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SEROLOGIC SURVEY FOR SELECTED DISEASE AGENTS IN WOLVES (*CANIS LUPUS*) FROM ALASKA AND THE YUKON TERRITORY, 1984–2000

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ABSTRACT: Wolves (*Canis lupus*) were captured in several geographic areas of Alaska (USA) and the Yukon Territory (Canada) during 1984–2000. Blood was collected from 1,122 animals. Sera were tested for antibodies against infectious canine hepatitis virus (ICH), canine distemper virus (CDV), canine parvovirus (CPV), *Francisella tularensis,* and serovars of *Leptospira interrogans.* Antibody prevalence for ICH was >84% for all areas. Area-specific prevalences of antibodies ranged from 12% to 70% for CPV, from 0% to 41% for CDV, and from 4% to 21% for *F. tularensis*. There was no evidence of CDV exposure at the two southernmost locations in Alaska. Prevalence of antibodies for ICH increased slightly during the 16-yr course of the survey. There was essentially no evidence of exposure to *L. interrogans.* Prevalences of antibodies for both CPV and CDV were age-specific, with higher values in the adult cohort compared with the pup cohort. There were no sex-specific differences in prevalence of antibodies for any of the five disease agents.

Key words: Alaska, infectious disease, serologic survey, wolf, Yukon Territory.

INTRODUCTION

Wolves (*Canis lupus*) are one of the primary predators in Alaska (USA) and the Yukon Territory (Canada). Their effects on prey populations make wolves a keystone species, both biologically and politically. Population dynamics of wolves can be influenced by numerous factors (Mech, 1970). The two primary factors are harvest by humans (Ballard et al., 1981) and availability of prey.

Infectious diseases can also serve as a source of mortality for wolves (Carbyn, 1982; Weiler et al., 1995). Wolves in Alaska and the Yukon have ample opportunity for interaction with other canid species such as red fox (*Vulpes vulpes*), coyote (*Canis latrans*), arctic fox (*Alopex lagopus*), and domestic dog. These other canids might serve as reservoirs for transmission of infectious diseases to wolves.

The following disease agents were included in this survey: infectious canine hepatitis virus, canine parvovirus, canine distemper virus, *Francisella tularensis,* and *Leptospira interrogans.* Serologic evidence of exposure to these agents has been reported previously for wolves from Alaska and northern Canada (Choquette and Kuyt, 1974; Zarnke and Ballard, 1987). In addition, clinical cases of disease have been reported in domestic dogs and wolves (Elton, 1931; Choquette and Kuyt, 1974; Dieterich, 1981).

The objective of this study was to determine the effect of age, sex, year of collection, and location on serum antibody prevalence of selected infectious disease agents in wolf populations from several areas of Alaska and the Yukon Territory.

METHODS

Wolves were captured by employees of the Alaska Department of Fish and Game, US Fish and Wildlife Service, National Park Service, and the Yukon Department of Renewable Resources during 1984–2000 in conjunction with studies of wolf ecology (Fig. 1). Most of these animals were alive at the time of sample collection. Some of the animals from the Yukon were killed in conjunction with a planned reduction in the wolf population. Pups (<1 yr) were differentiated from adults on the basis of physical characteristics (McNay et al., 1999). Age data were not available for all animals. Population estimates were only available for a few areas during limited time periods. Therefore, changes in population size (perhaps because of disease) could not be addressed.

Blood was collected and allowed to stand for 10–30 hr. Serum was removed and stored at

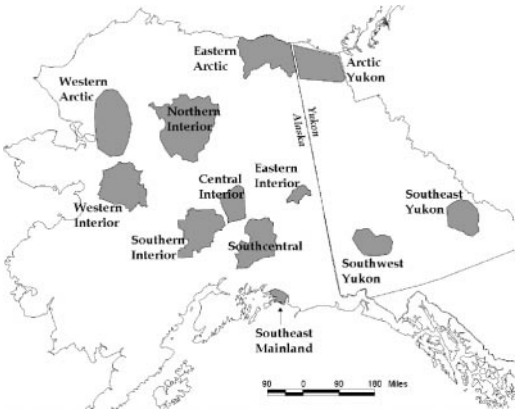

FIGURE 1. Capture areas for wolves included in serologic survey, 1984–2000.

−50 C for as long as 10 yr until the time of testing. The vast majority of sera were included in one of the two batches submitted to laboratories. Thus, testing conditions were consistent for all samples.

