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HEAVY METALS IN WHITE-TAILED DEER LIVING NEAR A ZINC SMELTER IN PENNSYLVANIA

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ABSTRACT: White-tailed deer (*Odocoileus virginianus* (Zimmermann)) shot within 20 km of the zinc smelters in the Palmerton, Pennsylvania area contained extremely high renal concentrations of cadmium (372 ppm dry weight (dw)) and zinc (600 ppm dw). The deer with the highest renal zinc concentration was shot 4 km from the smelters and had joint lesions similar to those seen in zinc-poisoned horses from the same area. The highest concentrations of lead in both hard and soft tissues were relatively low, 10.9 ppm dw in a sample of teeth, 17.4 ppm dw in a metacarpus, and 4.9 ppm dw in a kidney.

INTRODUCTION

The soils and vegetation surrounding the zinc smelters at Palmerton, Pennsylvania, are grossly contaminated with cadmium, zinc, and lead (Buchauer, 1973). Many kinds of organisms have been contaminated by these metals. Baker et al. (1977) found high concentrations of cadmium and lead in children's hair. Concentrations of cadmium and zinc in tissue of cattle and horses from farms near the smelters were very high, in some instances approaching toxic thresholds, and zinc poisoning was a cause of debility and death of foals (Chaney, pers. comm.; Gunson et al., 1982). In earlier studies personnel of the Patuxent Wildlife Research Center found that passerine birds had high lead burdens and depressed blood delta-aminolevulinic acid dehydratase (ALAD) activity, and that shrews (*Blarina* sp.) were severely contaminated with metals and had histological evidence of subclinical lead poisoning (Beyer, 1983). In the present study we collected tissues from white-tailed deer in the Palmerton area and from deer killed 100 km or more from Palmerton to determine the extent to which the Palmerton deer were contaminated with metals.

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Many reports exist of chronic cadmium, zinc, and lead poisoning in domestic and laboratory animals. High intakes of cadmium can cause anemia, enteropathy, and kidney damage (Anon., 1980). Signs of zinc toxicosis include anemia, poor bone mineralization, and arthritis (Anon., 1980). Lead toxicosis is characterized by abnormalities in hematological, neural, renal, or skeletal systems (Anon., 1980). Very little is known of the effects of these metals on wild mammals or the movement of these metals through wildlife food chains.

Tissues were collected from deer in the present study to provide several kinds of information. Livers, kidneys, and brains were analyzed to provide information on the metal contaminants in deer. The metal concentrations in the feces provided information on recent exposure from several possible sources of contamination, including food, soil or dust, and salt licks. Teeth were analyzed because they have been used to provide information on past dietary exposure to cadmium or lead in swine, rats, and children (Cousins et al., 1973; Shearer et al., 1980; Needleman et al., 1979). Witkowski et al. (1982) recently studied lead concentrations in deer teeth as a means of detecting environmental contamination. They are very durable, readily collected, and are a potential means of monitoring metals in deer. Our data indicate that many of the Palmerton deer were contaminated by zinc and cadmium.

MATERIALS AND METHODS

Tissues were collected from hunter-killed deer during the "antlered hunt" in December 1980 and the "antlerless hunt" in January 1981. Hunters were met on access roads as they entered and departed from the hunting area, and successful hunters located the kill site on topographical maps. Most hunters were very cooperative and personally collected feces and kidneys at the kill site, then saved the head when the carcass was butchered. We included data only when we were confident that the information given by the hunters was accurate. Some deer killed illegally or by auto collision were provided by personnel of the Pennsylvania Game Commission and the Patuxent Wildlife Research Center.

