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ENVIRONMENTAL UPTAKE OF LEAD IN BLACK-CROWNED NIGHT HERONS

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ABSTRACT

Samples of black-crowned night heron tissue were examined for lead using spectrophotometric analysis. Average lead content in tissues (in ppm wet weight) were 4.87 in eggshell, 3.6 in egg contents, 0.35 in developing embryos, and 18.3 in young heron tissues. These data suggest ontogenic dilution of lead and then dosage, presumably by food, in young herons. Heron foods contained lead. It is contended that lead from spent gunshot was dissolved, taken up by resident organisms, and then accumulated by young herons through the ingesting of food species. There are other pathways by which herons can take up lead. Though the amount of lead concentratable through the process described in this paper does not appear to be extraordinarily great, it is something about which to be aware, especially in this species whose breeding range and habitat seem to be decreasing.

1. INTRODUCTION

Historically, research on lead in natural systems has been concentrated on the ingestion of lead (in the form of spent gunshot) by waterfowl. However, there is some concern that lead from so-called environmental sources (i.e. lead which is introduced by surreptitious entrance into and throughout ecosystems) may have more widespread effects than lead from ingested shot. We are concerned here with certain, perhaps more vulnerable, environments.

It has been suggested that heavily hunted freshwater marshes may be especially vulnerable to environmental lead contamination by the introduction of spent lead shot and subsequent dissolution and uptake of lead by successively higher trophic levels (1), and there is compelling evidence that this does, in fact, occur. If this can be further demonstrated, the implications are impressive: 1969 data show that 6 * 10~6 kg of lead shot are deposited by hunters on waterfowl areas in the U.S. each year (2).

One of these waterfowl areas, Rush Lake in Winnebago Co., Wisconsin has for many years received heavy season-long hunting pressure. As much as 10^3 kg of lead shot has been delivered into Rush Lake on the opening day of waterfowl hunting season alone (3).

Chemically, lead is only mildly reactive in subtle reducing environments such as freshwater marshes. However, certain substances in biodegradable detergents readily dissolve lead, thus making it abundantly available for uptake by aquatic communities (4). Certain marine molluscs are able to concentrate lead from their environment (5). On Rush Lake, elevated levels of lead have been found in substrate sediments and in Chara, the dominant submergent plant of the lake (1). Further investigation of the Rush Lake biota has revealed marked lead contamination of algae other than Chara, mollusc shell, fish tissues (especially bone), and bird tissues (especially bone and eggshell).

Lead may be bioaccumulated in the Rush Lake biota much in the manner which has been shown for certain pesticides. Support for this idea is offered by data on bald eagles, which seem to accumulate more pesticide than golden eagles, presumably because of this increased length of the food chain for piscivorous balds (6). Thus, a focal (and most vulnerable) species may be the terminal consumers of aquatic systems. This idea has become well established. On Rush Lake one of the more conspicuous terminal consumers, the black-crowned night heron, was especially interesting for two other reasons. First, it has been found that avian predators consistently show greater heavy metal concentrations than mammalian predators (7). Second, these birds seem to have a somewhat precarious foothold in much of their breeding range. I, therefore, decided to examine these birds and their interaction with the lead pathways in this marsh system.

The major objective of this study was to determine the magnitude of lead contamination in the Rush Lake black-crowned night heron population. But because relatively high concentrations of lead had already been shown to be present in a wide variety of organisms in this system, and because herons ingest some of these lead-laden species as food, additional questions concerning those possibilities were asked. Specifically, what were the lead levels in heron foods? Was lead accumulated ontogenically in the offspring? Were there any possible interferences by lead in the reproductive success of adult herons? Finally, what were the possible dynamics and ramifications of lead uptake and deposition in these birds? I examined lead concentrations in eggs, young herons, and heron foods. I also considered the influence of lead on various aspects of the life history of this population.

In this paper I will discuss lead accumulation via environmental means in young night herons.

2. METHODS

I studied a colony of about 200 nesting herons during a 43-day observation period in the late spring of 1975. I

collected breeding biology data and salvaged broken and displaced eggs, regurgitae, and young herons which had died. I also monitored the growth of young herons.

