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EXPERIMENTAL LEAD POISONING IN TURKEY VULTURES (*CATHARTES AURA*)

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ABSTRACT: Lead-induced mortality appears to have been a major factor in the decline of the California condor (*Gymnogyps californianus*). We orally dosed turkey vultures (*Cathartes aura*) with BB-sized lead shot from January 1988 through July 1988 to determine physiologic response (delta-aminolevulinic acid dehydratase inhibition, erythrocyte protoporphyrin levels, anemia), diagnostic tissue lead concentrations (blood, liver, and kidney), and comparative sensitivity of this species. Two turkey vultures died and two became so intoxicated they were euthanized. Overall, responses of measured parameters were comparable to other species exposed to lead although there was considerable individual variation. Survival time (143–211 days), even with the large numbers of shot and constant redosing, was much longer than reported for other species of birds, suggesting considerable tolerance by turkey vultures to the deleterious effects of lead ingestion. Based on these observations, turkey vultures appear to be poor models for assessing the risk of lead poisoning to California condors or predicting their physiologic response.

Key words: California condors, *Cathartes aura*,, endangered species, *Gymnogyps californianus,* lead poisoning raptors, turkey vultures.

INTRODUCTION

Anthropogenic sources of lead have been implicated in the deaths of many avian species. Best known and documented in waterfowl (Sanderson and Bellrose, 1986), lead-induced mortality has been reported in a variety of birds (Eisler, 1988). All birds are vulnerable to the effects of lead but their response shows distinct intraspecific and interspecific differences (Pattee et al., 1981; Beyer et al., 1988). Lead poisoning is a special challenge to raptors because their populations are naturally low and the loss of a few individuals can affect population viability. Elevated lead levels have been reported in the following free-ranging raptors: marsh harriers (*Circus aeruginosis*) in France (Pain et al., 1997); ospreys (*Pandion haliaetus*) in Idaho (USA) (Henny et al., 1991); bald (*Haliaeetus leucocephalus*) and golden eagles (*Aquila chrysaetos*) in Idaho (Craig et al., 1990); bald and/or golden eagles in Alaska, Iowa, Illinois, Indiana, Michigan, Minnesota, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin in the US (Kramer and Redig, 1997); bald and golden eagles in Montana (USA; Harmata and Restani, 1995); bald eagles in British Columbia (Canada; Elliott et al., 1992); and golden eagles, turkey vultures (*Cathartes aura*), and California condors (*Gymnogyps californianus*) in California (USA; Janssen et al., 1986; Wiemeyer et al., 1986, 1988; Pattee et al., 1990).

Lead poisoning gained attention as a potentially significant factor for California condors during the 1970s and 1980s. A California condor that died in 1975 had elevated concentrations of lead in its bones (Wiemeyer et al., 1983). During the 1980s, lead poisoning was the cause of death in three of the five confirmed mortalities (Wiemeyer et al., 1988) and constituted 20–25% of all losses (Snyder, 1986). The

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probable source of the lead ingested by California condors was bullets and bullet fragments in the carrion consumed by the birds. Because lead poisoning appears to be an important mortality factor in California condors, and lead has been shown to be ubiquitous within their historic range (Pattee et al., 1990), controlled laboratory studies were undertaken to evaluate the extent of the hazard posed by lead ingestion. The turkey vulture was chosen for the initial study because it is a soaring scavenger commonly found within the range of the California condor and is known to have elevated tissue lead concentrations (Wiemeyer et al., 1986; Pattee and Wilbur, 1989). Reiser and Temple (1981) suggested, on the basis of one experimentally dosed turkey vulture, that they are especially susceptible to lead poisoning.

The objectives of our study were to assess turkey vulture susceptibility to lead poisoning, measure physiologic response, determine tissue lead concentrations, and use these data to design a study assessing similar parameters in Andean condors (*Vultur gryphus*).

