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PREVALENCE OF ANTIBODIES FOR SELECTED CANINE PATHOGENS AMONG WOLVES (*CANIS LUPUS*) FROM THE ALASKA PENINSULA, USA

Dominique E. Watts[1,3] and Anna-Marie Benson[2]

[1] US Fish and Wildlife Service, Alaska Peninsula and Becharof National Wildlife Refuges, PO Box 277, King Salmon, Alaska 99613, USA
[2] US Fish and Wildlife Service, 101 12th Ave., Room 216, Fairbanks, Alaska 99708, USA
[3] Corresponding author (email: dom_watts@fws.gov)

ABSTRACT: We collected blood samples from wolves (*Canis lupus*) on the Alaska Peninsula, southwest Alaska, US, 2006–11 and tested sera for antibodies to canine adenovirus (CAV), canine coronavirus (CCV), canine distemper virus (CDV), canine herpesvirus (CHV), canine parainfluenza (CPI), canine parvovirus (CPV), *Neospora caninum*, and *Toxoplasma gondii*. Detected antibody prevalence was 90% for CAV, 28% for CCV, 12% for CDV, 93% for CHV, 0% for CPI, 20% for CPV, 0% for *N. caninum*, and 86% for *T. gondii*. Prevalence of CCV antibodies suggested a seasonal pattern with higher prevalence during spring (43%) than in fall (11%). Prevalence of CCV antibodies also declined during the 6-yr study with high prevalence during spring 2006–08 (80%, n=24) and low prevalence during spring 2009–11 (4%, n=24). Prevalence of *N. caninum* and *T. gondii* antibodies were highly variable in the study area during 2006–11. Results suggested that some pathogens might be enzootic on the Alaska Peninsula (e.g., CAV and CHV) while others may be epizootic (e.g., CCV, *N. caninum*, *T. gondii*).

Key words: Alaska Peninsula, *Canis lupus*, serology, wolves.

INTRODUCTION

Wolves (*Canis lupus*) may regulate or limit ungulate populations (Van Ballenberghe and Ballard 1994; Bergerud and Elliott 1998) and have a strong influence on sympatric carnivore populations (Thurber et al. 1992; Arjo and Pletscher 1999; Berger et al. 2008). Through such direct and indirect effects at lower trophic levels, wolves also shape the structure and function of ecologic communities and are important for the maintenance of biodiversity (Berger et al. 2001; Ripple et al. 2001; Ray et al. 2005; Beschta and Ripple 2009). Wolves might also serve as important vectors or reservoirs for pathogens such as *Neospora caninum* and *Brucella suis*, which can affect ungulates and other mammal populations (Neiland and Miller 1981; Kreeger 2003; Gondim et al. 2004; Dubey et al. 2011). Accordingly, an understanding of factors that influence wolf population dynamics is important to the management and conservation of wolves and a variety of other wildlife.

Wolf population dynamics are primarily influenced by prey availability, intraspecific competition, and human harvest (Keith 1983; Fuller 1989; Fuller et al. 2003). With the exception of sarcoptic mange (*Sarcoptes scabiei*), parasitic diseases are generally not considered significant mortality factors (Todd et al. 1981; Kreeger 2003). In contrast, infectious viral diseases can serve as important morbidity and mortality factors and may influence wolf population demography through direct mortality and diminished recruitment (Ballard and Krausman 1997; Hedrick et al. 2003; Kreeger 2003; Mech et al. 2008). Rabies virus can be a significant source of mortality for wolves in Alaska (Weiler et al. 1995; Ballard and Krausman 1997) and Ontario (Theberge et al. 1994). Canine distemper virus (CDV) and canine parvovirus (CPV) are also important mortality factors for wolf populations in Manitoba (Carbyn 1982), in Montana and British Columbia (Johnson et al. 1994), and in Minnesota (Mech and Goyal 1995). Canine parvovirus was a significant factor retarding recolonization and population increase for wolves in Minnesota (Mech et al. 2008). Such potential pathogens may also

have important implications for captive (Hedrick et al. 2003) and reintroduced wolf populations (Mech et al. 1986). Information from these and other studies suggest that viral diseases can have population-level implications for wolves.

Zarnke et al. (2004) reported the results of an extensive serosurvey of wolf populations in central and northern Alaska (US) and the Yukon Territory (Canada), but no data were available for wolves in southwest Alaska. We addressed this information gap by collecting serologic data for selected canine pathogens from wolves on the Alaska Peninsula and Unimak Island (Fig. 1) and by investigating potential relationships among host parameters and antibody prevalence.

