

LEAD POISONING IN CAPTIVE WILD ANIMALS

Authors: ZOOK, B. C., SAUER, R. M., and GARNER, F. M.

Source: Journal of Wildlife Diseases, 8(3) : 264-272

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-8.3.264>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

LEAD POISONING IN CAPTIVE WILD ANIMALS

B. C. ZOOK,^[1] R. M. SAUER,^[2] and F. M. GARNER^[3]

Abstract: Lead poisoning was diagnosed post-mortem in 34 simian primates, 11 parrots, and 3 Australian fruit bats at the National Zoological Park. Diagnoses were made by the finding of acid-fast intranuclear inclusion bodies in renal epithelia or hepatocytes and, in most cases, by finding excess lead in samples of liver. The estimated prevalence of lead intoxication among autopsied primates and parrots was 44% and 50% respectively. Leaded paint was found in many animal enclosures at this zoo and it was available to all the lead-poisoned animals in this study.

The finding of renal intranuclear inclusion bodies in animals at several zoos, scattered reports of lead intoxication of animals dwelling in various zoos, the occurrence of leaded paint in many zoos and the high incidence of lead poisoning at this zoo, indicated that lead poisoning of zoo animals is much more common than was previously thought.

INTRODUCTION

Poisoning due to lead is all too common among domesticated creatures dwelling in our industrialized society. Children, young dogs and cattle are frequent victims, usually due to the ingestion of leaded paint. Wild animals, however, are seldom poisoned by lead — if the dearth of reports in the scientific literature is accepted at face value. An obvious exception is the vast numbers of waterfowl and some other game birds which have died due to the ingestion of spent lead shot from hunters' guns. The only other free-living wild animals of which we have found reports of lead poisoning are raccoons, a fox, and rats.

Hindle⁴ and Hindle and Stevenson¹⁰ reported that 90 to 95% of wild rats dwelling in London sewers or refuse dumps (but not country rats) had intranuclear inclusion bodies in renal tubular cells. These findings were substantiated

in Rochester, New York by Syverton, et al.¹⁷ More recently, Kilham, et al.¹¹ reported that nearly 100% of adult rats captured near a Hanover, New Hampshire, dump (but not laboratory rats) had similar inclusion bodies. The inclusions were found to be identical to those caused by lead poisoning by both staining characteristics and electron microscopy. Further, these inclusions were associated with renal tumors in some rats (lead is known to induce renal carcinomas in rats) and finally, the livers of those tested contained excessive lead.

Sanderson and Thomas¹¹ reported that 23 of 101 wild raccoons captured in Illinois were found to contain lead in amounts of 10 ppm^[4] or more in their livers. Erne, et al.⁸ reported that a red fox in Sweden was found to have 15 ppm lead in its kidneys.

Reports of lead poisoning in zoo animals are also uncommon. In 1954,

[1] Department of Pathology, George Washington University, School of Medicine, Washington, D.C. 20037, U.S.A.

[2] Division of Pathology, National Zoological Park, Smithsonian Institution, Washington, D.C. 20009, U.S.A.

[3] National Zoological Park, Smithsonian Institution, Washington, D.C. 20009, U.S.A.

[4] All values for lead in tissue are reported in parts per million (ppm) wet weight.

Address reprint requests to Dr. Zook, Division of Pathology, National Zoological Park, Smithsonian Institution, Washington, D.C. 20009, U.S.A.

This paper was presented at the 1971 Annual Wildlife Disease Conference, Ft. Collins, Colorado.

Fisher⁷ reported a case of non-fatal lead poisoning in a gorilla at the Lincoln Park Zoo due to the ingestion of leaded paint. In 1956, de Bisschop⁸ described a similar case in the Antwerp Zoo. In the same year, McIntosh¹³ reported that a polar bear, a kra monkey and an unspecified number of lorikeets and ferrets died of lead poisoning in a New Zealand Zoo. Cordy¹ reported two zoo-dwelling baboons (*Papio* sp.) had evidence of lead poisoning and osteodystrophia fibrosa concurrently in 1957. Hausman, et al.⁹ described a probable case of lead arsenate poisoning in a San Antonio Zoo orangutan (*Pongo pygmaeus*) in 1961 and also reported a confirmed case in a mandrill (*Mandrillus sphinx*) from a Chicago zoological park. In an annual report published in 1970 from the Chester Zoological Gardens in England, two parrots were listed as dying of lead poisoning from ingested lead oxide (a common paint pigment) on enclosures¹². Seventy-four captive waterfowl in the Slimbridge collection died after ingesting lead shot-gun pellets¹.