The jaw, liver, or kidney were collected from all the deer. Feces (formed pellets from the rectum), metacarpi, and brains were collected whenever possible. The tissues were transported on ice to the laboratory and then frozen until preparation for analysis. The capsule was stripped from the kidney and the entire organ was analyzed for cadmium, zinc, lead, and copper. Only the caudate process was excised from most of the livers, because the hunters generally considered the livers a prized food. Subsamples were later removed with a stainless steel scalpel and analyzed for cadmium, zinc, lead, and copper. Skin, fascia, and ligaments were stripped from a metacarpus and the distal half was analyzed for lead and cadmium. The calvaria and the intact brains were removed unless the brain case had been destroyed by the gunshot. A coronal segment of the cerebrum was analyzed for copper. Age was determined by the toothwear and replacement method (Taber, 1969). The right mandible was boiled for about 1 hr in distilled water to facilitate removal of the teeth. All the cheek teeth, both deciduous and permanent molars and premolars, were analyzed as one sample because of the difficulty of selecting an individual tooth which would represent each age class. Preliminary cleaning of the teeth was done with forceps and brush, then they were sonicated to remove all extraneous debris and submitted for lead and cadmium determination.

The tissues were shipped to Analytical Biochemistry Laboratories, Columbia, Missouri 65201, USA for analysis. Moisture content was determined by drying the samples 4 hr in a vacuum oven at 100 C and the tissues were then digested in nitric acid. Cadmium, zinc, and copper concentrations were determined by inductively coupled organ plasma spectroscopy. Lead was analyzed by atomic absorption spectroscopy by flame or graphite furnace techniques, dependent on sample concentration. The lower limit of reportable residues was 0.1 ppm for all metals. The distal halves of the metacarpi were cut longitudinally and the marrow removed before the moisture content was determined; to reduce the quantity of bone processed, only one of the longitudinal quarters of each metacarpus was analyzed. The other quarter was ashed in a muffle furnace at 600 C to determine ash content. A quarter-metacarpus from one deer was segmented into five sections and analyzed to determine variation in lead concentration over the length.

The distance from the smelters to the site where each deer was killed was measured and the data were placed in one of three categories: (1) less than 8 km, (2) 10 to 20 km, and (3) greater than 100 km from the smelters. Data on males were combined with those of females, since there were too few of each sex to analyze separately. The metal residue data were transformed to natural logs and statistically analyzed by using TECTRONIC (Beaverton, Oregon 97077, USA) programs for simple regression, analysis of variance (ANOVA), or analysis of covariance. Means were compared with Duncan's Multiple Range Test. In one instance 0.05 ppm was substituted for a concentration reported as less than 0.1 ppm. Bartlett's test for homogeneous variance showed no serious violations of the ANOVA assumptions. Data from our study are presented on a dry weight (dw) basis.

RESULTS

Tissues were collected from five deer within 8 km of the smelters, 13 deer within 10–20 km, and five deer more than 100 km away. Average moisture contents after frozen storage were feces 72%, livers 71%, kidneys 80%, cerebrums 78%, metacarpi 18%, and teeth 16%.

Concentrations of cadmium in feces were variable, but were generally much higher in deer killed within 20 km of the smelters ($P = 0.02$; Table 1). Renal concentrations of cadmium were very high in individual deer (215 and 372 ppm), and the averages were higher in deer killed near the smelters ($P = 0.001$; Table 1). Renal cadmium was also correlated with age regardless of the kill site ($P = 0.04$; Fig. 1). The average concentration of cad-

TABLE 1. Concentrations of heavy metals (ppm, dry weight) in feces, liver, and kidney samples from white-tailed deer collected near a zinc smelter in Pennsylvania.

