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TISSUE LEAD DISTRIBUTION AND HEMATOLOGIC EFFECTS IN AMERICAN KESTRELS (*FALCO SPARVERIUS* L.) FED BIOLOGICALLY INCORPORATED LEAD

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ABSTRACT: American kestrels were fed a diet containing 0.5, 120, 212, and 448 ppm (dry wt) biologically incorporated lead (Pb) for 60 days. The diet consisted of homogenized 4-wk-old cockerels raised on feed mixed with and without lead. No kestrels died and weights did not differ among treatment groups. The control group (0.5 ppm Pb) had the lowest mean concentration of lead and the high dietary group had the highest for the following tissues: Kidney, liver, femur, brain, and blood. Concentrations of lead were significantly correlated among tissues. There were no differences among treatment groups for packed cell volume, hemoglobin concentration, or erythrocyte count.

INTRODUCTION

Lead poisoning in carnivorous birds has been reported in the bald eagle, *Haliaeetus leucocephalus* (Jacobson et al., 1977; Redig et al., 1980), Andean condor, *Vultur gryphus* (Locke et al., 1969), king vulture, *Sarcorhampus papa* (Decker et al., 1979), and prairie falcon, *Falco mexicanus* (Benson et al., 1974; Redig et al., 1980). Most of these cases seemed to result from ingestion of lead shot present in food items, presumably shot embedded in tissues of game animals killed or crippled by hunters. Since lead poisoning is well documented in waterfowl (Bellrose, 1959; Trainer and Hunt, 1965; Howard and Penumarthy, 1979) and some upland game birds (Hunter and Rosen, 1965; Westemeir, 1966; Locke and Bagley, 1967), another source of lead to raptors is shot from the gizzards of birds that died of lead toxicosis.

Little information exists on the significance of biologically incorporated lead exposure to raptors. This could be an important issue, since management decisions concerning lead shot usage may in part depend on the relative contribution of biologically incorporated lead and lead shot to raptor mortality (Pattee and Hennes, 1983). Canada geese (*Branta canadensis*) and whistling swans (*Olor columbianus*), which succumbed in lead die-offs, had 102 and 64 ppm (wet wt) lead in their livers (Trainer and Hunt, 1965; Howard and Penumarthy, 1979). Stendell (1980) fed three American kestrels a diet of homogenized carcasses of lead-poisoned mallards (*Anas platyrhynchos*) containing an average of 29 ppm lead (wet wt) and noted no mortality or treatment-related weight loss. Reiser and Temple (1981) reported clinical signs in eight of nine raptors dosed with low levels of lead acetate; however, the comparative aspects of absorption and toxicity between lead salts and tissue bound lead are poorly understood, making it difficult to extrapolate from one form of exposure to another.

In the present study, day-old white leghorn cockerels were raised on a lead diet and subsequently fed to kestrels. We report the effects of biologically incorporated lead on survival, body weight, tissue lead distribution, and hematology in kestrels.

MATERIALS AND METHODS

On 15 December 1980, 10 1-yr-old female, 18 1-yr-old male, and 12 2-yr-old male kestrels were removed from outdoor pens and randomly assigned by treatment to individual indoor

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TABLE 1. Mean concentrations of lead (ppm, dry wt) in diets of captive American kestrels.

Treatment	Mean ppm Pb (n)	Range	% Moisture
Control	0.45 (5) A*	0.32–0.62	69.2
Low	119.7 (5) B	76.0–162.6	67.7
Medium	212.05 (5) C	112.7–256.1	67.2
High	448.3 (5) D	349.1–529.2	69.7

* Mean concentrations significantly different (Tukey multiple comparison method; overall, $\alpha = 0.05$) from one another do not share the same letters.

wire cages (1 m^3). Each cage was provided with a water bowl and a wooden perch. Birds were fed about 50 g of bird of prey diet (Nebraska®, Animal Spectrum, Inc., Lincoln, Nebraska 68516, USA) six times a week. Kestrels were divided into four treatment groups (Table 1). Because of the potential interactions between sexes and a shortage of females, we put the 10 females into two separate buildings; each building contained two control and three high treatment female kestrels. One- and 2-yr-old males were assigned to the control (2 1-yr-olds, 4 2-yr-olds), low treatment (6 1-yr-olds, 2 2-yr-olds), medium treatment (5 1-yr-olds, 3 2-yr-olds), and high treatment (5 1-yr-olds, 3 2-yr-olds) groups in six buildings. One control and four treated kestrels were assigned to each of six buildings.

