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Lead Poisoning of Bald (*Haliaeetus leucocephalus*) and Golden (*Aquila chrysaetos*) Eagles in the US Inland Pacific Northwest Region—An 18-year Retrospective Study: 1991–2008

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Abstract: To determine risk factors and seasonal trends of lead poisoning in bald (*Haliaeetus leucocephalus*) and golden (*Aquila chrysaetos*) eagles, blood lead levels were evaluated in eagles admitted from the inland Pacific Northwest region of the United States to the Raptor Rehabilitation Program, College of Veterinary Medicine at Washington State University from 1991 to 2008. Admissions were from Washington (32 bald eagles, 27 golden eagles), northern Idaho (21 bald eagles, 25 golden eagles), northeastern Oregon (5 bald eagles, 6 golden eagles), Montana (2 bald eagles), Alaska (1 bald eagle), and unrecorded (6 bald eagles, 5 golden eagles). In these birds, 48% (22/46) of bald and 62% (31/50) of golden eagles tested had blood lead levels considered toxic by current standards. Of the bald and golden eagles with toxic lead levels, 91% (20/22) and 58% (18/31) respectively, were admitted after the end of the general deer and elk hunting seasons in December. Coyote hunting intensifies with the end of the large game hunting seasons and coyote carcasses left in the field and contaminated with lead bullet fragments become readily available food sources, exposing scavenging bald and golden eagles to high risk of acute lead poisoning.

Key words: lead poisoning, coyote, bullet fragmentation, hunting ammunition, blood lead levels, inland Pacific Northwest, avian, bald eagle, *Haliaeetus leucocephalus*, golden eagle, *Aquila chrysaetos*, California condor, *Gymnogyps californianus*

Introduction

Lead poisoning from ingestion of hunting bullet fragments by avian scavengers such as bald eagles (*Haliaeetus leucocephalus*), golden eagles (*Aquila chrysaetos*), and California condors (*Gymnogyps californianus*) in many regions of the United States is of great concern to raptor biologists as well as to state and federal wildlife agencies.[1–7] It is widely accepted, with strong supporting evidence, that large game animals shot but not retrieved or lead-contaminated game animal offal discarded by hunters is a major and continuing source of lead poisoning in nontarget wildlife species.[3,5,7,8]

The purpose of this report is to provide information on seasonal trends and risk factors associated with lead poisoning in eagles as it relates to big game fall hunting seasons and winter shooting of coyotes in the inland Pacific Northwest region of the United States. Here we present data spanning an 18-year (1991–2008) retrospective study of lead poisoning in bald and golden eagles admitted to the Raptor Rehabilitation Program, Washington State University, College of Veterinary Medicine, Pullman, WA, from eastern Washington, northern Idaho, northeastern Oregon, western Montana, and Alaska.

Materials and Methods

Source of animals and sample collection

For several decades the Washington State University Raptor Rehabilitation Program has

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Figure 1. Immature (top) and adult (bottom) golden eagles with lead poisoning. Blood lead levels were 2.9 ppm (290 μg/dL; 14.1 μmol/L) and 0.87 ppm (87 μg/dL; 4.22 μmol/L) respectively. Clenched toes, inability to stand, and profound depression are evident. Both eagles were admitted to the Raptor Rehabilitation Program, Washington State University, in January from widely separated locations.

accepted injured and sick raptors from the US inland Pacific Northwest—which comprises eastern Washington, northern Idaho, northeastern Oregon, and western Montana—with the primary goal of rehabilitating and releasing them back into the wild. From 1991 to 1997, the policy was to test only eagles with clinical signs suggestive of lead poisoning (eg, depression, paralysis, or weakness of legs or wings) but no other clinical abnormalities (eg, gunshot wounds, electrical burns, trauma) (Figs 1 and 2). In 1998, we decided to screen all eagles admitted to the Raptor Rehabilitation Program for blood lead concentrations. From 1991 to 1997, 21 bald and 20 golden eagles were admitted; 6 and 11 birds, respectively, were tested. From 1998 to 2008, 46 bald and 43 golden eagles were admitted; 40 and 39 birds, respectively, were tested.

We radiographed all eagles to screen for metallic objects (lead) in the gastrointestinal tracts and obtained 3 rifle-shot coyote carcasses from a taxidermist to demonstrate bullet fragmentation and fragment dispersion by fluoroscopy.

