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LEAD POISONING IN CAPTIVE ANDEAN CONDORS (*VULTUR GRYPHUS*)

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ABSTRACT: Elevated lead in the tissues of raptors, especially those that scavenge, is a common occurrence, and lead poisoning appears to be a significant problem in the ongoing recovery effort for California condors (*Gymnogyps californianus*). Elevated blood lead levels have been found in released birds, and a number of birds have died of lead poisoning. In earlier work, we dosed turkey vultures (*Cathartes aura*) with lead shot but found them to be a poor model for lead poisoning. In this study, we dosed four Andean condors (*Vultur gryphus*) with lead shot and found them to be quite sensitive, as two of the birds died and the other two exhibit signs of lead poisoning within 50 days. All lead-responsive parameters were affected, and regurgitation of dosed shot occurred only once. The response of the Andean condors appeared to mimic California condors, suggesting that once exposed to lead, the possibility of survival is poor. This is consistent with observations in the wild, where otherwise healthy birds exposed to metallic lead quickly succumb. At the very least, the release program has to maintain constant surveillance and an active lead monitoring program.

Key words: Andean condors, California condors, endangered species, *Gymnogyps californianus*, lead poisoning, nontoxic lead substitutes, raptors, *Vultur gryphus*.

INTRODUCTION

Lead remains a hazard to free-ranging raptors, and there have been numerous reports of elevated lead in condors and eagles. The scientific literature is replete with cases of raptors exposed to or dying of lead poisoning. In the United States, egested pellets from bald eagles (*Haliaeetus leucocephalus*) were found to contain lead shot at study sites in the Midwest (Dunstan, 1974), Utah (Platt, 1976), Missouri (Griffin et al., 1980), and Arkansas (Nelson et al., 1989). Elevated blood lead levels have been reported in golden eagles (*Aquila chrysaetos*) throughout the range of the California condor (*Gymnogyps californianus*; Pattee et al., 1990) and in golden and bald eagles from Idaho (Craig et al., 1990), Alaska, Iowa, Illinois, Indiana, Michigan, Minnesota, Nebraska, North Dakota, Ohio, South Dakota, Wisconsin (Kramer and Redig, 1997), and Montana (Harmata and Restani, 1995; Miller et al., 1998). In Canada, elevated blood lead levels in golden and bald eagles have been found in the Canadian prairie provinces (Wayland and Bollinger, 1999), British Columbia (Elliot et al., 1992), and Saskatchewan (Miller et al., 1998).

Elevated lead levels in raptors may represent a global problem. Elevated tissue lead levels and lead-induced mortality in Steller's sea eagles (*Haliaeetus pelagicus*) and white-tailed sea eagles (*Haliaeetus albicilla*) have been reported from Hokkaido, Japan (Kim et al., 1999). Pain et al. (1995) determined liver lead levels in 424 moribund individuals representing 16 raptor species from Great Britain. Two individuals probably died of lead poisoning, and two others had levels

suggestive of but not definitive for lead poisoning. An additional 18 individuals of eight species had liver lead levels greater than 6 ppm dry weight. Liver lead concentrations were determined in 222 dead individuals of 16 species recovered in and around France (Pain and Amiard-Triquet, 1993). Liver lead levels greater than 6 ppm (dry weight) were reported in eight individuals drawn from three species.

Lead poisoning has been, and continues to pose, a significant threat to the establishment of viable and secure wild California condor populations. Actual and potential losses from lead poisoning were the driving force behind the decision to capture all known California condors (Pattee and Wilbur, 1989). Elevated blood lead levels were found in five of 14 wild California condors, and all 14 had detectable levels of lead in their blood (Wiemeyer et al., 1988). During this period (1980s), 12–15 condors died or disappeared; three were diagnosed as lead poisoned (Janssen et al., 1986; Wiemeyer et al., 1988), accounting for 20–25% of the known mortality (Snyder, 1986). Elevated blood lead levels have been found in the recently established wild California and Arizona condor subpopulations (Sorenson et al., 2000).