MATERIALS AND METHODS

As part of a large-scale study of wolf ecology on the Alaska Peninsula (2006–14), we chemically immobilized wolves with 572 mg Telazol® (Fort Dodge Laboratories Inc., Fort Dodge, Iowa, USA) using aerial darting. We fitted wolves with GPS radio collars and estimated age by using tooth eruption and wear techniques (Gipson et al. 2000). Blood samples were collected from the cephalic vein using Vacutainer serum separator tubes (BD, Franklin Lakes, New Jersey, USA) and were cold-stored in the field. Samples were centrifuged <10 h after capture and sera were extracted and stored below –20 C until testing. Capture and sampling procedures were approved by the Alaska Department of Fish & Game Division of Wildlife Conservation Animal Care and Use Committee (Protocol 06-19).

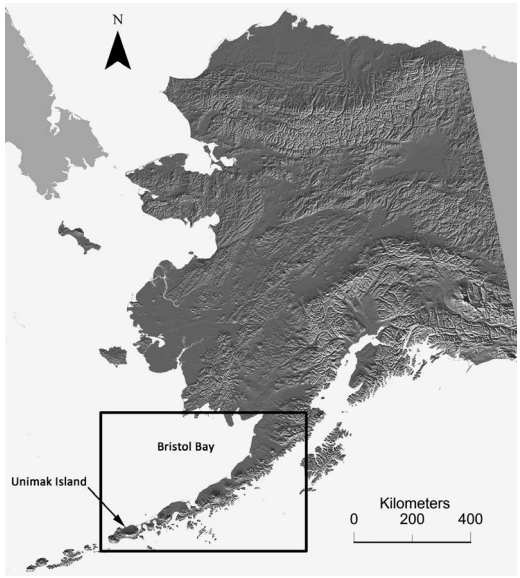

FIGURE 1. Study area (black rectangle) in southwest Alaska (USA) for serologic survey of selected canine pathogens during 2006–11. Serum samples were collected from wolves (*Canis lupus*) throughout the region (including Unimak Island) but primarily from the northern Alaska Peninsula.

Sera were analyzed for antibodies to CDV, canine adenovirus (CAV), canine coronavirus (CCV), canine herpesvirus (CHV), canine parainfluenza (CPI), CPV, *N. caninum*, and *Toxoplasma gondii* by Washington Animal Disease Diagnostic Laboratory, Pullman, Washington, USA. We considered samples with titers at or above pathogen-specific cutoff values to have had previous exposure to a pathogen (positive). All other samples were considered to have had no previous exposure (negative), though titers in some previously exposed individuals could have declined below cutoff values (false-negatives). Test methods and pathogen-specific positive cutoff values are listed in Table 1. Although maternally derived antibodies could have influenced results (false-positive titers), we assumed no influence from maternal antibodies because all pups were at least 5 mo old when sampled (Pollock and Carmichael 1982; Green et al. 1984). Samples from 39 wolves collected from the northern part of the study area during 2006–09 were provided for a statewide survey of *N. caninum* and *T. gondii* and results were reported by Stieve et al. (2010). To build on this work, 28 additional samples collected during 2010–11 were also tested for *N. caninum* and *T. gondii* (six of 28 were collected from recaptured individuals).

To examine factors that might influence the probability of detecting antibodies within the sampled wolf population, we fit generalized linear mixed models (Pinheiro and Bates 2002) to our binary (positive or negative) serologic test results using R (R Development Core Team 2012). These were essentially logistic regression models with the addition of a nested random effect to account for expected autocorrelation among individuals that were sampled twice (n=15) and among individuals within the same pack. By averaging over the distribution of the random effects, we obtained the mean response of the population (probability of detecting antibodies to a given pathogen), $\Pr(y_i=\text{Positive})$ characterized by the fixed effects. Candidate models took the form:

TABLE 1. Methodologies and positive cutoff-values used to detect pathogen exposure or infection. Tests were conducted at the Washington Animal Disease Diagnostic Laboratory, Pullman, Washington, USA, 2006–11.

