



Blood Lead Levels of Wild Raccoons (*Procyon lotor*) from the Eastern United States

Authors: Hamir, A. N., Galligan, D. T., Ebel, J. G., Manzell, K. L., and Rupprecht, C. E.

Source: Journal of Wildlife Diseases, 30(1) : 115-118

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-30.1.115>

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.

Blood Lead Levels of Wild Raccoons (*Procyon lotor*) from the Eastern United States

A. N. Hamir,¹ D. T. Galligan,¹ J. G. Ebel, Jr.,² K. L. Manzell,² and C. E. Rupprecht,³ ¹ The Departments of Pathobiology and Clinical Studies, School of Veterinary Medicine, University of Pennsylvania, New Bolton Center, Kennett Square, Pennsylvania 19348, USA; ² Department of Toxicology, Diagnostic Laboratory, Cornell University, Ithaca, New York 14850, USA; ³ Department of Microbiology & Immunology, Center for Neurovirology, Thomas Jefferson University, Philadelphia, Pennsylvania 19107, USA

ABSTRACT: We analyzed 161 raccoon (*Procyon lotor*) blood samples obtained from New Jersey ($n = 109$), rural Pennsylvania ($n = 29$) and laboratory confined animals ($n = 23$) in the USA for lead content; we found significantly higher levels in the New Jersey raccoons (mean = 4.4 $\mu\text{g}/\text{dl}$, SE = 2.9). There was no difference between the lead levels of raccoons from the other two groups (mean = 2.6, SE = 0.5 and mean = 2.5, SE = 0, respectively).

Key words: Blood lead, Pennsylvania, New Jersey, raccoons, *Procyon lotor*.

Lead is ubiquitous in the environment and traces of it have been found in all living tissues (Goyer, 1986; Hamir, 1986). Exposure to toxic amounts of lead produces pathological changes in a variety of organ systems (Hamir, 1986), including the immune system. Experimental exposure of inorganic lead results in impairment of immune function (Hemphill et al., 1971; Koller, 1973; Luster et al., 1978) and suppression of host defense mechanisms resulting in increased susceptibility to infectious agents (Hemphill et al., 1971).

Presently the eastern USA is undergoing an epizootic of raccoon rabies (Cartter et al., 1992). To control this disease, an oral vaccinia-rabies glycoprotein (V-RG) vaccine has been developed (Rupprecht et al., 1986). Experimental vaccination with the V-RG vaccine results in protective immunity in the vaccinated animals and protects them against challenge by street rabies virus for at least six months (Rupprecht et al., 1986). Under U.S. Department of Agriculture (USDA) approval, the V-RG vaccine has been field-tested in Virginia, Pennsylvania and New Jersey (USA). However, the use of vaccinia virus as a vaccine vector of other genetic material is a controversial issue (Karzon, 1985). Since

lead is widespread in the environment and has been shown to suppress the immune system, we were concerned about the risk for vaccine failures in wild vaccinated raccoons that have high but subclinical levels of lead.

In some areas of the USA, liver lead concentrations of raccoons have been found to be high (Sanderson and Thomas, 1961; Diters and Nielsen, 1978) and clinical cases of lead toxicosis have been documented (Diters and Nielsen, 1978; Morgan et al., 1991). However, background blood lead concentrations have not been reported. Since blood lead levels reflect the degree of lead exposure, we conducted a survey of blood lead levels of raccoons from two locations and compared the results to laboratory-confined raccoons.

Blood samples of free-ranging, wild raccoons of both sexes were obtained from New Jersey ($n = 109$) and Pennsylvania ($n = 29$), June to September, 1992. The raccoons were live-trapped in Tomahawk #207 traps (Tomahawk Live Trap Company, Tomahawk, Wisconsin, USA), and lightly anesthetized with a mixture of ketamine (10 mg/lb, Veterinary Products, Bristol Laboratories, Syracuse, New York) and xylazine (0.4 mg/lb, Haver, Miles Laboratory, Inc., Shawnee, Kansas, USA); 2 ml of blood was drawn from the jugular vein. All samples were collected in ethylenediaminetetra-acetic acid (EDTA) tubes. Twenty-three blood samples also were obtained after light anesthesia from captive raccoons that were kept under laboratory conditions for periods ranging from several months to over a year.

Raccoons from northeastern Pennsylvania were obtained from State Game

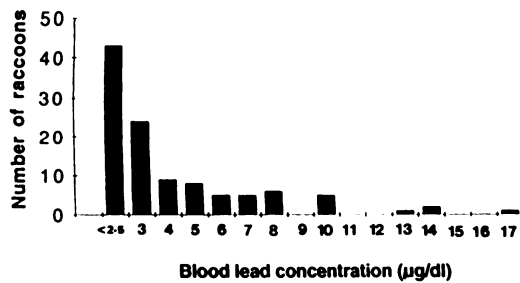


FIGURE 1. Blood lead levels of 109 raccoons from New Jersey, USA, June to September, 1992.

