Serologic Survey of Canine Coronavirus in Wild Coyotes in the Western United States, 1972–1982

Authors: Foreyt, W. J., and Evermann, J. F.

Source: Journal of Wildlife Diseases, 21(4) : 428-430

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-21.4.428

sea lion virus (Smith et al., 1980, Am. J. Vet. Res. 41: 1846–1850; Gelberg and Lewis, 1982, op. cit.), and in cats infected with feline calicivirus (Love and Baker, 1972, Aust. Vet. J. 48: 643). By contrast, we were unable to correlate the cerebral histopathology with the subsequent isolation of *E. coli*; possibly this represented postmortem contamination from another body site.

The douc langur is indigenous to the tropical rain forests of southeast Asia and is classified as an endangered species by both the International Union for Conservation of Nature and Natural Resources (I.U.C.N., 1972–1978, Red Data Book, Vol. I, Morges, Switzerland) and the United States Department of the Interior (Fed. Reg. 45: 33768–33781). It is impossible at present to assess the impact of caliciviruses (if they are present) on the douc langur in its natural habitat. It is apparent, however, that, as a group, the caliciviruses are pathogens and can produce a variety of disease manifestations in a number of animal species (Smith, 1983, op. cit.). We have yet to demonstrate an etiological link between PCV-Pan 1 and a specific disease entity. However, the documented presence of this agent within an established primate collection, its recognized capacity for spreading and establishing infections in several different species of primates within this collection (Smith et al., 1983, op. cit.; Smith et al., 1985, op. cit.), and the widening recognition of the role of caliciviruses in diverse disease processes of animals (Smith, 1983, op. cit.; Barlough et al., 1985, op. cit.), together suggest to us an underlying potential for disease production by this virus.

This work was supported by the Zoological Society of San Diego, San Diego, California 92112, and by the College of Veterinary Medicine, Oregon State University, Corvallis, Oregon 97331.

Journal of Wildlife Diseases, 21(4), 1985, pp. 428–430

Serologic Survey of Canine Coronavirus in Wild Coyotes in the Western United States, 1972–1982

W. J. Foreyt, Department of Veterinary Microbiology and Pathology, Washington State University, Pullman, Washington 99164, USA; **and J. F. Evermann,** Department of Veterinary Clinical Medicine and Surgery and Washington Animal Disease Diagnostic Laboratory, Washington State University, Pullman, Washington 99164, USA

Viral agents were first identified as causes of infectious canine enteritis in the early 1970's (Carmichael and Binn, 1981, Adv. Vet. Sci. Comp. Med. 25: 1–37). In 1979 canine parvovirus-2 (CPV-2) and canine coronavirus (CCV) were reported in captive juvenile coyotes (*Canis latrans*) with severe diarrhea and high mortality (Evermann et al., 1980, J. Am. Vet. Med. Assoc. 177: 784–786). Although the clinical significance of CCV could not be determined at that time, it was speculated that a concurrent infection with CPV-2 could result in a more severe case of enteritis (Evermann et al., 1980, op. cit.) in coyotes held in captivity. The multiple etiology of enteric infections in domestic dogs has been reported (Carmichael and Binn, 1981, op. cit.). The major route of CCV transmission is through fecal contamination. Therefore, crowding, unsanitary conditions and other environmental

Received for publication 20 July 1984.

stressors, such as the immunosuppressive viruses, CVP-2 and canine distemper virus (Olsen and Krakowka, 1984, Comp. Cont. Educ. 6: 422–427), increase the rate of CCV infection and severity of clinical signs in domestic dogs. Coyotes are therefore susceptible to CCV infections and may serve as a reservoir for transmission to other susceptible wild and domestic canids. The purpose of this study was to determine the prevalence of antibody to CCV in wild coyote populations in selected western states.

Serum was obtained from wild captured coyotes from the United States Fish and Wildlife Service, Center for Disease Control and from trappers. Numbers of samples and locations of samples are listed in Table 1. Specific collection areas included California (Monterey County), Colorado (Adams and El Paso counties), Texas (Borden, Crane, Gaines, Hale, Howard, Motley, Potter, Upton and Webb counties), Utah (Cache County), and Washington (Whitman County).

Sera were tested for CCV IgG antibody by the indirect immunofluorescence method (Helfer-Baker et al., 1981, Canine Pract. 7: 37–42). Antibody titers were expressed as the reciprocal of the highest serum dilution resulting in positive immunofluorescence. Antibody titers of 1:25 or greater were considered positive. Positive CCV antibody titers were present in 12 of 235 (5.1%) samples (Table 1). Positive samples were detected in 1975, 1976, 1977, 1979, and 1980. Seropositive coyotes were detected in Colorado (n = 2, 1977), Texas (n = 3, 1975; 1, 1976; 1, 1979, 1, 1980), Utah (n = 2, 1977; 1, 1979), and Washington (n = 1, 1980).

