Plasma Marking of Arctic Foxes with Iophenoxic Acid

Authors: Follmann, Erich H., Savarie, Peter J., Ritter, Donald G., and Baer, George M.

Source: Journal of Wildlife Diseases, 23(4) : 709-712

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-23.4.709

Journal of Wildlife Diseases, 23(4), 1987, pp. 709–712

Plasma Marking of Arctic Foxes with Iophenoxic Acid

Erich H. Follmann,[1] Peter J. Savarie,[2] Donald G. Ritter,[3] and George M. Baer[4] [1] Institute of Arctic Biology, University of Alaska, Fairbanks, Alaska 99775, USA; [2] USDA/APHIS/ADC, Denver Wildlife Research Center, Building 16, Denver Federal Center, Denver, Colorado 80225, USA; [3] State of Alaska NRL (Virology-Rabies Unit), Division of Public Health, Fairbanks, Alaska 99706, USA; [4] Viral and Rickettsial Zoonoses Branch, Division of Viral Diseases, Center for Infectious Diseases, Centers for Disease Control, P.O. Box 363, Lawrenceville, Georgia 30246, USA

ABSTRACT: Six arctic foxes (*Alopex lagopus*) were marked with iophenoxic acid (IA), a substance which elevates concentrations of protein-bound iodine in blood plasma. Buccal absorption of IA was determined by placing 20 mg IA dissolved in 100% ethyl alcohol on the tongue. Blood samples collected from 1 to 36 wk following exposure showed that all foxes were marked already at 1 wk and continued until 13 wk; two foxes were still marked at 36 wk. Clearance rates for iodine varied with initial dose response, and those foxes with high 1-wk iodine concentrations excreted iodine more rapidly than those with lower initial concentrations; by 13-wk excretion rates were similar.

Key words: Arctic fox (*Alopex lagopus*), iophenoxic acid, plasma marker, experimental marking.

The marking of wild animals has proven to be an extremely valuable technique in wildlife studies. Certain techniques use physiological markers which require recapture or killing of animals to detect the marker. These include the fluorescent marker tetracycline (Linhart and Kennelly, 1967; Crier, 1970; Evans and Griffith, 1973), dyes that are absorbed by internal tissues and/or excreted with feces or urine (Evans and Griffith, 1973; Cowan et al., 1984), and radioisotopes that mark tissue or urine and feces (Taylor and Quy, 1973).

As part of a program to evaluate an oral rabies vaccine in wild arctic foxes (*Alopex lagopus*) in Alaska, it is essential to simultaneously mark the animal so that subsequent surveys will show which animals consumed baits containing the vaccine. The marker selected for this study was iophenoxic acid (IA) (α-ethyl-3-hydroxy-2,4,6-triiodohydrocinnamic acid) which has already been tested on several species, including the red fox (*Vulpes vulpes*) (Larson et al., 1981; Baer et al., 1985). This substance elevates the protein-bound iodine (PBI) in blood plasma, thus identifying animals that have ingested a marked bait. Specifically, the iodine binds to serum albumin and remains in this form (Shapiro and Man, 1960) until it is uncoupled in the liver (Hall and VanderLaan, 1961), and voided in urine (Osol et al., 1967). For application in a field study, it is essential to first determine baseline iodine concentrations in a study area because this could vary regionally and seasonally, especially with the arctic fox where some segments of the Alaskan population depend heavily on marine organisms during all or part of the year.

The hypothesis for the rabies vaccine study is that arctic foxes can be immunized via the oral route, as has been shown for the red fox (Baer et al., 1971; Mayr et al., 1972; Winkler et al., 1975; Winkler and Baer, 1976; Hafliger et al., 1982; Steck et al., 1982). The use of the marker, IA, would identify which foxes ingested the vaccine containing baits and permit evaluation of the effectiveness of the vaccine by serological testing for rabies antibodies in foxes. Marked animals with rabies antibodies would indicate immunization whereas marked animals without rabies antibodies would indicate no immunization.

