

Comparison of Lead Levels in Bone, Feathers, and Liver of Herring Gull Chicks (*Larus argentatus*)

JOANNA BURGER,*† STEPHANIE M. REILLY† AND MICHAEL GOCHFELD†‡

*Department of Biology, Rutgers University, Piscataway, NJ 08855

†Environmental and Occupational Health Sciences Institute, Piscataway, NJ 08854

‡Environmental and Community Medicine, UMDNJ-Robert Wood Johnson Medical School,
Piscataway, NJ 08854

Received 16 September 1991

BURGER, J., S. M. REILLY AND M. GOCHFELD. *Comparison of lead levels in bone, feathers, and liver of herring gull chicks (*Larus argentatus*)*. PHARMACOL BIOCHEM BEHAV 41(2) 289-293, 1992. — Bone, feathers, and liver were analyzed for lead in herring gull chicks (*Larus argentatus*) of two different ages. The highest levels were found in the bone, evidence of chronic exposure. No differences were found within the bones. Differences occurred between different bones, with the ribs having twice the amount of lead than any other bone. These studies indicate that type of bone affects lead levels; thus researchers should clearly state which parts of which bones are examined. It is also suggested that for humans consistent location should be used for analysis by in vivo X-ray fluorescence.

Lead Bone Tissue deposition Gulls Feather Liver

LEAD is a common contaminant of the environment, whether from natural or anthropogenic sources. Mortality in wild birds has mostly been attributed to ingestion of lead shot (31,39), but there are many other sources of lead available. Contamination of the Coeur d'Alene Valley by mine wastes resulted in increased mortality of swans (*Cygnus*) and other waterfowl (14). Vehicle emissions, smoke from coal and refuse burning, and emissions from metallurgical and industrial processes are all sources of lead contamination (15).

Exposure to lead can cause a variety of pathological, physiological, and neurological effects including impairment of behavior functions (21,24). Barthalmus et al. (5) showed that the schedule-controlled response of the adult male White Carneaux pigeon is sensitive to chronic low-level exposure to lead. Avery and Cross (3) found a dose-related relationship in impairment of learning and memory in the rat. Snowdon (35) suggested that rats may be more sensitive to the behavioral effects of lead during the earliest developmental stages. Low concentrations of lead have been shown to retard learning in children (2,7,8,11,12,23,25,33).

There are data on the concentrations of lead in tissues of birds that have died from lead poisoning, but there is little information of the relationships of natural, nonlethal levels of lead in different tissues of wild birds that include feathers, bone, and liver.

Lead is unevenly distributed in the vertebrate body (4,41), with the skeleton containing over 90% of the total body burden of lead (32). For humans, 70% of lead in children is present in osseous tissue, and over 95% of lead in adults is in bone (26). Uptake of lead by bone is rapid and release is slow, so lead concentrations in bone reflect chronic exposure (38). The bone pool of lead is in dynamic equilibrium with the blood lead, and during chelation treatment for lead poisoning a dramatic decline in blood lead is followed by a rebound, when lead is mobilized from the skeleton and other organs (17,34). Humans and other animals continually exposed to low amounts of lead over extended periods of time accumulate high levels in their bones (39). By contrast, high concentrations of lead in the liver reflect recent acute exposure (31).

Recognition of the importance of the skeleton as a reservoir for lead and the rebound of blood lead levels in people undergoing chelation has focused attention on a better understanding of the skeletal blood pool and its potential utility as an indicator of exposure status. Although the affinity of lead for the skeleton has been recognized (26), there are few evaluations of skeletal lead toxicity in humans or animals (29). Lead indirectly alters bone cell function through changes in circulating levels of hormones, by perturbing the ability of bone cells to respond to hormonal regulation, and by impairing the ability of cells to synthesize or secrete components of the bone

¹ Requests for reprints should be addressed to Joanna Burger, Department of Biology, Rutgers University, Piscataway, NJ 08855.

matrix (29). Further, during periods of altered mineral metabolism (as in pregnancy) lead in the bone may be mobilized and transferred to the more bioavailable compartment of circulation (34). Nonetheless, because of its relative stability, lead in bone may be a more accurate dosimeter for chronic exposure (34). Since lead is one of the major public health problems in the United States (26), understanding lead deposition in bone is important.