Pathogen[a]	Method[b]	Positive cutoff
CDV	IFA (IgG only)	1:50
CAV	VN	1:4
CCV	IFA	1:25
CHV	VN	1:4
CPI	VN	1:4
CPV	IFA (IgG only)	1:25
N. caninum	IFA	1:50
T. gondii	IFA	1:64

[a] CDV = canine distemper virus; CAV = canine adenovirus; CCV = canine coronavirus; CHV = canine herpesvirus; CPI = canine parainfluenza; CPV = canine parvovirus; *N. caninum* = *Neospora caninum*; *T. gondii* = *Toxoplasma gondii*.

[b] IFA = indirect fluorescent antibody; IgG = immunoglobulin G; VN = virus neutralization.

$$\Pr(y_i = \text{Positive}) = \text{logit}^{-1}(\alpha_{jk[i]} + \beta_0 + \beta_1 \text{Age}_{[i]} + \beta_2 \text{Year}_{[i]} + \beta_3 \text{Sex}_{[i]} + \beta_4 \text{Season}_{[i]} + \varepsilon_{[i]}),$$

for i=1,. . .,n wolves, where the random intercepts $\alpha \sim N(\mu{=}0, \sigma 2\ \sigma^2_{jk})$ for j=1,. . .,n individuals nested in k packs $\varepsilon_{[i]} \sim N(0, \sigma^2)$. Age and Year were continuous variables and Sex (male or female) and Season (spring or fall) were categorical. All variables were centered for these analyses. Because no individuals were sampled twice for *T. gondii* antibodies, the random effects component of the model was modified such that the random effect was modeled as an intercept-only model with Pack as the random effect; this resulted in a difference in the degrees of freedom for these models.

A balanced model set comprised of 16 models was fit for each of the eight antibodies tested for. Models were compared using Akaike's information criterion for small sample sizes (AIC_c) (Burnham and Anderson 2002). We used model-averaging to account for model uncertainty using the natural-average method, where a parameter estimate for each predictor is averaged only over the models in which that predictor appears and is weighted by the summed weights of these models (Burnham and Anderson 2002; Bolker et al. 2009). We used 95% confidence intervals (CI) of model-averaged parameter estimates to evaluate which parameters influenced the probability of detecting antibodies to a given pathogen. We considered parameter estimates to be significant if CI did not overlap zero. The best model in each set was evaluated to make certain the models were not overdispersed and that the sum of the squared Pearson residuals were not different from a chi-squared distribution using program R and package lme4 (Bates et al. 2015).

RESULTS

We collected 100 samples from 85 individuals (46 male, 39 female) representing 15 packs from the Alaska Peninsula and Unimak Island (D.E.W. unpubl. data). Mean estimated age was ~2.5 yr (SD=2.3) but ranged 6 mo to 10 yr. Including all samples, detected prevalence of antibodies was 12% for CDV, 90% for CAV, 28% for CCV, 93% for CHV, and 20% for CPV. Prevalence did not change significantly if samples from recaptured individuals (n=15) were excluded (13% for CDV, 89% for CAV, 28% for CCV, 92% for CHV, and 19% for CPV). No wolves had detectable antibodies to CPI. A detailed summary of serologic test results is provided in Table 2. Because the study area was remote, prompt carcass collection was not practical and no direct evidence of morbidity or mortality due to infections with these pathogens was documented. One individual (W040805) was recaptured during 2011 and was emaciated and in poor physical condition. Female wolf W040805 died ~12 d postcapture and was serologically positive for CAV at >1:512, CHV at 1:16, and *T. gondii* at 1:1024 at capture.

For four of the eight pathogens (CAV, CHV, CPI, *N. caninum*), no model could be fit to the data because results were fundamentally homogenous (i.e., mostly positive or mostly negative). A detailed summary of model results and the model-averaged parameter estimates (95% CI) for the remaining four pathogens (CDV, CCV, CPV, *T. gondii*) is reported in Tables 3 and 4, respectively. The best model for predicting CDV antibody presence contained Season and Age (Table 3), but model-averaged parameter estimates showed no evidence that Age, Season, or Sex were useful predictors of CDV antibody

TABLE 2. Results of serologic survey for selected canine pathogens among wolves (*Canis lupus*) on the Alaska Peninsula, Alaska, USA, 2006–11. Results for *N. caninum* and *T. gondii* include only 2010–11 samples from the Alaska Peninsula. Results from the northern Alaska Peninsula during 2006–09 are reported by Stieve et al. (2010).

