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THE TOXICOLOGY OF INGESTED LEAD ACETATE IN RINGED TURTLE DOVES *STREPTOPELIA RISORIA*

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ABSTRACT

Adult male ringed turtle doves Streptopelia risoria *received lead acetate daily by intubation at the following dosage levels: 0, 25, 50 and 75 μg of lead per gramme (equivalent to mg kg^{-1}) body weight for 7 days. In the 75 μg g^{-1} dose level, the doves lost 17% of their original body weight as compared with 5 to 8% in other treatments; however, this effect was not statistically significant. At sacrifice (Day 7) haemoglobin was lower in the 75 μg g^{-1} treated birds and blood concentrations were greatly elevated (>300 μg%). Measurement of the enzyme, delta-aminolevulinic acid dehydratase (ALAD), proved to be a sensitive indication of lead ingestion at 5 h after the first lead dose. At 30 h and 168 h after the beginning of the experiment blood ALAD was also lowered by lead treatment. Lead concentrations in the liver, brain, and kidney were higher in the 75 μg g^{-1} treatment birds than doves that received 50 or 25 μg of lead per gramme of body weight. Brain lead averaged 12·41 μg g^{-1} in the 75 μg g^{-1} lead treatment and seizures were noted in these birds.*

INTRODUCTION

Waterfowl often ingest lead pellets spent during hunting activities, experience lead poisoning and die (Bellrose, 1959). Upland birds, such as mourning doves *Zenaida macroura*, ingest lead shot and have also been reported to be victims of lead poisoning (Locke & Bagley, 1967), the symptoms reported being a moribund, emaciated condition with acid-fast intranuclear inclusions in kidney cells. In contrast to waterfowl, the extent of lead poisoning in mourning doves would be difficult to evaluate as they are probably dispersed widely on land and are readily consumed by predators.

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The exposure of mourning doves to lead may be further compounded by their occupation and visitation of the lead-contaminated environment of roadsides (Goldsmith *et al.*, 1976; Kendall, 1980) where they consume lead-contaminated grit. Rock doves *Columba livia* were reported to have higher body burdens of lead when inhabiting urban compared with rural areas. Ohi *et al.* (1974) attributed the difference to be due to the ingestion of grit particles from roadsides with heavier traffic volumes with associated lead-contaminated exhausts. Hutton & Goodman (1980) also found rock doves had higher body burdens of lead when inhabiting urban areas compared with more rural sites. They attributed these increased body burdens of lead in rock doves to be primarily due to ingestion of food contaminated with roadside dust, particularly lead. Kendall & Scanlon (1979) reported that bone lead concentrations were higher in mourning doves when collected from areas of higher human population densities which included more roadside environments and higher traffic volumes using such roadways.

Lead has been demonstrated to be extremely deleterious as related to haeme biosynthesis (Chisholm, 1971). The blood enzyme, delta-aminolevulinic acid dehydratase (ALAD), important in haeme biosynthesis, has been shown to be almost totally inhibited in avians with elevated blood lead (Dieter *et al.*, 1976; Hutton, 1980). Bates *et al.* (1968) reported generalised hypochromic microcytic anaemia in peripheral blood of mallards *Anas platyrhynchos* poisoned by lead shot.

A noticeable effect of lead shot ingestion is weight loss in avians. At death, most waterfowl will have lost from 30 to 35% of their original body weight (Anderson, 1975). Salisbury *et al.* (1958) reported weight loss in lead intoxicated chicken hens *Gallus domesticus*.

Longcore *et al.* (1974) determined that a lead concentration exceeding $3\,\mu g\,g^{-1}$ wet weight (approximately $15\,\mu g\,g^{-1}$, dry weight) in the brain and a range of 6 to $20\,\mu g\,g^{-1}$ (approximately $24–80\,\mu g\,g^{-1}$ dry weight) in the kidney or liver, was indicative of acute lead exposure. A mourning dove dying of lead poisoning had $72\,\mu g\,g^{-1}$, wet weight lead in the liver (Locke & Bagley, 1967).

It is apparent that mourning doves have considerable potential exposure to lead from spent lead shot and from areas contaminated by exhaust of leaded gasoline. More information is needed concerning the toxicity of lead in this species. Information on the clinical signs, blood characteristics and tissue lead concentration in lead-poisoned ringed turtle doves could be of diagnostic value in identifying lead-poisoned mourning doves. Additionally, more information would become available on the toxicology of lead ingestion in avian systems. The present study was conducted to satisfy the above needs.

