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Heat Exposure and the Toxicity of One Number Four Lead Shot in Mallards, *Anas platyrhynchos*

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Lead poisoning from ingested shot is a major cause of mortality in waterfowl throughout the world. In North America, most waterfowl that die from lead poisoning succumb following the hunting season (Sanderson and Bellrose 1986). Cold and harsh winter weather is generally thought to exacerbate lead toxicity in birds. This has been in part confirmed by Kendall and Scanlon (1984) with ringed turtle doves (*Streptopelia risoria*); marked mortality and greater blood and liver lead concentrations were observed in doves exposed to 6°C compared with birds maintained at 21°C. Although largely undocumented, there is considerable opportunity for birds to ingest lead shot in the summer during periods of extreme heat. Substantial lead exposure in over 50% of the American black ducks (*Anas rubripes*) captured in late summer has been observed in areas of the Chesapeake Bay (D. J. Pain, *unpublished observations*). Moreover, investigations with rodents (Baetjer et al. 1960; Edwards and Beatson 1984), rabbits (Horiguchi et al. 1979), and man (Baetjer 1959) have demonstrated that the toxicity of lead is increased by high environmental temperature, possibly caused by elevated metabolic rate, dehydration, and impaired lead excretion. A recent study in which black ducks were dosed with a single number 4 lead shot suggested that toxicity may be enhanced during periods of extreme hot weather (D. J. Pain, *unpublished observations*). To further investigate this finding, we examined lead toxicity in mallards maintained at thermoneutral temperature (21°C) and at an

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elevated temperature (35°C) approaching the upper critical limit (Weathers 1981).

MATERIALS AND METHODS

Mallard drakes (6 months old) were housed in vinyl-coated wire cages (90 x 90 x 60 cm) within environmental chambers at 21 ± 1°C in a 12-L:12-D photoperiod for 30 days before the experiment. Ducks were provided a nutritionally-balanced diet (Duck Developer Mash, Beacon Milling Co., Cayuga, NY) and water ad libitum, and were randomly assigned to treatment groups. Ten days before dosing, temperature in one chamber was increased to 35 ± 1°C and the other remained at 21°C. On day 0, three ducks in each chamber were sham-dosed (intubated but not dosed) and eight in each chamber were dosed with a single preweighed number 4 lead shot (mean ± SD: 232 ± 15 mg). Ducks were weighed, fluoroscoped, and bled by jugular venipuncture 10 days predosing, before intubation on day 0, and on days 3, 7, 14, and 21 after sham- or lead-dosing. On day 21, ducks were killed with CO_2, necropsied, and liver and eroded shot were weighed.

Hematocrit and hemoglobin concentration (cyanomethemoglobin method) were determined from heparinized blood samples. Aliquots of whole blood and plasma were frozen at -70°C for subsequent quantification of red blood cell delta-aminolevulinic acid dehydratase activity (ALAD; Burch and Siegel 1971; optimized for mallards with pH 6.4 buffer) and plasma chemistries (alanine and aspartate aminotransferase, alkaline phosphatase, total protein, albumin, and uric acid with Baker Instrument Corporation reagent kits using a CentrifiChem®[1] 500 analyzer). The remainder of each whole blood sample was held for 48 hours at 4°C before measuring free-protoporphyrin (Roscoe et al. 1979).

Variables were tested for homogeneity of variance and transformed as appropriate. Variables were then compared using ANOVA techniques and Tukey's method of multiple comparison. Untransformed data are presented in the text.

RESULTS AND DISCUSSION

None of the mallards maintained on a balanced diet died following intubation of a single number 4 lead shot. Although rather variable, ingestion of one number 4 lead shot by free-ranging waterfowl ingesting natural foods (Bellrose 1959) or pen-reared birds maintained on an incomplete diet (Longcore et al. 1974a, b) can result in as much as 20% mortality. Analysis of variance for repeated measures (2 temperature regimens x sham- and lead-dosed x 6 times of sampling) revealed a slight decrease ($P < 0.10$) in body weight following shot intubation that was comparable at both

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[1] Mention of a trade name, proprietary product, or specific equipment does not constitute a guarantee or warranty by the United States Department of the Interior.

environmental temperatures (average loss of all dosed birds on days 3, 7, 14, and 21 postdosing was 2.0, 3.6, 7.7, and 10.6%). Fluoroscopy and necropsy revealed that seven of the eight dosed birds reared at 21° or 35°C retained shot, with 184 ± 31 and 172 ± 14 mg of lead eroded over 21 days, respectively. Body weight change, shot retention, and erosion rates were similar to those observed in other studies with mallards (Finley et al. 1976). Only three ducks (2 at 21°C and 1 at 35°C) became emaciated with bile-staining of the gizzard and intestine, both classical signs of lead poisoning. Liver:body weight ratio did not differ between dosed groups at necropsy (g liver/kg body weight: 1.85 ± 0.66 at 21°C vs 1.95 ± 0.39 at 35°C).