METHODS

Eight turkey vultures were captured using rocket nets between January 1987 and June 1987 in and around Beltsville, Maryland (USA; 39°03′14N, 076°48′59W). Birds were housed two or three per pen (2.5×6.5×1.9 m) where they were acclimated to captivity and a diet of whole chickens for a minimum of 6 mo. In January 1988, birds were radiographed to check for pre-existing lead shot and weighed, then housed individually in wire-mesh cages (2.0×4.0×1.9 m) elevated over a concrete slab. After 4 days they were switched to a diet of whole chicken breasts. On day 7, the diet was changed to cubes of chicken breast and the birds were captured and handled every other day to acclimate them to the experimental regime. A 5 ml blood sample was obtained from the basilic (brachial) vein on day 17. Birds were fasted on day 21 and every Sunday, Tuesday, Thursday, and Saturday, thereafter.

On day 22, birds were randomly assigned without regard to sex, age, or weight to three treatments: zero shot (n=2), one shot (n=3), three shot (n=3). Birds were weighed and a blood sample drawn, then fed the preweighed lead shot dose (BB sized; 0.35–0.49 g) hidden in a cube of chicken meat. Shot were sorted by weight and chosen to approximate doses of 0.192 g lead per kg of body weight (one shot dose) and 0.576 g lead per kg of body weight (three shot dose). Thereafter, the diet was whole chickens unless redosing with regurgitated shot was necessary. Birds were cared for and observed daily and the area under the pen searched for regurgitated and defecated shot. To facilitate the search, the cage size was reduced to 1.2×1.2×1.2 m on day 44. Birds were weighed and blood sampled 3 and 7 days postexposure and weekly thereafter. Recovered shot were weighed and the birds redosed with the shot the next time they were fed. On day 85 (63 days after the first dose), the birds receiving one shot were given nine additional preweighed BB-size shot, for a new treatment level of 10 lead shot (equivalent to 1.92g/kg). Birds remained on the experimental regime until they died (without prior evidence of severe clinical signs), were euthanized by CO_2 inhalation after becoming visibly intoxicated, or were euthanized to end the study. Intoxicated birds were lethargic, nonresponsive, emaciated, and lacked coordinated movement.

A postmortem examination was performed on all eight vultures. Tissues obtained for histologic 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 and acid-fast. Liver, kidney, and heart blood clots were also placed in glass jars with Teflon lid liners that had been cleaned in 10% nitric acid and rinsed with acetone, then hexane. Samples were stored at −15 C until analyzed for their lead content (DeStefano et al., 1991). Detection limit was 0.22 ppm (wet weight); recovery of spiked samples averaged 104.9%.

Immediately following blood collection, a heparinized 2 ml subsample was reserved for determining hematocrit (HCT), delta-aminolevulinic acid dehydratase (ALAD) activity, and erythrocyte protoporphyrin (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

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TABLE 1. Clinical outcome, mass change, lead eroded, and tissue lead levels (parts per million, wet weight) of turkey vultures experimentally dosed with 0, 3, or 10 BB-size lead shot.

Bird	Number of shot[a]	Outcome[b]	Days treated	Mass change (kg)	Mass change (%)	Lead eroded (mg)	Lead eroded per day (mg)	Liver lead (ppm[c])	Kidney lead (ppm[c])	Heart blood clot lead (ppm[c])
182	0	ES	211	+0.03	+1.3	0.0	0	0.10	0.30	0.02
183	0	ES	211	0.00	0.0	0.0	0	0.05	0.10	0.00
178	3	ES	211	−0.33	−16.3	112.0	0.53	2.22	28.92	1.87
194	3	ES	211	−0.14	−6.8	138.5	0.66	1.48	24.63	1.12
196	3	EW	170	−0.78	−37.3	121.8	0.72	18.71	180.00	25.66
184	10	EW	183	−1.05	−44.5	110.7	0.60	6.79	181.17	6.00
186	10	FD	148	−1.06	−46.7	178.6	1.21	20.73	245.89	—[d]
193	10	FD	143	−0.69	−33.2	247.0	1.73	33.78	226.02	29.56

[a] Birds 184, 186, and 193 were dosed with one shot for the first 61 days. This was changed to a dose of 10 shot on day 62.
[b] ES=Survivor euthanized at study end; EW=severe clinical signs, euthanized; FD=found dead.
[c] ppm=parts per million, wet weight.
[d] No sample.

and Siegel, 1971). The analysis was optimized for turkey vulture blood with a pH 6.4 buffer. The remainder of the sample was stored at 4 C for 48 hr before determining EEP 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 hexane. Samples were stored at −15 C until the end of the study. A subset of all the samples for each bird was selected for determination of lead concentrations. 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. Clotted heart blood was sonicated, then analyzed similarly to the whole blood samples. The lower limit of reportable, uncorrected residues was 0.02 ppm, wet weight. Recovery of spiked samples averaged 106.2%.