Beginning on 12 January 1981, kestrels in the four treatment groups were offered six times a week a diet of 50-g portions of homogenized chickens. Diets were prepared as follows: Day-old white leghorn cockerels were obtained from a local hatchery (Bowman's Hatchery, Westminster, Maryland 21157, USA) and housed in indoor pens with a photoperiod of 12 hr light per 24 hr. For treated chickens, lead acetate was dissolved in propylene glycol (1% of feed by wt) and mixed in a commercial poultry starter mash (28% protein) to approximate 2,000 ppm lead (dry wt). Chickens on control diet received clean starter mash. Feed and water were provided ad libitum for 4 wk, at which time feed was removed for 3 hr and chickens were killed with CO_2. Beaks, feet, and ends of primaries and secondaries were removed and discarded. Treated chickens were used as the high lead diet (Table 1), treated chickens and control chickens were used as the medium diet (1:1, wt:wt), and medium diet and control chickens were used as the low diet (1:1, wt:wt). All diets were mixed in a vertical mill blender.

Kestrels were maintained on treated and control diet for 60 days. Blood samples were collected by jugular venipuncture with heparinized plastic syringes, and birds were killed by halothane inhalation. Packed cell volume (PCV) was determined by the microhematocrit method, hemoglobin (Hb) was measured by the cyanomethemoglobin procedure (Hycel, Inc., Houston, Texas 77036, USA), and erythrocyte counts (RBC) were determined by a clinical laboratory (Bionetics Medical Laboratories, Kensington, Maryland 20795, USA). Duplicate samples were used for all determinations. Remaining whole blood was stored at −20 C before lead analysis.

Kidney, brain, liver, femur, and pectoral muscle were removed and stored at −20 C. Blood, tissues, and diet samples were submitted to a commercial laboratory (Hazleton Raltech, Inc., Madison, Wisconsin 53707, USA) for lead analysis according to the association of Official Analytical Chemists (1980), Methods 25.068–25.073 and 25.083–25.088. The lower level of detection for Pb was 0.5 μg. All residue data herein are reported as ppm dry weight except for blood samples, which are reported on a wet weight basis.

Statistical comparisons among means were made by using one-way analysis of variance. Multiple comparisons among groups were made by the Bonferroni method when sample sizes were not equal and by the Tukey method when sample sizes were equal (Neter and Wasserman, 1974). For samples in which no residues could be detected, a value of one-half the reportable limit was assigned before statistical analysis. In order to make the variance among treatment groups homogeneous, residue concentrations in tissues were log-transformed before statistical analysis and the antilogs (i.e., geometric means) are presented in Table 2.

RESULTS

Samples of lead-treated starter mash fed to chickens averaged 1,850 ppm lead (dry wt); two samples of untreated starter mash contained 1.54 and 1.21 ppm lead (dry wt). Hematological effects and lead levels in liver, kidney, and blood of lead-fed chickens are reported elsewhere (Franson and Custer, 1982). Concentrations of lead (ppm, dry wt) in the four homogenized chicken diets fed to kestrels averaged 0.45, 120, 212, and 448 ppm and were significantly different ($\alpha = 0.05$) from one another (Table 1).

No kestrels died and there were no be-

TABLE 2. Geometric mean concentrations of lead in tissues of American kestrels fed four diets of differing lead concentrations for 60 days.

Group	Sample size	Mean ppm lead[a] (range)					
		Dry weight					Wet weight
		Muscle	Kidney	Liver	Femur	Brain	Blood
Controls	10	0.20 (nd[b]–3.93)	1.35 A (nd–12.35)	0.42 A (nd–4.38)	1.67 A (nd–8.31)	0.70 AB (nd–10.0)	0.33 AB (nd–0.52)
Low	8	0.19 (nd–0.35)	7.47 B (nd–16.50)	4.47 B (3.33–5.69)	7.84 B (5.68–10.98)	0.66 A (nd–2.07)	0.43 B (nd–0.75)
Medium	8	0.33 (nd–2.07)	9.89 C (8.21–12.08)	6.77 BC (5.19–9.77)	16.60 C (11.25–38.89)	1.06 AB (nd–2.44)	1.13 AC (0.58–2.25)
High	14[c]	0.48 (0.26–1.45)	15.22 C (10.29–25.36)	10.59 C (6.90–15.83)	18.44 C (14.05–23.25)	2.35 B (1.07–10.16)	1.69 C (nd–9.39)
Average % moisture		68.39	67.69	63.33	9.57	76.49	77.72

[a] For each tissue, unweighted group means that are significantly different from each other (Bonferroni multiple comparison method, $\alpha = 0.05$) do not share the same superscript letters. Comparisons for age, sex, and treatment were done by using contrasts constructed to eliminate the bias introduced by missing cells.

[b] nd = not detected.

[c] Only 13 blood samples were in the high lead group.

tween-age ($n = 2$) or among-treatment ($n = 4$) differences for the weights of birds at the onset of treatment, weights at time of killing, or change in weights between these periods (1-way ANOVA, $\alpha = 0.05$). Females weighed more (1-way ANOVA, Bonferroni multiple comparison method, $\alpha = 0.05$) than males at the onset of treatment (139.1 g, $n = 10$ vs. 118.3 g, $n = 30$), but not at time of sacrifice (122.4 g vs. 115.6 g).