In the past, lead was assayed by biopsies of a small sample of bone, but a major question related to its representativeness (26). With the development of X-ray fluorescence as a means of assaying human skeletal lead in vivo (13,30), it is important to understand how lead levels vary among bones and within the parts of bones. We use an avian model to assess these differences using traditional AA spectrophotometry techniques.

We studied the distribution of lead in the avian skeleton in lead-exposed and control birds. The objectives were to compare lead levels in different parts of several bones in herring gulls (*Larus argentatus*) and compare lead levels in bone with those in liver and feathers. We chose liver and feathers because they are most often used for monitoring lead levels in wild birds.

METHODS

We performed three experiments with herring gull chicks:

1. comparison of lead levels in the radius and ulna of 30-day-old chicks;
2. comparison of lead levels in the liver, feathers, tibiotarsus, and radius-ulna of 2-day-old chicks; and
3. comparison of lead levels in the liver, feathers, skull, tibiotarsus, and radius-ulna of 15- to 20-day-old chicks.

All herring gull chicks (*L. argentatus*) were collected under appropriate federal and state permits. We analyzed bone, liver, and breast feathers. Acid-rinsed instruments were used for all dissections. After dissection, all bones were rinsed in distilled water and dried overnight at 100°C. The bones were ashed in a muffle furnace at 550°C for 8 h or until completely white (40). A section of the liver was removed and dried overnight. Breast feathers were washed three times with acetone and distilled water and then dried overnight. All samples were digested with 3 ml Ultrex nitric acid and then diluted to 10 ml. Samples were analyzed on a Perkin-Elmer graphite furnace atomic absorption spectrophotometer using a modification of methods described by the EPA (18).

In the first experiment, 22 1-day-old herring gull chicks were collected from Captree, Long Island. The chicks were randomly assigned to two groups: lead dosed or control (9). Dosed birds received 0.1 or 0.2 mg/g of a 50 mg/ml lead nitrate solution IP on day 2. All birds were fed fresh fish obtained from bait shops. At 30 days of age, the chicks were sacrificed and the radius and ulna were dissected out and divided into three parts. The radius was separated into the head, shaft, and styloid; the ulna into the styloid, shaft, and coronoid.

In the second experiment, 11 2-day-old chicks were collected. Liver, feathers, tibiotarsus, and radius-ulna were analyzed.

In the third experiment, we collected 13 15- to 20-day-old herring gull chicks. Liver, feathers, segments from the skull and the midsection of the tibiotarsus and radius-ulna, and six ribs were removed from each bird for analysis. Three anterior rib sections and three dorsal sections were analyzed. Two birds had two vertebrae and additional ribs analyzed.

TABLE 1
LEAD LEVELS (PPM DRY WEIGHT) IN RADIUS-ULNA OF HERRING GULL CHICKS (*L. ARGENTATUS*, AGE = 30 DAYS)

	N	Ulna	Radius	χ^2 (p)
Control				
Styloid	11	12.9 ± 9.7	39.4 ± 30.2	4.8 (0.03)
Shaft	11	19.6 ± 19.5	15.2 ± 9.8	NS
Coronoid/head	11	13.1 ± 19.5	25.8 ± 26.7	5.1 (0.02)
χ^2 (p)		NS	NS	
Dosed				
Styloid	11	111.0 ± 69.2	210.3 ± 126	5.1 (0.02)
Shaft	11	158.7 ± 117.5	161.3 ± 118.2	NS
Coronoid/head	11	120.4 ± 64.7	145.3 ± 80.2	NS
χ^2 (p)		NS	NS	

Given as mean ± SE; Kruskal-Wallis χ^2 ; NS, not significant.

