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EFFECTS OF CHRONIC DIETARY LEAD IN AMERICAN KESTRELS (*FALCO SPARVERIUS*)

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ABSTRACT: American kestrels were fed a diet containing 0, 10, or 50 ppm lead (Pb) powder for at least 5 mo. Blood δ-aminolevulinic acid dehydratase (ALAD) activity in birds receiving 50 ppm Pb was as low as 20% of controls but no significant effects were noted in packed cell volume (PCV) or hemoglobin concentration (Hb). Mean liver Pb residues in birds fed 50 ppm Pb were 1.3 and 2.4 ppm (dry wt) for males and females, respectively. Liver Pb residues in birds fed 10 ppm Pb were not significantly greater than controls. There was no significant correlation between blood ALAD activity and blood Pb concentration, no consistent histopathological lesions were noted, and body and organ weights were not affected.

INTRODUCTION

Lead (Pb) poisoning in waterfowl is well documented (Bellrose, 1959; Trainer and Hunt, 1965; Howard and Penumarthy, 1979) and has also been reported in ring-necked pheasants (*Phasianus colchicus*) (Hunter and Rosen, 1965), bobwhites (*Colinus virginianus*) (Westemeier, 1966), rock doves (*Columba livia*) (Hutton, 1980), and mourning doves (*Zenaida macroura*) (Locke and Bagley, 1967). Birds with clinical manifestations of Pb toxicosis would be easy prey, and there is evidence that predatory birds are exposed to Pb as a result of ingesting shot present in food items (Locke et al., 1969; Mulhern et al., 1970; Jacobson et al., 1977; Redig et al., 1980). Avian predators may also be periodically exposed to Pb by consuming tissues with elevated Pb levels. Up to 102 ppm Pb (wet wt) was found in livers of Canada geese (*Branta canadensis*) which died of Pb poisoning (Howard and Penumarthy, 1979), 64 ppm Pb (wet wt) was reported in whistling swan (*Olor columbianus*) livers (Trainer and Hunt, 1965), and Hunter and Rosen (1965) found 168 ppm Pb (wet or dry not stated) in the liver of a ring-necked pheasant which died of Pb toxicosis.

Previous work on Pb poisoning in bald eagles (*Haliaeetus leucocephalus*) using Pb shot has shown wide variations in shot retention and erosion (Pattee et al., 1981), resulting in variations in Pb exposure. Stendell (1980) found that kestrels usually regurgitated Pb shot the day after ingestion. Powdered Pb was used in the present study to ensure a more uniform exposure to metallic Pb throughout the course of the experiment. We report here the effects of chronic dietary Pb on blood δ-aminolevulinic acid dehydratase (ALAD) activity, packed cell volume (PCV), hemoglobin concentration (Hb), histopathology, and body and organ weights in American kestrels. Effects of Pb ingestion on reproduction and eggshell thickness will be reported elsewhere.

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MATERIALS AND METHODS

Forty-eight pairs of 1- to 6-yr-old pen-reared kestrels were randomly assigned on 7 November 1979 to outdoor pens (14.5 m × 3 m × 1.8 m) containing a nest box, two covered perches, two hanging perches, a covered feeding platform, and a waterbowl (Porter and Wiemeyer, 1970). Birds were fed 100 g of commercial bird of prey diet (Nebraska®, Animal Spectrum, Inc., Lincoln, Nebraska 68516, USA)/pair/day. Sixteen pairs were assigned to each of three treatment groups; control, 10 ppm Pb, and 50 ppm Pb. Diets were mixed with the appropriate amount of metallic Pb powder and supplemented with a vitamin/mineral formulation (Vionate®, E. R. Squibb & Sons, Inc., Princeton, New Jersey 08540, USA) (1%), calcium phosphate (0.5%), and furazolidone (0.0055%). Birds were maintained on these diets until females completed a clutch of eggs, 5–7 mo after initiation of the treated diets.

Upon clutch completion, pairs were bled via jugular venipuncture with heparinized plastic syringes. PCV was determined by the microhematocrit method, Hb was measured by the cyanomethemoglobin procedure (Hycel, Inc., Houston, Texas 77036, USA), and blood ALAD was assayed by the method of Burch and Siegel (1971). Duplicate samples were used for all determinations. Kestrels were killed by halothane inhalation, and 10 control and 17 birds receiving 50 ppm Pb were examined at necropsy. Carcass, liver, and kidney weights were recorded. Samples of heart, esophagus, proventriculus, gizzard, duodenum, liver, kidney, and spleen were fixed in 10% neutral buffered formalin and submitted to a commercial laboratory (American Histolabs, Inc., Rockville, Maryland 20852, USA) for sectioning and staining with hematoxylin and eosin (H&E). Duplicate kidney sec-

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TABLE 1. Packed cell volume (PCV), hemoglobin concentration (Hb), and blood δ-aminolevulinic acid dehydratase (ALAD) activity in American kestrels fed diets with 0, 10, or 50 ppm added lead (Pb) for at least 5 mo. Means ± SD.