Toxicologic testing

Determination of lead concentrations in fresh blood samples was done by the Analytical Sciences Laboratory, University of Idaho, Moscow, ID, USA.

We recorded 4 categories of blood lead levels by state, species, and quarter of year admitted. Blood lead levels of <0.2 ppm (<20 μg/dL; <0.97 μmol/L) were considered to be background and of no clinical significance; levels between 0.2 and 0.5 ppm (20–50 μg/dL; 0.97–2.43 μmol/L) were diagnosed as subclinical; levels between 0.51 and 1.00 ppm (51–100 μg/dL; 2.47–4.85 μmol/L) were classified as clinical lead poisoning; levels >1.00 ppm (>100 μg/dL; >4.85 μmol/L) were classified as severe clinical poisoning with recovery after treatment rare. Background and subclinical blood lead levels indicate chronic exposure whereas clinical and severe clinical poisoning indicate acute exposure.[1,2]

Data collection and statistical analysis

We identified all eagle admissions between 1991 and 2008 from case files of the Raptor Rehabil-

Figure 2. Mature bald eagle with lead poisoning admitted to the Raptor Rehabilitation Program, Washington State University, in February. The blood lead level was 2.3 ppm (230 μg/dL; 11.2 μmol/L). Fixed stare, unkempt feathers, and depression are pronounced.

itation Program and entered information from each record into a computer spreadsheet (Excel, Microsoft Inc, Redmond, WA, USA). Descriptive statistics and chi-square analysis were performed with Statistix 7.0 (Analytical Software, Tallahassee, FL, USA). Cases missing a value for a particular comparison were omitted from that analysis but were otherwise included.

Results

In the period from 1991 to 1997, the Raptor Rehabilitation Program admitted 21 bald and 20 golden eagles, and from 1998 to 2008, 46 bald and 43 golden eagles. Of these, 46 bald and 50 golden eagles were tested for blood lead levels. Eagles with toxic blood lead levels came from Washington (11 bald eagles, 18 golden eagles), northern Idaho (7 bald eagles, 11 golden eagles), northeastern Oregon (2 bald eagles, 2 golden eagles), Alaska (1 bald eagle), and unknown origin (1 bald eagle) (Figs 3 and 4).

Before the testing policy change, a higher proportion of golden eagles tended to have toxic blood lead levels than bald eagles (Fisher exact test, $P = .11$) but after the policy change the proportions were similar ($P = .65$) (Table 1). Of the eagles with background blood lead levels (<0.20 ppm; <20 μg/dL; <0.97 μmol/L) currently considered inconsequential, 6 bald and 10 golden eagles had blood lead concentrations ranging from 0.10 to 0.19 ppm (10–19 μg/dL; 0.48–0.92 μmol/L).

The admission of bald and golden eagles to the Raptor Rehabilitation Program was strongly seasonal (Fig 5). Of the bald eagles, 36 (54%) were admitted in the first quarter, (January–March), 14 (21%) in the second (April–June), 9 (13%) in the third (July–September), and 8 (12%) in the fourth (October–December). Of the golden eagles, 28 (44%) were admitted in the first, 5 (8%) in the second, 14 (22%) in the third, and 16 (25%) in the fourth quarter. Significantly more bald than golden eagles were admitted during the first 2 quarters than the last 2 ($P = .03$). Of the 22 bald eagles with subclinical, clinical, or severe clinical blood lead levels (n = 12, 3, and 7, respectively), 20 (91%) were admitted January–March; of the 31 golden eagles with subclinical, clinical, and severe clinical blood lead levels (n = 15, 5, and 11, respectively), 18 (58%) were admitted January–March and 10 (32%) October–December. For the 40 tested bald eagles admitted after the testing policy change, admission in the first quarter was significantly associated with toxic blood lead levels ($P = .03$) but the associations were not significant for the 39 golden eagles tested ($P = .08$).

None of the eagles had radiographic evidence of ingested lead particles in their gastrointestinal tracts.