Year[a]	*n*	Prevalence (%)[b]							
		CDV	CAV	CCV	CHV	CPI	CPV	*N. caninum*	*T. gondii*
2006	12	8	100	17	100	0	33	—	—
2007	20	15	95	100	95	0	10	—	—
2008	12	8	83	33	92	0	42	—	—
2009	28	7	82	0	89	0	18	—	—
2010	19	16	95	10	95	0	16	0	95
2011	9	22	89	100	75	0	11	0	67
All	100	12	90	28	93	0	20	0	86

[a] Results for 2006 include samples collected during fall only (no samples were available for spring 2006). Number of samples collected during spring and fall sampling periods varied and were not necessarily uniform among years. Year and All include samples collected from recaptured individuals (n=15).

[b] CDV = canine distemper virus; CAV = canine adenovirus; CCV = canine coronavirus; CHV = canine herpesvirus; CPI = canine parainfluenza; CPV = canine parvovirus; *N. caninum* = *Neospora caninum*; *T. gondii* = *Toxoplasma gondii*.

presence (Table 4). The best model predicting CPV antibody presence contained Year and Age but several models were also within 2 AIC_c values of this model (Table 3). Model-averaged parameter estimates did, however, indicate a significant Age effect with the probability of CPV antibody detection increasing with age (Table 4). The best model predicting *T. gondii* presence contained only Year, but two other models were also within 2 AIC_c values of this model (Table 3). Model-averaged parameter estimates indicated a significant Year effect with the probability of detecting *T. gondii* antibodies decreasing during the 6-yr study (Table 4).

Prevalence of CCV antibodies was higher during the spring (43%) than during fall (11%). Prevalence appeared to vary considerably among years during both spring and fall but samples sizes were limited during some years (Table 5). In addition, three wolves sampled during December 2010 were negative, one sampled during May 2007 was positive (1:25), and two sampled during June 2007 were positive (1:25). High CCV titers occurred in both spring (four of 20 positive wolves) and fall samples (three of five positive wolves). Eight of 20 CPV antibody-positive wolves were concurrently positive for CCV antibodies. When prevalence of CCV antibodies is expected to be low (fall), two of 10 CPV antibody-positive wolves were concurrently positive for CCV antibodies. During spring, when CCV antibody prevalence is expected to highest, six of 10 wolves were concurrently positive for both antibodies. Model results indicated that Season and Year were the most important factors for predicting CCV antibodies (Table 3). Model-averaged parameter estimates also indicated a higher probability of CCV antibody detection among wolves sampled during spring than those sampled during fall (Table 4). Overall prevalence of CCV antibodies declined during our study with samples collected during spring dropping from 80% during 2007–08 to 4% during 2009–11 (Table 5). Models also showed a significant decreasing Year effect with the probability of CCV antibody detection decreasing during the 6-yr study.

DISCUSSION

Canine distemper virus is an important infectious agent among wild carnivores that may cause acute and subacute disease with high case fatality in canids, particularly among juvenile and immunosuppressed individuals (Appel 1987; Williams 2001; Kreeger 2003; Greene and Appel 2006). Prevalence of CDV

TABLE 3. Candidate models and selection results for Akaike's information criterion for small sample sizes (AIC_c) analyses for selected pathogens in wolves (*Canis lupus*) from the Alaska Peninsula, Alaska, USA, 2006–11. Best models are depicted in bold.

		Pathogen[c]								
		CDV		CCV		CPV		*T. gondii*		
Model[a]	K[b]	ΔAIC_c	Weight	ΔAIC_c	Weight	ΔAIC_c	Weight	**K**[c]	ΔAIC_c	Weight
SeasonYearSexAge	8	4.5	0.02	1.80	0.15	1.75	0.09	7	8.44	0.00
SeasonSexAge	7	2.16	0.05	35.05	0.00	2.39	0.07	6	9.04	0.00
YearSeasonAge	7	2.27	0.05	0.95	0.22	1.15	0.12	6	5.04	0.02
SeasonYearSex	7	4.64	0.02	0.51	0.28	7.04	0.01	6	5.15	0.02
YearSexAge	7	4.93	0.01	19.41	0.00	0.78	0.15	6	5.10	0.02
SeasonAge	6	**0.00**	0.16	32.79	0.00	2.28	0.07	5	5.86	0.02
SeasonSex	6	2.36	0.05	34.49	0.00	6.81	0.01	5	6.97	0.01
SeasonYear	6	2.59	0.04	**0.00**	0.36	5.97	0.01	5	2.34	0.09
SexAge	6	2.62	0.04	45.53	0.00	1.90	0.08	5	5.96	0.02
YearAge	6	2.84	0.04	17.80	0.00	**0.00**	0.22	5	1.95	0.11
YearSex	6	4.17	0.02	20.71	0.00	5.32	0.02	5	2.59	0.08
Season	5	0.38	0.13	32.37	0.00	6.08	0.01	4	4.14	0.04
Age	5	0.58	0.12	43.27	0.00	1.56	0.10	4	3.14	0.06
Sex	5	1.91	0.06	47.24	0.00	5.49	0.01	4	4.01	0.04
Year	5	2.23	0.05	19.51	0.00	4.17	0.03	4	**0.00**	0.30
Null	4	0.02	0.15	45.06	0.00	4.66	0.02	3	1.47	0.15