The slope of the blood-lead by time regression line, fitted with the y-intercept at zero, was used to estimate the rate of lead uptake by each bird. The slopes of ALAD, EPP, and HCT regression lines over time (with intercepts allowed to be non-zero) also were used as measures of the rate of each bird's response to lead. Spearman's rank correlation was used to investigate relationships among variables of interest. The Jonckheere-Terpstra (J-T) test (Daniel, 1990) was used to evaluate differences between treatments in liver lead level, blood lead by time slope, ALAD by time slope, EPP by time slope, HCT by time slope, weight loss, heart blood clot lead concentration, and kidney lead concentration.

All J-T tests were run on the software package StatXact (Cytel Corporation, Cambridge, Massachusetts, USA), making it possible to calculate exact P-values even for small samples where asymptotic theory may be invalid (Daniel, 1990). An estimate of the median lethal dose (LD_{50}) and a 95% confidence interval (CI) was calculated by constructing a profile likelihood for dose under a binomial sampling model with a logit link function. The software package GLIM (Numerical Algorithms Group, Inc., Downers Grove, Illinois, USA) was used to fit the logistic regression model. A P value of 0.05 was chosen for tests of significance in all calculations.

RESULTS

Two of the high dose birds died (day 143 and 148) and two others (one high dose, one low dose) became quite weak (coordination problems, no appetite, general weakness) and were euthanized (day 170 and 183) (Table 1). The remaining two dosed birds failed to show signs of impairment or intoxication and were euthanized along with the controls after 211 days on treatment. At necropsy, the control vultures had prominent pectoral muscles, moderate subcutaneous fat, and abundant coelomic fat. The birds dosed with three shot had minimal to moderate pectoral muscles, minimal to moderate subcutaneous fat, and minimal to abundant coelomic fat. The birds receiving 10 shot had minimal to modest pectoral muscles and min-

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imal subcutaneous and coelomic fat. No other significant or consistent gross lesions were observed in any of the birds. Histologically, acid-fast lead inclusions were observed in hepatocytes and Kupffer's cells of one vulture (bird 193; highest liver lead in the group). Hemosiderin was present in hepatocytes and Kupffer's cells in three of six treated birds but not in control birds. All treated vultures had some degree of acute renal tubule degeneration. Acid-fast lead inclusions were observed in proximal tubule epithelial cell nuclei of the six birds dosed with lead shot. The inclusions tended to be more abundant in the higher-dosed birds. All treated birds had some degree of status spongiosis in the white matter of the cerebrum, the cerebellum, and the medulla oblongata, with the most pronounced lesions in the dosed birds. No significant or consistent lesions were observed in the other tissues.

The J-T test of differences between treatments showed that the rate of change in liver lead concentrations ($P=0.0054$), kidney lead concentrations ($P=0.0018$), and heart blood clot rate concentrations ($P=0.0143$) increased with time and treatment level (Table 1). In general, the higher the dose, the greater the rate of weight loss ($P=0.0054$) and the more precipitous the slope of the regression line (Fig. 1); both trends were significantly related to the treatments. The downward trend was accelerated in the 10 shot treatment group when the dose was increased from one shot to 10 shot 63 days after the start of the study. The two dosed birds that survived to the end of the study lost more weight and had higher tissue lead levels than controls (Table 1).