Lead fragments numbering into the hundreds of pieces in 1 of 3 rifle-shot coyote carcasses to less than 10 pieces in another carcass were visualized by fluoroscopy. Lead fragments were

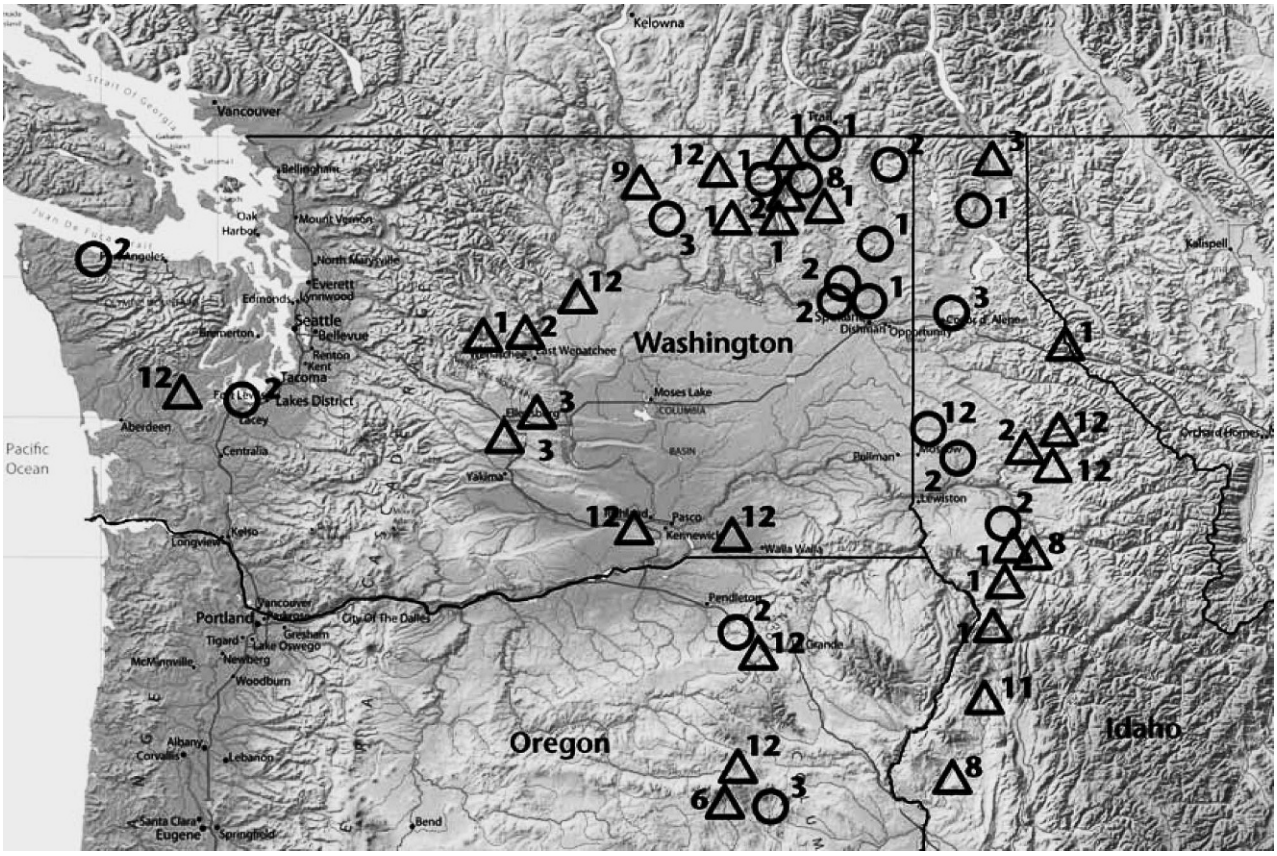

Figure 3. Known locations of bald and golden eagles from Washington, Idaho, and Oregon with lead poisoning submitted to the Washington State University Raptor Rehabilitation Program, 1991–2008. Circles indicate bald eagles; triangles, golden eagles; and numbers, month of hospital admittance.

widely distributed in the chest and abdominal cavities without obvious skeletal impact by the bullet (Fig 6).

