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Source: Journal of Wildlife Diseases, 37(1) : 133-137

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-37.1.133>

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## RABIES AND CANINE DISTEMPER IN AN ARCTIC FOX POPULATION IN ALASKA

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**ABSTRACT:** Two oil field workers were attacked by a rabid arctic fox (*Alopex lagopus*) in the Prudhoe Bay oil field (Alaska, USA) prompting officials to reduce the local fox population. Ninety-nine foxes were killed during winter 1994. We tested foxes for prevalence of rabies and canine distemper. Exposure to rabies was detected in five of 99 foxes. Of the five, only one fox had rabies virus in neural tissue as determined by the direct fluorescent antibody test. The other four foxes had been exposed to rabies, but had apparently produced antibodies and did not have an active infection. No evidence of canine distemper was detected as determined by the absence of distemper antibodies in serum and distemper virus in neural tissue.

**Key words:** Arctic fox, distemper, oil, rabies, survey.

### INTRODUCTION

Studies in the Prudhoe Bay oil field (Alaska, USA) in the 1970's suggested that oil development may have stabilized arctic fox (*Alopex lagopus*) populations in developed areas (Eberhardt et al., 1982, 1983a, b; Rodrigues et al., 1994). Increased food availability (i.e., refuse and feeding by oil field personnel) in oil field developed areas may stabilize food sources and result in increased fox productivity, increased density, and dampened population fluctuations. Eberhardt et al. (1982:188) concluded that "localized petroleum development activities did not appear to have an immediate, dramatic, deleterious effect on the resident arctic fox population." Bousfield and Syroechkovskiy (1985) indicated that the fox population on Wrangel Island (Russia) increased and became more stable after the establishment of a small settlement provided foxes with a relatively stable food source. Recent work by Burgess et al. (1993), Rodrigues et al. (1994), and Ballard et al. (2000a) support the hypothesis that an increase in food supply in developed areas may have stabilized or increased fox densities.

The probability of more dense and stable arctic fox populations at Prudhoe Bay led to concerns about the potential effects on humans and wildlife, particularly the threat of rabies transmission. Arctic foxes are the primary reservoir species for rabies in the Arctic, and the disease is endemic in its populations, even when obvious signs and mortality do not occur (Bannikov, 1970). Rabies epizootics often occur as fox populations periodically increase along with populations of microtine rodents (Chapman, 1978; Kaplan, 1985). However, epizootics also may occur when fox populations are low (Rausch, 1958). Epizootics are generally geographically specific, non-synchronous, seasonal (peaks during February), and cyclic (3 to 4 yr intervals) (Ritter, 1981; Follmann et al., 1992). The large distances traveled by individual arctic foxes (e.g., as reviewed by Garrott and Eberhardt, 1987) facilitates the transmission of rabies virus by increasing contact among foxes, particularly when they congregate around food sources (Follmann et al., 1992). The variable incubation period of rabies, that may be up to 6 mo in arctic fox (Rausch, 1958), allows infected animals

to survive long enough to infect other animals. Scavenging on animals that have died of the disease has been proposed as another source of infection. Crandell (1975) and Dieterich and Ritter (1982) suggested that this reservoir for rabies virus serves as a focus of infections during periods of high fox population densities. However, other mammalian species which occur in the Prudhoe Bay area such as red fox (*Vulpes vulpes*), caribou (*Rangifer tarandus*), reindeer, and polar bear (*Ursus maritimus*) also may become infected, but such occurrences in ungulates and polar bear are rare (Dieterich and Ritter, 1982; Loewen et al., 1990; Taylor et al., 1991).

Arctic foxes also have been diagnosed with canine distemper on the Alaskan Beaufort Sea coast (Rausch, 1953). Canine distemper affects wolves (*Canis lupus*), dogs, red foxes, grizzly bears (*Ursus arctos*), and polar bears (Zarnke, 1981; Follmann et al., 1996). Canine distemper, like rabies, is caused by a virus that may manifest itself at higher host population densities (Choquette and Kuyt, 1974). Some signs of distemper are similar to rabies, including neural disorders. However, distemper, unlike rabies, does not include aggressive behavior, and incubation periods (i.e., up to 10 days) are short (Siegmond et al., 1979).