		Females		
Added Pb	*n*	PCV (%)	Hb (g/dl)	ALAD (units)[a]
0	12	43.0 ± 3.2	12.95 ± 1.39	213 ± 29 A[b]
10 ppm	14	43.8 ± 2.5	12.97 ± 1.51	83 ± 21 B
50 ppm	14	42.6 ± 3.5	13.39 ± 1.39	42 ± 16 C

		Males		
Added Pb	*n*	PCV (%)	Hb (g/dl)	ALAD (units)
0	12	43.0 ± 3.0	13.07 ± 1.90	172 ± 19 A
10 ppm	14	42.2 ± 2.6	13.57 ± 1.53	100 ± 15 B
50 ppm	14	42.6 ± 2.9	13.39 ± 1.45	51 ± 17 C

[a] One unit of activity is an increase in absorbance at 555 nm of 0.100, with a 1.0-cm light path, per ml erythrocytes per hr, at 38 C.

[b] Means in the same column with different letters are significantly different (Bonferroni, $P < 0.05$).

tions were stained with the Kinyoun's acid-fast method (AF). Heparinized blood (1–2 ml) and livers from all birds were stored frozen in acid-washed glass jars before Pb analysis.

Blood samples for Pb analysis were thawed, weighed into a crucible, and dried in an 80 C oven overnight. Next, the sample was placed in a muffle furnace at 200 C, the temperature was raised to 550 C at the rate of 50 C/hr, and left overnight. The ash was dissolved in nitric and hydrochloric acids, then diluted to 5 ml with distilled deionized water. Pb determinations were made by atomic absorption spectrophotometry. Recoveries from duck blood spiked with Pb averaged 90%; residues were not corrected. The lower limit of detection was 0.3–0.6 ppm, depending on sample size. Livers and three samples from each diet were submitted to a commercial laboratory (Hazelton Raltech, Inc., Madison, Wisconsin 53707, USA) for Pb analysis according to the Association of Official Analytical Chemists (1980), Methods 25.068–25.073 and 25.083–25.088. A composite of the nine diet samples was analyzed by the same laboratory for protein, moisture, ash, calcium (Ca) and phosphorus (P). Analyses were according to the Association of Official Analytical Chemists (1980), as follows: protein, Method 2.057; moisture, Method 16.233; ash, Method 14.006. For Ca and P, sample preparation was according to Method 3.005 of the Association of Official Analytical Chemists (1980), and analyses were according to Dahlquist and Knoll (1978).

Multivariate analysis of covariance (Morrison, 1967), using age and duration of exposure as covariates, was used to test for differences in PCV, Hb and ALAD. The Bonferroni multiple comparison procedure (Morrison, 1967) was used to identify treatment and sex differences which were considered significant at $P < 0.05$.

TABLE 2. Liver lead (Pb) residues (ppm, dry wt) from American kestrels fed diets with 0, 10, or 50 ppm added Pb for at least 5 mo. Means ± SD.

Added Pb	*n*	Males	*n*	Females
0	15	0.43 ± 0.09 A[a]	15	0.33 ± 0.05 A
10 ppm	15	0.67 ± 0.76 A	15	0.76 ± 0.65 A
50 ppm	16	1.30 ± 1.00 B	14	2.40 ± 1.40 C

[a] Means with different letters are significantly different (Bonferroni, $P < 0.05$).

RESULTS

Blood ALAD activity was significantly higher in control females than control males, so hematological results for males and females were separated. There were no treatment or sex differences in PCV or Hb (Table 1), and there were no significant differences in PCV, Hb, or ALAD with respect to age or duration of exposure. For both sexes, ALAD was significantly reduced by increasing dietary Pb exposure (Table 1). Blood Pb was detected in five of 24 controls (0.64–3.00 ppm, mean = 1.38), eight of 28 birds fed 10 ppm Pb (0.39–14.00 ppm, mean = 2.27), and 14 of 28 birds receiving 50 ppm Pb (0.37–33.00 ppm, mean = 3.94). In birds receiving 50 ppm Pb, liver Pb residues were significantly higher in females than in males (Table 2). Liver residues from birds in the 50 ppm group were greater than residues in the two other treatment groups, but there was no significant difference between controls and 10 ppm birds (Table 2). There was no significant correlation between blood ALAD activity and blood Pb concentration. Feed samples contained the following mean Pb levels on a wet wt basis: 0.11 ppm (control); 9.67 ppm (10 ppm group); 54.0 ppm (50 ppm group). Moisture content was 56.7% and nutrient levels, as % dry matter, were: Ca, 3.39; P, 2.08; protein, 44.1; ash, 12.47.