Discussion

Lead poisoning in bald and golden eagles and other scavengers continues to be a major problem over wide regions of the United States.[1–4] To understand the risk factors, extent, and seasonality of lead poisoning in eagles in the US inland Pacific Northwest region, we undertook an 18-year retrospective study (1991–2008) of bald and golden eagles admitted to the Washington State University Raptor Rehabilitation Program. In the first 7 years (1991–1997) only eagles with clinical signs of lead poisoning (depression, paralysis, weakness of legs or wings) but without evidence of physical injuries were tested for blood lead levels. From 1998–2008, blood lead levels of all eagles admitted were determined whenever possible.

Our findings agree with others that lead poisoning of eagles is a major problem during the elk and deer hunting seasons from October to December.[4–8] Additionally our data document that the risk of lead poisoning of eagles extends beyond the big game hunting seasons into the first quarter of the following year. The small number of eagles tested from 1991 to 1997 (6 of 21 bald and 11 of 20 golden eagles) gave the impression that golden eagles (9 of 11) were at higher risk for lead poisoning than bald eagles (2 of 6). It also appeared that most cases of lead poisoning in bald (2 of 2) and golden eagles (8 of 9) occurred in the first quarter of the year. Extending the testing to all eagles admitted after 1998 gave us a clearer picture of the quarterly prevalence of lead poisoning in bald and golden eagles and allowed us to draw conclusions about the seasonal occurrence and differences between the 2 species.

Our documentation of the seasonal prevalence (October–March) of blood lead levels consistent with lead poisoning suggests that ingestion of ammunition lead is the major source. The temporal clustering of bald eagles (20 of 22, 91%) and the continued high prevalence of golden eagles (18 of 31, 58%) with toxic blood lead levels after the end