RESULTS

Although there was a difference overall between the dosed and control 30-day-old birds in lead levels in the radius and ulna, only the styloid and coronoid/head portions of the control birds and the styloid of the dosed birds showed any significant differences (Table 1). The dosed birds showed 11 of a possible 15 positive correlations in levels of lead among and within parts of the radius and ulna, a greater number than in the control birds (Table 2).

The above experiments indicated that there were some differences in lead levels in 30-day-old chicks among and within bone. These chicks, however, were raised in the laboratory. Thus in the next experiment we examined chicks from the wild

TABLE 2
CORRELATIONS OF LEAD LEVELS IN DIFFERENT PARTS OF THE RADIUS AND ULNA AMONG HERRING GULLS (30 DAYS, KENDALL TAU TEST)

	Ulna			Radius		
	Styloid	Shaft	Coronoid	Head	Shaft	Styloid
Control						
Ulna						
Styloid	—	0.382	-0.055	0.564	0.236	0.709
Shaft	NS	—	0.127	0.163	0.418	0.163
Coronoid	NS	NS	—	-0.200	-0.090	-0.055
Radius						
Head	0.016	NS	NS	—	-0.055	0.636
Shaft	NS	NS	NS	NS	—	0.018
Styloid	0.002	NS	NS	0.006	NS	—
Dosed						
Ulna						
Styloid	—	0.477	0.550	0.477	0.550	0.661
Shaft	NS	—	0.709	0.418	0.709	0.600
Coronoid	0.019	0.002	—	0.491	0.709	0.818
Radius						
Head	0.042	NS	0.036	—	0.273	0.309
Shaft	0.019	0.002	0.002	NS	—	0.745
Styloid	0.005	0.010	0.0005	NS	0.001	—

Above the diagonal is the Kendall's Tau; below is the level of significance; NS, not significant.

TABLE 3

LEAD LEVELS (ppm) IN THE LIVER, FEATHERS, TIBIOTARSUS, RADIUS-ULNA, AND SKULL OF 2-DAY-OLD AND 15 TO 20-DAY-OLD HERRING GULL CHICKS

	N	2 Days Old	N	15-20 Days Old	χ^2 (p)
Liver	11	0.79 ± 0.92	13	0.27 ± 0.10	4.47 (0.0344)
Feathers	11	6.32 ± 5.74	13	0.81 ± 0.31	16.21 (0.0001)
Tibiotarsus	10	13.48 ± 7.20	13	19.85 ± 8.35	NS
Radius/ulna	9	38.90 ± 19.14	13	13.82 ± 7.65	11.22 (0.0008)
Skull	—	—	13	18.66 ± 10.70	—
F value (p)		NS		18.5 (0.0004)	

Given are mean ± SE, Wilcoxin χ^2 , F value; NS, not significant.

that were exposed to environmental lead through their food at two different ages (Table 3). In this experiment, the 2-day-old chicks had significantly higher levels of lead in the liver, feathers, and radius-ulna, compared to the 15- to 20-day-old chicks. One of the 2-day-old samples showed a level of 295 ppm lead; this unusually high level was excluded from analysis. There were no significant differences between the tissue samples of the 2-day-old chicks.

Among the 15- to 20-day-old chicks, however, the liver and feathers were significantly different from the bone tissues ($\chi^2 = 18.78$; $p < 0.001$), but there were no differences among the bones. The only differences occurred between the tibiotarsus and the radius-ulna ($\chi^2 = 4.28$, $p = 0.0485$).

There were no correlations between the bone and liver levels in 2-day-old chicks (Table 4). Among the older chicks, however, levels in skull, tibiotarsus, and radius-ulna were all positively correlated with each other, but not with liver levels or weight (Table 4).