Sex and age did not significantly (1-way ANOVA, $\alpha = 0.05$) affect mean concentrations of lead in six tissues of kestrels fed four concentrations of lead (Table 2). Treatment did not significantly affect concentration of lead in the muscle; however, there were significant differences among treatment groups for kidney, liver, femur, brain, and blood. With the exception of brain, controls had the lowest mean concentration of lead and the high dietary group had the highest. Because of increased variability caused by one outlier in the control group, mean brain lead for controls was not significantly different from means of any other treatment group; however, mean brain lead concentration in the low treatment group was significantly less than that in the high treatment group.

Concentrations of lead were significantly correlated among all tissues (Table 3) except brain-kidney ($P = 0.06$) and brain-muscle ($P = 0.11$). When one outlier in each of the kidney, brain, and muscle samples was omitted, all intercorrelations among tissues were significant at $P \leq 0.001$. No significant treatment differences were found for PCV, HB, or RBC (1-way ANOVA, $\alpha = 0.05$).

DISCUSSION

Kestrels tolerated about 450 ppm (dry wt) dietary lead for 60 days with no weight loss, anemia, or other clinical signs. Assuming an average daily consumption of 25 g (wet wt) of homogenized chicken diet, a 120-g kestrel would have ingested about 28 mg of biologically incorporated

TABLE 3. Correlation matrix of lead concentrations in American kestrel tissues. All correlations are significant at $P \leq 0.05$ unless indicated by footnote.

	Pearson correlation coefficient[a]				
Tissue	Brain	Femur	Kidney	Liver	Muscle
Blood	0.549	0.670	0.593	0.659	0.456
Brain		0.365	0.305[b]	0.408	0.253[c]
Femur			0.847	0.914	0.525
Kidney				0.927	0.373
Liver					0.407

[a] Sample size = 40, except for blood = 39.
[b] Significant at $0.10 > P > 0.05$.
[c] $P > 0.10$.

lead per kg of body weight daily. This is nearly 10 times the dosage of lead acetate used by Reiser and Temple (1981) in red-tailed (*Buteo jamaicensis*) and rough-legged (*B. lagopus*) hawks, which resulted in clinical signs in eight of nine birds. Although their study extended over a longer time period (30 wk), one might expect a 10-fold increase in dosage to show some effects within 60 days. However, kestrels in the high dose group of our study accumulated 10.6 ppm (dry wt) liver lead, essentially equal to the 9.8 ppm (range = 2.8–22.7 ppm dry wt) reported in livers of red-tailed and rough-legged hawks fed lead acetate (Reiser and Temple, 1981). Liver lead residues in kestrels fed homogenized mallard carcasses (29 ppm, wet wt) were about 0.4 ppm (wet wt) (Stendell, 1980); for kestrels in the low dietary treatment group in the present study, dietary and liver lead concentrations were roughly comparable to those reported by Stendell (1980).

Considerable differences exist with regard to availability of different forms of lead. Stone et al. (1981) found biologically incorporated lead in oyster meat to be 69–75% as available as lead acetate to Japanese quail (*Coturnix coturnix japonica*). Availability of lead in the homogenized chicken diet was probably much less, because lead in liver and kidney contributed only small amounts of lead to total homogenate (Franson and Custer, 1982). Most of the lead was probably incorporated in bones, rendering it less available than soft-tissue bound lead. Some of the lead in the cockerels was probably in the form of lead acetate that had not passed through the digestive system. Because food was withheld from the cockerels for 3 hr prior to sacrifice, however, the amount of lead acetate in the digestive system was probably low. Mean liver lead of kestrels fed 50 ppm (wet wt) lead powder was about half (Franson et al., 1983) that of kestrels fed the lower treatment diet in the present study (39 ppm, wet wt). These data support the assumption that biologically incorporated lead is more absorbable than lead powder; substantial amounts of particulate lead probably pass through the gastrointestinal tract with little absorption. The toxicity range of these forms of lead is probably lead acetate > biologically incorporated lead > lead powder.

Based on a literature review and experimental evidence, Pattee and Hennes (1983) concluded that most cases of lead-poisoned bald eagles could be attributed to the ingestion of lead shot. The high dose diet in our study contained more lead (136 ppm, wet wt) than reported from tissues of lead-poisoned birds (Trainer and Hunt, 1965; Howard and Penumarthy, 1979; Stendell, 1980). Because no detectable adverse effects were noted over the 60 day exposure period, our study provides further evidence that lead poisoning in rap-

tors is probably due to ingestion of lead shot, not the ingestion of biologically incorporated lead. Biologically incorporated lead undoubtedly contributes to the lead burden of carnivorous birds; however, our study suggests that biologically incorporated lead alone is unlikely to cause clinical lead poisoning.

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