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SERUM ANTIBODY PREVALENCE OF MALIGNANT CATARRHAL FEVER VIRUSES IN SEVEN WILDLIFE SPECIES FROM ALASKA

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ABSTRACT: Blood samples were collected from seven species of free-ranging ungulates in Alaska. Sera were tested for evidence of exposure to malignant catarrhal fever viruses (MCFV) by means of a competitive enzyme-linked immunosorbent assay. Antibody prevalences were as follows: muskox (*Ovibos moschatus*) 100 positive samples of 104 tested (96%); Dall sheep (*Ovis dalli*) 212 of 222 (95%); elk (*Cervus elaphus*) 14 of 51 (27%); bison (*Bison bison*) 34 of 197 (17%); caribou (*Rangifer tarandus*) nine of 232 (4%); Sitka black-tailed deer (*Odocoileus hemionus sitkensis*) one of 49 (2%); and moose (*Alces alces*) three of 219 (1%). Antibody prevalence in a bison population from the Interior was stable over a 5 yr period. These results indicate that at least one virus in the MCF group is enzootic in Dall sheep and muskox in Alaska. Lower antibody prevalences in the other species in this survey suggest that MCFV are latent or subclinical in these free-ranging ruminants. Whole blood samples were collected from 14 Dall sheep and subjected to a polymerase chain reaction assay. Fragments of ovine herpesvirus-2 DNA were detected in six of the samples. The significance of these findings for the health of free-ranging ungulates in Alaska is unknown.

Key words: Alaska, malignant catarrhal fever virus, serology, wildlife.

INTRODUCTION

Malignant catarrhal fever (MCF) is a complex disease syndrome of ruminants involving numerous organs and tissues (Metzler, 1991). Malignant catarrhal fever is often fatal, with severe, widespread inflammatory and degenerative changes. It typically has a short, dramatic clinical course characterized primarily by high fever, severe depression, swollen lymph nodes, salivation, diarrhea, dermatitis, neurologic disorders, and ocular lesions often leading to blindness (Plowright et al., 1990). Histopathologic hallmarks of MCF include widespread lymphoproliferation and infiltration, inflammation of mucosal surfaces of gastrointestinal and urinary tracts, ophthalmitis, lymphocytic meningoencephalitis, vascular inflammation, necrosis, and intense perivascular infiltration (Sanford et al., 1977).

The MCF syndrome is caused by members of a group of closely related gammaherpesviruses (Reid and Buxton, 1989). These viruses exist in nature as enzootic subclinical infections in certain ruminant species. Viruses and these natural hosts are well adapted to each other. The two most prominent carriers are wildebeest (*Connochaetes* spp.) and domestic sheep. Wildebeest are hosts of alcelaphine herpesvirus-1 (AlHV-1), which causes the classic 'African' form of MCF (Seal et al., 1989). Many domestic and exotic breeds of sheep are hosts for the other major form of MCF virus (MCFV). This virus is known as ovine herpesvirus-2 (OvHV-2). Recently, other previously undescribed members of the group have been recognized (Li et al., 2000, 2001a). It appears that many species of ruminants may harbor viruses that are closely related to the MCFV group at a genetic and antigenic level. Detailed information about the prevalence, epizootiology, and pathogenicity of these emerging viruses is as yet very sketchy.

Malignant catarrhal fever is an escalating problem for zoos, exotic game farms, and other mixed-species operations. Transmission of MCFV from domestic sheep and goats to susceptible domestic and exotic ruminants often leads to catastrophic losses, particularly in cervids (Li et al., 1999). When captive wild ruminants are

