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Source: Journal of Wildlife Diseases, 19(3) : 170-174

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-19.3.170>

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## SEROLOGIC AND EXPERIMENTAL INVESTIGATIONS OF CONTAGIOUS ECTHYMA IN ALASKA

Randall L. Zarnke,<sup>1</sup> Robert A. Dieterich,<sup>2</sup> Kenneth A. Neiland,<sup>1</sup> and Georgeanne Ranglack<sup>1,3</sup>

**ABSTRACT:** Serologic evidence of contagious ecthyma (CE) was found in domestic sheep (*Ovis aries*), domestic goats (*Capra hircus*), Dall sheep (*Ovis dalli*), and muskox (*Ovibos moschatus*) in Alaska. A moose (*Alces alces*) calf and a caribou (*Rangifer tarandus*) fawn were susceptible to experimental infection and both developed antibody titers as a result. CE virus was isolated from lesions of Dall sheep which were involved in a natural outbreak of the disease.

### INTRODUCTION

Contagious ecthyma (CE) is primarily a disease of domestic sheep and domestic goats (Beck and Taylor, 1974). However, it has also been diagnosed in humans and several species of wildlife from various parts of the world (Connell, 1954; Carr, 1968; Blood, 1971; Samuel et al., 1975; Hebert et al., 1977; Falk, 1978; Kummeneje and Krogsrud, 1978, 1979; Dieterich et al., 1981; Lance et al., 1981). An outbreak of CE occurred in captive muskox and Dall sheep in Alaska during 1976–1977 (Dieterich et al., 1981). As a result of these episodes and because of a continuing concern over the possibility of disease transmission from domestic animals to wildlife, a multi-faceted investigation was begun to clarify various aspects of the epizootiology of CE in Alaska. The objectives of the investigation were: (1) to determine if there was any evidence of past CE infection in wild and/or domestic animals in Alaska, (2) to determine if moose and/or caribou are susceptible to infection with CE virus, and (3) to evaluate the potential threat posed by CE to wildlife populations.

### MATERIALS AND METHODS

Blood samples from mountain goats (*Oreamnos americanus*) on the Kenai Peninsula and free-ranging Dall sheep near Dry Creek, Sheep Creek and on the Kenai Peninsula (Fig. 1) were collected by Alaska Department of Fish and Game (ADF&G) personnel when animals were captured for population studies. Samples from Dall sheep in the Brooks Range and muskox on Nunivak Island were obtained from hunt-

er-killed animals. Specimens from captive Dall sheep near Fairbanks, captive muskox near Unalakleet, and from domestic sheep and domestic goats near Fairbanks, Delta Junction, and Tok were collected by ADF&G personnel specifically for the present study. Serum was collected by aspiration after clots had formed and was frozen until tested.

The majority of sera were tested by the serum neutralization test (Rossi and Kiesel, 1971) at the Washington Animal Disease Diagnostic Laboratory (Washington State University, Pullman, Washington 99164, USA). Based upon past experience with this virus, a serum antibody titer of eight or greater was considered indicative of past CE infection (Vander-Schalie, pers. comm.). Dall sheep, muskox, and mountain goat sera collected during 1979 and 1980 were analyzed for antibody by the complement-fixation test (CF) (Erickson et al., 1975) at the National Veterinary Services Laboratories (NVSL) (Veterinary Services, APHIS, USDA, Ames, Iowa 50010, USA). Based upon previous experience, serum antibody titers of 20 or greater were considered indicative of past infection (Pearson, pers. comm.). The change in laboratories took place when it became necessary to process samples in a more timely manner than previous commitments at WSU would allow. CF is the method of choice at NVSL. Sera with evidence of past CE infection may hereafter be referred to as "positive."

In September 1981, a wildlife photographer observed a lamb Dall sheep with bleeding hoof lesions in Denali National Park. The animal was killed and examined at necropsy. Samples of the lesions were collected, preserved in 10% formalin, and processed for histologic examination. Tissue samples were frozen until tested in cell culture (Erickson et al., 1975) for CE virus at the National Veterinary Services Laboratories. In mid-November, Park Service personnel observed a band of sheep in the same vicinity where the earlier lamb had been seen. There were eight lambs in the band, four of which had easily detectable hoof lesions. Within 10 days of the initial report, one lamb was found dead and another was killed because of the severity of its lesions. Both of these animals were examined at necropsy and specimens collected as before.

