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Source: Journal of Wildlife Diseases, 22(3) : 418-420

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-22.3.418

may have lost detectable antibody due to natural decline or to chronic stress occurring during the last several years. The sheep could have then become susceptible to infections with these agents, predisposing them to bacterial (*Pasteurella*) pneumonia. The exact role of this respiratory syncytial virus or of PI-3 virus in the pathogenesis of illness of sheep of the Ouray herd could not be elucidated, but further serologic testing should help to clarify their roles.

Viruses were first implicated as being a possible predisposing factor to bacterial pneumonia in bighorn sheep in the mid 1960's (Howe et al., 1966, Bull. Wildl. Dis. Assoc. 2: 34–37). The first respiratory virus isolated from bighorn sheep was PI-3 virus from a captive herd in Wyoming (Parks et al., 1972, J. Wildl. Dis. 6: 669–672). Later PI-3 virus was isolated from free-ranging bighorn lambs from Colorado (Spraker, 1979, Ph.D. Thesis, Colorado State University, Fort Collins, Colorado, 232 pp.). Respiratory syncytial virus has been isolated from domestic sheep (Evermann et al., 1985, Am. J. Vet. Res. 46: 947–952) and pneumonic lesions have been induced experimentally in sheep using challenges of both respiratory syncytial virus and *Pasteurella haemolytica* (Al-Barraji et al., 1982, Am. J. Vet. Res. 43: 236–240). Isolation of a respiratory syncytial virus from this 8-mo-old bighorn lamb and serological evidence of this virus within the herd documents the presence of another respiratory virus of bighorn sheep. The primary role of this bighorn sheep respiratory syncytial virus in the pathogenesis of bacterial bronchopneumonia observed in these two sheep and in producing the rhinitis and coughing in the herd was undetermined.

Journal of Wildlife Diseases, 22(3), 1986, pp. 418–420

Serologic Studies of Select Infectious Diseases of Moose (*Alces alces* L.) from Alaska

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Few serologic studies have been conducted on moose from Alaska. Serologic reactivity has, however, been demonstrated in moose from Alaska to select arboviruses (Zarnke et al., 1983, J. Wildl. Dis. 19: 175–179) and antibody to contagious ecthyma was detected in an experimentally exposed moose calf (Zarnke et al., 1983, J. Wildl. Dis. 19: 170–174). Sera of moose from Alaska were also positive for antibodies to bovine viral diarrhea virus and infectious bovine rhinotracheitis virus (Dieterich, 1981, *In* Alaskan Wildlife Diseases, Dieterich (ed.), Univ. of Alaska Press, Fairbanks, pp. 28–29). The present serologic survey was designed to determine the prevalence of certain infectious agents of free-ranging moose from Alaska.

Serum samples were obtained between 1974 and 1982 from 110 free-ranging moose from Alaska. Samples were obtained from one location on the Alaska Peninsula (12 samples), three locations

Received for publication 15 July 1985.

around Anchorage or on the Kenai Peninsula (58 samples) and three areas in the Suscitna River area (40 samples). Serologic testing was for antibodies to bluetongue virus (BTV), infectious bovine rhinotracheitis virus (IBRV), bovine viral diarrhea virus (BVDV), epizootic hemorrhagic disease virus (EHDV), *Brucella abortus*, and several *Leptospira* serovars.

Antibodies to *B. abortus* were detected by the rapid card agglutination test conducted by the State–Federal Brucellosis Laboratory in Oklahoma City. Tests were read as either positive or negative. All other tests were conducted by the National Veterinary Services Laboratory, U.S. Department of Agriculture, Ames, Iowa. The microscopic agglutination test was used for detection of antibodies to *Leptospira interrogans* with the following serovars: *canicola*, *grippotyphosa*, *hardjo*, *icterohaemorrhagiae*, and *pomona*. The minimum titer used to indicate past exposure was 1:100. Antibodies for BTV and EHDV were detected by the immunodiffusion test. Tests were read as either positive or negative. The virus neutralization test in cell culture was used to detect antibodies to IBRV and BVDV. The minimum titer used to indicate past exposure was 1:16.

No serologic reactivity was detected in any samples for *Brucella*, BTV, or EHDV. The absence of antibody to BTV or EHD virus may be due to the apparent low prevalence of these viruses in northern North America (Sekers, 1981, *In* Virus Diseases of Food Animals, Academic Press, New York, pp. 567–584). BTV infection has not been documented in wild or domestic animals in Alaska (Zarnke, 1981, *In* Alaskan Wildlife Diseases, Dieterich (ed.), Univ. of Alaska Press, Fairbanks, pp. 38–39). However, 6% of 39 Alaskan moose with EHDV antibodies have been reported (Zarnke et al., 1983, op. cit.). Likewise, the absence of serologic reactivity to *B. abortus* in moose was expected since there have been no reports of *B. abortus* or *B. ovis* type 4 infections in Alaskan moose (Zarnke et al., 1983, op. cit.). There have been however, two documented cases of clinical brucellosis in moose from the continental United States (Zarnke et al., 1983, op. cit.; Fenstermacher and Olsen, 1942, Cornell Vet. 32: 241–254) and studies from Alberta have detected antibody in apparently normal moose (Fenstermacher and Olsen, 1942, op. cit.; Corner and Connell, 1958, Can. J. Comp. Med. 22: 9–20). Some investigators believe that the absence of seropositive moose in free-ranging populations may be due to their susceptibility to the *Brucella* organism and the high fatality rate that ensues (Corner and Connen, 1958, op. cit.; Jellison et al., 1953, J. Wildl. Manage. 17: 217–218; Dieterich, 1981, op. cit., pp. 53–58).