R. Wilson, director of the Detroit Zoological Park, confirmed lead poisoning post-mortem in two rheas, two sea lions, one gray seal, and one kori bustard during the past 13 months (personal communication). Samples of kidney and liver combined contained 17.3, 549, 56, 12.2, 23 and 100 ppm lead respectively. Although some paint containing 5% lead was discovered on a fence available to the rheas, no leaded paint was found accessible to the other animals. Throw-away portions of self-developing camera film were frequently seen within animal enclosures. Portions of these "throw-aways" were found to contain over 16% lead and were determined to be a significant source of lead.

E. P. Dolensek (personal communication) of the Bronx Zoo diagnosed lead poisoning in two island gray foxes which had tremors, convulsions, and posterior paralysis with high levels of lead in

blood. The foxes recovered, but remain partially blind although no lesions are discernable in the optic fundi. Two black panthers in the Staten Island Zoo (E. P. Dolensek, personal communication) were diagnosed as having lead poisoning, by the presence of typical signs including Burtonian lines, high blood or fecal lead values and positive response to chelation therapy. The panthers were believed to have ingested paint containing approximately 10% lead. I. M. Budinger (personal communication) diagnosed lead poisoning in five wanderoo monkeys (*Macaca silenus*) which died in the Bronx Zoo in 1963. These primates had typical signs, high levels of tissue lead and were exposed to heavily leaded paint in their cages.

We were unable to find other documented reports of lead poisoning in zoo animals (other than recent publications emanating from this zoo^{15,16,10}). There is, however, evidence (discussed later) that lead intoxication may be much more frequent among captive wild animals than is commonly believed. The purpose of this report is to describe the high frequency of lead poisoning in several species at a large zoological park and to suggest that lead poisoning may be quite common among certain species of wild animals exposed to lead paint.

MATERIALS AND METHODS

Poisoning due to lead was first recognized in a nonhuman primate at this zoological park in October 1969. This finding precipitated a retrospective study of primate autopsy material (from 1955 to August 1968) in a search for additional cases. Sections of kidney and sometimes liver were examined histologically from all simian primates in which brain lesions or in which renal or hepatic inclusion bodies were previously recorded. Formalin-fixed samples of liver (when available) were analyzed⁶ for lead content in monkeys found

⁸ Unpublished communication to the International Union Zoological Society Directors, Chicago, Illinois, 1956.

⁶ Specimens of liver were analyzed by a dithizone technique by Mr. Boylen, Massachusetts Institute of Technology, Boston, Mass.

to have acid-fast intranuclear inclusion bodies in their kidneys or liver.

In an effort to evaluate the incidence of lead poisoning at this zoo, sections of kidneys and liver available from all primates autopsied from September 1968 to December 1970 were examined for the presence of typical inclusion bodies. When such inclusions were found, liver (when available) was analyzed for lead content. Similarly, available tissues were studied from all parrots autopsied from January 1969 to May 1971.

Diagnoses of lead poisoning were based on the findings of multiple acid-fast intranuclear inclusion bodies in renal proximal tubular epithelial cells or hepatocytes. Such inclusion bodies are generally considered nearly pathognomonic for lead poisoning. In most of the animals, formalin-fixed specimens of liver were analyzed for lead content and found to contain 3 or more ppm lead. Samples of formalin-fixed liver from 10 apparently non-lead-poisoned simian primates from this zoo contained 0.3, 0.4, 1.1, 1.2, 1.3, 1.6, 1.6, 1.8, 2.5 and 2.5 ppm lead. These monkeys died of trauma, inanition or an infectious disease and did not have intranuclear inclusion bodies typical of lead poisoning. In addition to these findings, paint was analyzed⁽⁷⁾ from every cage where lead-poisoned animals were confined, and found to contain hazardous (over 3%) amounts of lead. Additionally, post-mortem lesions, especially those in the brain, were generally consistent with lead poisoning. Complete necropsy findings will be published later.