In order to determine if moose and caribou are susceptible to CE infection, one individual of each species was experimentally exposed to a strain of CE which had been previously isolated from a naturally infected Dall sheep. These were newborn animals

Received for publication 24 May 1982.

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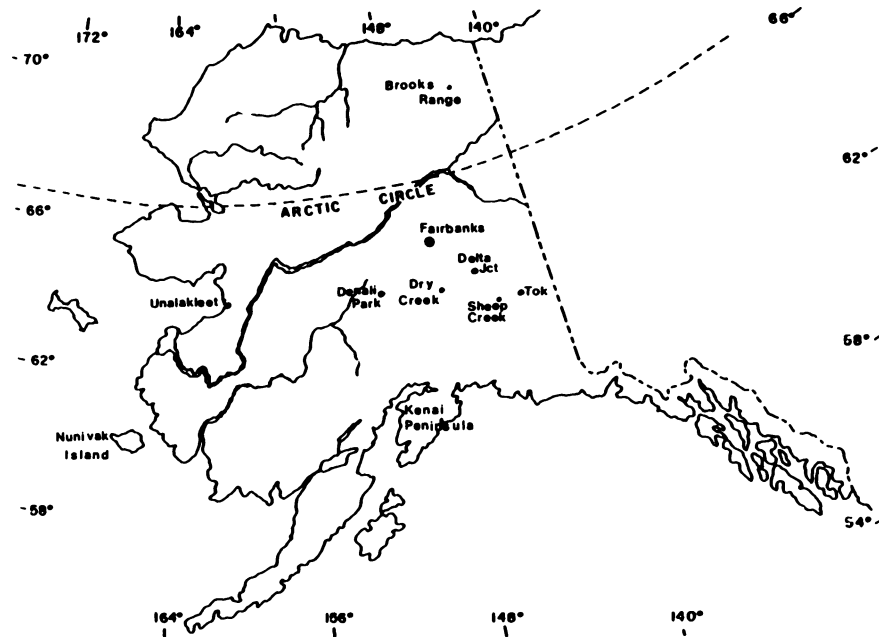


FIGURE 1. Sites in Alaska at which samples were collected for investigations of contagious ecthyma.

which were reared in isolation to prevent prior exposure to CE. An adult domestic sheep with no historic or serologic evidence of past CE infection served as a control. It was obtained from a flock never known to have experienced clinical CE. Pre-inoculation blood samples were drawn from all three animals on the day of exposure. They were exposed to the virus by scarification of the upper lip and application of a CE suspension. Blood samples were drawn from all three animals every 1–3 days for 3 wk and again on day 27 post-infection (PI). Biopsy specimens of lesions were excised and stored in 50% glycerol or frozen until tested for CE virus. Other portions of the biopsy samples were fixed in 10% formalin and processed for histologic examination.

### RESULTS

Results of the serologic survey are presented in Table 1. Antibody to CE was present (although uncommon) in domestic sheep and goats in Alaska's Interior. Nearly half (10/22) of the wild muskox which were shot by hunters during 1978 had CE antibody, whereas prevalence declined to zero in 1979 and 1980. Surprisingly, no antibody was found in the captive muskox during 1978. Antibody prevalence was high (9/11) in the captive Dall sheep in 1977, but declined to much lower levels in 1978 (1/9) and 1979 (1/10). Sero-positive free-ranging Dall sheep were found in the Interior as early as

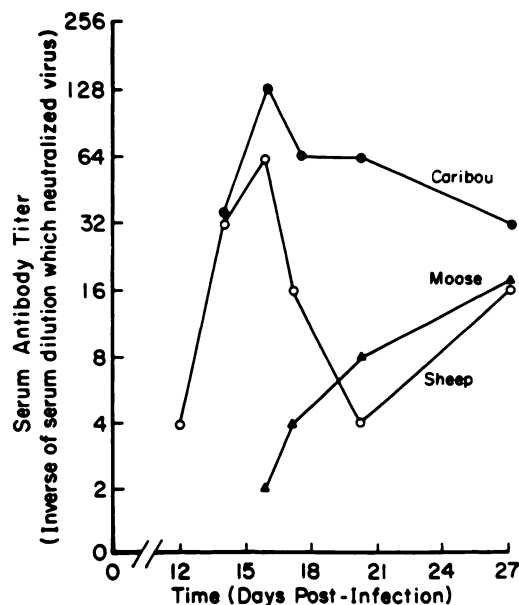


FIGURE 2. Antibody titers for moose, caribou, and domestic sheep inoculated with contagious ecthyma virus.