Table 5 shows the lead levels (ppm) in the ribs of the 15- to 20-day-old herring gull chicks. These levels were much higher than those found in the other bones of the same chicks ($\chi^2 = 36.2$, $df = 12$, $p < 0.0001$). The ribs showed only 4 correlations of a possible 21, but these were for anterior with anterior and dorsal with dorsal (Table 6).

TABLE 4

CORRELATIONS OF LEAD LEVELS IN THE LIVER, FEATHERS, SKULL, TIBIOTARSUS, RADIUS-ULNA, AND WEIGHT IN 2-DAY-OLD AND 15 TO 20-DAY OLD HERRING GULL CHICKS (KENDALL TAU TEST)

	Liver	Feathers	Tibiotarsus	Radius/Ulna	Weight
2-day-old					
Liver	—	0.018	0.244	-0.156	-0.037
Feathers	NS	—	-0.111	0.111	0.147
Tibio	NS	NS	—	0.156	-0.467
Radius/ulna	NS	NS	NS	—	-0.333
Weight	NS	NS	NS	NS	—
15-20 Day Old					
Liver	—	0.013	0.039	-0.116	0.194
Feathers	NS	—	0.821	0.615	0.256
Skull	NS	0.0001	—	0.590	0.333
Tibio	NS	0.003	0.005	—	0.538
Radius/ulna	NS	0.003	0.005	0.010	—
Weight	NS	NS	NS	NS	—

NS, not significant.

TABLE 5

LEAD LEVELS (ppm DRY WEIGHT) IN THE FRONT AND BACK RIBS OF 15- TO 20-DAY-OLD HERRING GULL CHICKS

Rib	N	Front	Back	χ^2
1	13	60.9 ± 77.2	49.7 ± 44.2	NS
2	13	58.8 ± 127.8	54.7 ± 75.1	NS
3	13	57.7 ± 64.4	41.5 ± 43.4	NS

Given mean ± SE, Kruskal-Wallis χ^2 test; NS, not significant.

DISCUSSION

Because the principle organ of accumulation of lead in humans and other animals is bone (26), the measure of skeletal lead concentrations can provide an index of cumulative exposure. Therefore, it is important to understand where lead is deposited in the skeleton. Consistent differences in lead deposition should influence selection of sample sites for analysis (37). This is particularly true for people, where one possible objective is to quantify the skeletal stores using in vivo x-ray fluorescence (13). These levels would be an important variable in the understanding of the long-term human health effects of lead (34,36). It is also necessary to identify the importance of sample site selection when examining previous research. Randomly selecting bones for analysis is not adequate. Consistency in results is required for drawing conclusions. Therefore, knowledge of how site affects lead deposition will influence interpretation of past results.

The lack of significant differences within parts of the radius or ulna suggest that within a particular bone there may be no distinction where bone samples are taken. However, there were differences in lead levels in different bones. The ribs contained twice the levels of lead found in any other bone taken from the herring gull chicks. This is a significant result because ribs are not commonly chosen for lead analysis. The high levels found here may suggest that lead can preferentially deposit in portions of the axial skeleton, rather than the appendicular. The results indicate that lead deposition is homogeneous within a particular bone, but variations occur between different bones.

TABLE 6

CORRELATIONS OF LEVELS IN THE FRONT AND BACK RIBS AND WEIGHT IN 15- TO 20-DAY-OLD HERRING GULL CHICKS (KENDALL TAU TEST)

	Front Rib			Back Rib			Weight (g)
	1	2	3	1	2	3	
Front							
1	—	0.513	0.576	0.205	0.051	0.205	0.103
2	0.01	—	0.424	0.128	0.077	0.077	0.282
3	0.009	NS	—	0.424	0.272	0.091	0.061
Back							
1	NS	NS	NS	—	0.590	0.333	0.026
2	NS	NS	NS	0.005	—	0.487	0.385
3	NS	NS	NS	NS	0.02	—	0.333
Weight	NS	NS	NS	NS	NS	NS	—

Above the diagonal is the Kendall tau; below is the level of significance; NS, not significant.