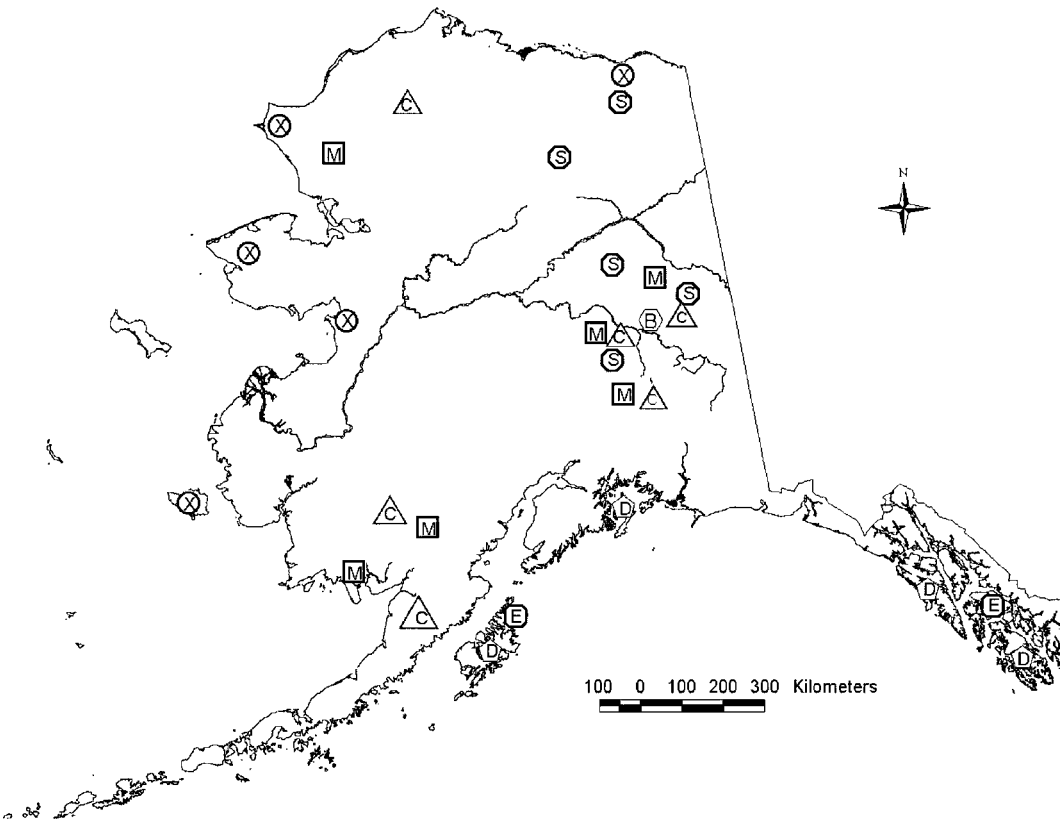

FIGURE 1. Study areas where blood samples were collected for malignant catarrhal fever serologic survey. B=bison, C=caribou, D=black-tailed deer, E=elk, M=moose, S=Dall sheep, and X=muskox.

exposed to MCFV, serious losses can result. However, reports of clinical disease in exotic species have dealt predominantly with captive rather than free-ranging populations (Heuschele, 1982; Metzler, 1991). Therefore, the impact of MCF on free-ranging wild ruminant populations is largely unknown.

The objective of the current survey was to determine the serum antibody prevalence of MCFV in seven species of free-ranging ruminants from Alaska.

MATERIALS AND METHODS

Most specimens were collected by personnel of the Alaska Department of Fish and Game, U.S. Fish and Wildlife Service, or the National Park Service from numerous locations throughout Alaska (Fig. 1). Hunters and hunting guides also provided specimens for this study. Blood samples were initially stored at either ambient or refrigerated temperatures for 12–36 hr. Sera were removed and stored temporarily at −15 C. Long-term storage was at −55 C for 1–20 yr until the time of testing.

Sera were tested for evidence of exposure to MCFV by means of a competitive enzyme-linked immunosorbent assay (Li et al., 1994, 2001b). Briefly, this assay utilizes a monoclonal antibody that reacts with an epitope which is present on all known members of the MCFV group of ruminant rhadinoviruses. Sera were scored based on their ability to inhibit binding of this monoclonal antibody to AlHV-1 antigens coated onto microtiter plates. Sera that inhibited binding of the monoclonal antibody greater than or equal to three standard deviations higher than negative control sera were considered indicative of previous natural exposure to the viruses. These samples will be referred to as "positive."

Whole blood was collected from 14 free-ranging Dall sheep (*Ovis dalli*) during 1997–2000. Deoxyribonucleic acid (DNA) was extracted, purified, and amplified by means of polymerase chain reaction (PCR) (Li et al., 1995).

TABLE 1. Serum antibody prevalence of malignant catarrhal fever viruses in seven selected species of wildlife from Alaska.

Species	Prevalence (%)
Muskox	100/104 (96)[a]
Dall sheep	212/222 (95)
Elk	14/51 (27)
Bison	34/197 (17)
Caribou	9/232 (4)
Deer	1/49 (2)
Moose	3/219 (1)

[a] Number positive/number tested (%).