TABLE 1. Prevalence of sera from various Alaskan animals which were positive for contagious ecthyma antibody.

Species	Location	Year(s) collected	No. positive/ no. tested	% Prevalence	Range of titers
Domestic sheep	Fairbanks, Delta Jct., Tok	1978	2/32	6	16-64
Domestic goat	Fairbanks, Delta Jct., Tok	1978	3/87	3	8-128
Muskox	Nunivak Island	1978-1980	10/45	22	8-16
Muskox (captive)	Unalakleet	1978	0/9	0	—
Dall sheep	Dry Creek, Sheep Creek	1971, 1978, 1979	46/124	37	8-≥320
Dall sheep (captive)	Fairbanks	1977-1979	11/30	37	8-≥320

1971, with highest prevalence occurring in 1978. No positives were found in small numbers of Dall sheep from the Brooks Range or the Kenai Peninsula nor in mountain goats from the latter location.

All the Dall lambs collected in Denali Park had crusty lesions on the mouth and/or hooves. CE virus was isolated from the lesions of all three animals.

All three animals (moose, caribou, and domestic sheep) which were experimentally exposed to CE virus developed oral lesions which were grossly and histologically compatible with a diagnosis of CE. Affected areas were raised 1-2 mm with a pink, granular appearance averaging 1 cm in diameter and were limited to one or two sites on the lips. Their lesions did not interfere with normal feeding. Attempts to isolate CE virus from lesion biopsy specimens were unsuccessful. Results of serum antibody assays for these animals are presented in Figure 2. The antibody titers depict a typical mammalian response ("sero-conversion") to a viral infection.

#### DISCUSSION

Results of the serologic survey (Table 1) indicated that domestic sheep and domestic goats with previous exposure to CE virus were present in Interior Alaska. Although the disease appeared to be geographically widespread within Alaska, serum antibody prevalence in these domestic animals was not high. We believe that the CE antibody titers were indicative of natural exposure and were not due to purposeful

vaccination by the owners. Several goat owners in Interior Alaska have experienced recurrent episodes of a disease with signs which were compatible with clinical CE. However, there have been no confirmed reports of clinical CE to the State Veterinarian or Federal Veterinarian in Alaska (Gore, pers. comm.).

The absence of significant serum antibody titers in the nine members of the captive Unalakleet muskox herd was of special interest. Three of the nine animals were known to have been infected with CE within 1 yr prior to the time the samples were collected. A fourth individual had exhibited classic CE lesions within 2 yr prior to the sampling date. Thus, serum antibody titer decline is apparently quite rapid in muskox. On the other hand, based upon the absence of disease in adult muskoxen at Unalakleet, previous CE infection apparently did confer immunity in this species. The death of five of 39 young captive animals as a result of CE infection (Dieterich et al., 1981) indicated that this disease was more severe in younger individuals (Kummeneje and Krogsrud, 1978) and can contribute to population reduction.

Based upon results of serologic tests (Table 1), CE was present in the free-ranging muskox on Nunivak Island. Suspicious "warty lesions on the noses and mouths" of the original Nunivak Island stock were reported in 1931 (Bell, 1931; Palmer and Rouse, 1963). Apparently, CE has been present in the herd for quite some time, and the animals have not suffered noticeable losses as a result.

As would be expected, antibody prevalence

and titers were high in the captive Dall sheep shortly after signs were first observed in 1977. Antibody prevalence was low in both 1978 and 1979. However, titers were high in those specific animals which were considered serologically positive. Thus, apparently the environment in which these sheep were held was still contaminated with CE virus, or latent CE infections had been reactivated.

The presence of measurable CE antibody in sera from three of 10 Dall sheep captured during 1971 at Dry Creek indicates that the virus was present in wild populations prior to the outbreak in captive muskox and Dall sheep near Fairbanks. In the early 1960's, a Dall sheep lamb was found dead at Dry Creek by an ADF&G employee (unpubl. data). Lesions on the muzzle were suggestive of CE. In addition, CE virus was isolated from mammary gland lesions of an adult ewe Dall sheep which was captured at Sheep Creek in 1979 (Smith et al., 1982). These incidents lend support to the hypothesis that CE was present in wild sheep prior to the 1976–1977 outbreak in captive animals. Perhaps the disease has been enzootic in wild sheep since prehistoric times. Alternatively, occasionally domestic sheep and goats are allowed to graze in remote areas of Alaska near wild sheep habitat. These domestic animals could serve as sources of CE (and other diseases) for wild animal populations. It is believed that there was no spread of CE virus from either the captive muskox or captive Dall sheep to any wild animals. Thus, the finding of CE antibody in the free-ranging Dall sheep populations at Sheep Creek and Dry Creek in 1978 and Sheep Creek in 1979 further substantiates the belief that CE is enzootic in these populations.