MATERIAL AND METHODS

Twenty (five birds per treatment) adult male ringed turtle doves ($\bar{X} = 150{\cdot}5$ g body weight) from a captive colony in the Department of Fisheries and Wildlife Sciences, Virginia Polytechnic Institute and State University, Blacksburg, Virginia, USA,

were used in this experiment. Lead was administered in the form of lead acetate (Fisher Scientific Co., Pittsburgh, PA, USA). Lead dosages to the birds were calculated as μg of lead per gramme of body weight and were as follows: (1) 0·0, (2) 25·0, (3) 50·0 and (4) 75·0. Birds given no lead received a sodium acetate solution (25 μg of Na per gramme of body weight). The experiment was a completely randomised design. Each bird was dosed daily between 0900 and 0930 h. Body weights were taken on days 1, 4 and 7.

The birds were housed individually in suspended 6·35 mm mesh stainless steel cages (24 × 18 × 18 cm). Quail Laying Mash and water were provided *ad libitum*. The laboratory was maintained at 21° ± 2 °C with a controlled light regimen (14L: 10D).

For the lead treatments, lead acetate was dissolved in deionised water at a concentration of 10,000 μg of lead per millilitre (corrected for acetate moiety) and confirmed by atomic absorption spectrophotometry. Dosage was accomplished with a 1 cc syringe equipped with an 8 cm stainless steel stomach pump needle (Popper and Sons, Inc., New Hyde Park, NY, USA). The smallest dosage was 0·36 ml while the largest was 1·3 ml (2 × 0·65 ml). These volumes were tolerated by the doves.

Blood samples, at 5 and 30 h after initial lead treatment, were taken by venipuncture (brachial vein). The blood collection technique involved removing the feathers from the brachial vein area, cleaning the area and carefully puncturing the vein with a 22 gauge needle. The blood came out of the vein in droplets which were readily collected in small, plastic heparinised Unopette blood collection systems (Becton-Dickinson, Rutherford, NJ, USA). Blood flow was terminated by compression. Approximately 0·50 ml of blood was collected from each bird at each of the first two bleedings. At sacrifice (Day 7) all birds were decapitated and blood was collected in heparinised test tubes.

Delta-aminolevulinic acid dehydratase activity (Burch & Siegel, 1971) was determined for each blood sample. With sufficient blood available, at sacrifice haemoglobin was determined colorimetrically by the formation of cyano-methaemoglobin with Hycel, Inc. (Houston, TX, USA) reagents (No. 116) and standard (No. 117). Lead concentrations in brain, liver and kidney were measured by atomic absorption spectrophotometry as previously described in Scanlon *et al.* (1980). Lead in blood was measured by the Delves cup technique (Delves, 1970).

A multivariate one-way analysis of variance (Morrison, 1976) was performed on weight and ALAD activity data. If an initial profile analysis on these variables (Wilks' Criterion Statistic) indicated a significant treatment × day interaction, an analysis of variance (one-way classification; Steel & Torrie, 1960) was performed on data collected on each individual day. Blood, kidney, liver and brain lead concentrations and haemoglobin data were analysed utilising analysis of variance (one-way classification). Duncan's Multiple Range Test (Steel & Torrie, 1960) was used to locate significant differences in treatment means. Multiple comparisons between days of weight and ALAD data within treatments were accomplished using

Hotelling's T^2 statistic. Means ($\pm$SE) were computed on all variables. Statistical tests attaining an alpha level of $p < 0{\cdot}05$ were considered statistically significant. Data were analysed using the Statistical Analysis System (SAS76, Barr *et al.*, 1976).

RESULTS

Lead treatment did not significantly affect ringed turtle dove body weight (Table 1). Treatments did not interact with time over the three weighing periods. Ringed turtle dove weight data were pairwise tested; Days 1 and 4 and Days 1 and 7 mean weights were compared. A significant difference was indicated (Table 1) in ringed turtle dove

TABLE 1
MEAN ($\pm$SE) BODY WEIGHTS ON DAYS 1, 4 AND 7 OF RINGED TURTLE DOVES THAT RECEIVED SEVEN DAILY DOSES OF LEAD ACETATE (μg OF LEAD PER GRAMME OF BODY WEIGHT)