In the lab I measured eggshells and dissected the salvaged young into tissue groups of special interest.

All biological materials were weighed to the nearest 0.0001 g. Samples were then dried for 24 hours and ashed in a furnace at 800 degrees C. Ashed samples were digested in 4 N nitric acid and brought to a 25 ml volume by water dilution. The samples were then assayed for lead by atomic absorption spectrophotometry. Lead concentrations are reported on a wet-weight basis, unless otherwise indicated.

I performed a posteriori statistical tests of hypotheses using data analysis. The validity of assumptions on the distribution characteristics of the data as well as the validity of specific hypotheses was tested. The statistical procedures used in this study are outlined by Sokal and Rolff (8). Data are presented by the mean followed by the standard error.

3. RESULTS

A total of 224 biological samples was analyzed for lead content. Tissues averaged 6.2 + 3.2 ppm lead (n=224) on a wet weight basis, and ranged from 0.1 ppm in embryonic tissue to 123.1 ppm in young heron bone tissue. The two most significant features of these data are the seemingly ubiquitous nature of lead and the great variation of values encountered.

In an effort to determine where the variability of observed values lay, I divided tissues into the following groups: eggshell, egg contents, embryos, and young herons. A summary of the lead residues found in night herons is given in Table 1. All sampled tissues were positive for lead.

Were there actual differences in lead concentrations among these four groups? Statistical comparisons were made of lead amounts in tissues within and among life history groups (eggs, embryos, and young). Tissues among these groups differed significantly in lead concentration (F=13.81, KW statistic =

Table 1. Summary statistics of lead concentrations in tissues of black-crowned night herons. (ppm fresh weight)

Statistic	Eggshell	Egg contents	Embryos	Young *
N	135	35	6	12
Mean	4.78	3.6	0.35	18.35
Range	1.0 - 40.1	0.2 - 14.9	0.1 - 0.7	0.3 - 123.1
SD	2.40	3.20	0.25	27.84
SE	0.21	0.54	0.11	5.46

*Refers to bone, liver, and feathers of young herons, collectively.

Table 2. A summary of comparisons of lead concentrations in tissues between and among life history stages of black-crowned night herons. All figures are significant at the p<0.001 level unless otherwise indicated.

Comparison	ANOVA	T-Test(2)	U-Test
All tissues	13.81		2045(3)
Within stages			
Eggs			
Shell vs. contents	5.75(b)	2.4(b)	2.78(c)
Young heron tissues			
Liver vs. bone vs. feathers	1.30(a)		
Liver vs. bone	0.01(a)	1.02(a)	28(a)
Liver vs. feathers	5.39(b)	1.42(a)	35(a)
Bone vs. feathers	2.10(a)	1.45(a)	63(a)
Among stages(1)			
Shell vs. embryo vs. young	17.39		
Contents vs. embryo vs. young	6.07(c)		
Shell vs. young	31.72	2.53(c)	2.66(c)
Contents vs. young	9.72	2.74(c)	3.07(c)
Embryo vs. young	2.45(a)	3.17(c)	3.45
Embryo vs. shell	19.79	19.29	4.14
Embryo vs. contents	7.32(c)	5.91	3.45

(a) p>0.05 (1) Young heron tissues pooled
(b) p<0.05 (2) Unequal variance
(c) p<0.01 (3) Kruskal-Wallis test

2045, p<0.001). Further comparisons were made in order to clarify the nature of the variability among specific groups. A summary of these tests is given in table 2.

Results of the analysis of lead amounts in tissues within life history stages were divergent. The two egg stage tissues (eggshell and egg contents) differed significantly (F=5.75; t=2.4; U=2.78; 0.05>p<0.001) while young heron samples showed no such difference on a parametric basis (p>0.05). The application of analysis of variance was probably not appropriate for young heron tissues as their sample size may have precluded the validity of application of a parametric assumption set. Thus, the significance of the F statistic above (Table 2, liver vs. feathers) may be misleading. Also, even if the samples could have been shown to satisfy the parametric assumption set, it is quite possible that an added variance component (such as age) may have affected the variability of encountered lead concentrations in this case.