Metal	Distance from smelters (km)	Feces	Liver	Kidney
Cadmium	<8	12.2 (3) A[a]	11.6 (4) A	93.9 (4) A[c]
		2.1–71.0[b]	6.6–20.1	22.0–400
	10–20	13.4 (5) A	4.2 (13) B	55.9 (9) A
		3.4–52.8	3.1–5.8	11.6–270
	>100	0.9 (5) B	1.9 (5) C	15.5 (5) B
		0.2–3.5	0.6–1.5	3.1–78
Zinc	<8	577 (3)	256 (4) A	310 (4) A
		185–1,797	178–368	211–454
	10–20	574 (5)	167 (13) B	274 (9) A
		238–1,384	137–205	212–355
	>100	185 (5)	132 (5) B	145 (5) B
		77–445	95–182	103–205
Lead	<8	15.6 (3)	0.8 (4) A	1.8 (4) A
		6.5–37.3	0.4–1.8	1.1–3.1
	10–20	23.9 (5)	0.5 (13) A	1.4 (9) AB
		12.2–46.9	0.3–0.7	1.0–2.0
	>100	8.0 (5)	0.2 (5) B	0.8 (5) B
		4.1–15.7	0.1–0.4	0.5–1.3
Copper	<8	20.8 (3)	190 (4)	18.2 (4)
		13.4–32.4	80–448	15.3–21.6
	10–20	18.1 (5)	82 (13)	20.6 (9)
		12.9–25.5	51–131	18.4–23.1
	>100	21.3 (5)	106 (5)	18.2 (5)
		15.1–30.0	49–229	15.6–21.3

[a] Geometric mean (n); means with different capitalized letters within a given metal group are statistically different by Duncan's Multiple Range Test, which was performed only when the ANOVA was significant at $P \leq 0.05$.

[b] Lower and upper limits of the 95% confidence interval.

[c] Means of concentrations of cadmium in the kidney samples were compared by analysis of covariance with age as the covariate.

mium in the five samples of teeth collected within 8 km of the smelters was 0.21 ppm. Concentrations of cadmium greater than the detection limit were measured in five of 12 samples collected 10–20 km from the smelters; the average concentration of these five was 0.18 ppm. Only one of the tooth samples collected 100 km from the smelter contained a detectable concentration of cadmium (0.19 ppm). Concentrations of cadmium in the metacarpi were below the detection limits.

The average concentration of zinc in feces from deer collected near the smelters (Table 1) was not significantly different ($P = 0.13$) from those of deer collected farther from the smelters. Mean hepatic and renal concentrations of zinc were significantly higher in deer killed near the smelters ($P = 0.03$, $P = 0.01$). By fortuitous circumstance, the hind legs of a deer killed 4 km from the smelter were examined. There was a 1-cm hemorrhagic erosion in the articular cartilage of the centroquartal bone and a similar lesion on the opposing metatarsal (Figs. 2, 3). This deer also had the highest renal concentration of zinc (600 ppm).

Mean concentrations of lead in feces in deer killed near the smelters were not statistically different ($P = 0.07$; Table 1) from those from deer collected farther away from the smelters. Both hepatic and renal concentrations of lead were higher in deer

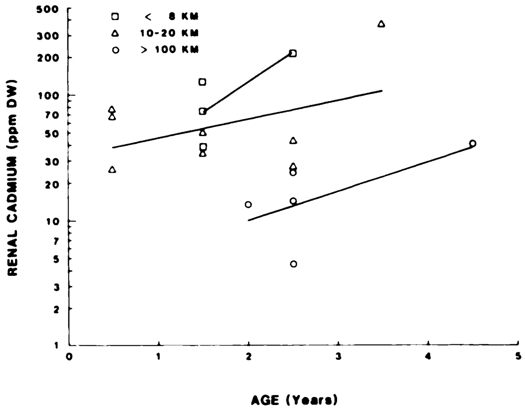

FIGURE 1. Relation between age and concentrations of cadmium in kidneys of white-tailed deer collected 8 km, 10–20 km, or more than 100 km from zinc smelters in Pennsylvania. Renal cadmium concentration was dependent on both age ($P = 0.04$) and distance from the smelters ($P = 0.001$). The slopes for each region were not significantly different ($P = 0.67$).