Lands #13 (41°18'N; 76°20'W) and the New Jersey raccoons were from the southern part of the state (39°15'N; 75°00'W). The former mountainous site is sparsely inhabited by humans whereas the latter site has a relatively high human population density. Raccoons at the New Jersey site were predominantly trapped at municipal dump sites.

Blood lead levels were determined by Zeeman atomic absorption spectrophotometry using the graphite furnace method (Pruszkowska et al., 1983). The results were analyzed by statistical method using Duncan's multiple means test (Bruning and Kintz, 1977). A level of $\alpha = 0.05$ was accepted as significant.

The mean blood lead level of all the raccoons was 3.8 µg/dl (SE = 2.5). There were no significant differences in blood lead levels of raccoons from Pennsylvania and the laboratory-confined raccoons (mean = 2.6 µg/dl, SE = 0.5; and mean = 2.5 µg/dl, SE = 0, respectively). All animals from the laboratory and 26 of 29 raccoons from the Pennsylvania group had <2.5 µg of lead per 100 ml of blood. The three raccoons from the latter group had blood lead levels of 2.9, 3.0, and 5.0 µg/dl. These values were considered to be within the normal range.

Raccoons from New Jersey had mean (\pm SE) values of 4.4 (\pm 2.9) µg/dl (Fig. 1). Many (66 of 109) of these raccoons had blood lead levels <2.5 µg/dl and nine had blood lead levels which ranged from 10 to 17 µg/dl. As a group, the New Jersey raccoons had significantly higher levels ($P =$

<0.05) than those from Pennsylvania and the laboratory-confined group, though the mean was not higher than the normal range for other species (Bratton and Kowalczyk, 1989).

Lead toxicosis affects many organ systems including central and peripheral nervous, hemopoetic, skeletal, gastrointestinal, reproductive and urinary systems (Goyer, 1986). In dogs the clinical signs of lead intoxication are predominantly gastrointestinal and nervous (Hamir, 1986). Clinical signs of lead toxicosis may be difficult to distinguish from canine distemper and rabies (Zook et al., 1969; Hamir et al., 1982).

For the detection and diagnosis of lead poisoning, most procedures rely on determination of concentration of lead in blood. The blood lead level indicates lead absorption by the animal and is the commonly used index of lead exposure in medical, scientific, and legal contexts (Hamir, 1986). According to Bratton and Kowalczyk (1989), analysis of blood for lead content is the single best index for establishing a diagnosis of lead intoxication. Since normal blood lead values for raccoons are not available, we had to rely on data that were available from other species. In dogs, blood lead levels of >25 µg/dl may indicate lead intoxication (Bratton and Kowalczyk, 1989). This level, however, is considered rather high for humans, for whom values as low as 10 to 15 µg/dl are considered evidence of potential lead toxicity (U.S. Department of Health and Human Services, 1988). In our study, many of the New Jersey raccoons had high lead levels and nine raccoons had lead levels between 10 and 17 µg/dl. Although the effects of low levels of lead exposure on raccoons has not been documented, and certainly the immunosuppression induced by lead has only been documented in laboratory animals and wild birds, the possibility of lead modulation of immune functions should be of concern to authorities responsible for the management and control of wildlife diseases.

The source of lead for the raccoons in southeastern New Jersey is unknown. Perhaps these animals obtained lead from their omnivorous diet. Maurer and Nielson (1981) proposed that scavenging of garbage by raccoons provides a source of lead for the animals that show clinical lead toxicosis.

The finding of higher blood lead levels in the New Jersey raccoons was not a surprise, as similar findings have been reported in dogs that reside in rural and urban areas (Hamir et al., 1986). Since lead-exposed animals may have suppressed immune functions, such animals may not be able to mount an adequate immune response when challenged with naturally occurring or introduced microbial agents. Therefore, when considering vaccination of animals in the wild, it is essential that the target animals have adequately functioning immune systems; otherwise vaccine failures may result in a proportion of animals, which could lead to an unsuccessful disease control in an area.

Based on our results, we propose that raccoons in some urban areas of the eastern USA have higher blood lead levels than raccoons from relatively uninhabited areas. We are in the process of evaluating the pathologic effects of experimental administration of low levels of lead to raccoons and the effects of V-RG oral vaccine on such animals.

This work was supported by funds from the Pennsylvania Department of Agriculture, the United States Department of Agriculture (formula funds), and the Department of Pathobiology, University of Pennsylvania. We thank R. Buchanan and D. Diehl for their technical expertise.