The rate of change in concentration of blood lead and the slope of the regression line increased as the treatment level increased ($P=0.030$), generally showing an abrupt rise prior to death (Fig. 2). The slope of the ALAD activity regression line (Fig. 3) decreased as the dose increased ($P=0.0054$). ALAD activity exhibited a gradual decline in the controls; whereas

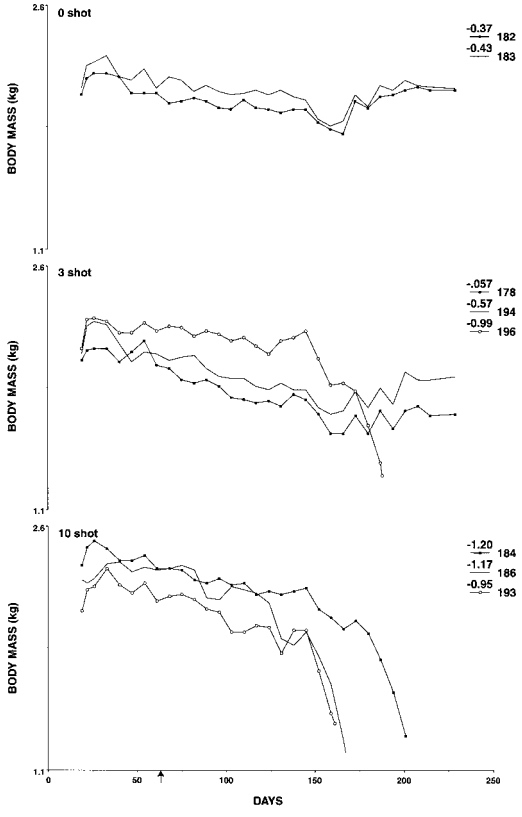


FIGURE 1. Mass change (kg) over time and the slope of the regression line in turkey vultures dosed with 0, 3, or 10 BB-sized lead shot. Dose in 10 shot group changed from one shot to 10 shot 63 days into the study as indicated by arrow. Numbers on the right side refer to change in individual birds.

the dosed bird's ALAD activity declined sharply and remained depressed throughout the study. Vulture 194 had two peaks of increased ALAD activity strong enough to yield a positive slope for the regression line; whereas all the other treated birds had negative slopes. EPP activity increased with shot dose ($P=0.0054$) (Fig. 4). The slope of the HCT regression line decreased with increasing shot dose ($P=0.0018$) and dropped precipitously prior to death in the birds found dead or in a seriously weakened condition (Fig. 5).

Liver lead, kidney lead, heart blood clot lead, mg of lead eroded, slope of the blood lead concentrations, slope of the ALAD activity, slope of the EPP activity, and slope of the HCT values were correlated

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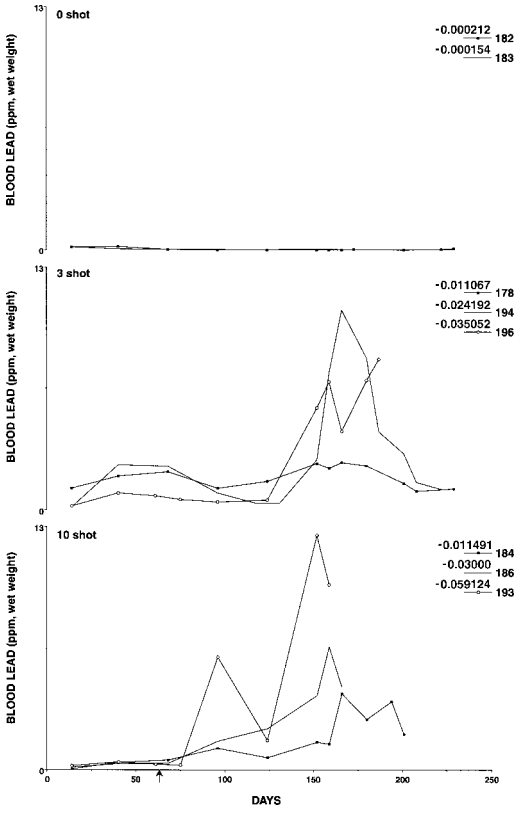


FIGURE 2. Blood lead levels (ppm wet weight) over time and the slope of the regression line in turkey vultures dosed with 0, 3, or 10 BB-sized lead shot. Dose in 10 shot group changed from one shot to 10 shot 63 days into the study as indicated by arrow. Numbers on the right side refer to change in individual birds.