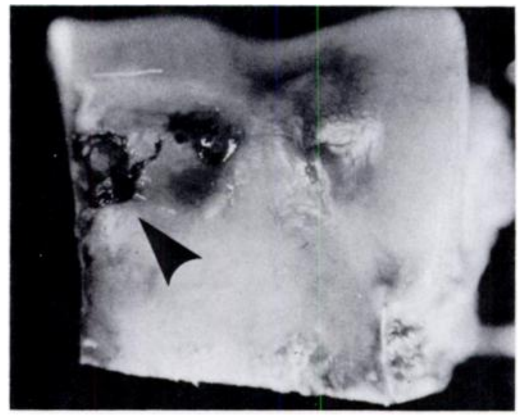

FIGURE 2. Centroquartal bone from the hind leg of a white-tailed deer shot 4 km from a zinc smelter in Pennsylvania. Note the depressed, hemorrhagic 1-cm erosion in the articular cartilage (arrow). The left side of the bone was cut away.

killed near the smelters ($P = 0.02$, $P = 0.05$), but the differences were not as marked as for cadmium and zinc, did not approach toxic concentrations, and were similar to published normal values (Table 3). Average concentrations of lead in teeth from deer collected near the smelters were not significantly different ($P = 0.08$) from those collected farther away (Table 2), and the concentrations were comparable to published normal values (Table 3). Average ash content and average lead concentration in the metacarpi did not vary with distance from the smelters (Table 2, $P = 0.50$, $P = 0.40$). In the single metacarpal sampled segmentally (from a 2½-yr-old female) the lead concentration decreased gradually from 3.1 ppm at the distal epiphysis to 1.7 ppm 20 cm toward the midshaft.

Concentrations of copper were not correlated ($P > 0.05$) with the distance from the smelters for any of the tissues (Tables 1, 2). All of the concentrations were comparable to published values for normal deer (Table 3).

DISCUSSION

The high concentrations of zinc and cadmium detected in feces of some of the deer from Palmerton showed that they were exposed to very high amounts of these metals. Whereas the concentration of cadmium in deer feces from Palmerton was about 13 ppm, the rumen cadmium content of roe deer (*Capreolus capreolus*) from a clean environment was 0.22 ppm, and that from a contaminated environment was 1.23 ppm (Partschefeld et al., 1977). Feces from one of the deer from Palmerton contained 3,600 ppm zinc; feces of horses experimentally poisoned with zinc (180 mg/kg body weight) contained 4,200 ppm zinc (Willoughby et al., 1972). Fecal analysis was a useful way to detect gross exposure, and showed that some deer living close to the smelters were ingesting potentially dangerous quantities of zinc and cadmium.

Cadmium accumulated with age in the kidneys of the Palmerton deer. This has been shown also in other deer herds (Munshower and Neuman, 1979; Kocan et al., 1980). In many species a concentration of 200 ppm wet weight in the kidney cortex is associated with tubular damage (Anon.,

FIGURE 3. Erosion (arrow) in the articular cartilage of the metatarsal of a white-tailed deer which opposed the centroquartal shown in Figure 2.

TABLE 2. Concentrations of heavy metals in metacarpal, teeth, and brain samples from white-tailed deer collected near a zinc smelter in Pennsylvania.

Distance from smelter (km)	Lead in metacarpal $\left(\frac{\text{ppm, dw}}{\text{\% ash}} \times 100\right)$	Lead in teeth (ppm, dw)	Copper in brain (ppm, dw)
<8	8.5 (4)[a]	5.9 (5)	8.2 (5)
	4.4–16.5[b]	3.2–11.0	6.2–11.0
10–20	8.7 (7)	3.8 (12)	7.8 (10)
	5.3–14.3	2.5–5.6	6.3–9.5
>100	5.8 (5)	2.2 (5)	9.6 (5)
	3.2–10.5	1.2–4.1	7.2–12.8

[a] Geometric mean (n).

[b] Lower and upper limits of the 95% confidence interval.

1980). Because the kidneys were relatively small, the entire kidney was analyzed. We do not know the whole-kidney concentration associated with renal dysfunction, but it seemed that some of the deer from Palmerton had renal concentrations of cadmium that approached those associated with early damage. We were surprised that cadmium was below the detection limits in many of the samples of bone and teeth. Cousins et al. (1973) and Shearer et al. (1980) found much higher concentrations in teeth of rats and swine fed cadmium at 30 ppm or more in the diet. The dietary exposure of the Palmerton deer may have been too low for cadmium to accumulate in hard tissue. It seems that for deer, as for other species, most of the body burden of cadmium is in soft tissues, particularly liver and kidney.