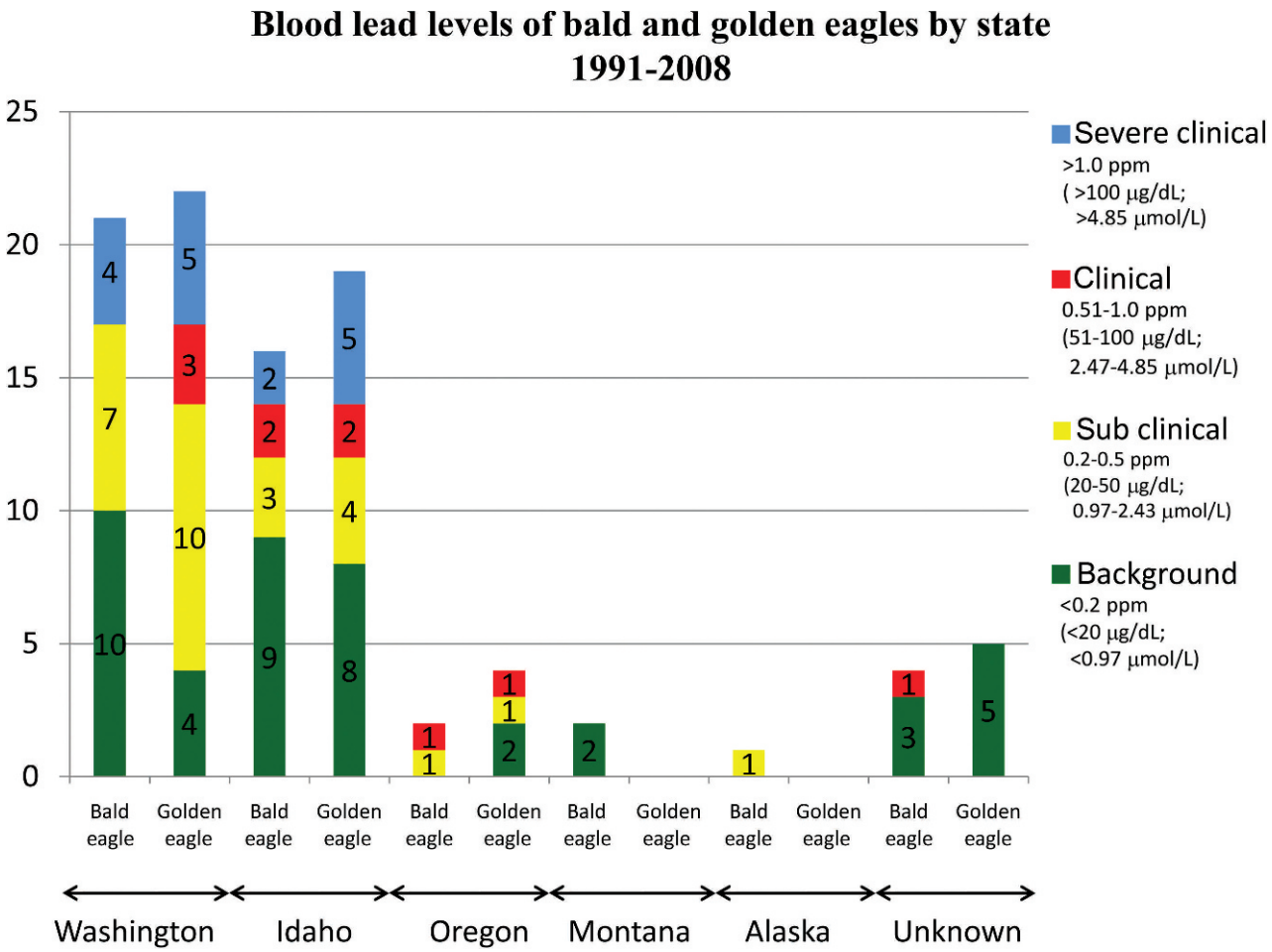

Figure 4. Blood lead levels in bald and golden eagles admitted to the Washington State University Raptor Rehabilitation Program, 1991–2008, by state.

of the big game hunting seasons specifically points to the importance of shot coyotes in eagle poisoning from January to March.

Although golden eagles are common throughout the region year round, bald eagles are not regularly seen far from water until bodies of water freeze. This forces bald eagles from a predominantly fish and waterfowl diet to forage over terrestrial habitat where rifle-shot coyotes become a ready food source. It is unlikely that elk and deer remains, covered by winter snow, are important sources of lead for this time of year.

Table 1. Blood lead levels of bald eagles and golden eagles admitted to the Washington State University Raptor Rehabilitation Program, 1991–1997 and 1998–2008.

	1991–1997		1998–2008	
	Bald eagles	Golden eagles	Bald eagles	Golden eagles
Birds admitted	21	20	46	43
Birds tested	6	11	40	39
Lead level[a]				
Background	4 (66%)	2 (18%)	20 (50%)	17 (44%)
Subclinical	2 (33%)	4 (36%)	10 (25%)	11 (28%)
Clinical	_	1 (10%)	3 (8%)	4 (10%)
Severe clinical	_	4 (36%)	7 (17%)	7 (18%)

[a] Background = <0.2 ppm (20 µg/dL; 0.97 µmol/L); subclinical = 0.2–0.5 ppm (20–50 µg/dL; 0.97–2.43 µmol/L); clinical = 0.51–1.0 ppm (51–100 µg/dL; 2.47–4.85 µmol/L); severe clinical = >1.0 ppm (>100 µg/dL; >4.85 µmol/L).