RESULTS

Occurrence by species, age, sex and season and length of time in this zoo

Lead poisoning has been diagnosed in 34 simian primates, 11 parrots and three Australian fruit bats to date (Table 1). A pet South American crab-eating rac-

coon and a pet squirrel monkey died of lead poisoning a short time after they were given to this zoo. They are not, however, included in this report because neither animal had obvious access to lead while in the zoo, and, in the case of the raccoon, paint from its previous dwelling was found to contain 5% lead.

The estimated incidence of lead poisoning in this zoo is given for primates (Table 2) and parrots (Table 3). All but four primates were Old World monkeys. Fourteen affected primates were juveniles, and varied from 6 to 30 months of age; the remaining 21 were adults, the oldest being 24 years of age. All but one of the parrots were adults; the immature parrot was about 2 months of age. The poisoned Australian fruit bats were young adults. There was no obvious sex predilection in any species studied. A seasonal occurrence was evident among the primates: 74% (25 of 34) died between June and November. The three fruit bats also died in these months, but no seasonal variance was found among the parrots.

The poisoned animals had resided in this zoo for various lengths of time prior to death. About half of the animals dwelt here less than 1 year, the shortest period of time being 2 months and the longest, 18 years.

Clinical signs

Clinical signs were often of sudden onset, generally non-specific in character, or no abnormalities were noticed prior to sudden and unexplained death. The duration of clinical illness varied from 0 days (no signs noticed before death) to 4 years. The majority of animals were reported to be ill for less than 1 week. The nutritional state of the bodies at autopsy varied, but half of them were thin or emaciated, suggesting that the duration of illness was longer in some cases than was observed clinically.

⁽⁷⁾ Paint was tested by a spot test⁽⁶⁾ and in most cases also by atomic absorption spectroscopy by the Health Services Administration, Division of Chemistry, Bureau of Laboratories, Washington, D.C.

TABLE 1. Captive Wild Animals Found to Have Lead Poisoning at the National Zoological Park.

| Common Name | Scientific Name | Date of Autopsy (mo/yr) | Acid-fast Intranuclear Inclusion Bodies Kidney | Lead Content of Liver (ppm) | Lead Content of Paint on Cage (%) | Age | Sex | Length of Time in Zoo |
|---------------------------|---------------------------------|-------------------------|--|-----------------------------|-----------------------------------|----------|-----|-----------------------|
| Golden baboon | <i>Papio cynocephalus</i> | 12/55 | + | 106 | — | 4 yrs | ♀ | 2 yrs |
| Philippine hybrid macaque | <i>Macaca sp.</i> | 1/60 | + | — | 3.0 | immature | — | — |
| Javan macaque | <i>Macaca fascicularis</i> | 6/60 | + | 3.8 | 10.0 | 7 mos | — | 7 mos |
| Drill | <i>Mandrillus leucophaeus</i> | 7/60 | — | 69 | 21.6 | 2 yrs | ♂ | 8 mos |
| DeBrazza's guenon | <i>Cercopithecus neglectus</i> | 11/60 | + | 45 | 3.0 | 6 mos | — | 6 mos |
| Barbary ape | <i>Macaca sylvana</i> | 4/61 | + | 6.7 | 13.6 | 9 mos | ♀ | 9 mos |
| Lion tail macaque | <i>Macaca silenus</i> | 3/64 | + | 0*** | 6.9 | 18 mos | ♂ | 3.5 mos |
| Lion tail macaque | <i>Macaca silenus</i> | 4/64 | + | 5.6 | 2.6 | 18 mos | ♂ | 3.5 mos |
| Lion tail macaque | <i>Macaca silenus</i> | 10/64 | + | 3.6 | 2.6 | 25 mos | ♂ | 10 mos |
| Rhesus monkey | <i>Macaca mulatta</i> | 9/65 | + | 5.3 | 13.6 | 2 yrs | — | — |
| Barbary ape | <i>Macaca sylvana</i> | 7/67 | + | 59 | 13.6 | 2 yrs | ♂ | 2 yrs |
| Red-faced macaque | <i>Macaca speciosa</i> | 6/68 | + | 65 | 0.5/18.2 | adult | ♂ | 11.5 mos |
| Lesser spot-nosed guenon | <i>Cercopithecus petaurista</i> | 10/68 | + | 10 | 3.0 | adult | ♂ | 2 yrs |
| Woolly monkey | <i>Lagothrix lagotricha</i> | 11/68 | + | 6.8 | 3.0 | 6½ yrs | ♂ | 5½ yrs |
| White-handed gibbon | <i>Hylobates lar</i> | 11/69 | + | 6.7 | 6.5 | 4 yrs | ♀ | — |
| Drill | <i>Mandrillus leucophaeus</i> | 12/68 | + | 3.2 | 21.6 | 10 yrs | ♂ | 5 yrs |
| Red-faced macaque | <i>Macaca speciosa</i> | 12/68 | + | 360**** | 2.6 | 1 yr | ♂ | 2 mos |