On 25 January 1994, two oil field personnel were attacked and bitten by a rabid fox. There also were numerous sightings of foxes behaving abnormally. These incidents and the apparently high densities of arctic foxes in the Prudhoe Bay oil field (Rodrigues et al., 1994; Ballard et al., 2000a) prompted a reduction in fox numbers. Our objectives in this study were to determine the prevalence of rabies and canine distemper in the arctic fox population at Prudhoe Bay.

#### MATERIALS AND METHODS

The study was conducted in the Prudhoe Bay oil field and included the Prudhoe Bay Unit (PBU) (within 147°50' to 149°10'N, 70°25' to 70°10' W). The area has been described by Pollard et al. (1996). The PBU is a system of

oil production facilities and supporting infrastructure including 44 producing oil wells, 19 non-producing wells, seven gathering centers, two gravel landing strips for jet aircraft, and two base camps for workers from British Petroleum Exploration (Anchorage, Alaska, USA), ARCO Alaska, Inc. (Anchorage) and oil field contractors. All facilities are supported by gravel pads and are connected by a network of primary and secondary (access) gravel roads totaling 278 km in length.

During January and February 1994 arctic foxes were live trapped with Havahart® traps (Ben Meadows Co., Atlanta, Georgia, USA) and then shot. Ninety-nine foxes were trapped and 92 and 80 were tested for rabies and canine distemper, respectively.

The heads of 92 foxes were collected for both rabies and distemper diagnosis. Serum from 80 foxes was collected by centrifuging whole blood collected by cardiac puncture. The direct fluorescent antibody test (FAT; Kissling, 1975) was used to detect rabies virus in the hippocampus, pons, and cerebellum, and the rapid fluorescent focus inhibition test (RFFIT; Thomas, 1975) to detect rabies virus antibodies in serum. Rabies antibody titers were considered positive at dilutions >1:5. The RFFIT was performed at the Department of Veterinary Diagnosis (Kansas State University, Manhattan, Kansas, USA).

Diagnosis for canine distemper virus was made on cerebellar tissue (92 animals) using FAT. The serum of 80 foxes was analyzed for distemper antibodies by serum neutralization. Serum was heat inactivated for 30 min at 56 C and various dilutions incubated for 1 hr at 25 C with 100 TCID<sub>50</sub> of the Rockborn strain of CDV. Vero monkey kidney cells were added following incubation and the microtiter plates incubated at 36 C for 5 days in a humidified incubator containing 5% CO<sub>2</sub>. Serum dilutions of ≥1:5 were considered positive. These analyses were conducted at the Washington Animal Disease Diagnostic Laboratory (Washington State University, Pullman, Washington, USA).

#### RESULTS

Of 92 foxes tested, evidence of exposure to rabies on the basis of neutralizing antibody presence was identified in four (4%) foxes, with titers of 1:11, 1:17, 1:25, and 1:45. One fox of the 92 tested (1% infected) had rabies virus present in brain tissue. None of the four foxes with rabies antibodies in serum had rabies virus in brain tissue. All five foxes were young males.

No evidence for serum neutralizing antibodies or virus was detected for canine distemper in either serum ( $n = 80$ ) or brain tissue ( $n = 92$ ), respectively. None of the sera were reported to be cytotoxic by either diagnostic lab which conducted analyses for rabies (KSU) and distemper (WSU) antibodies.