The skeletal levels found in this study are comparable to those previously reported. Lead in the radii-ulnae of four species of waterfowl showed levels ranging from <0.5–345 ppm (40). Stendell et al. (39) found levels up to 127 ppm lead in bones of sora rails (*Porzana carolina*) that had ingested lead shot. Lead levels (40.38 ppm) in skeletal tissue of 13 swans found dead in the Mission Lake area of Idaho were attributed to ingestion of lead-contaminated vegetation (6).

DiGiulio and Scanlon (16) examined the ulnar bones for lead in 12 species of waterfowl from the Chesapeake Bay. The means for most of the species were lower than these, but ranged from <0.5–331.4 µg/g. White and Stendall (40) analyzed wingbones from waterfowl from 11 U.S. Fish and Wildlife Service refuges on Sauvie Island, an Oregon Wildlife Management Area. Mallards, *Anas platyrhynchos*, from three refuges (San Luis and Sacramento, CA, and Sauvie Island, OR) contained mean lead levels of 16.45, 18.58, and 27.85 ppm, respectively, similar to those found in the herring gulls, but birds from other areas had much lower levels. Pattee et al. (28) dosed bald eagles (*Haliaeetus leucocephalus*) with no. 4 lead shot. After death, the femur, humerus, and tibia were analyzed. The bone lead levels were higher in the dosed eagles than in the controls, although the levels in the dosed birds were slightly lower than those in the herring gulls. The controls were definitely lower than the gull controls in this study. No trends were observed among the different bones in the bald eagles.

Among the other tissues we analyzed, the liver exhibited the lowest level in both the 2- and 15- to 20-day-old chicks. This was anticipated since hepatic lead concentrations indicated recent exposure, which was not expected to be high. The levels found in the 2-day-old chicks are noteworthy since the

only source of lead for these chicks was the egg. Pattee (27) found eggs from clutches of American kestrels fed metallic lead all had levels below 0.5 µg/g. He concluded that little lead was transferred from the female to the eggshell or egg content. Nonetheless, transfer of heavy metals from females to their eggs has been demonstrated in a number of cases (10,19,20,22).

The levels of lead found in the 2-day-old chicks must have come from the egg since the time after hatching was not enough to allow for such levels to be deposited in the bone. If the chicks obtained lead from their food in the first 2 days alive, the lead levels in the liver should have been higher than they were.

There were also differences in levels between the chicks of different ages. In the liver, feathers, and radius-ulna, the levels reported in the younger chicks were significantly higher than in the older chicks. Just-hatched chicks may have an initial dose of lead that is very high, with maturation improving the ability to metabolize and excrete such metals.

The data in this article explore correlations between the different sites of lead deposition in bone, liver and feathers. Further studies will be necessary to reveal the definitive relationships of a larger variety of bone. Nonetheless, our results show significant differences in levels of lead in different bones, suggesting the importance of bone selection in assessing lead levels in humans and other animals.

ACKNOWLEDGEMENTS

The authors thank T. Shukla and D. Bertrand for help with analyses. This study was funded in part by a NIEHS grant (ESO 5022) to EOSHI of Rutgers University and UMDNJ-Robert Wood Johnson Medical School, and a grant from NJ DEP to S.R.