Shortly after the tissues were collected from the deer, zinc poisoning was diagnosed in foals from farms in the vicinity of several of the deer-kill sites (Gunson et al., 1982). This disease was characterized clinically by lameness, swollen joints, unthriftiness, and a renal cortical zinc concentration of 150 ppm ww (about 750 ppm dw). The salient lesion was generalized osteochondrosis. Osteoporosis and nephrocalcinosis also occurred in the foals and were attributed to cadmium toxicity. Based on the extremely high renal zinc concentration (600 ppm dw) and an articular lesion similar to that seen in the zinc-poisoned horses, we concluded that the deer shot 4 km from the smelters was suffering from zinc poisoning.

It seems that even though the soils were greatly contaminated with lead, relatively little of it became incorporated into the deer's diet. Concentrations of lead in the feces from deer were lower than those in feces from cattle on farms 2 km (41 ppm dw) and 10 km (29 ppm dw) from the smelters (Chaney, pers. comm.). Cattle graze, and may ingest contaminated soil, to which browsing deer are not exposed. Also the high dietary exposure to zinc may have prevented accumulation of lead in bone. After feeding lead and zinc simultaneously to horses, Willoughby et al. (1972) noted that zinc enhanced retention of lead in soft tissues but retarded deposition in bone.

Gunson et al. (1982) discussed the possibility of zinc-induced derangements of

TABLE 3. Reported concentrations of cadmium, zinc, lead, and copper in tissues of normal Cervidae.

Tissue	Species	Location	*n*	ppm	Base[a]	Author
			Cadmium			
Liver	Whitetail	Ohio, USA	32	0.27	ww	Lynch (1973)
	Whitetail	Illinois, USA	190	0.37	dw	Woolf et al. (1982)
	Mule deer[b]	Colorado, USA	10	0.7	dw	Kienholz (1977)
	Mule deer	Montana, USA	30	0.51	fw	Munshower and Neuman (1979)
	Reindeer[b]	Finland	50	0.19	ww	Salmi and Hirn (1981)
Kidney	Whitetail	Ohio, USA	57	0.7	ww	Lynch (1973)
	Whitetail	Oklahoma, USA	64	0.7	ww	Kocan et al. (1980)
	Mule deer	Montana, USA	24	2.7	fw	Munshower and Neuman (1979)
	Reindeer	Finland	50	0.88	ww	Salmi and Hirn (1981)
Bone	Whitetail	Ohio, USA	41	0.56	ww	Lynch (1973)
Teeth	Roe deer	Czechoslovakia	9	5.2	dw	Mankovska (1980)
			Zinc			
Liver	Whitetail	Illinois, USA	190	70	dw	Woolf et al. (1982)
	Mule deer	Montana, USA	30	113	fw	Munshower and Neuman (1979)
	Red deer	DDR	40	111	dw	Anke et al. (1980b)
Kidney	Mule deer	Montana, USA	24	97	fw	Munshower and Neuman (1979)
	Red deer	DDR	40	131	dw	Anke et al. (1980b)
			Lead			
Liver	Whitetail	Illinois, USA	190	4.4	dw	Woolf et al. (1982)
	Whitetail	Ohio, USA	11	0.74	ww	Lynch (1973)
	Mule deer	Montana, USA	27	0.9	fw	Munshower and Neuman (1979)
Kidney	Whitetail	Oklahoma, USA	64	0.57	ww	Kocan et al. (1980)
	Whitetail	Ohio, USA	33	1.06	ww	Lynch (1973)
	Mule deer	Montana, USA	21	0.7	fw	Munshower and Neuman (1979)
Bone	Whitetail	Ohio, USA	38	4.8	ww	Lynch (1973)
	Whitetail	Pennsylvania, USA	48	36.2	aw	Witkowski et al. (1982)
Teeth	Roe deer	Czechoslovakia	9	5.5	dw	Mankovska (1980)
	Whitetail	Pennsylvania, USA	48	36.4	aw	Witkowski et al. (1982)
			Copper			
Liver	Whitetail	Illinois, USA	190	109	dw	Woolf et al. (1982)
	Mule deer	Colorado, USA	10	120	dw	Kienholz (1977)
	Mule deer	Colorado, USA	18	73	dw	Stelter (1980)
	Mule deer	Montana, USA	29	46	fd	Munshower and Neuman (1979)
Kidney	Mule deer	Montana, USA	24	30	fd	Munshower and Neuman (1979)
	Mule deer	Colorado, USA	18	28	dw	Stelter (1980)
Brain	Roe deer	DDR	70	5.6	dw	Anke et al. (1980a)
	Red deer	DDR	39	7.7	dw	Anke et al. (1980a)
	Fallow deer	DDR	15	9.7	dw	Anke et al. (1980a)