TABLE 1. (continued)

| Common Name | Scientific Name | Date of Autopsy (mo/yr) | Inclusion Bodies Kidney | Inclusion Bodies Liver | Lead Content of Liver (ppm) | Lead Content of Paint on Cage (%) | Age | Sex | Length of Time in Zoo |
|---------------------|----------------------------------|-------------------------|-------------------------|------------------------|-----------------------------|-----------------------------------|-----------|-----|-----------------------|
| Siamang gibbon | <i>Symphalangus syndactylus</i> | 2/69 | + | 0 | 3.8 | 2.6/11.8 | adult | ♀ | 1 yr |
| Barbary ape | <i>Macaca sylvana</i> | 2/69 | + | + | 110 | 13.6 | 22.5 mos | ♀ | 22.5 mos |
| Barbary ape | <i>Macaca sylvana</i> | 6/69 | + | 0 | 3.0 | 13.6 | 6-7 yrs | ♂ | 6-7 yrs |
| Vervet guenon | <i>Cercopithecus aethiops</i> | 6/69 | + | 0 | 20 | 2.6 | adult | ♂ | 8 yrs |
| White-faced saki | <i>Pithecia pithecia</i> | 6/69 | + | + | 7.1 | 2.0 | adult | ♂ | — |
| Barbary ape | <i>Macaca sylvana</i> | 7/69 | + | 0 | — | 13.6 | adult | — | — |
| Ring-tailed lemur | <i>Lemur catta</i> | 8/69 | + | 0 | 3.2 | 2.6/11.8 | adult | ♀ | 5 yrs |
| Ring-tailed lemur | <i>Lemur catta</i> | 8/69 | + | + | 8.9 | 2.6/11.8 | adult | ♀ | 2 yrs |
| Spectacled langur | <i>Presbytis obscurus</i> | 8/69 | + | 0 | — | 2.6/18.2 | 1½ yrs | ♀ | — |
| Ring-tailed lemur | <i>Lemur catta</i> | 8/69 | + | 0 | 5.1 | 2.6/11.8 | adult | ♂ | 3.5 yrs |
| Ring-tailed lemur | <i>Lemur catta</i> | 8/69 | + | 0 | — | 2.6/11.8 | adult | ♂ | 8½ yrs |
| Squirrel monkey | <i>Saimuri sciureus</i> | 6/69 | + | + | 3.1 | — | 2.5 yrs | ♂ | 1 yr |
| Hybrid gibbon | <i>Hylobates sp.</i> | 11/69 | + | 0 | 6.7 | 6.5 | 4 yrs | ♀ | 4 yrs |
| Gelada baboon | <i>Theropithecus gelada</i> | 7/70 | + | 0 | 15 | 21.6 | 8 yrs | ♀ | 8 yrs |
| White-handed gibbon | <i>Hylobates lar</i> | 8/70 | + | 0 | 18.1 | 6.5 | 24-25 yrs | ♂ | 18 yrs |
| Night monkey | <i>Aotus trivirgatus</i> | 9/70 | + | + | 4.8 | 2.6 | adult | ♀ | 15 mos |
| Sooty mangabey | <i>Cercocebus torquatus atys</i> | 9/70 | + | — | — | — | 6 mos | ♂ | 6 mos |