Thirteen samples (12%) were seropositive to various *Leptospira* serovars; eight (20%) from the Susitna area and five (42%) from the Alaska Peninsula. *Leptospira* serovars represented were: *L. grippotyphosa*—three (8%) from the Susitna area; *L. hardjo*—two (5%) from the Susitna area and two (17%) from the Alaska Peninsula; *L. pomona*—three (25%) from the Alaska Peninsula; *L. canicola*—three (8%) from the Susitna area; and *L. icterohaemorrhagiae*—five (13%) from the Susitna area. The presence of antibodies to *Leptospira* reported in this study is not unlike reports of similar studies in moose in North America (Diesch et al., 1972, U.S. Anim. Health Assoc. 76: 645–657; McGowan et al., 1963, Trans. N. Am. Wild. Nat. Resour. Conf. 28: 199–206; Bourque and Higgins, 1984, J. Wildl. Dis 20: 95–99). The serovar *grippotyphosa* was found to be the most prevalent for moose in Quebec and Minnesota (Bourque and Higgins, 1984, op. cit.). The reports of serovars *harjo*, *icterohaemorrhagiae*, and *canicola* appear to be the first for moose from Alaska. The significance of these findings, as they relate to moose health, is presently unknown.

Thirteen of 110 (12%) samples were seropositive for BVDV with four samples

(33%) originating from the Alaska Peninsula and nine samples (23%) from the Susitna area. Positive titers ranged from 1:16 to 1:128. Six samples (6%) were seropositive to IBRV, with four (10%) being from the Susitna area and two (17%) from the Alaska Peninsula. All positive titers were at the 1:16 dilution. Antibodies to IBRV in moose have been reported previously for 14% of 14 moose sampled in Alberta (Zarnke and Yuill, 1981, J. Wildl. Dis. 17: 453–461) although none were found in a previous study (Thorsen and Henderson, 1971, J. Wildl. Dis. 7: 93–95). Unpublished studies on serologic reactivity to IBRV and BVDV apparently have shown seroreactive moose in Alaska (Dieterich, 1981, op. cit.) although evaluation of 73 Alaskan Dall's sheep (*Ovis dalli*) failed to detect serologic reactivity to either virus (Foreyt et al., 1983, J. Wildl. Dis. 19: 136–139) nor did an evaluation of 39 moose from Alaska result in the detection of antibodies to BVDV or IBRV (Zarnke et al., 1983, op. cit.). The detection of serologic reactivity to these two viruses in the present study indicates that contact with domestic cattle or other seropositive wild ruminants may have occurred. Further studies appear warranted to determine the role of moose in the epidemiology of these infectious diseases and the significance of these findings to moose health.

Journal of Wildlife Diseases, 22(3), 1986, pp. 420–422

Isolation of a Poxvirus from a House Finch, *Carpodacus mexicanus* (Müller)

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Avian pox has been reported in many bird species (Kirmse, 1967, J. Wildl. Dis. 3: 14–20). On two occasions pox lesions have been observed on the house finch (*Carpodacus mexicanus* Müller), but virus was not isolated. Warner (1969, Condor 70: 101–120) first observed pox lesions on house finches introduced to Hawaii. He reported that nearly half of the house finches trapped had lesions at the bend of the wing, lores, and/or tarsal joint. None of the lesions were severe and it was suggested that the house finch was resistant to the virus. Power and Human (1976, Condor 78: 262–263) described an outbreak of disease among house finches frequenting bird feeders in the Santa Barbara, California area during the winter of 1971–1973. Of the total number of house finches captured, 17% (7/42) died with lesions. The authors noted that some of these birds had lesions severe enough to have caused mortality in the wild. In one of the captive birds, a minor lesion near one eye progressed in 3 wk to the point of completely closing both eyes. We report here the first isolation of poxvirus from the house finch.

In February 1983, two house finches found dead at a bird feeder in Boise, Idaho were submitted to the U.S. Fish and Wildlife Service, National Wildlife Health Laboratory, Madison, Wisconsin. Lesions were present near the beak and on the legs of both birds (Fig. 1). In both cases, the lesions on the beak were extensive enough to obstruct vision.

Received for publication 26 March 1985.