[a] Year and Age were modeled as continuous variables. Season (fall or spring) and Sex (male or female) were categorical variables.

[b] K = number of parameters. For CDV, CCV, and CPV, the random effects part of the model contributed 3 additional parameters (residual variance, intercept for Pack, and intercept for Individual nested within Pack). Individual was nested within Pack to account for the inclusion of samples from recaptured wolves (n=15). Because no data from recaptured wolves were available for *T. gondii*, random effects were modeled as an intercept-only model with Pack as the random effect, contributing 2 additional parameters to the model.

[c] CDV = canine distemper virus; CCN = canine coronavirus; CPV = canine parvovirus; *T. gondii* = *Toxoplasma gondii*.

TABLE 4. Model-averaged parameter estimates and 95% confidence intervals (CI) for selected pathogens in wolves (*Canis lupus*) from the Alaska Peninsula, Alaska, USA, 2006–11. Estimates that were considered significant (based on CI that did not overlap zero) are depicted in bold.

	Pathogen[a]											
	CDV			CCV			CPV			*T. gondii*		
		95% CI			95% CI			95% CI			95% CI	
Parameter	Estimate	Lower	Upper	Estimate	Lower	Upper	Estimate	Lower	Upper	Estimate	Lower	Upper
Intercept	1.13	1.04	1.22	1.28	1.21	1.35	1.18	1.06	1.31	0.86	0.73	0.99
Age	0.10	−0.03	0.24	0.09	−0.06	0.23	**0.18**	**0.04**	**0.33**	0.14	−0.13	0.42
Season (spring)	−0.08	−0.20	0.04	**0.34**	0.18	0.50	−0.06	−0.18	0.06	−0.04	−0.32	0.23
Sex (male)	−0.03	−0.17	0.10	−0.09	−0.24	0.05	0.09	−0.04	0.23	−0.07	−0.33	0.20
Year	0.01	−0.11	0.13	**−0.46**	−0.60	−0.32	−0.12	−0.24	0.01	**−0.30**	**−0.56**	**−0.02**

[a] CDV = canine distemper virus; CCV = canine coronavirus; CPV = canine parvovirus; *T. gondii* = *Toxoplasma gondii*.

TABLE 5. Seasonal prevalence (no. positive/no. tested) of antibodies to canine coronavirus in wolves (*Canis lupus*) from the Alaska Peninsula, Alaska, USA, during 2006–11. No samples were available for spring 2006.

	Antibody prevalence (%)					
	2006	2007	2008	2009	2010	2011
Fall	17 (2/12)	100 (2/2)	0 (0/3)	0 (0/16)	9 (1/11)	0 (0/2)
Spring	—	100 (15/15)	44 (4/9)	0 (0/12)	20 (1/5)	0 (0/7)
Total	17	100	33	0	13	0

antibodies in Alaskan wolf populations is typically ≤12% but may range from 0–64% (Zarnke and Ballard 1987; Zarnke et al. 2004). Overall CDV antibody prevalence on the Alaska Peninsula was relatively low (12%). Given these results and information from previous studies, we speculate that exposure to CDV was low in the study area during 2006–11 and that CDV may be enzootic on the Alaska Peninsula.