RESULTS

A summary of results for each species is presented in Table 1. Antibody prevalence was very high for both muskox (*Ovibos moschatus*) and Dall sheep. Two distinct populations of elk (*Cervus elaphus*) were represented in the 51 samples (Fig. 1). Prevalence for the population in the southeastern portion of the state was 0/11. Prevalence for the population from the southwestern region was 14/40 (35%). Therefore, the reported prevalence of 27% (14/51) for the combined sample was somewhat misleading. The 197 bison (*Bison bison*) samples all came from a single free-ranging herd during five consecutive years. Prevalence in bison was stable throughout the period. Prevalence was quite low for caribou (*Rangifer tarandus*), Sitka black-tailed deer (*Odocoileus hemionus sitkensis*), and moose (*Alces alces*). Six of nine positive caribou sera came from a herd in the northwestern region of the state. Fragments of OvHV-2 DNA were detected in six of 14 Dall sheep whole blood samples.

DISCUSSION

Antibody prevalence for muskox in the current survey was 96% (100/104). These values were significantly higher than the 40% (8/20) reported previously for Alaska (Li et al., 1996). There are several possible explanations for the apparent discrepancies between the results of these surveys. First, sensitivity of the assay has been improved since the 1996 study (Li et al., 2001b). Secondly, muskox in the current study were all adults, whereas half of the muskox in the 1996 study were <1 yr old. Antibody prevalence of MCFV in domestic sheep and goats is directly related to age (Li et al., 1996). Perhaps the same age relationship influences prevalence in muskox. Thirdly, muskox in the 1996 study were captive; whereas the muskox in the current study were free-ranging. Perhaps the free-ranging herds are exposed to reservoirs that have no contact with captive muskox. A discrepancy in contact with known reservoirs could explain the observed difference in antibody prevalence between the two studies.

Antibody prevalence for Dall sheep in the current survey was 212/222 (95%). This is the first report of natural MCFV infection in free-ranging Dall sheep. Serologic evidence of exposure to MCFV was previously reported for three of nine captive Dall sheep (Heuschele et al., 1984). However, those animals were held in proximity to exotic host species. Therefore, that report does not necessarily reflect the MCFV exposure status of free-ranging Dall sheep populations.

The 95% prevalence reported here for Dall sheep is substantially higher than the 37% (124/339) reported for bighorn sheep (*Ovis canadensis*) from five western states in the contiguous United States (Li et al., 1996). In that previous survey, antibody prevalence for bighorns was higher than for other free-ranging species in the same vicinity. In the current survey, antibody prevalence was high for Dall sheep and muskox compared to other species of Alaskan wildlife.

Detection of an OvHV-2 DNA fragment in Dall sheep blood confirms the presence of the domestic sheep strain of MCFV in free-ranging Dall sheep. OvHV-2 is the major cause of MCF outbreaks in North America. Contact between known reservoir species, including Dall sheep, and clinically-susceptible species (whether domestic or free-ranging) should be avoided.

There was a disparity in antibody prevalence for elk from two locations in this survey. Based on results of Fisher's exact test ($P=0.1024$) and standard chi-square analysis ($P=0.1325$), these prevalences were not significantly different. Both populations of elk were introduced into Alaska. The population that currently lives in southwestern Alaska was introduced from Washington in 1929. The population in southeastern Alaska came from Oregon in 1986. Neither population has contact with domestic sheep. Perhaps MCFV infection occurred in the source population for the 1929 translocation, but did not occur in the source population for the 1986 operation. Alternatively, perhaps MCFV was introduced into the southwestern Alaska elk population since the relocation. There are no muskox, Dall sheep, or domestic sheep in either locale.

The stable antibody prevalence for bison over a 5 yr period suggests that exposure of bison to MCFV is a long-term relationship. If this were a new introduction, antibody prevalence might still be increasing as a largely susceptible population is exposed to the new agent. Similar antibody prevalences were reported for captive bison in other regions of North America (Li et al., 2001b). Mechanisms for transmission and maintenance of MCFV in bison populations remains unknown.

The low antibody prevalence in caribou, moose, and Sitka black-tailed deer reflect similar low prevalences in deer from five western states in the contiguous United States (Li et al., 1996). Obviously, either MCFV exposure rates for members of the cervid family are relatively low or most exposed cervids develop clinical disease and die following exposure. These low prevalences may reflect (a) low rates of exposure to known reservoir species, or (b) species-specific differences in response to infection. One major aspect of the epizootiology that remains unknown is the method(s) of intraspecific transmission (if any) within susceptible cervid species. All available information is consistent with the theory that intraspecies transmission does not occur in cervids.