REFERENCES

- Anderson, W. L. Lead poisoning in water fowl at Rice Lake, Illinois. *J. Wildl. Manage.* 39:246–270; 1975.
- Angell, N. F.; Weiss, B. Operant behavior of rats exposed to lead before or after weaning. *Toxicol. Appl. Pharmacol.* 63:62–71; 1982.
- Avery, D. D.; Cross, H. A. The effects of tetraethyl lead on behavior in the rat. *Pharm. Biochem. Behav.* 2:473–479; 1974.
- Barry, P. S. I. A comparison of concentrations of lead in human tissues. *Bri. J. Indust. Med.* 32:119–139; 1975.
- Barthalmus, G. T.; Leander, J. D.; McMillan, D. E.; Mushak, P.; Krigman, M. R. Chronic effects of lead on schedule controlled pigeon behavior. *Toxicol. Appl. Pharm.* 42:271–284; 1977.
- Benson, W. W.; Brock, D. W.; Gabica, J.; Loomis, M. Swan mortality due to certain heavy metals in the Mission Lake area, Idaho. *Bull. Environ. Contam. Toxicol.* 15:171–174; 1977.
- Bornschein, R.; Pearson, D.; Reiter, L. Behavioral effects of moderate lead exposure in children and animal models: Part 1, clinical studies. *CRC Crit. Rev. Toxicol.* 7:43–99; 1980.
- Bornschein, R.; Pearson, D.; Reiter, L. Behavioral effects of moderate lead exposure in children and animal models: Part 2, animal studies. *CRC Crit. Rev. Toxicol.* 7:101–152; 1980.
- Burger, J.; Gochfeld, M. Effects of lead on growth in young herring gulls (*Larus argentatus*). *J. Toxicol. Environ. Health* 25:227–236; 1988.
- Burger, J.; Gochfeld, M. Cadmium and lead in common terns (*Aves: Sterna hirundo*): Relationship between levels in parents and eggs. *Environ. Monit. Assess.* 16:253–258; 1991.
- Bushnell, P. J.; Bowman, R. E. Effects of chronic lead ingestion on social development in infant rhesus monkeys. *Neurobehav. Toxicol.* 1:207–219; 1979.
- Bushnell, P. J.; Bowman, R. E. Persistence of impaired reversal learning in young monkeys exposed to low levels of dietary lead. *J. Toxicol. Environ. Health* 5:1015–1023; 1979.
- Chettle, D. R.; Scott, M. C.; Somerville, L. J. Lead in bone: Sampling and quantation using K x-rays excited by ¹⁰⁹Cd. *Environ. Health Perspect.* 91:49–56; 1991.
- Chupp, N. R.; Dalke, P. D. Waterfowl mortality in Coeur d'Alene River Valley, Idaho. *J. Wildl. Manage.* 28:692–702; 1964.
- Day, J. P.; Hart, M.; Robinson, M. S. Lead in urban street dust. *Nature* 253:343–345; 1975.
- DiGiulio, R. T.; Scanlon, P. F. Heavy metals in tissues of waterfowl from the Chesapeake Bay, USA. *Environ. Poll. (Series A)* 35:29–48; 1984.
- Drasch, G.; Bohm, J.; Baur, C. Lead in human bones. *Sci. Total Environ.* 64:303–315; 1987.
- EPA. Interim methods for sampling and analysis of priority pollutants in sediments and fish tissue. U.S. Environmental Protection Agency, EPA 600/4-81-055, Cincinnati, OH, 1981.
- Fimreite, N.; Brerik, E. M.; Trop, R. Mercury and organochlorines in eggs from a Norwegian gannet colony. *Bull. Environ. Contam. Toxicol.* 28:58–60; 1982.
- Fimreite, N.; Brum, E.; Frosli, A.; Fredericksen, P.; Gundersen, N. Mercury in eggs of Norwegian seabirds. *Astark* 1:71–75; 1974.
- Gochfeld, M.; Burger, J. Effects of lead on growth and feeding behavior of young common terns (*Sterna hirundo*). *Arch. Environ. Contam. Toxicol.* 17:513–517; 1988.
- Haseltine, S. D.; Mulhern, B. M.; Stafford, C. Organochlorine and heavy metal residues in Black duck eggs from the Atlantic flyway, 1978. *Pest Monitor. J.* 14:53–57; 1980.
- Landrigan, P. T.; Whitmorth, R. N.; Baloh, R.; Staehling, N. W.; Barthel, W. F.; Rosenblum, B. T. Neuropsychological dysfunctions in children with chronic low-level lead absorption. *Lancet* 1:708–712; 1975.
- Nation, J. R.; Bourgeois, A. E.; Clark, D. E. Behavioral effects of chronic lead exposure in the adult rat. *Pharm. Biochem. Behav.* 18:833–840; 1983.
- Needleman, H. L.; Gunnoe, C.; Leviton, A.; Reed, R.; Peresie,