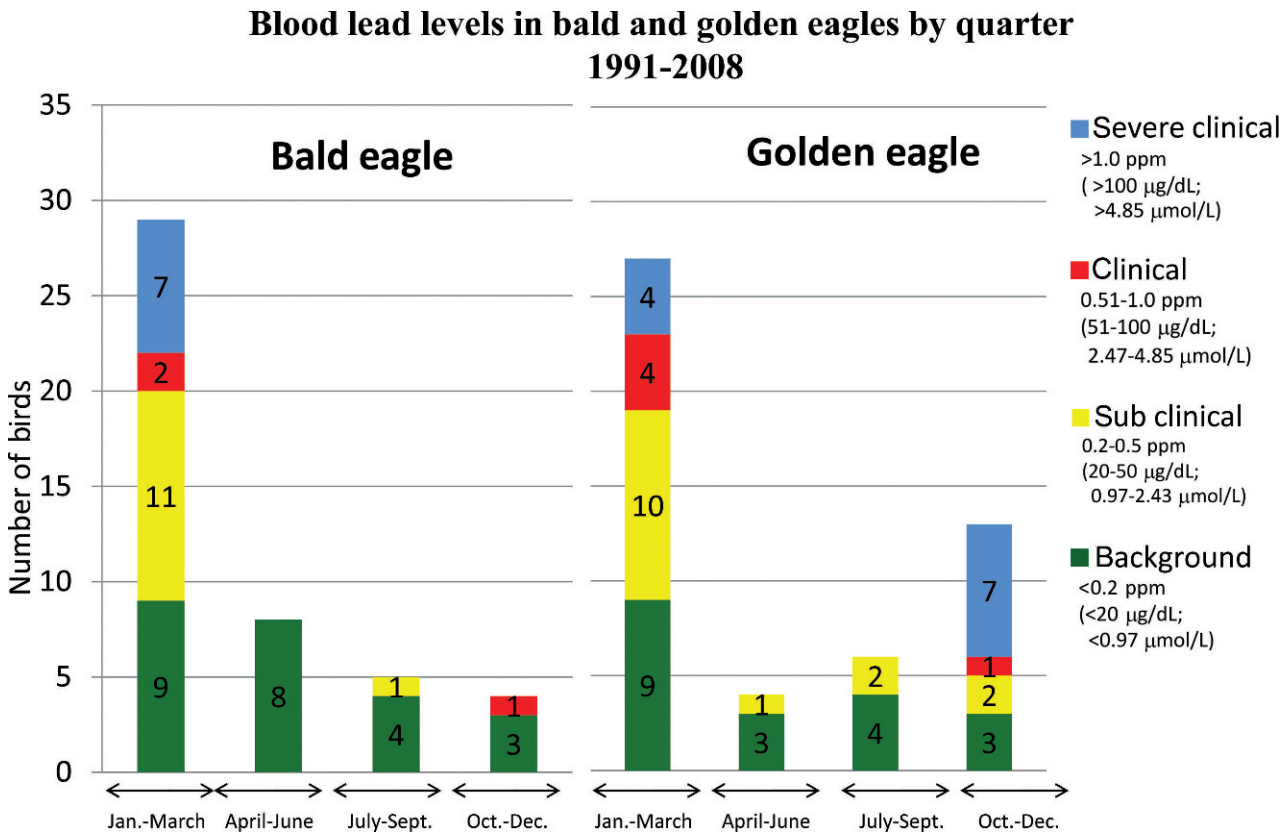

Figure 5. Blood lead levels in bald and golden eagles admitted to the Washington State University Raptor Rehabilitation Program, 1991–2008, by annual quarter.

After December, when big game hunting ends, the shooting of coyotes intensifies. Less caution on the part of coyotes during the mating season, combined with greater visibility for hunters when snow covers the ground and hunters' habit of shooting coyotes from motorized vehicles, make them easy targets for predator hunters. (W. Westberg, oral communication, February 2008; L. Tompkins, written communication, March 2008; T. Grimm, C. Hickey, written communication, November 2008; M. Flamm, written communication, February 2009).

Personal observations of shot coyotes left in open fields and fed on by eagles as well as statements by ranchers who observe eagles feeding on winter-shot coyote carcasses support the view that coyote shooting contributes to lead poisoning of eagles during this time period. Additional risks for lead poisoning in eagles arise as the snow melts and rodents emerge from hibernation and are shot for recreational purposes.[6,9] One biologist from Montana reported seeing 19 bald eagles on the ground in a pasture near their roosting place where 1.5 hours previously men had been shooting ground squirrels. (G. Pampush, written communication, March 2008.)

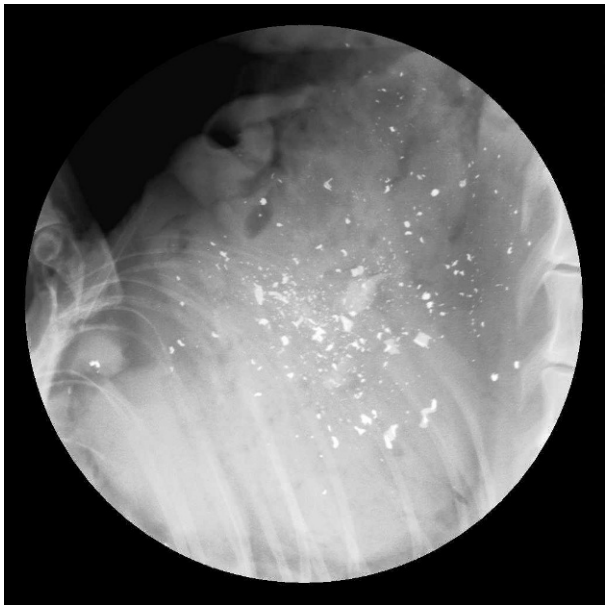

Figure 6. Visible lead fragments in carcass of rifle-shot coyote scanned by fluoroscopy. Note the extensive fragmentation without visible impact on bone.