There is no evidence to indicate that Alaska wildlife species are experiencing clinical signs of MCF. Due to the vast area of the state and the secretive nature of free-ranging animals, clinical disease and/or death of wildlife species is rarely observed or investigated. Therefore, the impact on free-ranging animals is unknown.

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LITERATURE CITED

HEUSCHELE, W. P. 1982. Malignant catarrhal fever in wild ruminants—A review and current status report. Proceedings of the United States Animal Health Association 86: 552–570.

———, H. R. FLETCHER, J. OOSTERHUIS, D. JANSSEN, AND P. T. ROBINSON. 1984. Epidemiological aspects of malignant catarrhal fever in the USA. Proceedings of the United States Animal Health Association 88: 640–651.

LI, H., D. T. SHEN, D. P. KNOWLES, J. R. GORHAM, AND T. B. CRAWFORD. 1994. Competitive-inhibition enzyme-linked immunosorbent assay for antibody in sheep and other ruminants to a conserved epitope of malignant catarrhal fever virus. Journal of Clinical Microbiology 32: 1674–1679.

———, ———, D. O'TOOLE, D. P. KNOWLES, J. R. GORHAM, AND T. B. CRAWFORD. 1995. Investigation of sheep-associated malignant catarrhal fever virus infection in ruminants by PCR and competitive inhibition enzyme-linked immunosorbent assay. Journal of Clinical Microbiology 33: 2048–2053.

———, ———, D. A. JESSUP, D. P. KNOWLES, J. R. GORHAM, T. THORNE, D. O'TOOLE, AND T. B. CRAWFORD. 1996. Prevalence of antibody to malignant catarrhal fever virus in wild and domestic ruminants by competitive-inhibition ELISA. Journal of Wildlife Diseases 32: 437–443.

———, W. C. WESTOVER, AND T. B. CRAWFORD. 1999. Sheep-associated malignant catarrhal fever in a petting zoo. Journal of Zoo and Wildlife Medicine 30: 408–412.

———, N. DYER, J. KELLER, AND T. B. CRAWFORD. 2000. Newly recognized herpesvirus causing malignant catarrhal fever in white-tailed deer (*Odocoileus virginianus*). Journal of Clinical Microbiology 38: 1313–1318.

———, J. KELLER, D. P. KNOWLES, AND T. B. CRAW-

FORD. 2001a. Recognition of another member of the malignant catarrhal fever virus group: An endemic gammaherpesvirus in domestic goats. Journal of General Virology 82: 227–232.

———, T. C. MCGUIRE, D. U. MULLER-DOBLIES, AND T. B. CRAWFORD. 2001b. A simpler, more sensitive competitive-inhibition ELISA for detection of antibody to malignant catarrhal fever viruses. Journal of Veterinary Diagnostic Investigation 13: 361–364.

METZLER, A. E. 1991. The malignant catarrhal fever complex. Comparative Immunology, Microbiology and Infectious Diseases 14: 107–124.

PLOWRIGHT, W., Z. DINTER, AND B. MOREIN. 1990. Malignant catarrhal fever virus. *In* Virus infections of ruminants, Z. Dinter and B. Morein (eds.). Elsevier Science Publishers B.V., Sweden, pp. 123–150.

REID, H. W., AND D. BUXTON. 1989. Malignant catarrhal fever and the *Gammaherpesvirinae* of Bovidae. *In* Herpesvirus diseases of cattle, horses, and pigs, G. Wittmann (ed.). Kluwer Academic Publishers, Boston, Massachusetts, pp. 116–162.

SANFORD, S. E., P. B. LITTLE, AND W. A. RAPLEY. 1977. The gross and histopathologic lesions of malignant catarrhal fever in three captive sika deer (*Cervus nippon*) in southern Ontario. Journal of Wildlife Diseases 13: 29–32.

SEAL, B. S., W. P. HEUSCHELE, AND R. B. KLIEFORTH. 1989. Prevalence of antibodies to alcelaphine herpesvirus-1 and nucleic acid hybridization analysis of virus isolated from captive exotic ruminants. American Journal of Veterinary Research 50: 1447–1453.

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