Female whole body, kidney, and liver weights were greater than males, but there were no significant differences related to Pb dosage (Table 3). Neither were there any differences between treatment groups in ratios of kidney and liver weights to whole body weights (Table 3).

No significant gross lesions were noted in the kestrels examined at necropsy. Microscopically, medullary ray edema was seen in one kestrel fed 50 ppm Pb. No acid-fast intranuclear inclusions were noted in kidney sections. Tibial

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TABLE 3. Carcass and organ weights (g), and ratios of organ weights to carcass weights for American kestrels fed diets with 0 or 50 ppm added lead (Pb) for at least 5 mo. Means ± SD.

		Females				
Added Pb	*n*	Carcass	Liver	Kidney	Liver/carcass	Kidney/carcass
0	5	139.8 ± 7.9	3.30 ± 0.32	1.45 ± 0.44	0.024 ± 0.001	0.010 ± 0.004
50 ppm	8	150.5 ± 41.4	3.10 ± 0.39	1.25 ± 0.19	0.022 ± 0.004	0.008 ± 0.001
		Males				
Added Pb	*n*	Carcass	Liver	Kidney	Liver/carcass	Kidney/carcass
0	5	114.0 ± 11.1	2.17 ± 0.16	0.88 ± 0.07	0.019 ± 0.002	0.008 ± 0.001
50 ppm	9	112.4 ± 8.6	2.19 ± 0.39	0.95 ± 0.18	0.020 ± 0.004	0.008 ± 0.002

erythropoiesis, medullary bone deposition, and reproductive osteoclasis in three female kestrels on the Pb diet were similar to controls.

DISCUSSION

Depression of ALAD activity occurs rapidly after lead exposure (Finley et al., 1976; Hoffman et al., 1981), so kestrels in the present study may have withstood blood ALAD activities as low as 20% of normal for at least 5 mo, with no significant effects on Hb or PCV. Hoffman et al. (1981) reported that similar ALAD reduction in bald eagles resulted in significant depression of both PCV and Hb. This suggests there may be interspecies variation in raptors regarding the relationship between ALAD depression and anemia. Blood ALAD activity is a sensitive indicator of Pb exposure, but its value as an indicator of Pb toxicity should be established for individual species by correlation with clinical signs. The absence of a correlation between blood ALAD activity and blood Pb concentration is contrary to what has been reported elsewhere in ducks (Dieter et al., 1976; Finley et al., 1976; Dieter and Finley, 1979). This may be due in part to less sensitive Pb detection in the present study, since the lower detectable limit of 0.3–0.6 ppm was much higher than the lower limit of 0.02 ppm reported by Dieter and Finley (1979). It is surprising that clinical signs and hematologic effects were not observed in two kestrels with blood Pb concentrations of 14 and 33 ppm. In a study of chronic Pb acetate ingestion in red-tailed hawks (*Buteo jamaicensis*), rough-legged hawks (*Buteo lagopus*), and a golden eagle (*Aquila chrysaetos*), Reiser and Temple (1981) reported blood Pb levels of 5–8 ppm at the onset of clinical signs which resulted in death in four of nine birds. Redig et al. (1980) reported a blood Pb concentration of 11 ppm in a Pb-poisoned prairie falcon (*Falco mexicanus*) with a history of weight loss, anorexia, and anemia.

The significant sex difference in blood ALAD activity in control birds is of interest. In earlier studies with mallards (*Anas platyrhynchos*) and canvasbacks (*Aythya valisineria*) no differences were found between males and females in blood ALAD (Finley et al., 1976; Dieter et al., 1976), but the females apparently were not laying. Haeger-Aronsen et al. (1971) reported higher blood ALAD in human females than in males, but attributed it to lower Pb exposure in females. The reason for greater ALAD activity in laying female kestrels is unknown. Perhaps it was related to reproductive status.

Gross and microscopic examination indicated no consistent treatment-related lesions, although medullary ray edema in one kestrel fed 50 ppm Pb may have been related to Pb exposure. Normal medullary bone deposits and reproductive osteoclasis in three Pb-fed females which had laid five eggs each suggests that the dietary Pb did not prevent medullary bone deposition. The absence of pathology is supported by the relatively low liver Pb residues. Liver Pb residues of 10 ppm (wet wt) or greater have been shown to be associated with toxicosis leading to death in bald eagles (Pattee et al., 1981). Urban rock doves had liver Pb levels of up to 21 ppm dry wt (Hutton and Goodman, 1980), nearly 10 times what was found in the kestrels receiving 50 ppm Pb.

Results of this study indicate that dietary Pb exposure for at least 5 mo, resulting in blood ALAD levels as low as 20% of normal, had no

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adverse effects in American kestrels as measured by indices of anemia, body and organ weights, and histopathology.

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