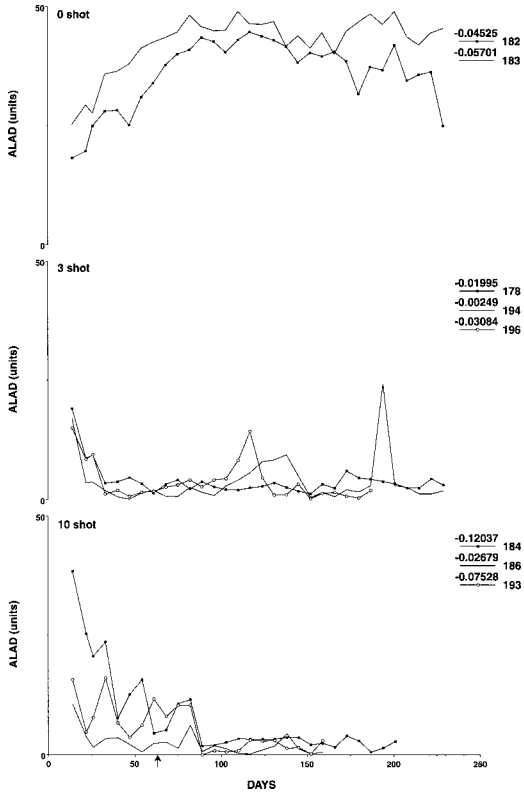


FIGURE 3. Delta-aminolevulinic acid dehydratase (ALAD) activity over time and the slope of the regression line in turkey vultures dosed with 0, 3, or 10 BB-sized lead shot. Dose in 10 shot group changed from one shot to 10 shot 63 days into the study as indicated by arrow. Numbers on the right side refer to change in individual birds.

(Table 2) with the following exceptions; heart blood clot lead and the slope of the ALAD activity were not significantly correlated with the mg of lead eroded from the dosed lead shot. The LD_{50} was estimated to be 3.1 shot with a 95% confidence limit of 0.95 to 8.6 shot.

DISCUSSION

Four turkey vultures succumbed to lead poisoning after prolonged exposure and constant redosing with regurgitated or defecated shot. They either died or became so weakened that they were euthanized. Conversely, two of the dosed birds never exhibited any overt signs of lead poisoning even after 211 days of constant exposure. Pattee et al. (1981) saw a similar pattern in experimentally lead-dosed bald eagles and attributed the differences in response to amount of lead eroded and individual susceptibility. The first turkey vulture did not die in this study until 143 days after the initial dose. 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). There were no gross lesions pathognomonic for lead intoxication. Microscopic lesions in liver, kidney, and central nervous system were not sufficiently different

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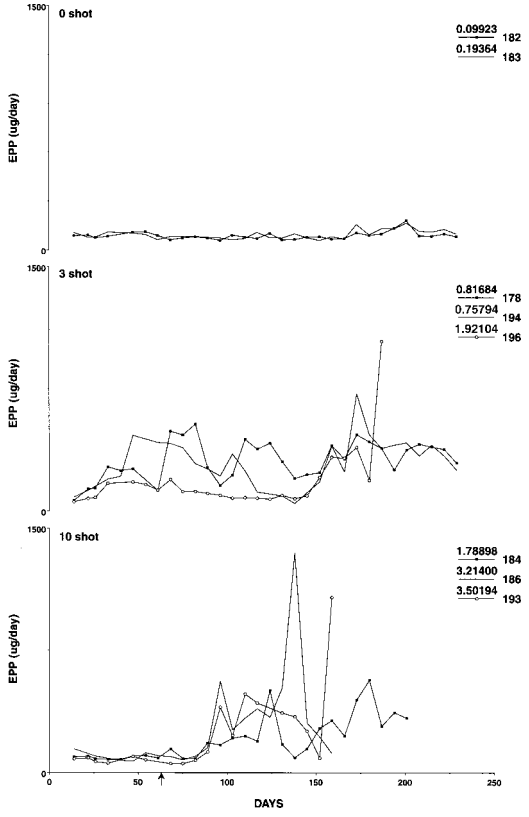


FIGURE 4. Erythrocyte protoporphyrin (EPP) activity over time and the slope of the regression line in turkey vultures dosed with 0, 3, or 10 BB-sized lead shot. Dose in 10 shot group changed from one shot to 10 shot 63 days into the study as indicated by arrow. Numbers on the right side refer to change in individual birds.