LITERATURE CITED

- BRATTON, G. R., AND D. F. KOWALCZYK. 1989. Lead poisoning. *In* Current veterinary therapy, R. W. Kirk (ed.). W. B. Saunders, Philadelphia, Pennsylvania, pp. 152-159.
- BRUNING, J. L., AND B. L. KINTZ. 1977. Duncan's multiple-range test. *In* Computational handbook of statistics, 2nd ed. Scott, Foresman and Co., Glenview, Illinois, pp. 116-119.
- CARTTER, M. L., J. L. HADLER, M. G. SMITH, F. E. SORHAGE, K. C. SPITALNY, J. G. DEBBIE, D. L. MORSE, J. L. HUNTER, J. N. MACCORMACK, K. A. SMITH, T. J. HALPIN, S. R. JENKINS, AND L. E. HADDY. 0000. Extension of the raccoon rabies epizootic—United States, 1992. Morbidity and Mortality Weekly Report, Number 41. Centers for Disease Control, U.S. Department of Health and Human Services, The Massachusetts Medical Society, Waltham, Massachusetts, pp. 661-664.
- DITERS, R. W., AND S. W. NIELSEN. 1978. Lead poisoning of raccoons in Connecticut. *Journal of Wildlife Diseases* 14: 187-192.
- GOYER, R. A. 1986. Toxic effects of metals. *In* Casarett and Doull's Toxicology, 3rd ed. C. D. Klaassen, M. O. Amdur, and J. Doull (eds.). Macmillan Publishing Co., New York, New York, pp. 582-605.
- HAMIR, A. N. 1986. Review of lead poisoning in dogs. *Veterinary Bulletin*, Number 56, C.A.B. International, Weybridge, England, pp. 1059-1070.
- , J. S. WILKINSON, AND P. D. HANDSON. 1982. Lead poisoning and canine distemper. *Journal of Small Animal Practice* 23: 301-305.
- , P. D. HANDSON, N. D. SULLIVAN, AND G. ANDERSON. 1986. Lead tissue levels of dogs from rural and urban areas of Victoria, Australia. *The Veterinary Record* 118: 77-78.
- HEMPHILL, F. E., M. L. KAEBERLE, AND W. B. BUCK. 1971. Lead suppression of mouse resistance to *Salmonella typhimurium*. *Science* 172: 1031-1032.
- KARZON, K. T. 1985. Vaccinia viruses as vectors for vaccine antigens. *In* Proceedings of the workshop on vaccinia viruses as vectors for vaccine antigens, G. V. Quinnan (ed.). Elsevier, New York, New York, pp. 231-240.
- KOLLER, L. D. 1973. Immunosuppression produced by lead, cadmium, and mercury. *American Journal of Veterinary Research* 34: 1457-1458.
- LUSTER, M. I., R. E. FAITH, AND C. A. KIMMEL. 1978. Depression of humoral immunity in rats following developmental lead exposure. *Journal of Environmental Pathology and Toxicology* 1: 397-402.
- MAURER, K. E., AND S. W. NIELSEN. 1981. Neurologic disorders in the raccoon in northeastern United States. *Journal of the American Veterinary Medical Association* 179: 1095-1098.
- MORGAN, R. V., F. M. MOORE, L. K. PEARCE, AND T. ROSSI. 1991. Clinical and laboratory findings in small companion animals with lead poisoning: 347 cases (1977-1986). *Journal of the American Veterinary Medical Association* 199: 93-97.
- PRUSZKOWSKA, E., G. R. CARNRISK, AND W. SLAVIN. 1983. Blood lead determination with the platform furnace technique. *Atomic Spectroscopy* 4: 59-61.
- RUPPRECHT, C. E., T. J. WIKTOR, D. H. JOHNSTON,

- A. N. HAMIR, B. DIETZSCHOLD, W. H. WUNNER, L. T. GLICKMAN, AND H. KOPROWSKI. 1986. Oral immunization and protection of raccoons (*Procyon lotor*) with a vaccinia-rabies glycoprotein recombinant virus vaccine. Proceedings of Natural Academy of Science, United States of America 83: 7947-7950.
- SANDERSON, G. C., AND R. M. THOMAS. 1961. Incidence of lead in livers of Illinois raccoons. The Journal of Wildlife Management 25: 160-168.
- U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES. 1988. The nature and extent of lead poisoning in children in the United States: A report to congress. U.S. Public Health Service, Agency for Toxic Substance and Disease Registry, Atlanta, Georgia, pp. 1-17.
- ZOOK, B. C., J. L. CARPENTER, AND E. B. LEEDS. 1969. Lead poisoning in dogs. Journal of the American Veterinary Medical Association 155: 1329-1342.

Received for publication 6 November 1992.