The objectives of this study were to assess Andean condor (*Vultur gryphus*) susceptibility to lead poisoning, measure physiological response, determine tissue lead concentrations, and use these data to better understand the response of California condors to lead. Andean condors have been used at the Patuxent Wildlife Research Center in lieu of California condors since 1965 and have proven to be exceptional surrogates for developing husbandry and breeding protocols for the California condor program. The similarities between the two condor species led to a consensus that experimental work with Andean condors would also provide a means to understand lead poisoning in California condors, and this study was identified as priority research by the California Condor Recovery Team. An Endangered Species Act Section 7 consultation on the use of Andean condors in a terminal study was conducted by the Office of Endangered Species, US Fish and Wildlife Service (USFWS), which determined that the study was necessary; the research was also approved by the Patuxent Wildlife Research Center Institutional Animal Care and Use Committee.

METHODS

Six immature Andean condors were selected for this study. Two birds served as undosed controls and were returned to the breeding colony (Laurel, Maryland, USA: 39°03′14″N, 76°48′59″W) at the conclusion of the study. The controls provided baseline data on pen conditions and physiological parameters. The other four Andean condors were captive-hatched and reared and were behaviorally unsuited for release or captive propagation. To evaluate the health of the condors prior to the study, all six birds were given a physical examination and weighed, bled via basilic venipuncture, and a survey radiograph was obtained. All birds were fat and had a body condition score of 4 to 4+ (based on a score of 1–5, with 1 being emaciated and 5 being grossly overweight). There was no evidence of ingested lead. Blood samples were obtained weekly during the study to determine blood lead, hematocrit (HCT), delta-aminolevulinic acid dehydratase (ALAD) activity, and erythrocyte protoporphyrin (EPP) levels. On 3 January 1989, the six condors were individually housed in wire-mesh cages (2.0 × 4.0 × 1.9 m) elevated over a concrete slab. The birds were allowed to acclimate to pen conditions and a diet of cubed chicken breast for 2 wk. On 17 January 1989, each of two birds was dosed with zero, two, or six number 00 lead shot. Shot were weighed and sorted and then used to produce shot dosages corresponding to 0.575 g/kg (two shot) and 1.92 g/kg (six shot); shot ranged from 7.5 mm to 8.5 mm in diameter. Birds were weighed and a 5 ml blood sample was taken from the basilic vein prior to dosing and then weekly thereafter. Birds were fed on Sunday, Wednesday, and Friday and fasted on the other days. The area under the pens was searched daily for regurgitated or passed shot. Although the condors were observed daily, more intensive observations were conducted on the three

feeding days. On these days, birds were observed for 2 min before and 3 min after feeding for any changes in behavior that might have reflected a clinical disease.

Birds remained on the experimental regime until they died or were euthanized after displaying clinical signs of toxicosis (lethargy, weight loss, and uncoordination). A postmortem examination was performed on the four lead-dosed condors immediately following death. Tissues obtained for histological evaluation included cerebrum, cerebellum, medulla oblongata, optic lobe of brain, heart, lung, trachea, thyroid, parathyroid, thymus, liver, gall bladder, spleen, pancreas, kidney, gonad, adrenal, crop/esophagus, proventriculus, gizzard, small and large intestines, cloaca, bursa of Fabricius, peripheral nerves and ganglia associated with other tissues, skin, skeletal muscle, and bone marrow. All tissues were placed in 10% buffered formalin, then embedded in paraffin, and stained with hematoxylin and eosin (H&E) and with an acid-fast stain.

Liver and kidney samples were placed in glass jars with Teflon lid liners cleaned in 10% nitric acid and rinsed with acetone and then hexane. Liver and kidney samples were stored at −15 C until analyzed for their lead content (DeStefano et al., 1991). The detection limit for kidney and liver lead was 0.22 ppm (wet weight); recovery of spiked samples averaged 104.9%. A heparinized 2 ml blood sample was set aside for the determination of HCT, ALAD activity, and EPP levels. Hematocrit was determined by measuring the packed cell volume of whole blood in capillary tubes centrifuged at 13,460 × G for 5 min. An aliquot of the subsample was stored at −70 C for subsequent quantification of ALAD (Burch and Siegel, 1971); the analysis was optimized for Andean condor blood with a pH 6.8 buffer. The remainder of the sample was stored at 4 C for 48 hr before determining EPP concentration with a hematofluorometer (AVIV Biomedical, Inc., Lakewood, New Jersey, USA) as modified by Roscoe et al. (1979).