TABLE 1. (continued)

| Common Name | Scientific Name | Date of Autopsy (mo/yr) | Acid-fast Intranuclear Inclusion Bodies Kidney | Acid-fast Intranuclear Inclusion Bodies Liver | Lead Content of Liver (ppm) | Lead Content of Paint on Cage (%) | Age | Sex | Length of Time in Zoo |
|---------------------------|---------------------------------|-------------------------|--|---|-----------------------------|-----------------------------------|-------|-----|-----------------------|
| Rainbow lorikeet | <i>Trichoglossus haematodus</i> | 1/69 | + | 0 | — | 12.0/5 | adult | ♂ | 1 yr+ |
| Philippine hanging parrot | <i>Loriculus philippensis</i> | 3/69 | + | 0 | — | 12.0/0.6 | adult | ♂ | 2 yrs |
| Kea parrot | <i>Nestor notabilis</i> | 8/69 | + | 0 | 15 | — | adult | — | 13 mos |
| White-fronted amazon | <i>Amazona leucocephala</i> | 8/69 | + | 0 | 3.1 | 12.0/5 | 2 mos | — | 2 mos |
| Turquoise parakeet | <i>Neophema pulchella</i> | 10/69 | + | + | — | 12.0/— | — | — | 4 mos |
| Lessor vasa parrot | <i>Coracopsis nigra</i> | 12/69 | + | + | 5.9 | 12.0/5 | adult | ♂ | 2.5 yrs |
| Yellow-backed lory | <i>Domicella garrula</i> | 12/69 | + | 0 | 10 | 12.0/5 | adult | ♂ | 1 yr |
| Sulphur-crested cockatoo | <i>Kakatoe galerita</i> | 1/70 | + | 0 | 3.6 | 12.0/5 | adult | ♂ | 1 yr |
| Kea parrot | <i>Nestor notabilis</i> | 8/70 | + | + | 6.1 | 12.0/— | — | ♀ | — |
| Sulphur-crested cockatoo | <i>Kakatoe galerita</i> | 1/71 | + | 0 | — | — | adult | — | — |
| Kea parrot | <i>Nestor notabilis</i> | 5/71 | + | 0 | — | — | — | ♀ | — |
| Australian fruit bat | <i>Pteropus poliocephalus</i> | 8/69 | + | + | 500 | 4.5 | adult | ♀ | 15 mos |
| Australian fruit bat | <i>Pteropus poliocephalus</i> | 9/69 | + | + | 500 | 4.5 | adult | ♀ | 16 mos |
| Australian fruit bat | <i>Pteropus poliocephalus</i> | 10/69 | + | 0 | — | 4.5 | adult | ♂ | 17 mos |

* Positive.

** Undetermined.

*** Negative.

**** Analysis for lead done on formalin-fixed, paraffin-embedded liver specimen.

TABLE 2. Estimated Incidence of Lead Poisoning in Simian Primates Autopsied at the National Zoological Park from August 1968 to December 1970.

| Year | Primate Deaths (No.) | Cases Studied (No.) | Cases Studied Having Typical Inclusion Bodies (No.) | (%) |
|--------------|----------------------|---------------------|---|-----|
| 1968 (5 mo.) | 21 | 14 | 4 | 29 |
| 1969 | 33 | 25 | 13 | 52 |
| 1970 | 13 | 10 | 5 | 50 |
| Totals | 67 | 49 | 22 | — |
| Mean | — | — | — | 44 |

TABLE 3. Estimated Incidence of Lead Poisoning in Parrots Autopsied at the National Zoological Park from January 1969 to May 1971.