Lead treatment[a]	N	*Weight 1*[b]	*Weight 4*[b]	*Weight 7*[b]
0	5	$161{\cdot}2 \pm 7{\cdot}0$	$147{\cdot}2 \pm 7{\cdot}2$	$146{\cdot}8 \pm 8{\cdot}1$
25 $\mu g\,g^{-1}$ daily	5	$149{\cdot}2 \pm 3{\cdot}3$	$136{\cdot}2 \pm 8{\cdot}0$	$139{\cdot}2 \pm 4{\cdot}9$
50 $\mu g\,g^{-1}$ daily	5	$142{\cdot}6 \pm 3{\cdot}7$	$133{\cdot}4 \pm 3{\cdot}7$	$135{\cdot}2 \pm 4{\cdot}3$
75 $\mu g\,g^{-1}$ daily	5	$149{\cdot}6 \pm 4{\cdot}7$	$131{\cdot}3 \pm 13{\cdot}1$[c]	$125{\cdot}3 \pm 9{\cdot}6$[c]

[a] No significant treatment effects.
[b] Ringed turtle dove weight data were pairwise tested to elucidate any time effects: weights on Day 4 were significantly ($p < 0{\cdot}05$) lower than dove weights on Day 1; weights on Day 7 were significantly lower ($p < 0{\cdot}05$) than dove weights on Day 1.
[c] $N = 4$.

weight data on Day 1 compared with Day 4 ($p < 0{\cdot}05$) and Day 1 compared with Day 7 ($p < 0{\cdot}05$). Both controls and treatment animals lost weight during the experiment. However, the 75 $\mu g\,g^{-1}$ birds had approximately a 17 % weight loss by Day 7 of the experiment as compared with 9 % in controls. The 25 and 50 $\mu g\,g^{-1}$ treatments had losses of 7 and 5 %, respectively. Thus, there was a trend towards increased weight loss in the 75 $\mu g\,g^{-1}$ treatment. These latter birds exhibited seizures and appeared not to be consuming food.

There was a highly significant ($p < 0{\cdot}001$) lead effect on ALAD activity over the three blood collection times (Table 2). The ALAD values of control birds varied little over the experiment. At the 5 h blood collection, ALAD was found to be significantly ($p < 0{\cdot}05$) lower in lead-treated birds. The ALAD activities of lead-treated birds were decreased ($p < 0{\cdot}05$) at the 30 h and 168 h blood collection times (Table 2). At 168 h (sacrifice), ALAD activity was 7 % of that of the controls in the 25 $\mu g\,g^{-1}$ treatment. Those of the 50 and 75 $\mu g\,g^{-1}$ treatments were 7 and 7·5 %, respectively. Apparently, the units of activity had reached a minimum value as all of the lead treatments approached activity that was 7 % those of controls.

Haemoglobin was lowered ($p < 0{\cdot}05$) by lead dosing in the ringed turtle doves given 75 $\mu g\,g^{-1}$ daily (Table 3). Blood lead increased ($p < 0{\cdot}05$) in all treatment groups when a determination was made at 5 h after the first lead dose (Table 3).

TABLE 2

BLOOD DELTA-AMINOLEVULINIC ACID DEHYDRATASE ACTIVITY (ALAD, $\bar{X} \pm$ SE) OF RINGED TURTLE DOVES THAT RECEIVED SEVEN DAILY DOSES OF LEAD ACETATE (μg OF LEAD PER GRAMME OF BODY WEIGHT)

Treatment	N	*ALAD at*[ab] *5 h*	*ALAD at*[ab] *30 h*	*ALAD at*[ab] *168 h*
0	5	$213{\cdot}50 \pm 44{\cdot}71^{c}$	$221{\cdot}42 \pm 52{\cdot}16^{c}$	$218{\cdot}23 \pm 55{\cdot}22^{c}$
25 $\mu g\,g^{-1}$ daily	5	$51{\cdot}63 \pm 11{\cdot}67^{d}$	$24{\cdot}62 \pm 1{\cdot}32^{d}$	$14{\cdot}42 \pm 1{\cdot}23^{d}$
50 $\mu g\,g^{-1}$ daily	5	$45{\cdot}50 \pm 6{\cdot}54^{d}$	$22{\cdot}30 \pm 0{\cdot}98^{d}$	$14{\cdot}73 \pm 1{\cdot}74^{d}$
75 $\mu g\,g^{-1}$ daily	5	$39{\cdot}96 \pm 3{\cdot}26^{d}$	$22{\cdot}38 \pm 0{\cdot}78^{de}$	$16{\cdot}41 \pm 1{\cdot}25^{de}$