Sera were tested for presence of antibody to the following disease agents (not all samples were tested against all agents): canine distemper virus at the Wyoming State Veterinary Laboratory (Laramie, Wyoming, USA) by means of serum neutralization (Appel and Robson, 1973) with a threshold titer of 16; serovars of *L. interrogans* at the Wyoming State Veterinary Laboratory by means of a microscopic agglutination test (National Veterinary Services Laboratory, 1987) with a threshold titer of 100; canine parvovirus and infectious canine hepatitis virus at the National Veterinary Services Laboratory (Ames, Iowa, USA) by means of a serum neutralization test (Appel and Robson, 1973) with a threshold titer of 36; and *F. tularensis* at the Alaska Department of Fish and Game (Fairbanks, Alaska, USA) by means of a rapid plate agglutination test (Owen, 1970) with a threshold titer of 20. Specimens with titers that met or exceeded thresholds were considered indicative of previous natural exposure. These samples will be referred to as "positive." All other samples will be referred to as "negative."

A generalized linear model, with a logit link (McCullagh and Nelder, 1989) and a binomial distribution, was used to determine whether there was significant dependence of antibody prevalence on age, sex, year, and location. Serologic test result is a binary response variable. Age was treated as a categorical variable with two classes: pups and adults. Sex and geographic location were treated as categorical variables. Year was treated as a continuous variable. All main and interaction effects of these variables were examined. During the modeling process, all higher order terms were removed from the model if they did not substantially ($P>0.05$) increase the fit of the model on the basis of the deviance function compared with a chi-squared distribution (McCullagh and Nelder, 1989). The GENMOD procedure of the SAS statistical software package (version 6.12, SAS Institute, Cary, North Carolina, USA) was used to fit the model with maximum likelihood parameter estimates.

Pups were only sampled in some geographic units. Therefore, data were analyzed two ways. One data set included all geographic units in which pups occurred. Models for these data evaluated the effects of location, sex, year, and age on antibody prevalence. Another data set included all geographic units. However, only the data for the adult animals were used. The models for these data evaluated the effects of location, sex, and year (not age) on antibody prevalence.

RESULTS

A summary of serologic test results is presented in Table 1. Antibody prevalences varied significantly ($P<0.05$) between study sites for canine parvovirus (CPV), canine distemper virus (CDV), and *F. tularensis*. Antibody prevalences varied significantly ($P<0.05$) between pup and adult age cohorts for infectious canine hepatitis virus (ICH; pups: 138/162, 85%; adults: 495/547, 90%), CPV (pups: 29/162, 18%; adults: 211/530, 40%), and CDV (pups: 0/166, 0%; adults: 73/545, 13%). Prevalence increased significantly ($P<0.05$) during the course of the study for ICH (Table 2). There were no sex-specific differences in prevalence for any of the agents included in the survey.

DISCUSSION

Clinical signs of ICH infection in captive red foxes can include rhinitis, ataxia, anorexia, blood in feces, ocular keratitis, and occasionally convulsions leading to paralysis and death (Woods, 2001). Infected animals shed virus in one or more of saliva, urine, and feces. Transmission occurs via direct contact with these materials (Woods, 2001).

Previous serologic surveys of wolves

TABLE 1. Serum antibody prevalence of five infectious disease agents in wolves (*Canis lupus*) from 12 areas of Alaska, USA, and the Yukon Territory, Canada, 1984–2000.

Area	Prevalence[a]				
	Infectious canine hepatitis virus	Canine parvovirus	Canine distemper virus	*Francisella tularensis*	*Leptospira interrogans*
Southeast Mainland	16/19 (84)	2/16 (13)	0/20 (0)	0/5 (0)	0/26 (0)
Southcentral	14/16 (88)	10/16 (63)	0/16 (0)	1/6 (17)	0/10 (0)
Central Interior	154/158 (97)	94/157 (60)	1/159 (1)	8/32 (25)	1/177 (1)
Southern Interior	183/213 (86)	58/204 (28)	70/214 (33)	28/135 (21)	0/126 (0)
Eastern Interior	128/141 (91)	47/137 (34)	2/136 (1)	2/30 (7)	0/138 (0)
Western Interior	28/31 (90)	21/30 (70)	13/32 (41)	3/30 (10)	0/3 (0)
Northern Interior	43/50 (86)	17/51 (33)	17/54 (31)	7/48 (15)	0/29 (0)
Western Arctic	73/77 (95)	42/77 (55)	4/77 (5)	5/75 (7)	0/0 (0)
Eastern Arctic	38/40 (95)	10/40 (25)	16/46 (35)	2/45 (4)	0/32 (0)
Southwestern Yukon	12/14 (86)	3/14 (21)	8/15 (53)	4/13 (31)	0/2 (0)
Southeastern Yukon	60/76 (79)	20/74 (27)	32/83 (39)	26/64 (41)	0/11 (0)
North Slope/Yukon	22/23 (96)	16/21 (76)	14/22 (64)	6/18 (33)	0/1 (0)

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

from Alaska reported high antibody prevalence for ICH (Stephenson et al., 1982; Zarnke and Ballard, 1987). Current results continue that pattern (Table 1). Apparently, this virus is enzootic in wolf populations throughout the region. Antibody prevalence for ICH was slightly higher in the adult cohort (495 of 547, 90%) compared with the pup cohort (138/162, 85%). This minor difference might reflect greater likelihood of exposure to the virus during additional years of life.

