercury detected in Bald Eagle feathers in the Great Lakes (GM=21.1 ppm, range: 3.6–48) as merely "elevated." Wood et al. (1996) considered feather GM level of 3.23 ppm ($n=61$) in Florida Bald Eagle nestlings as "background."

None of the captured Golden Eagles exhibited symptoms of selenium poisoning, and although all had levels above background for selenium in blood of birds studied (>0.20 ppm; Eisler, 1985), all were under the level considered toxic for waterfowl and quail (*Coturnix* spp.) (2.0 ppm; Eisler, 1985). Selenium levels in both blood and feathers of Golden Eagles (Table 1) were half or less of those found in Bald Eagles captured in the same area during the same time period (GM=~0.7 ppm, $n=88$, and GM=~1.6 ppm, $n=91$, respectively; Harmata, 2011). Selenium is considered efficacious for detoxifying mercury in birds (Yoneda and Suzuki, 1997; Berry and Ralston, 2008) and lower selenium may further reflect the relative absence of mercury in the diet of Golden Eagles. Moderate blood selenium levels over time suggested that the values reported from Montana may be normal for healthy Golden Eagles.

ACKNOWLEDGMENTS

LITERATURE CITED

Bedrosian B, Parish CN, Craighead D. 2009. Differences between blood lead level detection techniques: Analysis within and among three techniques and four avian species. In: *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*, Watson RT, Fuller M, Pokras M, Hunt WG, editors. The Peregrine Fund, Boise, Idaho, pp. 287–288.

Berry MJ, Ralston NVC. 2008. Mercury toxicity and the mitigating role of selenium. *EcoHealth* 5:456–459.

Bloom PH, Clark WS. 2001. Molt and sequence of plumages of Golden Eagles and a technique for in-hand ageing. *N Am Bird Bander* 26:97–116.

Boening DW. 2000. Ecological effects, transport, and fate of mercury: A general review. *Chemosphere* 40:1335–1351.

Bowerman WW. IV, Evans ED, Giesy JP, Postupalsky S. 1994. Using feathers to assess risk of mercury and selenium to Bald Eagle reproduction in the Great Lakes region. *Arch Environ Contam Toxicol* 27:294–298.

Braune BW, Gaskin DE. 1987. Mercury levels in Bonaparte's gull (*Larus philadelphia*) during autumn molt in the Quoddy region, New Brunswick, Canada. *Arch Environ Contam Toxicol* 16:539–549.

Burger J. 1994. Metals in avian feathers: Bioindicators of environmental pollution. *Rev Environ Toxicol* 5:203–311.

Burger J, Gochfeld M. 2009. Comparison of arsenic, cadmium, chromium, lead, manganese, mercury and selenium in feathers in Bald Eagles (*Haliaeetus leucocephalus*), and comparison with Common Eider (*Somateria mollissima*), Glaucous-winged Gull (*Larus glaucescens*), Pigeon Guillemot (*Cepphus columba*), and Tufted Puffin (*Fratercula cirrhata*) from the Aleutian

Chain of Alaska. *Environ Monit Assess* 152:357–367.

Burgess NM, Evers DC, Kaplan JD. 2005. Mercury and other contaminants in Common Loons breeding in Atlantic Canada. *Ecotoxicology* 14:241–252.

Church ME, Gwiazda R, Risebrough RW, Sorenson K, Chamberlain CP, Farry S, Heinrich W, Rideout BA, Smith DR. 2006. Ammunition is the principal source of lead accumulated by California Condors re-introduced to the wild. *Environ Sci Technol* 40:6143–6150.

Craighead D, Bedrosian B. 2008. Blood lead levels of Common Ravens with access to big-game offal. *J Wildl Manag* 72:240–245.

Eisler R. 1985. Selenium hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 85(1.5), Contaminant Hazard Reviews, Report No. 5, US Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland, 41 pp.

Eisler R. 1987. Mercury hazards to fish, wildlife and invertebrates: A synoptic review. Biological Report 85 (1.10), Report No. 10, US Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland, 63 pp.