These comparisons point to a high within group variation for young heron tissues, but not so great that young herons could not be considered a separate entity in terms of lead concentration.

I conclude that there are four distinct tissue populations with respect to lead load. Egg tissues (both eggshell and egg contents) contained greater concentrations of lead than embryonic tissues, and young heron tissues contained more lead than either other group.

The proportion of variability accounted for among groups is between 86 and 96 percent (see Table 2, ANOVA column, among groups comparisons). The relationships of lead concentrations among life history groups (eggs > embryos < young herons) suggest ontogenic dilution of lead during incubation and then dosage by food in the young herons.

It is clear that eggs, embryos and young herons differed in the amounts of lead contained in their respective tissues. It is suspected that some factor or factors other than heritability from the adult female (present but probably of decreasing importance as ontogeny progressed) operated on this system to create these differences. It is possible that there was some very small transfer of lead from adults to offspring (embryos and young herons), but the very large amounts of lead in young herons cannot be accounted for by heritability to any measureable degree. It appears that some dilution of lead occured during embryonic development and it seems nearly conclusive that relatively large amounts of lead seem to have appeared in offspring after hatching. We now look to a suspected dosage medium (i.e. heron foods) for further elucidation of this problem.

The favored food of this population appeared to be fish (see Table 3, column C) but herons ate a variety of foods during the study period. The average amount of lead present in all heron food items was 2.72 + 2.2 ppm.

Statistically, lead concentrations in heron foods differed

Table 3. Foods summary for black-crowned night herons on Rush Lake in 1975.

	(A)	(B)	(C)	(D)	(E)
Mammals	12.3	4.3	1.3	0.57	0.23
Birds	2.6	15.2	1.1	1.20	0.23
Amphibians	12.4	8.7	2.7	2.51	2.02
Fish	60.9	58.7	91.0	3.52	95.55
Invertebrates	11.8	13.1	3.9	1.56	1.81

(A) % total of sampled mass based on collected samples of regurgitated items and proventricular contents of salvaged birds (n=20).
(B) % frequency occurrence of all food items based on samples listed in (A) in addition to field observations of foods which were not collected (n=64).
(C) adjusted % of total dietary mass.
(D) average lead load per food type (in ppm).
(E) % of total lead intake per source.

significantly from adult derived tissue lead (eggshell: $F=6.66$, $p<0.05$; contents: $t=2.13$, $p< 0.05$), young heron tissue lead ($t=2.02$, $p<0.05$), and embryo lead ($U=48$, $p<0.01$). Embryos contained less lead than did food items while both eggs and young herons contained more.

In sum, heron foods contained lead, and feeding herons (both young and probably adults, based on possible adult lead contributions to eggshell and egg contents) contained more lead than their foods. Only the nonfeeding component of the population (embryos) contained less lead than heron foods. It is therefore highly probable that lead is passed to herons through their foods and that the bulk of the lead found in heron tissues is attributable to their food sources.

There seems to be ample experimental evidence which indicates that lead is retained in organisms which chronically ingest lead contaminated foods (9). The same evidence indicates that the amounts of lead animals will accumulate is a function of ingested vs. excreted lead. It follows that those animals which ingest lead contaminated foods for the longest time will have larger amounts of retained lead in their tissues. If that is so, then older herons should have higher lead loads than younger herons. I tested the relationships between age of young herons and their respective tissue lead loads. One significant correlation was found between ranked age and feather lead ($Rho = 0.66$, $p<0.05$). This may be an indicator that young herons accumulate lead, but there was no other evidence in this study which supports such a contention.

4. DISCUSSION

Lead studies in wild populations have generally fallen into two major groups: those that report acute toxic episodes resulting from ingestion of spent shot, and those that report baseline information on lead loads, presumably environmentally accumulated. Consequently, published figures for lead concentrations have been, predictably, divergent (10). Generally, it appears that lead residues found in young Rush Lake herons exceed concentrations found in other studies of (perhaps) environmentally contaminated species and fall within the range, and in some cases exceed the upper limit, of values found in acutely lead-poisoned wildlife.