[a] ww = wet weight; dw = dry weight; fw = freeze dried weight; aw = ash weight.
[b] Mule deer (*Odocoileus hemionus*); reindeer (*Rangifer tarandus*).

copper metabolism in the pathogenesis of osteochondrosis in zinc-poisoned horses. Lysyl oxidase, a copper-dependent enzyme involved in collagen cross-linking may be the connection between excessive dietary zinc and articular lesions. Partschefeld et al. (1977) attributed low copper concentrations in livers of roe deer and in livers and cerebrums of mouflon (*Ovis musimon*) to environmental cadmium. We considered these possibilities but found no indication of copper deficiency. Copper concentrations were independent of the distance from the smelters.

There was no appreciable cadmium or lead in the metacarpi. Therefore, contamination of meat by bone dust during butchering for human consumption is unlikely. However, deer livers are eaten by many hunters, and those livers having the highest concentrations of cadmium should probably be considered unfit for human consumption.

There is a good opportunity at Palmerton to study the deleterious effects of metal pollutants on herd health. A thorough examination of the deer would include serological, radiological, and histopathological work, as well as chemical analyses. Urinary metallothionein or B_2 microglobulin might be measured to detect damage in deer kidneys with more than 300 ppm cadmium. Blood tests might show lead-induced ALAD depression or increased free erythrocytic protoporphyrin. Serum ceruloplasmin determination might detect induced alterations in copper metabolism. Determinations of the age structure of the herd might also be informative. From Figure 1 it can be estimated that the kidneys from deer living near the smelters would have over 400 ppm cadmium by the time a deer was 3 yr old. It may be that there are no old deer in the herd since they succumb to cadmium poisoning. Zinc, on the other hand, is more toxic to young animals (Willoughby et al., 1972) which might be reflected in the younger cohorts of a life table.

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LITERATURE CITED

ANONYMOUS. 1980. Mineral Tolerance of Domestic Animals. National Academy of Sciences, Washington, D.C., 577 pp.

ANKE, M., B. GROPPEL, S. PRIEN, L. BRIEDERMANN, AND S. MEHLITZ. 1980a. The supply of wild ruminants with major elements and trace elements. 4. The copper content of winter grazing and the copper status of red deer, fallow deer, roes, and mouflons. Arch. Tierernaehr. 30: 707–721.

———, E. RIEDEL, E. BRUCKNER, AND G. DITTRICH. 1980b. The supply of wild ruminants with major elements and trace elements. 3. The zinc content of winter grazing and the zinc status of red deer, fallow deer, roes and mouflons. Arch. Tierernaehr. 30: 479–490.

BAKER, E. L., JR., C. G. HAYES, P. J. LANDRIGEN, J. L. HANDKE, R. T. LEGER, W. J. HOUSWORTH, AND J. M. HARRINGTON. 1977. A nationwide survey of heavy metal absorption in children living near primary copper, lead, and zinc smelters. Am. J. Epidemiol. 106: 261–273.