Canine adenovirus can cause infectious canine hepatitis in infected canids with clinical symptoms including vomiting, diarrhea, depression, seizures, and death in severely affected individuals (Greene 1998). Previous studies suggest that CAV antibody prevalence may range from 13–95% and that CAV is enzootic among wolves throughout much of interior Alaska and Canada (Choquette and Kuyt 1974; Stephenson et al. 1982; Zarnke and Ballard 1987). High antibody prevalence (90%) among wolves in this study suggests that CAV may also be enzootic on the Alaska Peninsula. Causes for the high prevalence detected among wolves in this study are unknown but data are suggestive of high transmission rates or high antibody persistence (or both). Transmission generally occurs through direct contact with saliva, urine, or feces (Cabasso 1981). Greene (1998) suggested that CAV can remain stable in the environment for long periods under cold conditions, which might partially explain the high prevalence on the Alaska Peninsula. Sympatric carnivores could also serve as reservoirs that perpetuate high CAV prevalence in wolves (Zarnke et al. 1997; Dalerum et al. 2005; Almberg et al. 2009).

Canine coronavirus is a highly contagious intestinal virus that typically causes outbreaks of enteritis (Tennant et al. 1993). The primary source of infection is contact with feces from infected individuals that may shed the virus periodically over long periods (Pollock et al. 1980; McCaw and Hoskins 2006). Zarnke et al. (2001) reported a distinct seasonal pattern in CCV antibody prevalence among wolves from interior Alaska and suggested that: 1) serum CCV antibody decay may be rapid, 2) re-exposure during summer is rare, and 3) peak transmission occurs during February–March because all high titers were found in spring samples. Alaska Peninsula wolves showed a similar seasonal pattern with higher CCV antibody prevalence during spring compared to fall; however, prevalence varied considerably among years during both seasons (Table 5). High CCV antibody titers also occurred in both spring and fall samples, which could suggest greater variation in transmission rates on the Alaska Peninsula compared to interior populations. Declines in overall prevalence and model results suggested high variation in transmission rates or that CCV could be epizootic in the study area. Further, because wolf abundance appeared to be relatively stable during our study (D.E.W. unpubl. data), the observed declines in prevalence could also indicate that conspecifics are not the primary source of infection. Sympatric carnivores could also serve as reservoirs for CCV (Foreyt and Evermann 1985; Dalerum et al. 2005), and changes in their populations might also influence transmission rates. Additionally, CCV is environmentally labile and variation in CCV antibody

prevalence could also be related to varying environmental conditions during our study.

Canine herpesvirus is a typical alphaherpesvirus found in canids (Carmichael and Greene 1998). Although infection is typically asymptomatic in adults, clinical signs include abortion, respiratory infection, ataxia, anorexia, vomiting, and depression (Carmichael and Greene 1998). The prevalence and effects of CHV in wild canid populations and the pathogenicity of CHV remain poorly understood. Carmichael and Greene (1998) suggested that CHV infection may cause acute mortality or neurologic disorders in domestic dog pups that are infected in utero or within 3 wk of parturition if lacking maternally derived immunity. Evermann et al. (1980) also reported high mortality rates among CHV-infected coyote (*Canis latrans*) pups, which suggest that CHV might also have population-level implications for wolves. The high antibody prevalence (93%) we observed suggests that CHV could be enzootic on the Alaska Peninsula.

Canine parvovirus is a highly contagious pathogen and is primarily transmitted through contact with infected feces (Pollock et al. 1980; McCaw and Hoskins 2006). Canine parvovirus may cause enteric disease in canids and is often fatal in young or immunosuppressed individuals (Mech et al. 1986; McCaw and Hoskins 2006). For wolf packs in Montana and British Columbia in which pups died, adults exhibited high CPV antibody titers, suggesting that CPV may have been an important factor limiting pup survival and recruitment (Johnson et al. 1994). Mech et al. (2008) also reported that CPV was an important factor retarding population growth and expansion among wolves in Minnesota. Previous studies suggest that CPV antibody prevalence is commonly $\geq$30% and that CPV may be enzootic throughout much of interior Alaska (Zarnke and Ballard 1987; Johnson et al. 1994; Zarnke et al. 2004; Mech et al. 2008). Prevalence of CPV antibodies on the Alaska Peninsula (20%) was slightly lower than reported for other regions of Alaska. Although the effects of CPV in Alaskan wolf populations are currently unknown, previous work suggests that CPV infection is probably not a major factor limiting wolf abundance in the state (Zarnke et al. 2004). Relatively low prevalence and model results herein suggest that CPV may be enzootic on the Alaska Peninsula and could potentially have population-level effects in the region. Further, pathogens such as CPV may not significantly affect morbidity or mortality alone, but the synergistic effects of concurrent infections (e.g., increased physiologic stress and immunosuppression) may have important consequences. Concurrent infections of CPV and CCV may cause acute enteritis in other canids such as coyotes (Evermann et al. 1980). Eight of 20 CPV antibody-positive wolves in this study were concurrently positive for CCV antibodies.