Hematocrit, hemoglobin concentration, red blood cell ALAD activity, and free-protoporphyrin levels were measured as indices of lead exposure and toxicity. Following shot intubation of 21° and 35°C test groups, hematocrit and hemoglobin concentration declined ($P < 0.01$), log-transformed ALAD activity was inhibited ($P < 0.01$) by over 60% through day 21, and log-transformed protoporphyrin levels increased ($P < 0.01$) to day 14 and then seemed to decline (Fig. 1). The magnitude and time course of these changes did not differ between the 21° and 35°C groups and, in general, closely resembled responses of mallards in other lead shot studies (Del Bono and Braca 1973; Finley et al. 1976; Roscoe et al. 1979). Physiological condition of ducks was assessed by quantifying enzyme activities suggestive of liver and bone marrow damage, plasma protein levels indicative of liver function, and uric acid concentration reflecting nucleotide metabolism and kidney function. Although several of these blood chemistry measurements have been reported to be altered by lead shot exposure in waterfowl (e.g., elevated alanine and aspartate aminotransferase, inhibited alkaline phosphatase) (Rozman et al. 1974; Hoffman et al. 1981), none were affected in ducks receiving sublethal doses of lead and exposed to 21° or 35°C.

In conclusion, elevated environmental temperature did not appear to dramatically affect the toxicity of one number 4 lead shot to mallards fed a nutritionally-balanced diet. Although this finding is compromised by relatively small sample size, the marked increase in lead toxicity induced by mild cold exposure (>70% mortality; Kendall and Scanlon 1984) suggests that elevated temperature may have only minimal effects on lead toxicity in birds. This is in contrast to observations in mammals, wherein heat exposure enhanced lead toxicity, as evidenced by increased lethality, greater weight loss and anemia, higher lead concentration in tissues, and lower lead excretion rates compared to thermoneutral lead-dosed controls (Baetjer et al. 1960; Horiguchi et al. 1979). Further investigations using moderately higher doses of lead in heat-stressed waterfowl seem warranted.

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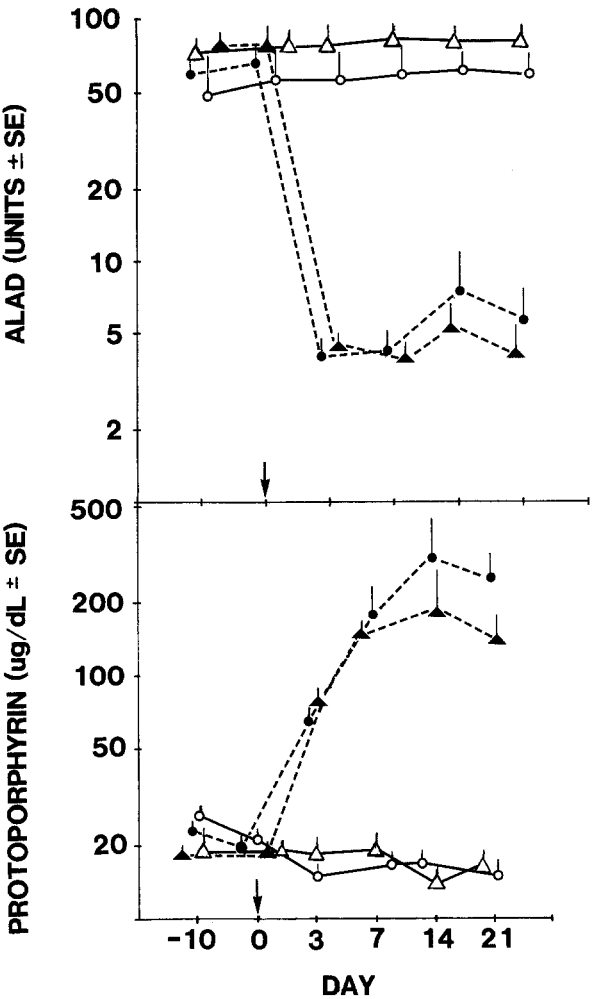

Figure 1. Whole blood ALAD activity and free-protoporphyrin concentration in sham-dosed (o----o = 21°C; Δ----Δ = 35°C) and one number 4 lead shot-dosed (●----● = 21°C; ▲----▲ = 35°C) mallards maintained at various temperatures. Arrow denotes day of sham- and lead-dosing.

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