[a] Units of ALAD activity expressed as the increase in absorbance of 0·100 per hour per millilitre of erythrocytes measured at 38C at 555 Nm.
[b] The ALAD data were pairwise tested for any time effects: ALAD at 5 h was not significantly different from the 30 h reading; ALAD at 5 h was significantly ($p < 0{\cdot}05$) higher than the 168 h reading.
[c,d] Means in the same column with different superscript letters are significantly different ($p < 0{\cdot}05$).
[e] Sample size = 4.

TABLE 3

MEAN ($\pm$SE) BLOOD LEAD (Pb) CONCENTRATIONS (μg PER CENT) AND HAEMOGLOBIN (Hb, g dl^{-1}) OF RINGED TURTLE DOVES THAT RECEIVED SEVEN DAILY DOSES OF LEAD ACETATE (μg OF LEAD PER GRAMME OF BODY WEIGHT)

Treatment	N	*Blood Pb*** (*5 h*)	*Blood Pb**** (*30 h*)	*Blood Pb** (*168 h*)	*Hb**** (*168 h*)
0	5	$18{\cdot}75 \pm 6{\cdot}84(4)^{a}$	$11{\cdot}40 \pm 2{\cdot}66(5)^{a}$	$18{\cdot}50 \pm 3{\cdot}50(2)^{a}$	$14{\cdot}94 \pm 0{\cdot}31(5)^{a}$
25 $\mu g\,g^{-1}$ daily	5	$169{\cdot}00 \pm 22{\cdot}19(3)^{b}$	$88{\cdot}00 \pm 12{\cdot}70(4)^{b}$	$80{\cdot}67 \pm 19{\cdot}80(3)^{a}$	$14{\cdot}43 \pm 0{\cdot}47(5)^{a}$
50 $\mu g\,g^{-1}$ daily	5	$187{\cdot}50 \pm 31{\cdot}43(4)^{b}$	$211{\cdot}40 \pm 29{\cdot}29(5)^{c}$	$121{\cdot}67 \pm 38{\cdot}52(3)^{a}$	$15{\cdot}15 \pm 0{\cdot}26(5)^{a}$
75 $\mu g\,g^{-1}$ daily	5	$311{\cdot}80 \pm 63{\cdot}76(5)^{b}$	$324{\cdot}75 \pm 26{\cdot}92(4)^{d}$	$395{\cdot}00 \pm 100{\cdot}56(4)^{b}$	$12{\cdot}08 \pm 0{\cdot}66(4)^{b}$

[a,b] Means in the same column followed by different superscript letters are significantly different $p < 0{\cdot}05$ (Duncan's New Multiple Range Test).
* Significant treatment effect ($p < 0{\cdot}05$).
** Significant treatment effect ($p < 0{\cdot}01$).
*** Significant treatment effect ($p < 0{\cdot}001$).
( ) Numbers in parentheses indicate sample size.

TABLE 4

MEANS ($\pm$SE) OF LIVER, BRAIN AND KIDNEY LEAD CONCENTRATIONS ($\mu g\,g^{-1}$, DRY WEIGHT) OF RINGED TURTLE DOVES THAT RECEIVED SEVEN DAILY DOSES OF LEAD ACETATE (μg OF LEAD PER GRAMME OF BODY WEIGHT) AND WERE SACRIFICED

Treatment	N	*Liver**	*Brain***	*Kidney***
0	5	$1{\cdot}17 \pm 0{\cdot}32^{a}$	$3{\cdot}03 \pm 1{\cdot}02^{a}$	$8{\cdot}23 \pm 3{\cdot}61^{a}$
25 $\mu g\,g^{-1}$ daily	5	$10{\cdot}71 \pm 3{\cdot}36^{a}$	$4{\cdot}15 \pm 0{\cdot}59^{a}$	$84{\cdot}47 \pm 17{\cdot}52^{a}$
50 $\mu g\,g^{-1}$ daily	5	$10{\cdot}49 \pm 1{\cdot}51^{a}$	$6{\cdot}13 \pm 0{\cdot}90^{a}$	$186{\cdot}38 \pm 27{\cdot}75^{a}$
75 $\mu g\,g^{-1}$ daily	5	$28{\cdot}59 \pm 10{\cdot}71^{b}$	$12{\cdot}41 \pm 3{\cdot}36^{b}$	$456{\cdot}91 \pm 153{\cdot}86^{b}$

[a,b] Means in the same column followed by different superscript letters are significantly different $p < 0{\cdot}05$ (Duncan's New Multiple Range Test).
* Significant treatment effect ($p < 0{\cdot}05$).
** Significant treatment effect ($p < 0{\cdot}01$).