The observations of diseased animals and the virus isolations from animals in Denali National Park indicated that the infection was most common and most severe in lambs. This is the case in domestic species (Beck and Taylor, 1974) and captive muskox and Dall sheep (Dieterich et al., 1981). The captive Dall sheep lambs near Fairbanks which developed severe infections (Dieterich et al., 1981) were infected at age 2–3 mo. The lambs from Denali Park were 2–5 mo older than these captive lambs, and the majority were in apparently healthy condition. However, the individual which was found dead had essentially no fat deposits and had apparently starved to death as a result of CE infection. The animal

which was killed due to the severity of its lesions was in relatively good physical condition with substantial subcutaneous, pericardial, perirenal, and mesenteric fat deposits. Perhaps this individual was in an earlier stage of the disease compared to the other lamb.

Results of the experimental exposure of caribou and moose demonstrate that these species are susceptible to CE infection. These animals were in good physical condition at the time of inoculation and remained so throughout the experiment. Lesions were mild. This observation agrees with previously published reports on the severity of CE lesions in domesticated reindeer (Falk, 1978; Kummeneje and Krogsrud, 1978). Serum antibody titers to CE virus reached significant levels. The small sample size in this experiment precludes any statements on the quantitative interpretation of serologic surveys involving free-ranging moose or caribou. Based upon the absence of any published reports of CE infection in free-ranging moose or caribou and the relatively mild lesions in our experimentally exposed young animals, CE probably does not pose any serious threat to healthy individuals of these species in the wild.

#### ACKNOWLEDGMENTS

The authors wish to acknowledge the assistance of E. A. Carlbrey, J. Pearson, D. Burger, W. E. Heimer, L. Nichols, Jr., and the staff of Denali National Park. Financial support for these studies was provided, in part, by Federal Aid in Wildlife Restoration Projects W–22-R.

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*Journal of Wildlife Diseases*, 19(3), 1983, p. 174  
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## BOOK REVIEW . . .

**A Host-Parasite Catalogue of the Avian Haematozoa**, Gordon F. Bennett, Madonna Whiteway and Carla Woodworth-Lynas. Memorial University of Newfoundland Occasional Papers in Biology #5. International Reference Centre for Avian Haematozoa, Department of Biology, Memorial University of Newfoundland, St. John's, Newfoundland A1B 3X9, Canada. 1982. 243 pp. \$15.00 Canadian.

This book is the culmination of a dozen years of original and literature research. It is a vast storehouse of information and it should be in the possession of anyone working on the biology of the blood parasites of birds. It is well organized and nicely keyed into the original literature references through citations to the two volumes of bibliography still available from the same Centre.

The first 200 pages contain an alphabetical listing of avian species arranged by host family. The parasites recorded in the literature or as unpublished records in the International Reference Centre for Avian Haematozoa are listed following each species name. Species that have been examined, but found to be uninfected are reported also. Synonyms are given where the host's name has been changed since the information was published.

A total of 3,816 avian species are listed representing 152 of 172 (84%) of the extant avian families. Species of *Haemoproteus* are recorded from 1,732 species representing 67% of the infected avian species. Other genera of blood parasites occurred as follows:

microfilariae (48%), *Plasmodium* (42%), *Leucocytozoon* (39%) and *Trypanosoma* (30%). Other infrequently encountered hematozoa are listed.

The number of species infected with the various hematozoan genera is summarized by avian family. It is interesting to note that nearly all species of certain families are susceptible to at least one genus of blood parasite, whereas species of other families are rarely, if ever, found positive. This is only a superficial indication due to the vast disparity in the numbers of individuals examined, but in some cases, these findings correspond nicely to recent reviews based on the prevalence of blood parasites in birds from entire continents.

There are also listings of the species of *Haemoproteus*, *Plasmodium*, *Leucocytozoon* and *Trypanosoma* that occur in birds. Each species of parasite is followed by the authority for the species and the type host and family.

An index to the families and genera of avian hosts greatly assists in the location of the desired avian species.

To reiterate my feelings about this volume as mentioned in the beginning of this review, it is a bargain for the price and is going to be invaluable to parasitologists from around the world.

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