Figure 7. Carcass of an adult female golden eagle pointed to by a hunting dog in the sagebrush desert in central Washington in mid-January. Lead analysis of tissues were 4.8 ppm (480 μg/dL; 23.3 μmol/L) (wet weight) for liver, 1.8 ppm (180 μg/dL; 8.7 μmol/L) (wet weight) for kidneys, and 28.0 ppm (2800 μg/dL; 135.8 μmol/L) (dry weight) for bone, indicating severe clinical poisoning.[11] Photo courtesy of Steve deJong.

The risk of lead poisoning for scavenging eagles depends on the amount of lead in animal remains or carcasses. With game, the sources of lead are either shot animals not retrieved or, more commonly, tissues (ie, entrails, heart, lungs, liver, blood-shot meat) discarded by hunters after field dressing. Lead contamination of those tissues can be expected because bullets expand widely along wound channels to a diameter of 40 cm around the bullet path, and fragments become embedded in tissues not kept for human use.[8,10] Animals shot with varmint bullets designed to fully disintegrate in their target without leaving the body, as seen in the radiograph of the rifle-shot coyote carcass with extensive bullet fragmentation and dispersal, undoubtedly pose a much greater risk of lead poisoning because the entire carcass is available for consumption.[11] Lead particles imbedded in the food source are readily ingested by scavengers, resulting in the formation and absorption of lead salts before toxic effects become evident. Ingested lead is rarely seen in the digestive tract by radiography or retrieved from eagles with lead poisoning, but a direct cause-effect relationship in rifle-shot animals and blood lead levels in eagles and California condors has been established by comparisons of lead isotope ranges.[5,7,12]

We believe that eagles admitted to our Raptor Rehabilitation Program represent only a small percentage of lead-exposed birds in the region of our study, which is either unpopulated or sparsely populated. Most eagles suffering from lead poisoning would never be seen or found alive for diagnosis and medical treatment (Fig 7).

Lead has no known biologic functions and safe exposure limits for most species have not been established. Clinical implications of lead poisoning in wild birds with blood levels >0.20 ppm

(>20 μg/dL; >0.97 μmol/L) have been documented and described.[1,2] Effects on the immediate well being of eagles are not expected at background concentrations of 0.10–0.19 ppm (10–19 μg/dL; 0.49–0.92 μmol/L) but emerging information on blood lead concentrations in the range previously considered inconsequential are associated with harmful effects in long-lived species,[13] with possible implications for eagles.

The gravity of lead poisoning in eagles in the US inland Pacific Northwest region is highlighted by the fact that 22 of 46 (48%) bald and 31 of 50 (62%) golden eagles tested had toxic blood lead levels of 0.20 ppm (20 μg/dL; 0.97 μmol/L) or higher. Inclusion of values in the 0.10–0.19 ppm (10–19 μg/dL; 0.49–0.92 μmol/L) range would increase the numbers to 28 of 46 (61%) for bald and 41 of 50 (82%) for golden eagles.

It is disheartening that in a society that prides itself for its love of wildlife in general and for the admiration of (bald) eagles in particular, lead poisoning continues at a level similar to previous decades. As long as hunting with lead-containing bullets is accepted and continues, the problem of lead poisoning will persist.[2,5,13] A small step to reduce the opportunity of lead poisoning in eagles and other wildlife can be taken by hunters in the safe disposal (eg, burial, burning) of lead-contaminated animal parts or ''sport-hunted'' animals or voluntary lead reduction commitments by hunters.[2,14–16] Testing more moribund or sick eagles for lead poisoning to prove the danger of lead in the environment is not needed; instead, we need to realize that lead must be removed from all hunting ammunition without delay. Lead-free bullets for hunting purposes are readily available.[2,16] Federal and state agencies, the hunting community, and the public must reach agreements on how to most effectively and swiftly make the transition from lead to no-lead ammunition. Countries, states, communities, and private entities with a progressive outlook have taken this important step for protecting nontarget wildlife species but ultimately also for the well-being of their own citizens.[2,14–23]

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