| Year | Parrot Deaths (No.) | Cases Studied (No.) | Cases Studied Having Typical Inclusion Bodies (No.) | (%) |
|--------------|---------------------|---------------------|---|-----|
| 1969 | 19 | 15 | 6 | 40 |
| 1970 | 7 | 3 | 2 | 67 |
| 1971 (5 mo.) | 3 | 2 | 2 | 100 |
| Totals | 29 | 20 | 10 | — |
| Mean | — | — | — | 50 |

Primates usually had non-specific signs of ill health such as partial anorexia and lethargy. Vomiting and diarrhea were seen in several, but six primates had no observed signs. Neurological disorders were manifest in 14 of 29 primates for which histories were available. Convulsions were the most common neurologic sign. Paralysis or paresis of limbs was common, but usually transient. Apparent blindness, tremors and ataxia were also seen. In most instances neurologic disorders could be correlated with the amount of lead found in the liver; 10 ppm or more of lead was nearly always associated with nervous signs. There were several monkeys, however, which had convulsions, but only 4 to 7 ppm liver lead.

Clinical signs were observed in only five parrots. These were convulsions in two, inability to fly or ataxia in two and lethargy was the only sign noticed in one. Six parrots were found dead. In one

ataxic kea, lead poisoning was suspected and blood found to contain 40 μ g lead/100 ml whole blood. No other hematologic abnormalities were found. The kea was treated for a total of 9 days (3 days on treatment followed by 3 to 4 days off) with calcium disodium ethylenediaminetetraacetate at 27 mg/kg given intraperitoneally. The kea seemed to improve temporarily, but never completely recovered and it was euthanized several months later.

Two of the three poisoned fruit bats were observed to remain apart from and be harassed by their cagemates shortly before they were found dead on the floor of their cage. No signs were noticed in the other bats. Two bats had ruptured livers, perhaps the result of fighting with cagemates, or acquired from a fall; the floor where they were found was 2.7 m from the ceiling screen from which they hung upside down.

Paint samples were analyzed from 75 different cages in this zoo. Thirty of these contained 2% or more lead, an amount generally considered hazardous. The highest amounts of lead in paint were found on outside metal cage bars; the highest being 66.6% lead.

Discussion

Despite the dearth of reports of lead poisoning in zoo-dwelling animals, there is evidence that led us to suspect that this disease is much more common and widespread than previously thought. In support of this are published reports of finding intranuclear inclusion bodies in renal proximal tubular epithelia of zoo animals. In 1930-1932 Hindle^{9,10} reported that such inclusions were commonly found in rhesus and cercopithecus monkeys that had died at the London Zoological Park, but were not found in recently imported primates. Several years later, Cowdry³ found that a variety of animals (including many primates and parrots) in the Philadelphia Zoo had "type B" intranuclear inclusion bodies in renal proximal tubular cells. Cowdry reported that these inclusions were probably "normal" for wild animals. This report by a distinguished scientist may have caused others to disregard the importance of similar inclusion bodies in wild animals. Hindle's and Cowdry's reports antedate the first association of intranuclear inclusion bodies with lead poisoning made by Blackman² in 1936. We have yet to find in zoo animals renal intranuclear inclusions that did not retain acid-fast stains and was not associa-

ted with excess lead (> 3 ppm) in visceral organs.

Van Bogaert³ described a disease in Old World zoo primates which we suspect may be caused by lead intoxication. This disease, acute amaurotic epilepsy, was reported in 16 primates (seven of which died and were autopsied) at the Antwerp Zoo. The neurologic signs, species affected (Old World monkeys), seasonal occurrence, and above all, the histologic changes in the nervous system are nearly identical to those of lead poisoning in our primates^{5,11}. Fourteen of the affected primates in this study were originally diagnosed as having this disease. We believe, and hope to prove, that these two diseases are one and the same.

Exposure of zoo animals to leaded paint is probably common, for many zoos have old painted metalwork animal enclosures, and lead is likely to be present in one or more layers of paint or primer. Leaded paint accessible to animals has been found in several zoos, e.g., a Chicago zoological park¹², Lincoln Park Zoo⁷, Antwerp Zoo³, Staten Island Zoo, Bronx Zoo (J. M. Budinger and E. P. Dolensek, personal communications), a New Zealand Zoo¹³, and an unnamed zoo¹.