Neospora caninum and *T. gondii* are important apicomplexan parasites that infect a wide range of hosts and may cause neurologic diseases, abortion, birth defects, encephalitis, blindness, or even death in immunosuppressed individuals (Dubey et al. 1999; Zarnke et al. 2000; Labelle et al. 2001; Dubey 2003; Dubey et al. 2003). Canids such as coyotes and wolves are definitive hosts for *N. caninum* (McAllister et al. 1998; Gondim et al. 2004; Dubey et al. 2011). Stieve et al. (2010) reported overall antibody prevalence of 20–29% for *N. caninum* among wolves from the northern part of the study area during 2006–09. Interestingly, none of the additional samples collected during 2010–11 (n=28) were positive for *N. caninum* antibodies, including those from six recaptured individuals that were antibody positive when previously sampled (Stieve et al. 2010). Though data over only 6 yr are presented here, such variation might suggest that *N. caninum* is epizootic among wolves in the region.

Zarnke et al. (2000) reported that *T. gondii* antibody prevalence was 9% among 125 wolves sampled elsewhere in Alaska. Stieve et al. (2010) reported overall prevalence of 46% for *T. gondii* antibodies among 39 wolves from the northern Alaska Peninsula sampled during 2006–09. In our study (2010–11), *T. gondii* antibody prevalence was nearly double (86%) that reported by Stieve et al. (2010) and

was considerably higher than has been reported for Alaskan wolves. Stieve et al. (2010) suggested that wolves on the Alaska Peninsula contract *T. gondii* by consuming infected marine mammals (see also Watts et al. 2010). Consumption of brown bear carcasses is also common and represents another potential source of exposure (D.E.W. pers. obs.). However, felids are the only known definitive hosts of *T. gondii* and wolves may become infected by consuming infected lynx or their feces. The GPS location data for wolves reported in Stieve et al. (2010) showed that comparatively few (~22% of *T. gondii* antibody positive wolves, n=18) had access to the coast, but most (~78%) lived inland where lynx are more abundant (D.E.W. pers. obs.). Based on this information and the relatively high prevalence among wolves from interior Alaska (Zarnke et al. 2001; Stieve et al. 2010; Simon et al. 2013), we speculate that lynx could be the primary source of *T. gondii* infection among wolves on the Alaska Peninsula. If this holds true, one potential explanation for the variation in *T. gondii* antibody prevalence observed during 2006–11 could be related to the cyclical population dynamics of lynx (Elton and Nicholson 1942; Stieve et al. 2010).

The presence of antibodies to pathogens indicates exposure but does not confirm disease in individuals or populations. Similarly, the absence of positive serologic test results (e.g., for CPI in this study) does not necessarily confirm that wolves have not been exposed because infected individuals may not maintain detectable antibody levels for long periods. Few studies document direct mortality related to disease in wild populations, and the population-level effects of these pathogens are poorly understood. Results of this study show that wolves on the Alaska Peninsula and Unimak Island have been exposed to several canine pathogens that are known to cause significant morbidity and mortality in wild canid populations. Consistent with previous work suggesting that the persistence of these pathogens does not necessarily limit wolf populations (Brand et al. 1995; Mech and Goyal 1995; Kreeger 2003; Zarnke et al. 2004), preliminary estimates of wolf population densities (~6.5–7.5 wolves/1000 km^2) and observed recruitment among packs (D.E.W. unpubl. data) suggest that wolves generally persisted at densities typical of Alaskan wolf populations and exhibited typical recruitment rates (Fuller et al. 2003). The presence of these pathogens does, however, suggest that wolf population dynamics in the region could be periodically influenced by these pathogens, particularly where they are epizootic. Although long-term datasets are necessary to fully comprehend pathogen endemicity, results suggested that some pathogens might be enzootic in the study area (e.g., CAV and CHV) while others could be epizootic (e.g., CCV, *N. caninum*, *T. gondii*).

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