#### DISCUSSION

Crandall (1991) indicated that rabies predominately affects young male foxes, consistent with our results. Presence of antibodies in serum indicates that these animals had been exposed to the rabies virus antigen sometime in the past, but survived, at least up to the time of capture. These individuals probably were protected against the disease. This assumption is based on previous experiments evaluating oral rabies vaccines in captive arctic foxes (Follmann et al., 1988, 1992). In those studies all vaccinated foxes seroconverted prior to challenge with a large dose of the Arctic variant of the rabies virus. It is assumed that the significant anamnestic responses that were noted in all cases following challenge were responsible for protecting the vaccinated foxes. This was corroborated by the absence of rabies virus in neural tissue harvested after foxes were euthanized. In the present survey we assume that had the four foxes having antibody encountered the rabies virus, their immune systems would have responded similarly, with a rapid and profound proliferation of antibodies to neutralize the newly-acquired virus. This should protect the animal and inactivate the virus before it enters the central nervous system. Therefore, clinical manifestations of the disease would not occur because the brain would not become infected with the virus. It is likely that these four animals would be unable to transmit the virus. However, dogs that survived experimental infection with rabies virus have been shown to shed virus up to 305 days after recovery (Fekadu and Baer, 1980; Fekadu et al., 1981).

The five foxes reflecting previous expo-

sure to rabies virus were collected from the Base Operations Center (BOC) trapping site. More foxes were collected at the BOC ( $n = 56$ ) than elsewhere in the oil field. Everard and Everard (1985) suggested that rabies appeared to be a density-dependent disease. No doubt higher fox densities provide more opportunities for transmission of the virus. The BOC area is attractive to foxes through availability of shelter and food, and had a relatively high density of foxes which may explain why it was the only area in which rabies virus or antibodies were detected.

The prevalence of rabies virus in Prudhoe Bay arctic foxes was similar to that reported from other areas where the disease has been quantified. Excluding Banks Island (Northwest Territories, Canada) which had a prevalence of 22% (Secord et al., 1980), prevalence has ranged from 0 to 1% in Norway (Prestrud et al., 1992), Siberia (Syuzyumova, 1968), and the Canadian High Arctic (Secord et al., 1980). The higher prevalence of rabies virus in the Banks Island (Northwest Territories, Canada) study was no doubt due to the foxes being collected during an epizootic. All of the other studies were conducted when a rabies epizootic was not in progress. During 1971 through 1998, 307 of 619 arctic foxes (50%) examined in northwestern Alaska were diagnosed as rabies positive, but this sample was biased because most foxes that were collected had been exhibiting abnormal behavior and were suspected as rabid.

Our results on the prevalence of arctic fox exposure to rabies appears consistent with that reported from other arctic areas in that exposure rates were relatively low. However, in industrial areas where human risk to exposure may be high, prevalence of fox exposure to rabies should be periodically monitored, and when potential exposure is high, fox populations may need to be managed for public safety concerns.

The absence of evidence of distemper exposure in arctic foxes in this sample is of interest. Little is known about this dis-

ease in arctic foxes other than it has been reported in this species by Rausch (1953). Also, it is known to be present in wolves, red fox, grizzly bears, polar bears, dogs, and caribou/reindeer in Alaska (Zarnke, 1981; Follmann et al., 1996). Thus, there should be ample opportunity for arctic foxes to be exposed to this virus. Two explanations for detecting neither distemper virus or antibody in our sample are plausible. Since 89% of foxes were aged as juveniles (Ballard et al., 2000b) perhaps these animals had not as yet been exposed to this virus or, if they had, the time of exposure prior to their harvest was too short to allow production of a detectable antibody titer, or for adequate viral replication in neural tissue. Alternatively, had foxes been exposed previously with subsequent antibody production sufficient to prevent entry of the virus into neural tissue, no virus would be found using FAT. In this latter scenario, with our present lack of understanding of distemper infection in arctic foxes, it is possible that at the time of harvest antibody titers had declined below level of detection using serum neutralization techniques.

#### ACKNOWLEDGMENTS

This study was funded by British Petroleum Exploration (Alaska) Inc. (BPXA) with additional funding by ARCO Alaska, Inc. We thank M. Guilders for coordination of the project. We also thank E. Arnold, W. Stubblefield, L. E. Noel, and B. J. Pierson for technical and analytical support. J. Selinger, K. Waggoner, and personnel from BPXA and ARCO Environmental and Security Department assisted in the field. This is Texas Tech University College of Agricultural Sciences and Natural Resources technical publication T-9-845.

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*Received for publication 16 September 1999.*