Six arctic foxes (two males and four females) were captured in late November 1983 on the central Arctic Coastal Plain of Alaska using baited box traps. Foxes were maintained in outdoor cages in Fairbanks, and fed a combination of commercial dry dog food (Eukanuba, The IAMS Company, Lewisburg, Ohio 45338, USA) and a canned cat food (Blue Mountain, The Hervin Company, Tualatin, Oregon

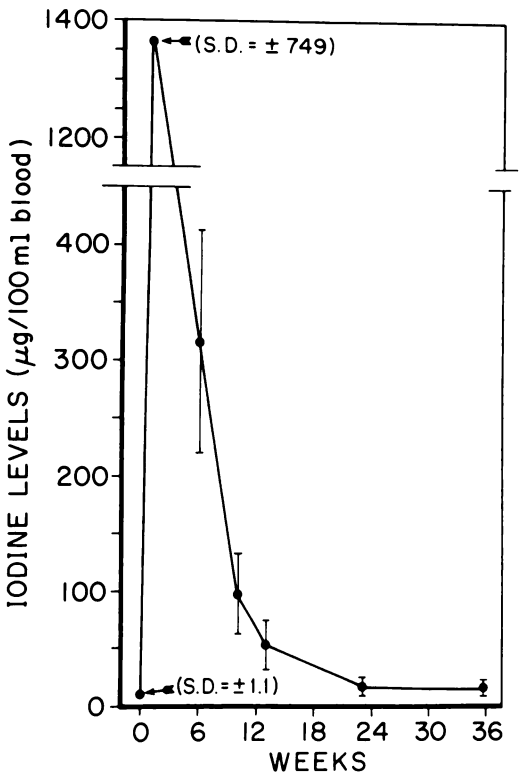

FIGURE 1. Mean iodine concentrations (μg/100 ml) in plasma of six arctic foxes administered 20 mg iophenoxic acid orally in liquid form.

97062, USA), and provided water ad libitum. In January 1984, in order to determine baseline concentrations of iodine, foxes were fasted for 24 hr (but were provided water), immobilized with 20 mg xylazine hydrochloride (Rompun, Bayvet Division, Miles Laboratories, Inc., Shawnee, Kansas 66201, USA) and a blood sample was drawn from the jugular vein. Samples were collected in heparinized tubes and centrifuged. Plasma was frozen and sent to the Denver Wildlife Research Center (U.S. Fish and Wildlife Service) for iodine determinations using the technique described by Larson et al. (1981).

Field tests with an oral rabies vaccine may require the vaccine and blood marker to be in liquid form to facilitate absorption through the buccal mucosa. To test the buccal absorption of IA, nonimmobilized foxes were administered 20 mg IA dissolved in 1 ml 100% ethyl alcohol by dropping the liquid onto the tongue with a small syringe. Blood samples for iodine determinations were taken as described above from foxes at 1, 6, 10, 13, 23, and 36 wk postexposure.

All six foxes responded to the administration of IA by a marked increase in iodine concentrations (Fig. 1). The 1 wk postexposure concentrations of four foxes were already much above the control mean (9.5 μg I/100 ml), demonstrating a high absorption of the marker, while two foxes had lower iodine concentrations but were still at least 44× baseline. A marked drop in iodine concentrations occurred 6 wk postexposure, but concentrations were still considerably above baseline (Fig. 1). If iodine concentrations twice the baseline value are considered to represent "marked" animals, most foxes would not be considered marked at 23 wk and only two would be considered marked at 36 wk. One was male and the other female, suggesting that the slower excretory rate of iodine from the blood of these foxes was not sex-related.

It is of interest that the two foxes still marked at 36 wk exhibited lower iodine concentrations (606 and 424 μg I/100 ml) 1 wk after administration of IA than did the other four foxes, the lowest of which had a concentration of 1,480 μg I/100 ml. The reasons for this initial variation are not clear. The total time of exposure would have been the same since it is known that the iodine in IA is absorbed via the alimentary canal (Baer et al., 1985). Food in the gut and differences in water intake should have been uniform for all foxes since none was fed prior to the marker application nor immediately after, and spilled water was not replaced in the cages until approximately 1 hr after marker application. Hence, different exposure times to IA should not be a factor.

The higher iodine concentrations at 36 wk in the two foxes that initially exhibited

the lowest concentrations at 1 wk postexposure to IA, suggest that the clearance rates were less over the duration of the experiment than in those foxes which exhibited higher initial dose responses. Lower clearance rates would prolong the loss of iodine thus maintaining a "marked" condition longer.

It was apparent that foxes responded physiologically to high concentrations of iodine intake by rapidly excreting it up to 13 wk, after which the rate of excretion decreased (Fig. 1). Astwood (1957) reported similar results in human patients administered IA as a radiographic contrast medium. Foxes responded with variable rates up to 13 wk, after which excretion rates decreased and were relatively uniform for all foxes (Fig. 1). Animals apparently adjusted their clearance rates in response to the amount of IA absorbed.