TABLE 2. Annualized serum antibody incidence of infectious canine hepatitis virus in wolves (*Canis lupus*) from Alaska, USA, and the Yukon Territory, Canada, 1984–99.

Year	Prevalence[a]
1984	7/8 (88)
1985	5/6 (83)
1986	14/18 (78)
1987	49/57 (86)
1988	54/61 (89)
1989	51/64 (80)
1990	68/74 (92)
1991	62/77 (81)
1992	74/79 (94)
1993	42/49 (86)
1994	21/23 (91)
1995	56/69 (81)
1996	67/72 (93)
1997	142/151 (94)
1998	124/133 (93)
1999	78/86 (91)

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

Antibody prevalence to ICH increased slightly during the course of the study ($P=0.011$; Table 2). Sample sizes were small during the first 3 yr of the survey. However, this did not appreciably affect the overall pattern. Antibody prevalences for ICH have been high in northern wolf populations for many years (Choquette and Kuyt, 1974; Stephenson et al., 1982; Zarnke and Ballard, 1987). Therefore, the minor increase observed in the current survey could not be explained by either 1) introduction of the agent into an immunologically naïve population or 2) increases and decreases in prevalence related to acute epizootics. Some of the sera collected in the early years of this survey were stored for 10 yr prior to testing. Perhaps a small proportion of antibody denatured during this storage period. There is no other readily apparent explanation for the slight increase in antibody prevalence during the 16 yr of this survey.

Rates of morbidity and mortality are difficult to assess in free-ranging species. If the high antibody prevalence rates reported here were combined with a significant

case fatality rate, presumably there would be a long-term negative effect on the regional population. No evidence of a long-term downward trend has been observed in any of the wolf populations included in this survey. Therefore, apparently the mortality rate attributable to ICH is low. This conclusion is in concurrence with a study of a free-ranging coyote population in which the effect of ICH was believed to be minimal (Trainer and Knowlton, 1968).

Clinical signs of CPV infection in domestic dogs can include leukopenia, diarrhea, dehydration, and depression. Signs of disease are more common and more severe in pups compared with adults (Pollock et al., 1980). Lesions might include enteritis, myocarditis, or both (Appel et al., 1978; Pollock et al., 1980). Transmission occurs via contact with virus shed in feces (Pollock et al., 1980). Clinical CPV disease was first confirmed in domestic dogs in 1978 (Appel et al., 1978). Transmission to free-ranging carnivores was documented shortly thereafter (Mech et al., 1986; Zarnke and Ballard, 1987). Coyotes with enteritis are often infected with both CPV and canine coronavirus (CCV) (Evermann et al., 1980). Many of the samples included in the current survey were also tested for evidenced CCV exposure (Zarnke et al., 2001). Antibody prevalence averaged 25% in autumn and 70% in spring. There was no detectable antibody in 4–5-mo-old pups. By age 9–10 mo, prevalence had risen to 60%.

Previous surveys of free-ranging canids from North America reported antibody prevalences to CPV ranging from 40% to 60% (Barker et al., 1983; Thomas et al., 1984; Zarnke and Ballard, 1987). Current results cover a much broader range, from 13–76% (Table 1). There was no apparent geographic or chronologic pattern. There is no readily apparent explanation for the geographic differences in antibody prevalence. Prevalence was high near human settlements where dogs are found. However, prevalence was even higher in remote areas.

Antibody prevalence to CPV was significantly higher in adults (211/530, 40%) compared with pups (29/162, 18%). This difference might reflect greater likelihood of exposure to the virus during additional years of life. Alternatively, perhaps some pups exposed to the virus succumb to clinical disease and are thus removed from the population.

Our results cover a broad geographic range (Table 1). There is no readily apparent explanation for the geographic differences in prevalence. Antibody prevalence was high near human settlements where dogs are often found. However, prevalence was even higher in remote areas.

The effect of CPV on populations of free-ranging wolves is unknown. An experimental study involving captive wolves under controlled laboratory conditions suggested CPV could be a significant source of morbidity and mortality (Zuba, pers. comm.). Clinical signs and postmortem lesions were similar to those reported for domestic dogs. Observations of wolves in a large enclosure suggested that CPV could be a significant source of mortality (Mech et al., 1986). Thus, CPV is theoretically capable of causing direct mortality in free-ranging wolves. Presumably, pups would be affected most, and entire litters could be lost. However, there have been no widespread declines in pack productivity or major population declines in any of the study areas included in this survey. Therefore, the high antibody prevalences reported here do not implicate CPV as a major factor in wolf population dynamics on a broad geographic scale.