Table 4 presents lead concentrations ($\mu g\ g^{-1}$, dry weight, $\bar{X} \pm SE$) in liver, brain and kidney. Lead concentrations in all three tissues were increased significantly by lead dosage.

DISCUSSION

Measurement of ALAD indicated that, 5 h after the first lead dose, treatment birds had at least a 76 % reduction in ALAD activity of that of controls. Dieter *et al.* (1976) showed that canvasback ducks *Aythya valisineria* exhibited a 75 % reduction in ALAD activity when blood lead was elevated. Hutton (1980) reported that rock doves with elevated blood lead had reduced blood ALAD activity. At 5 h after the first lead dose, ringed turtle doves had 169·00 μg % mean blood lead in the 25 $\mu g\ g^{-1}$ treatment group. It was apparent that lead was capable of entering the system very quickly and that ALAD activity was rapidly reduced.

Elevated blood lead disrupts ALAD activity in humans and can induce a reduction in haemoglobin (Goyer & Rhyne, 1973). At sacrifice, ringed turtle doves in the 75 $\mu g\ g^{-1}$ treatment group exhibited highly elevated blood lead (> 300 μg %) and lowered haemoglobin. Roscoe (1978) detected reduced haemoglobin in lead poisoned mallards. Since lead can impair a number of steps (e.g. ALAD and haeme synthetase) important in the biosynthesis of haeme and can cause red blood cell destruction, anaemia is a characteristic symptom of lead poisoning cases (Beliles, 1975).

Ringed turtle doves of the 75 $\mu g\ g^{-1}$ treatment group exhibited seizures and probably reflected a toxicological threshold as related to the other lead treatments. Mean brain lead was elevated to 10 $\mu g\ g^{-1}$, dry weight, in the 75 $\mu g\ g^{-1}$ treatment group. Presumably, these lead concentrations in the brain were associated with the seizures observed. Longcore *et al.* (1974) have proposed that brain lead concentrations of 3 $\mu g\ g^{-1}$, wet weight, or greater be considered as indicative of an advanced state of intoxication. Conversion of 3 $\mu g\ g^{-1}$, wet weight, to a dry weight basis indicates a concentration of approximately 15 $\mu g\ g^{-1}$, dry weight, which is very similar to the brain lead concentrations of ringed turtle doves experiencing seizures.

Many investigators have determined that liver lead concentrations provide an accurate measure of acute lead intoxication. A bald eagle *Haliaeetus leucocephalus* that was reported dying of lead poisoning had 22·9 $\mu g\ g^{-1}$ liver lead, wet weight (Jacobson *et al.*, 1977), Canada geese *Branta canadensis* had 0·5–32 $\mu g\ g^{-1}$, liver lead, wet weight before succumbing to lead poisoning (Cook & Trainer, 1966) and mallard ducks had 0·6–20 $\mu g\ g^{-1}$ liver lead, wet weight, having experienced lead intoxication (Longcore *et al.*, 1974). Locke & Bagley (1967) reported that a mourning dove dying of lead poisoning had 72 $\mu g\ g^{-1}$ wet weight liver lead. Ringed turtle doves in the 75 $\mu g\ g^{-1}$ treatment averaged 28·59 $\mu g\ g^{-1}$ dry weight liver lead.

In this group, one of the treatment birds succumbed to lead poisoning. These liver lead data appear to be diagnostically important in identifying lead poisoned ringed turtle doves.

Ringed turtle doves that ingested lead acetate provided important diagnostic criteria for identifying lead poisoning in avians. Activity of the ALAD enzyme was rapidly reduced a short time after ingestion of lead while blood lead increased. A 7-day period of elevated blood lead in the 75 $\mu g\,g^{-1}$ treatment group resulted in reduced haemoglobin. Liver and brain lead concentrations were of diagnostic significance in identifying lead poisoned avians. Body weight loss does occur during lead poisoning in doves but this characteristic is of questionable importance for diagnostic purposes.

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