Blood samples for lead determination were placed in vials cleaned in 10% nitric acid and rinsed with acetone and then hexane. Samples were frozen and stored at −15 C until the end of the study. Some samples were lost because of vial breakage. Lead analysis followed the methods of Fernandez and Hilligoss (1982) using a Perkin-Elmer (Norwalk, Connecticut, USA) HGA-400 graphite furnace at a wavelength of 283.3 nm for the analysis with deuterium arc background correction. The lower limit of reportable, uncorrected lead residues was 0.02 ppm (wet weight). Recovery of spiked samples averaged 100.4%.

RESULTS

During the first 14 days postdosage, all birds exhibited normal behavior. No change in behavior was noted in the two control birds during the study; both birds remained active, alert, curious, and responsive/defensive. On day 14, both of the low-dose condors were slightly lethargic, depressed, and slower in movement. Although there was a gradual decline in attitude and activity during this study, the birds were alert/curious and responsive until one died and the second bird was euthanized (Table 1). There also were several days during the latter phase of the study when both birds seemed to have a marked improvement in attitude and activity. One of the two high-dose birds began showing behavioral changes (droopy wings, opening mouth frequently, and reduced activity) on day 28. There was a gradual increase in lethargy and depression, which became more pronounced before death on day 39. The second high-dose bird began showing clinical signs of disease (reduced activity, postural change, lethargy, and depression) on day 21. Although there was a gradual decline in the attitude and activity level of this bird, the bird remained fairly alert and somewhat active until it was euthanized on day 41 (Table 1). One low-dose bird regurgitated two shot while being handled on day 7. It was immediately redosed. No shot was passed or regurgitated by any other bird and all the dosed shot were recovered from the birds during necropsy. At necropsy, the two low-dose condors had a body condition score of 1+ and 2+, subcutaneous fat was low or absent, and there was minimal abdominal and coronary fat. The body condition score of the two high-dose birds was 1+ and 2, subcutaneous fat was minimal, and the abdominal and coronary fat was minimal. Weights of the six condors were obtained

TABLE 1. Fate, exposure level (mg of lead eroded from dosed shot), and tissue lead levels (ppm, wet weight) of Andean Condors experimentally dosed with number 00 (7.3–8.5 mm diameter) lead shot equivalent to 0.575 g (two shot) or 1.92 g (six shot) lead per kg of body weight.

Tag number	Treatment	Days treated	Fate[a]	Pb eroded (mg)	Liver Pb (ppm)	Kidney Pb (ppm)
165	0	49	SV	0.00	N/A	N/A
85025	0	49	SV	0.00	N/A	N/A
166	2	46	FD	290.2	45.46	124.16
85021	2	49	SW	125.7	49.88	114.74
150	6	39	FD	602.6	58.52	229.94
85024	6	41	EW	527.4	109.09	179.09

[a] SV = undosed control; EW = found severely weakened, euthanized; FD = found dead; SW = survived but obviously weakened when euthanized; N/A = no sample available.

at necropsy (four treated birds) or at termination of the study (two control birds). The control birds, low-dose birds, and high-dose birds lost 7% and 10%, 21% and 27%, and 28% and 33% of their body weight, respectively.

Tissues from the four condors were examined histologically. All birds showed similar lesions of perivascular and perineuronal edema throughout the cerebrum. Sections of the cerebellum had spongiosis at the interface between the molecular and granular layer, and the lesions were more severe towards the tips of the folia. Purkinje cells within the affected areas of spongiosis were pyknotic. Three birds had similar lesions of the cerebellar folia, while one of the high dose birds was more severely affected. Liver sections from the four birds were similar and had a mild periportal infiltrate of lymphocytes and macrophages. Abundant yellow brown pigment compatible with hemosiderin was present within hepatocytes and in Kupffer cells. The pigment was positive for iron with Perl's Prussian Blue stain. Hemosiderin-laden macrophages were also present in the spleens of all the birds. All four of the dosed birds had mild acute diffuse nephrosis. Acid-fast staining of the kidney showed numerous acid-fast positive intranuclear inclusions within the tubular epithelium. Total bilirubin increased from an average of 0.25 mg/dl to 1.05 mg/dl in the four treated birds over the course of the study.

Liver and kidney lead concentrations increased as treatment dose increased (Table 1). Blood lead concentrations increased over time and peaked 2–3 wk before death (Table 2); EPP activity (Table 2) also increased over time and peaked 1–2 wk before death in the birds dosed with two shot. A precipitous decrease in ALAD activity (Table 2) was observed in the first week following dosage and remained depressed until the birds died or were euthanized. A gradual decline was observed with HCT (Table 2) throughout the study period, reaching maximum depression at the time of death.