- H.; Maher, C.; Barrett, P. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N. Engl. J. Med.* 300:689-695; 1979.
26. Nordberg, G. F.; Mahaffey, K. R.; Fowler, B. A. Introduction and summary, International Workshop on Lead in Bone: Implications for dosimetry and toxicology. *Environ. Health Perspect.* 91:3-8; 1991.
27. Pattee, O. H. Eggshell thickness and reproduction in American kestrels exposed to chronic dietary lead. *Arch. Environ. Contam. Toxicol.* 13:29-34; 1984.
28. Pattee, O. H.; Wiemeyer, S. N.; Mulhern, B. M.; Sileo, L.; Carpenter, J. W. Experimental lead-shot poisoning in bald eagles. *J. Wildl. Manage.* 45:806-810; 1981.
29. Pounds, J. G.; Long, G. J.; Rosen, J. F. Cellular and molecular toxicity of lead in bone. *Environ. Health Perspect.* 91:17-32; 1991.
30. Rosen, J. F.; Markowitz, M. E.; Bijur, P. E.; Jenks, S. T.; Wielopolski, L.; Kalef-Ezra, J. A.; Slatkin, D. N. Sequential measurements of bone lead content by L x-ray fluorescences in CaNa₂ EDTA-treated lead toxic children. *Environ. Health Perspect.* 91:57-62; 1991.
31. Scanlon, P. F.; Stotts, V. D.; Oderwald, R. G.; Dietrick, T. J.; Kendall, R. J. Lead concentrations in livers of Maryland waterfowl with and without ingested lead shot present in gizzards. *Bull. Environ. Contam. Toxicol.* 25:855-860; 1980.
32. Schroeder, H. A.; Tipton, I. H. The human body burden of lead. *Arch. Environ. Health* 17:965-978; 1968.
33. Schwartz, J.; Otto, D. Blood lead, hearing thresholds, and neuro-behavioral development in children and youth. *Arch. Environ. Health* 42:153-160; 1987.
34. Silbergeld, E. K. Lead in bone implications for toxicology during pregnancy and lactation. *Environ. Health Perspect.* 91:63-70; 1991.
35. Snowdon, C. T. Learning deficits in lead-injected rats. *Pharmacol. Biochem. Behav.* 1:599-603; 1973.
36. Somerville, L. J.; Chettle, D. R.; Scott, M. C. *In vivo* measurement of lead in bone using x-ray fluorescence. *Phys. Med. Biol.* 30:929-943; 1985.
37. Somerville, L. J.; Chettle, D. R.; Scott, M. C.; Tennant, D. R.; McKiernan, M. J.; Skilbeck, A.; Trethowan, W. N. *In vivo* tibia lead measurements as an index of cumulative exposure in occupationally exposed subjects. *Br. J. Indust. Med.* 45:174-181; 1988.
38. Stendell, R. C. Dietary exposure of kestrels to lead. *J. Wildl. Manage.* 44:527-530; 1980.
39. Stendell, R. C.; Artmann, J. W.; Martin, E. Lead residues in sora rails from Maryland. *J. Wildl. Manage.* 44:525-527; 1980.
40. White, D. H.; Stendell, R. C. Waterfowl exposure to lead and steel shot on selected hunting areas. *J. Wildl. Manage.* 41:469-475; 1977.
41. Wittmers, L. E.; Wallgren, J.; Alich, A.; Aufderheide, A. C.; Rapp, G. Lead in bone. IV. Distribution of lead in the human skeleton. *Arch. Environ. Health.* 43:381-391; 1988.