Clinical signs of CDV infection in captive red foxes can include oral icterus and ulceration, swollen feet, anorexia, ataxia, dyspnea, and neurologic abnormalities (Williams, 2001). Transmission occurs via aerosol droplet or direct contact between infected and susceptible animals (Williams, 2001).

Antibody prevalence for CDV differed significantly between areas (Table 1), but

TABLE 3. Annualized serum antibody incidence of canine distemper virus in wolves (*Canis lupus*) from Southern Interior, Alaska, USA, 1986–99.

Year	Prevalence[a]
1986	1/14 (7)
1987	5/13 (38)
1988	6/10 (60)
1989	3/14 (21)
1990	2/13 (15)
1991	14/33 (42)
1992	16/20 (80)
1993	3/16 (19)
1994	7/16 (44)
1995	6/23 (26)
1996	0/13 (0)
1997	2/28 (7)
1998	2/14 (14)
1999	3/9 (33)

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

there was no apparent geographic pattern. For each area with overall prevalence >30%, there was a chronologic pattern of 2 yr with high prevalence (>30%) followed by 1 or 2 yr with lower prevalence (<20%). The best example of this phenomenon was in the Southern Interior study area (Table 3). This pattern gives the impression of short-term epizootics followed by interepizootic periods. The results of the current survey were inadequate to determine whether epizootics in the various study areas were synchronous.

Antibody prevalences against CDV in previous surveys were ≤12% (Choquette and Kuyt, 1974; Stephenson et al., 1982; Zarnke and Ballard, 1987). Thus, the higher prevalences reported here (>30%) were unexpected. There were no known outbreaks of clinical CDV disease in domestic dogs during the course of this survey.

Antibody prevalence against CDV was 0% for the pup cohort (0/166). This result concurs with previous studies (Choquette and Kuyt, 1974; Stephenson et al., 1982; Zarnke and Ballard, 1987). Antibody prevalence in coyote pups was also lower than prevalence in the adult cohort (Guo et al., 1986). Young domestic dogs (Gorham, 1966) and coyotes (Williams, 2001) are susceptible to CDV infection. Prognosis is poor for clinically affected animals (Williams, 2001). Perhaps wolf pups that are exposed to CDV in the wild invariably succumb. Under this scenario, only pups that have not been exposed remain in the population.

Tularemia is an acute, febrile, plaguelike disease caused by the bacterium *F. tularensis* (Mörner and Addison, 2001). In Alaska, snowshoe hares (*Lepus americanus*) are the primary host. Clinical signs of tularemia in hares include ataxia and loss of fear (Mörner and Addison, 2001). Hare population density rises and falls in a predictable 10-yr pattern (Keith, 1963). Ticks (*Hemaphysalis leporispalustris*) serve as the primary vector for intraspecific transmission in hares, particularly when hare density is increasing (Zarnke and Ballard, 1987). Predators are exposed when they feed on infected hares.

A previous serologic survey for evidence of tularemia reported an antibody prevalence of 25% for wolves from the South-central study area (Zarnke and Ballard, 1987). In the current survey, antibody prevalences ranged from 0% to 41% for the various study areas. There was no apparent geographic pattern.

For several areas, antibody prevalence for tularemia in the wolf population peaked in 1991 and 1992. In most of the study areas, the snowshoe hare population peaked 1 or 2 yr earlier. Population-scale effects on hare predators typically lag 1 yr behind changes in the hare population (Keith, 1963). Perhaps prevalence of clinical tularemia in hares peaked in conjunction with the hare population density. Wolves preying on hares during peak hare numbers would show serologic evidence of exposure to *F. tularensis* in subsequent years.

Leptospirosis can cause chronic kidney infections, hepatitis, and abortion in a broad spectrum of domestic and wildlife species (Leighton and Kuiken, 2001). Leptospires are shed in urine. Transmission to carnivores can occur via exposure to contaminated urine or feeding on infected

prey (Reilly et al., 1970). Recognized free-ranging canid hosts include red fox (Clark, 1960), gray fox (*Urocyon cinereoargenteus*; Clark et al., 1961), and coyote (Marler et al., 1979; Drewek et al., 1981).

Antibody prevalence for *L. interrogans* was very low (Table 1). These results concur with a previous survey of wolves in Alaska (Zarnke and Ballard, 1987). Leptospires do not typically elicit a strong immune response (Leighton and Kuiken, 2001). In addition, antibody titers are often short-lived. Perhaps, natural exposure in wolves elicits similar low transient titers. Alternatively, exposure of wolves to leptospires might simply be rare. Clinical leptospirosis does not appear to be a significant source of morbidity or mortality for wolves.

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