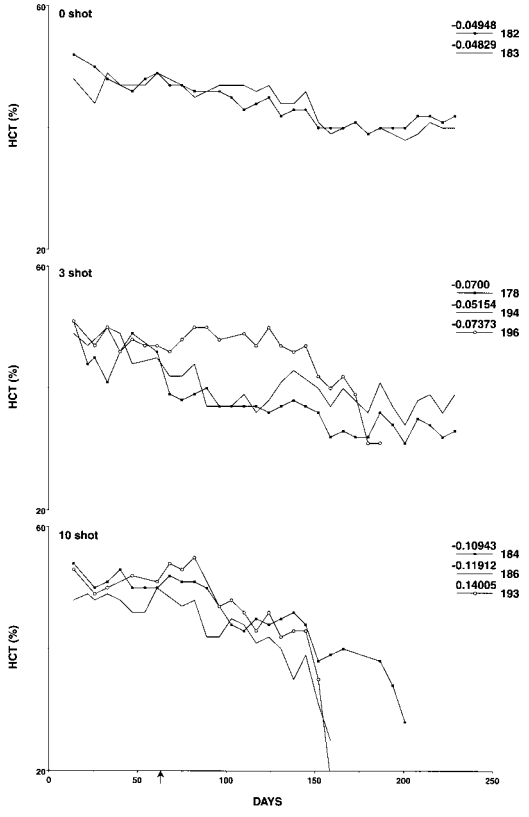


FIGURE 5. Hematocrit (HCT) values and the slope of the regression line in turkey vultures dosed with 0, 3, or 10 BB-sized lead shot. Dose in 10 shot group changed from one shot to 10 shot 63 days into the study as indicated by arrow. Numbers on the right side refer to change in individual birds.

from many other metabolic or toxic changes to make a definitive diagnosis of lead poisoning. The lone exception to this being acid-fast inclusion bodies in liver and kidney. Diagnosis of lead toxicity in this study was based on the presence of lead inclusions in liver and/or kidney cells or significant lead levels from tissue chemical analysis.

Liver lead concentrations in turkey vultures were similar to those reported by Franson (1996) as indicative of lead poisoning. Kidney lead concentrations were high when compared to reported concentrations in other Falconiformes (Franson, 1996). However, lead concentrations found in other avian orders diagnosed as lead poisoned were comparable to the vultures. Blood lead concentrations increased with exposure time, rising just prior to death. Bird 194, however, had a sudden rise in blood lead to concentrations exceeding 10 ppm, then decreased. The bird never exhibited overt clinical signs. Franson (1996) suggested blood lead concentrations greater than 1 ppm (wet weight) as indicative of lead toxicosis and levels greater than 5 ppm (wet weight) were fatal. Blood lead concentrations were greatest in the vultures that died and lowest in the controls and were approximately 1 ppm in the two dosed survivors at the termination of the study. Blood lead concentrations were 0.8 ppm (wet weight) after 24 hr and 5.4 ppm (wet weight) after 14

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TABLE 2. Spearman rank correlation coefficients for tissue lead levels, slope of the regression line for select blood parameters, and amount of lead eroded in turkey vultures dosed with lead shot (correlation coefficient/probability>[R] under Ho : Rho=0/8).

Parameter	Liver lead	Kidney lead	Heart clot lead	Slope: blood lead	Slope: ALAD[a]	Slope: EPP[b]	Slope: hematocrit
Lead eroded	0.82636 0.0114 8	0.75450 0.0305 8	0.73877 0.0579 7	0.89822 0.0024 8	−0.51498 0.1915 8	0.82636 0.0114 8	−0.77846 0.0229 8
Liver lead		0.95238 0.0003 8	1.00000 0.0 7	0.90476 0.0020 8	−0.83333 0.0102 8	0.97619 0.0001 8	−0.97619 0.0001 8
Kidney lead			0.96429 0.0005 7	0.78571 0.0208 8	−0.83333 0.0102 8	0.92857 0.0009 8	−0.97619 0.0001 8
Heart blood clot lead				0.89286 0.0068 7	−0.89286 0.0068 7	0.96429 0.0005 7	−0.96429 0.0005 7
Slope: blood lead					−0.71429 0.0465 8	0.88095 0.0039 8	−0.83333 0.0102 8
Slope: ALAD[a]						−0.80952 0.0149 8	0.88095 0.0039 8
Slope: EPP[b]							−0.95238 0.000 8