Ellis DH, Lish JW, Kery M, Redpath SM. 2006. Short-term oscillations in avian molt intensity: Evidence from the Golden Eagle *Aquila chrysaetos*. *J Avian Biol* 37:642–644.

Finkelstein ME, George D, Scherbinski S, Gwiazda R, Johnson M, Burnett J, Brandt J, Lawrey S, Pessier AP, Clark M, et al. 2010. Feather lead concentrations and $^{207}Pb/^{206}Pb$ ratios reveal lead exposure history of California Condors (*Gymnogyps californianus*). *Environ Sci Technol* 44:2639–2647.

Forrer R, Gautschi K, Stroh A, Lutz H. 1998. Direct determination of selenium and other trace elements in serum samples by ICP-MS. *J Trace Elem Med Biol* 12:240–247.

Furness RW, Muirhead SJ, Woodburn M. 1986. Using bird feathers to measure mercury in the environment: Relationship between mercury content and moult. *Mar Pollut Bull* 17:27–37.

Goulle JP, Mahieu L, Castermant J, Neveu N, Bonneau L, Laine G, Bouige D, Lacroix C. 2005. Metal and metalloid multi-elementary ICP-MS validation in whole blood, plasma, urine and hair. *Forensic Sci Int* 153:39–44.

Harmata AR. 2011. Environmental contaminants in tissues of Bald Eagles sampled in southwestern Montana, 2006–2008. *J Raptor Res* 45:119–135.

Harmata AR, Montopoli G. 2013. Morphometric sex determination of North American Golden Eagles. *J Raptor Res* 47, In press.

Harmata AR, Restani M. 1995. Environmental contaminants and cholinesterase in blood of

vernal migrant Bald and Golden Eagles in Montana. *Intermt J Sci* 1:1–15.

Hunt WG, Burnham W, Parish CN, Burnham KK, Mutch B, Oaks JL. 2006. Bullet fragments in deer remains: Implications for lead exposure in avian scavengers. *Wildl Soc Bull* 34:167–170.

Hunt WG, Watson RT, Oaks JL, Parish CN, Burnham KK, Tucker RL, Beltoff JR, Hart G. 2009. Lead bullet fragments in venison from rifle-killed deer: Potential for human dietary exposure. In: *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*, Watson RT, Fuller M, Pokras M, Hunt WG, editors. The Peregrine Fund, Boise, Idaho, pp. 144–153.

Kelly TR, Bloom PH, Torres SG, Hernandez YZ, Poppenga RH, Boyce WM, Johnson CK. 2011. Impact of the California lead ammunition ban on reducing lead exposure in Golden Eagles and Turkey Vultures. *PLoS ONE* 6:e17656.

Knopper LD, Mineau P, Scheuhammer AM, Bond DE, Mckinnon DT. 2006. Carcasses of shot Richardson's ground squirrels may pose lead hazards to scavenging hawks. *J Wildl Manag* 70:295–299.

Kramer JL, Redig PT. 1997. Sixteen years of lead poisoning in eagles, 1980–95: An epizootiologic view. *J Raptor Res* 31:327–332.

Lambertucci SA, Donázar JA, Huertas DAD, Jiménez B, Sáez M, Sanchez-Zapata JA, Hiraldo F. 2011. 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.

Martinez-Lopez E, Martínez JE, María-Mojica P, Peñalver J, Pulido M, Calvo JF, García-Fernández AJ. 2004. Lead in feathers and delta-aminolevulinic acid dehydratase activity in three raptor species from an unpolluted Mediterranean forest (southeastern Spain). *Arch Environ Contam Toxicol* 47:270–275.

Miner NR. 1975. Montana Golden Eagle removal and translocation project. In: G*reat Plains wildlife damage control workshop proceedings*. University of Nebraska, Lincoln, Nebraska, 8-11 December 1975. Paper 201, pp. 155–162. digitalcommons.unl.edu/gpwdcwp/201/. Accessed October 2012.