Lead poisoning was found in 10 of 20 (50%) parrots and 22 of 49 (43%) primates that died at this zoo during specified lengths of time (Table 2, 3). The cases studied were screened for acid-fast intranuclear bodies in sections of kidney. It is known that not all lead-poisoned animals develop such inclusion bodies and it is therefore possible that the true frequency is even higher.

LITERATURE CITED

1. BEER, J. V. and STANLEY, P. 1963. Lead poisoning in the Slimbridge Wildfowl Collection. The Wildfowl Trust, 16th Annual Report, pp. 30-34.
2. BLACKMAN, S. S. 1936. Intranuclear inclusion bodies in the kidney and liver caused by lead poisoning. *Bull. Hopkins Hosp.* 58: 384-404.
3. BOGAERT, VAN L. 1962. Acute Amaurotic Epilepsy. In *Comparative Neuro-pathology*. J. R. M. Innes and L. Z. Saunders, Eds., Academic Press, New York, N.Y.
4. CORDY, D. R. 1957. Osteodystrophia fibrosa accompanied by visceral accumulations of lead. *Cornell Vet.* 47: 480-490.

5. COWDRY, E. V., LUCAS, A. M. and FOX, H. 1935. Distribution of nuclear inclusions in wild animals. *Amer. J. Pathol.* 11: 237-253.
6. ERNE, K. and BORG, K. 1969. Lead poisoning in Swedish wildlife. In: *Meta's and Ecology; a symposium*. Bulletin No. 5, Research Committee, Swedish National Science Research Council, p. 31-33.
7. FISHER, L. E. 1954. Lead poisoning in a gorilla. *J. Amer. vet. med. Assoc.* 125: 478-479.
8. HAUSMAN, R. STURTEVANT, R. A. and WILSON, W. J. 1961. Lead intoxication in primates. *J. Forensic Sci.* 6: 180-196.
9. HINDLE, E. 1932. A new kidney virus. *Nature* 129: 796.
10. HINDLE, E. and STEVENSON, A. C. 1930. Hitherto undescribed intranuclear bodies in the wild rat and monkeys, compared with known virus bodies in other animals. *Trans. Roy. Soc. Trop. Med. & Hyg.* 23: 327.
11. KILHAM, L., LOW, R. J., CONTI, S. F. and DALLENBACH, F. D. 1962. Intranuclear inclusions and neoplasms in the kidneys of wild rats. *J. Nat. Cancer Inst.* 29: 863-885.
12. LYON, D. G. 1970. Veterinary Laboratory Annual Report, North of England Zoological Soc., Chester Zoological Gardens, p. 9
13. McINTOSH, I. G. 1956. Lead poisoning in animals. *Vet. Rev. & Anat.* 2: 57-60.
14. SANDERSON, G. C. and THOMAS, R. M. 1961. Incidence of lead in livers of Illinois raccoons. *J. Wildlife Man.* 25: 160-168.
15. SAUER, R. M., ZOOK, B. C. and GARNER, F. M. 1970. Demyelinating encephalomyelopathy associated with lead poisoning in nonhuman primates. *Science* 169: 1091-1093.
16. SAYRE, J. W. 1970. A spot test for detection of lead in paint. *Pediatrics* 46: 783-785.
17. SYVERTON, J. T. and LARSON, C. L. 1947. Intranuclear inclusion bodies in the kidneys of wild rats. *Arch. Path.* 43: 541-552.
18. ZOOK, B. C. 1971. An animal model for human diseases—Lead poisoning in nonhuman primates. *Comparative Pathology Bull.* 3: 3-4.
19. ZOOK, B. C., SAUER, R. M., and GARNER, F. M. 1970. Lead poisoning in Australian fruit bats (*Pteropus poliocephalus*). *J. Amer. vet. med. Assoc.* 157: 691-694.

Received for publication January 24, 1972