[a] ALAD=delta-aminolevulinic acid dehydratase.
[b] EPP=erythrocyte protoporphyrin.

days in experimentally dosed bald eagles (Hoffman et al., 1981); three of the five eagles died within 20 days. A turkey vulture treated by Platt et al. (1999) had a blood lead level of 2.27 ppm at death.

Delta-aminolevulinic acid dehydratase immediately dropped to near zero and remained depressed in all dosed birds. The slope of the regression line for ALAD activity over time was correlated ($P<0.05$) with the slope of the blood lead regression line. This is similar to the response of dosed bald eagles (Hoffman et al., 1981), American kestrels (*Falco sparverius*) (Franson et al., 1983; Hoffman et al., 1985), and eastern screech owls (*Otus asio*) (Beyer et al., 1988). 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).

Erythrocyte protoporphyrin increased over time. These results and reported activity levels are similar to those reported by Franson et al. (1986) in lead shot-dosed canvasbacks (*Aythya valisineria*) and by Beyer et al. (1988) for eastern screech owls. The initial EPP activity and magnitude of the response by turkey vultures was greater than reported by Rattner et al. (1989) in lead shot-dosed black ducks (*Anas rubripes*) and mallards (*A. platyrhynchos*).

At the beginning of the study, HCTs were comparable to the mean value (49.8%) reported by Coleman et al. (1988) for wild turkey vultures. In this study, HCT declined precipitously to <30% in the two birds that died. All of the exposed birds, regardless of fate, eventually had HCTs <40% whereas the two controls stayed at ≥40%. Coleman et al. (1988) reported HCT <31% in two sick/dying turkey vultures and suggested an HCT below 40% indicated a bird that was sick or in

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poor condition. Platt et al. (1999) recorded a HCT of 23% in a sick turkey vulture. The response of HCT in this study was similar to that reported by Beyer et al. (1988) in six avian species experimentally dosed with lead, by Hoffman et al. (1981) in lead-dosed bald eagles, and by Hoffman et al. (1985) in lead-dosed American kestrels.

Clinical response of individual turkey vultures was related to physiologic response to ingested lead more than to the level or severity of the lead exposure. Turkey vultures appear relatively tolerant to lead poisoning in comparison to other species dosed with lead shot. Even the most susceptible turkey vulture survived longer (143 days versus 133 days) while eroding more lead from the shot (247 mg versus 129 mg) than the most tolerant bald eagle (Pattee et al., 1981). Reiser and Temple (1981) found the turkey vulture they dosed with lead acetate accumulated lead more rapidly and excreted it more slowly than other raptors they dosed. This evidence of increased lead uptake was not reflected in increased sensitivity to lead intoxication. The initial exposure must therefore be quite high and maintained at a high level to cause acute toxicity. The birds that were found dead eroded 1.2–1.7 mg of lead per day and the two that were euthanized eroded 0.6–0.7 mg of lead per day. In contrast, the five bald eagles dosed with lead shot had erosion rates of 0.9–7.8 mg lead per day (Pattee et al., 1981). The shorter survival times were associated with the higher erosion rates.

Turkey vultures apparently can tolerate a substantial burden of lead. Whether they die or survive reflects the sensitivity of the individual, mediated by retention time of the lead object in the proventriculus and intestine, and frequency and nature of the reexposure to lead. Lead projectiles still appear to be the primary means of exposure. Contrary to Platt et al. (1999), only waterfowl hunting loads use non-toxic shot. Shotgun shells containing lead are extensively used and almost all rifle and pistol loads have lead-containing bullets. In conclusion, turkey vultures did die of lead poisoning. Tissue lead levels, enzyme activity, and histology are all within the range reported in other avian studies. However, it required more ingested lead to reach those levels and to cause mortality than reported in other avians. Consequently, turkey vultures are probably poor physiologic models for Andean or California condor lead studies.

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