DISCUSSION

All of the dosed Andean condors ($n=4$) developed lead poisoning; two birds died and two were euthanized. The high-dose bird that was euthanized displayed multiple signs of lead toxicosis. One low-dose bird that displayed early signs of lead toxicosis was euthanized based on observed responses of the other three dosed birds. The slower response of this individual may have been related to a slower erosion of the lead shot in this individual (164.5–476.9 mg more lead was eroded from dosed shot in the other birds); however, the lead levels exhibited in this bird equaled or exceeded levels reported from other birds (Beyer et al., 1988) and were well above the thresholds suggested to cause mortality (Franson, 1996). Al-

TABLE 2. Average blood parameters ($n=2$) of Andean condors dosed with zero, two, or six 00 lead shot equivalent to 0.575 or 1.92 mg of lead per kg of body weight.

	Dose	Day 7	Day 14	Day 21	Day 28	Day 35	Day 42	Day 49
Blood lead (ppm, weight wet)								
0 shot	0.02[a]	0.16[a]	0.07	—[b]	0.06	—[b]	0.02	0.00[a]
2 shot	0.15[a]	11.1[a]	9.05	—[b]	17.16	11.00	9.46	—[b]
6 shot	0.08	—[b]	13.04	—[b]	16.41	12.02	—[b]	—[b]
Hematocrit (percent)								
0 shot	43.0	43.5	44.0	45.5	42.5	42.0	43.0	43.5
2 shot	45.0	42.0	41.5	40.5	35.5	33.0	31.5	23.0[a]
6 shot	42.0	41.0	38.5	34.5	28.5	31.0	—[b]	—[b]
Erythrocyte protoporphyrin (μg/day)								
0 shot	44.5	42.0	41.5	39.5	38.5	36.5	44.0	43.5
2 shot	41.0	57.5	335.5	228.5	231.0	856.0	664.5	229.0[a]
6 shot	37.0	101.0	161.0	380.0	579.0	944.5	—[b]	—[b]
Delta-aminolevulinic acid dehydratase (units)								
0 shot	75.92	72.94	78.64	80.02	87.39	88.01	92.90	94.90
2 shot	56.94	1.41	1.31	2.00	1.10	3.23	2.80	1.11[a]
6 shot	112.9	2.11	2.53	1.82	2.62	3.92	—[b]	—[b]

[a] One sample lost.

[b] Samples lost or not taken.

though the low-dose condor was euthanized on day 49 without displaying the full suite of overt signs, the bird's survival was unlikely and further suffering unnecessary.

Histologically, the lesions of hepatic and splenic hemosiderosis, as well as acid-fast inclusions in the tubular nuclei of the kidney, were consistent with lead toxicity (Franson, 1996). There was no evidence of myocardial degeneration or fibrinoid necrosis of the vasculature. The lesions seen in the brain must be interpreted cautiously because increased perineuronal and perivascular space can occur due to postmortem change.

Lead poisoning is driven by three interacting factors: nutritional status, genetic predisposition, and lead availability. Ingestion is the primary pathway for lead exposure, but absorption is mediated and strongly influenced by the chemical environment of the lumen, age of the animal, and iron stores (i.e., the nutritional status of the subject). Dietary components such as sodium citrate, amino acids, vitamin D, protein, fat, and lactose bind lead and increase its solubility and enhance absorption (DeMichele, 1984). Total food intake, percent dietary fat, and dietary intakes of calcium, iron, zinc, lactose, and vitamin E are also known to influence lead toxicity and susceptibility (DeMichele, 1984). There also is a genetic component. Three polymorphic genes have been identified in mammals that apparently influence the bioaccumulation and toxicokinetics of lead and in all probability dictate not only individual susceptibility but species vulnerability as well (Fleming et al., 1998; Onalaja and Claudio, 2000). This genetic component has yet to be investigated in birds.

In a previous study, four of six lead-dosed turkey vultures succumbed to lead poisoning after prolonged exposure and constant redosing with regurgitated or defecated shot (Carpenter et al., 2003). Two of the dosed birds never exhibited overt signs of lead poisoning, even after 211 days of constant exposure. Pattee et al. (1981) reported similar results in experimentally lead-dosed bald eagles and attributed the differences to the interaction of several factors, including shot retention time, number of shot retained, amount of lead eroded, and individual susceptibility. Beyer et al.