The validity of making comparisons among studies of lead toxicity is called into question on a number of points. Variations among studies may be attributable to analytical technique (11) or differences in species, sex, stress, disease, age, path of absorption, and length of exposure (12).

There is some indication that lead concentrations in indigenous species are a reflection of the amount of lead present as a proportion of the earth's crust in that area (13). If that is so, Rush Lake organisms would then accumulate lead from at least four sources: the soil as a natural occurrence, atmospheric lead, lead deposited into the water by outboard motors which burn leaded fuel, and dissolved lead shot. The

ultimate importance of a determination of relative contributions of these sources lies in the implications this study may have on the multiple use nature of Rush Lake. It essentially makes no difference as to what, exactly, is the source of lead. The fact remains that these herons have lead in their tissues, and so do their foods, sometimes in relatively high concentrations.

Because lead was found in these young night herons, it must be assumed that they ate it as part of their food, that they were shot, or that they absorbed it through some body surface. The young herons studied herein had not been shot. No test was performed in order to assess gaseous lead presence, but as the only conceivable source of airborne lead would have been automobile exhaust, it seems unlikely that herons would have absorbed this lead to a great degree. It is possible that herons may have absorbed water-borne lead through body surfaces or as incidental to the ingestion of food. Herons do not drink a great deal of water, and the concentration of lead in Rush Lake water is less than 1 ppm (1). Herons may absorb lead through body contact with the water but if that were true one might expect the lead concentrations in each of the separate tissue groups to be more closely clustered. Thus the largest likely source of lead for herons is their food. Evidence presented in this study indicates that large amounts of lead are available for ingestion via this mode.

Night heron foods have been shown to be a statistically distinct entity with respect to lead values. Fish were the most important food items for herons during the study period. Because lead concentrations in fish vary as to tissue type (14), a knowledge of portions of foods eaten by herons is important. Lead residues concentrate in specific tissues, especially bone, liver, and kidney (10). Heron regurgitae contained whole small fish but larger fish (with soft parts partially eaten) were observed in the heronry.

Mallards accumulate approximately 0.02 percent of ingested lead when dosed with highly lead-contaminated foods (15). On the basis of this evidence, young herons must either have selected more highly contaminated portions (a contention with no data base) or accumulated lead in inordinately high proportions. This latter idea is supported by evidence in young rats, which accumulated lead three times faster than adults of the same species (16).

Observations of night herons revealed that young are fed between 4 and 6 times per day (17). Based on this information young herons in this study would have stored about 10 percent of the ingested lead. This seems somewhat high but may be explained on the basis of selection for more highly contaminated foods (i.e. fish) and the evidence indicating the susceptibility for growing young to store inordinately high amounts of lead.

It has been demonstrated that even very small amounts of lead may have detrimental effects on a variety of physiological functions (18). Recently, the enzyme delta-aminolevulinic acid dehydratase (ALAD) was found to be significantly depressed by lead in canvasback ducks (19). Since this enzyme is involved in

erythrocyte synthesis, the implications of even very low lead levels may be more serious than previously thought. The effects of low lead levels on dysfunction in any system are subtle and may not be detectable or statistically implicative in short term studies as factors which contribute to the possible decline of wild populations, but they may be exceedingly important in a long term sense. Populations of black-crowned night herons as terminal trophic level consumers and environmental accumulators of lead will require special monitoring if we are to prevent any problems which may (already be) develop(ing).

5. CONCLUSIONS

a) Young black-crowned night herons have larger concentrations of lead in their tissues than heron embryos and adult-derived egg tissues.

b) Heron foods contain lead.

c) The largest and most likely source of lead for young herons is their food.

d) It is probable that lead is passed through food chains.

e) Black-crowned night herons may be especially vulnerable because (i) lead is present in their systems, (ii) it affects subtle physiological functions, and (iii) these herons have declining breeding ranges.

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