BEYER, N. 1983. The smoke that settled over Palmerton. N.J. Aud. Mag. 9: 14–16.

BUCHAUER, M. J. 1973. Contamination of soil and vegetation near a zinc smelter by zinc, cadmium, copper and lead. Environ. Sci. Technol. 7: 131–135.

COUSINS, R. J., A. K. BARBER, AND J. R. TROUT. 1973. Cadmium toxicity in growing swine. J. Nutr. 103: 964–972.

GUNSON, D. E., D. F. KOWALCZCK, C. R. SHOOP, AND C. F. RAMBERG, JR. 1982. Environmental zinc and cadmium pollution associated with generalized osteochondrosis, osteoporosis, and nephrocalcinosis in horses. J. Am. Vet. Med. Assoc. 180: 295–299.

KIENHOLZ, E. W. 1977. Effects of environmental molybdenum levels upon wildlife. *In* Molybdenum in the Environment, Vol. 2, The Geochemistry, Cycling and Industrial Uses of Molybdenum, W. R. Chappel and K. K. Peterson (eds.). Marcel Dekker, Inc., New York, New York, 812 pp.

KOCAN, A. A., M. G. SHAW, W. C. EDWARDS, AND J. H. EVE. 1980. Heavy metal concentrations in the kidneys of white-tailed deer in Oklahoma. J. Wildl. Dis. 16: 593–596.

LYNCH, D. W. 1973. Selected toxic metals in Ohio's upland wildlife. M.S. Thesis. Ohio State Univ., Columbus, Ohio, 93 pp.

MANKOVSKA, B. 1980. The concentration of four toxic elements in the teeth of roe deer from the area of an aluminum plant. Biologia (Bratisl.) 35: 819–822.

MUNSHOWER, F. F., AND D. R. NEUMAN. 1979. Metals in soft tissues of mule deer and antelope. Bull. Environ. Contam. Toxicol. 22: 827–832.

NEEDLEMAN, H. L., C. GUNNOE, A. LEVITRON, R. REED, H. PERESIE, C. MAHER, AND P. BARRETT. 1979. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N. Engl. J. Med. 300: 689–695.

PARTSCHEFELD, M., B. GROPPEL, M. ANKE, AND M. GRUN. 1977. The Cd-exposure of game grazing and of roes, red deer, fallow-deer and moufflons in the GDR. *In* M. Anke and H. J. Schneider: Kadmiumstoffwechsel. Wiss. Beitrage der Friedrich-Schiller-Univ. Jena 2: 258–262.

SALMI, A., AND J. HIRN. 1981. Der Cadmiumgehalt des Muskels, der Leber und der Nieren finnischer Pferde und Rentiere. Fleischwirtschaft 61: 1199–1201.

SHEARER, T. R., J. L. BRITTON, AND D. J. DESART. 1980. Influence of post-developmental cadmium on caries and cariostosis by fluoride. Environ. Health Perspect. 34: 219–221.

STELTER, L. H. 1980. Baseline levels of selected trace elements in Colorado oil shale region animals. J. Wildl. Dis. 16: 175–179.

TABER, R. D. 1969. Criteria of sex and age. *In* Wildlife Management Techniques, 3rd Ed., R. H. Giles, Jr. (ed.). The Wildlife Soc., Washington, D.C., pp. 388–389.

WILLOUGHBY, R. A., E. MACDONALD, B. J. MCSHERRY, AND G. BROWN. 1972. The interaction of toxic amounts of lead and zinc fed to young growing horses. Can. J. Comp. Med. 36: 348–359.

WITKOWSKI, S. A., S. R. AULT, AND R. W. FIELD. 1982. Lead concentrations in white-tailed deer mandibles and teeth. Bull. Environ. Contam. Toxicol. 28: 561–565.

WOOLF, A., J. R. SMITH, AND L. SMALL. 1982. Metals in livers of white-tailed deer in Illinois. Bull. Environ. Contam. Toxicol. 28: 189–194.