Although the natural mortality in wild coyotes due to CCV is unknown, the virus is known to infect these animals based on this and other seroepidemiologic studies (Evermann et al., 1980, op. cit.; Green et al., 1984, J. Wildl. Dis. 20: 6–11). In captive coyotes maintained at the U.S. Sheep Experiment Station in Dubois, Idaho, 61% of the 46 unvaccinated adult coyotes had antibody to CCV (Green et al., 1984, op. cit.) indicating the widespread nature of the virus in that facility. Our data indicated a low seroprevalence of CCV (5.1%) in wild coyotes, indicating that the infection is not as widespread in wild coyotes as it is in coyotes kept in a kennel situation. This observation is in agreement with the epizootiology of CCV in domestic dogs, where between 55 and 70% of the dogs in high risk situations (boarding kennels and humane facilities) have been exposed to the virus on the basis of serologic surveys (Greene, 1984, *In* Clinical Microbiology and Infectious Diseases of the Dog and Cat, Greene (ed.), W. B. Saunders Co., Philadelphia, Pennsylvania, pp. 453–455). It is unlikely that intestinal infections by CCV alone are an important cause of mortality in adult coyotes (Green et al., 1984, op. cit.), or in juvenile coyotes 15–17 wk of age (Foreyt et al., unpubl. data). However, CCV may be an important etiologic agent of enteric disease in younger coyotes or in coyotes when other intestinal pathogens are present. Studies are cur-

TABLE 1. Prevalence of antibodies to canine coronavirus in wild coyotes (1972–1982).

Year	Location[a]	Number of coyotes	Number of coyotes with CCV antibody titers (%)
1972	UT	11	0
1973	UT	10	0
1974	UT	11	0
1975	TX	12	3 (25.0)
1976	TX, UT	19	1 (5.3)
1977	CA, CO, TX, UT	74	4 (5.4)
1978	TX, UT, WA	20	0
1979	TX, UT, WA	23	2 (8.7)
1980	TX, UT, WA	26	2 (7.7)
1981	TX	9	0
1982	TX	20	0
Total		235	12 (5.1)

[a] UT = Utah, TX = Texas, CA = California, CO = Colorado, WA = Washington.

rently underway in our laboratory to determine the distribution and prevalence of viral infections in coyotes in the western United States (Thomas et al., 1984, J. Am. Vet. Med. Assn. 185: 1283–1287; Evermann et al., 1985, Am. J. Vet. Med. Res. 46: 218–220).

The authors thank P. K. Bergstrom for assisting with the serologic testing and Kris Foreyt for raising the coyote pups. This project was supported in part by the Agricultural Research Service, the U.S. Department of Agriculture, U.S. Sheep Experiment Station, Dubois, Idaho 83423, USA.

Journal of Wildlife Diseases, 21(4), 1985, pp. 430–433

Spontaneous Poxviral Dermatitis and Keratoconjunctivitis in Free-Ranging Mule Deer (*Odocoileus hemionus*) in Wyoming

E. S. Williams and V. M. Becerra, Wyoming State Veterinary Laboratory, University of Wyoming, Box 950, Laramie, Wyoming 82070, USA; **E. T. Thorne,** Wyoming Game and Fish Department, Research Laboratory, University Station, Box 3312, Laramie, Wyoming 82071, USA; **T. J. Graham,** Box 478, Basin, Wyoming 82410, USA; **M. J. Owens,** Wyoming State Veterinary Laboratory, University of Wyoming, Box 950, Laramie, Wyoming 82070, USA; **and C. E. Nunamaker,** Plant Science Division, University of Wyoming, Laramie, Wyoming 82071, USA

Poxviruses infect a variety of mammalian and avian hosts, causing many diseases of public health or economic importance (Lane et al., 1981, *In* Handbook Series in Zoonoses, Section B: Viral Zoonoses, Vol. II, Steele (ed.), CRC Press, Inc. Boca Raton, Florida, pp. 365–385; Tripathy et al., 1981, *In* Comparative Diagnosis of Viral Diseases, Vol. III, Vertebrate Animal and Related Viruses, Part A—DNA Viruses, Kurstak and Kurstak (eds.), Academic Press, New York, pp. 267–346). Poxviral diseases are well studied in domestic animals and humans. Although the list of wildlife hosts is long (Nakano, 1977, *In* Comparative Diagnosis of Viral Diseases, Vol. I, Human and Related Viruses, Part A, Kurstak and Kurstak (eds.), Academic Press, New York, pp. 287–330), relatively little is known about pox infections in wildlife. Five reports document poxviral infection of cervids; two describe experimental contagious ecthyma caused by a parapox virus. Lance et al. (1983, J. Wildl. Dis. 19: 165–169) produced small proliferative lesions in the mucocutaneous tissue of the oral cavity of young mule deer, white-tailed deer (*O. virginianus*), and elk (*Cervus elaphus nelsoni*) by inoculation of lesion material from a bighorn sheep (*Ovis canadensis*) with contagious ecthyma. Lesions in all species were mild and regressed by 19 days postexposure. In a similar study, Zarnke et al. (1983, J. Wildl. Dis. 19: 170–174) exposed a moose calf (*Alces alces*) and a caribou fawn (*Rangifer tarandus*) to contagious ecthyma virus isolated from a naturally infected Dall sheep (*Ovis dalli*). Small lesions of contagious ecthyma developed on the lips of both animals.

Spontaneous contagious ecthyma has been described in domesticated reindeer (*Rangifer tarandus tarandus*) in Norway by Kummeneje and Krogsrud (1979, Vet. Rec. 105: 60–61), but the virus was not isolated. Lesions were mild and limited to

Received for publication 18 March 1985.