The results of this study in arctic foxes indicated that oral administration of IA in liquid form elevated iodine concentrations for at least 13 wk, which is adequate time to evaluate an orally-administered rabies vaccine. The rate of iodine loss varied considerably and depended on the initial dose response; foxes with high iodine concentrations excreted iodine much more rapidly than foxes with a lower initial dose response. The excretory rate was reduced after 13 wk. It appears that iodine concentrations are relatively uniform among foxes 3 mo or longer after administration of IA, regardless of the initial dose response.

We thank the University of Alaska's Office of the Vice-Chancellor for Research and Advanced Studies and Institute of Arctic Biology, the Alaska Department of Health and Social Services' Division of Public Health, and ARCO Alaska Inc. for support of various aspects of this project. Thanks are due D. J. Hayes for iodine analysis, L. K. Miller and two anonymous reviewers for review of early drafts of the manuscript, and D. A. Borchert for preparation of the figure.

LITERATURE CITED

ASTWOOD, E. B. 1957. Occurrence in the sera of certain patients of large amounts of a newly isolated iodine compound. Transactions of the Association of American Physicians 70: 183–191.

BAER, G. M., M. K. ABELSETH, AND J. G. DEBBIE. 1971. Oral vaccination of foxes against rabies. American Journal of Epidemiology 93: 487–490.

———, J. H. SHADDOCK, D. J. HAYES, AND P. SAVARIE. 1985. Iophenoxic acid as a serum marker in carnivores. The Journal of Wildlife Management 49: 49–51.

COWAN, D. P., J. A. VAUGHAN, K. J. PROUT, AND W. G. CHRISTER. 1984. Markers for measuring bait consumption by the European wild rabbit. The Journal of Wildlife Management 48: 1403–1409.

CRIER, J. K. 1970. Tetracyclines as a fluorescent marker in bones and teeth of rodents. The Journal of Wildlife Management 34: 829–834.

EVANS, J., AND R. E. GRIFFITH, JR. 1973. A fluorescent tracer and marker for animal studies. The Journal of Wildlife Management 37: 73–81.

HAFLIGER, U., P. BICHSEL, A. WANDELER, AND F. STECK. 1982. Oral immunization of foxes against rabies: Stabilization and use of bait for the virus. Zentralblatt fuer Veterinaermedizin Reihe B 29: 604–618.

HALL, R. R., AND W. P. VANDERLAAN. 1961. Effects of iophenoxic acid on tests of thyroid function. Journal of the American Medical Association 174: 648–649.

LARSON, G. E., P. J. SAVARIE, AND I. OKUNO. 1981. Iophenoxic acid and mirex for marking wild, bait-consuming animals. The Journal of Wildlife Management 45: 1073–1077.

LINHART, S. B., AND J. J. KENNELLY. 1967. Fluorescent bone labeling of coyotes with demethylchlortetracycline. The Journal of Wildlife Management 31: 317–321.

MAYR, A., H. KRAFT, O. JAEGER, AND H. HAACKE. 1972. Orale Immunisierung von Fuchsen gegen Tollwut. Zentralblatt fuer Veterinaermedizin Reihe B 19: 615–625.

OSOL, A., R. PRATT, AND M. D. ALTSCHULE. 1967. Iophenoxic acid. The United States Dispensary and Physicians Pharmacology, J. B. Lippincott Co., Philadelphia, Pennsylvania, 621 pp.

SHAPIRO, R., AND E. B. MAN. 1960. Iophenoxic acid and serum-bound iodine values. Journal of the American Medical Association 173: 1352.

STECK, F., A. WANDELER, P. BICHSEL, S. CAPT, AND L. SCHNEIDER. 1982. Oral immunization of foxes against rabies. A field study. Zentralblatt fuer Veterinaermedizin Reihe B 29: 372–396.

TAYLOR, K. D., AND R. J. QUY. 1973. Marking systems for the study of rat movements. Mammal Review 3: 30–34.

WINKLER, W. G., AND G. M. BAER. 1976. Rabies immunization of red foxes (*Vulpes fulva*) with vaccine in sausage baits. American Journal of Epidemiology 103: 408–415.

———, R. G. MACLEAN, AND J. C. COWART. 1975. Vaccination of foxes against rabies using ingested baits. Journal of Wildlife Diseases 11: 382–388.

Received for publication 10 February 1986.