(1988) found major differences in the six avian species they dosed with lead; one-half of the red-winged blackbirds (*Agelaius phoeniceus*) died in 61 days, whereas 111 days were required for half the eastern screech owls (*Otus asio*) to die (death in the other four species occurred between these extremes).

Weight loss is a typical response to lead poisoning and has been previously reported in many avian species dosed with lead (Pattee et al., 1981; Beyer et al., 1988). Weight loss was reported in the three California condors believed to have died of lead poisoning (Janssen et al., 1986). In our study, differences in body condition (weight, pectoral muscle mass, and subcutaneous and coelomic fat) between lead-shot dosed and undosed birds were associated with the degree of anorexia. The definitive diagnosis of lead toxicity in this study was based on the presence of lead inclusions in liver and/or kidney cells or the presence of significant lead levels from tissue chemical analysis.

Liver lead concentrations in our dosed birds were higher than those reported as indicative of lead poisoning (Franson, 1996). Kidney lead concentrations were also higher when compared to reported concentrations in other falconiform birds (Franson, 1996). Blood lead concentrations increased with shot exposure duration, dropping just before death. Franson (1996) suggested that blood lead concentrations greater than 1 ppm (wet weight) are indicative of lead toxicosis, and concentrations greater than 5 ppm (wet weight) support lead-mediated mortality. In experimentally dosed bald eagles, blood lead concentrations were reported to be 0.8 ppm (wet weight) after 24 hr and 5.4 ppm (wet weight) after 14 days; three of the five eagles died within 20 days (Hoffman et al., 1981). A dying turkey vulture had a blood lead level of 2.27 ppm (Platt et al., 1999). The condors in our study had blood levels between 16 and 19 ppm wet weight.

Delta-aminolevulinic acid dehydratase is a sensitive measure of exposure but can stay depressed over an extended period in an otherwise apparently healthy bird (Franson et al., 1983). In our study, ALAD declined to essentially zero activity. Our EPP results were similar to those reported by Franson et al. (1986) in lead shot-dosed canvasbacks (*Aythya valisineria*) and by Beyer et al. (1988) for red-winged blackbirds, brown-headed cowbirds (*Molothrus ater*), common grackles (*Quiscalus quiscula*), mallards (*Anas platyrhynchos*), northern bobwhites (*Colinus virginianus*), and eastern screech owls. However, our initial EPP activity and the magnitude of the response were greater than reported in lead-dosed black ducks (*Anas rubripes*) and mallards (Rattner et al., 1989).

The control birds maintained a relatively flat profile, with an HCT above 40%, whereas the HCT of the dosed condors declined following lead dosage. Coleman et al. (1988) reported HCT below 31% in two sick/dying turkey vultures and suggested an HCT below 40% indicated a bird that was sick or in poor condition. Platt et al. (1999) recorded an HCT of 23% in a dying turkey vulture. The response of HCT in our study was similar to that reported in six different avian species experimentally dosed with lead (Beyer et al., 1988), in lead-dosed bald eagles (Hoffman et al., 1981), and in lead-dosed American kestrels (*Falco sparverius*) (Hoffman et al., 1985). Our study produced results and responses to lead that appear to be similar to the California condor. This includes retention of the lead once swallowed, fast dissolution of the lead object, and absorption of the dissolved lead and rapid onset of lead toxicosis. As a general rule, only metallic lead (fishing sinkers, lead shot, bullet fragments) are capable of inducing death in free-ranging birds; the only exceptions known to the authors are the avian mortalities associated with the mine tailings at Coeur d'Alene, Idaho, where sediments have lead residues measurable

in percents (Henny et al., 1991), and lead-containing paint chips on Midway atoll (Sileo and Fefer, 1987). As for California condors, bullet fragments seem most probable. Work in determining lead origins based on their isotope ratios (Scheuhammer and Templeton, 1998) will help to resolve the issue as to the primary source of the lead killing condors. Considering the scope of the problem, with even the remote areas of Arizona not safe from the lead poisoning problem, a lead exposure surveillance program is essential. Samour and Naldo (2002) have shown that aggressive therapy and treatment can prevent the mortality associated with lead ingestion, and a similar program has been instituted in the California condor program (Sorenson et al., 2000). This therapy is essential to the survival of California condors.

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