Parkhurst DF. 1998. Arithmetic versus geometric means for environmental concentration data. *Environ Sci Technol* 32:92A–98A.

Pattee OH, Bloom PH, Scott JM, Smith MR. 1990. Lead hazards within the range of the California Condor. *Condor* 92:931–937.

Pauli JN, Buskirk SW. 2007. Recreational shooting of prairie dogs: A portal for lead entering wildlife food chains. *J Wildl Manag* 71:103–108.

Redig PT, Smith DR, Cruz-Smith L. 2009. Potential sources of lead exposure for Bald Eagles: A retrospective study. In: *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*, Watson RT, Fuller M, Pokras M, Hunt WG, editors. The Peregrine Fund, Boise, Idaho, pp. 210–218.

Rodrigues SM, Henriques B, Ferreira Da Silva E, Pereira ME, Duarte AC, Römkens PFAM. 2010. Evaluation of an approach for the characterization of reactive and available pools of twenty potentially toxic elements in soils: Part I—The role of key soil properties in the variation of contaminants' reactivity. *Chemosphere* 81:1549–1559.

Rose M, Knaggs M, Owen L, Baxter M. 2001. A review of analytical methods for lead, cadmium, mercury, arsenic and tin determination used in proficiency testing. *J Anal At Spectrom* 16:1101–1106.

Scheuhammer AM, Graham JE. 1999. The bioaccumulation of mercury in aquatic organisms from two similar lakes with differing pH. *Ecotoxicology* 8:49–56.

StatSoft, Inc. 2003. STATISTICA (data analysis software system), version 6.0, http://www.statsoft.com. Accessed January 2011.

Stauber E, Finch N, Talcott PA, Gay JM. 2010. Lead Poisoning of Bald (*Haliaeetus leucocephalus*) and Golden Eagles (*Aquila chrysaetos*) in the US Inland Pacific Northwest region—An 18-year retrospective study: 1991–2008. *J Avian Med Surg* 24:279–287.

Steenhof K, Kochert MN, Doremus JH. 1983. Nesting of subadult Golden Eagles in southwestern Idaho. *Auk* 100:743–747.

Todd AC, Wetmur JG, Moline JM. 1996. Unraveling the chronic toxicity of lead: An essential priority for environmental health. *Environ Health Perspecy* 104 (suppl 1): 141–146.

Wahlen R, Evans L, Turner J, Hearn R. 2005. The use of collision/reaction cell ICP-MS for the determination of elements in blood and serum samples. *Spectroscopy* 20:84–90.

Walters JR, Derrickson SR, Fry DM, Haig SM, Marzluff JM, Wunderle JM. 2010. Status of the California Condor (*Gymnogyps californianus*) and efforts to achieve its recovery. *Auk* 127:969–1001.

Wayland M, Bollinger T. 1999. Lead exposure and poisoning in Bald Eagles and Golden Eagles in the Canadian prairie provinces. *Environ Pollut* 104:341–350.

Wayland M, Wilson LK, Elliott JE, Miller MJR, Bollinger T, Mcadie M, Langelier K, Keating J, Froese JMW. 2003. Mortality, morbidity, and lead poisoning of eagles in western Canada, 1986–98. *J Raptor Res* 37:8–18.

Wood PB, White JH, Steffer A, Wood JH, Facemire CF, Percival HF. 1996. Mercury concentrations in tissues of Florida Bald Eagles. *J Wildl Manag* 60:178–185.

Yoneda S, Suzuki KT. 1997. Detoxification of mercury by selenium by binding of equimolar

Hg-Se complex to a specific plasma protein. *Toxicol Appl Pharmacol* 143:274–280.

Zhang ZW, Shimbo S, Ochi N, Eguchi M, Watanabe T, Moon CS, Iked M. 1997. Determination of lead and cadmium in food and blood by inductively coupled plasma mass spectrometry: A comparison with graphite furnace atomic absorption spectrometry. *Sci Total Environ* 205:179–187.