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Serologic Investigations of Canine Parvovirus and Canine Distemper in Relation to Wolf (*Canis lupus*) Pup Mortalities

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ABSTRACT: Twenty-one serum samples from 18 wolves (*Canis lupus*) were collected from 1985 to 1990 from northwestern Montana (USA) and southeastern British Columbia, Canada, and evaluated for antibodies to canine parvovirus (CPV), canine distemper (CD), infectious canine hepatitis, and Lyme disease; we found prevalences of 13 (65%) of 19, five (29%) of 17, seven (36%) of 19, and 0 of 20 wolves for these diseases, respectively. Pups died or disappeared in three of the eight packs studied. In these three packs, adult pack members had CPV titers ≥1,600 or CD titers ≥1,250. In packs that successfully raised pups, CPV and CD titers were low. We propose that CPV or CD may have caused some pup mortalities.

Key words: Serology, wolves, *Canis lupus*, canine parvovirus, canine distemper.

Diseases of concern in wolf populations include canine parvovirus (CPV), canine distemper (CD), infectious canine hepatitis (ICH) (Zarnke and Ballard, 1987), and Lyme disease (LD) (Thieking et al., 1992). All four diseases occur in both domestic and wild canids.

In 1983, CPV killed 11 of 12 wolf pups and yearlings in a captive wolf colony just north of Minneapolis, Minnesota (USA) (Mech and Fritts, 1987). The actual role of CPV in free-ranging wolf populations is not well-documented; however, small populations could be significantly affected by CPV mortality (Mech and Goyal, 1993). Canine distemper also may cause mortality in canids and could potentially have a negative impact on recolonizing populations of wolves.

Serologic surveys of wolf populations have been conducted in Minnesota (Goyal et al., 1986), Wisconsin (USA) (Thieking et al., 1992), Alaska (USA) (Zarnke and Ballard, 1987), and Northwest Territories (Canada) (Choquette and Kuyt, 1974). Except for Mech and Goyal (1993), previous investigators have not compared antibody prevalence in wolves to general population trends or to specific observations of neonatal mortality. Our objectives were to determine the prevalences of serum antibodies to CPV, CD, ICH, and LD in a wolf population centered in northwestern Montana (USA) and southeastern British Columbia (Canada) (48°40′ to 49°20′N, 114°00′ to 114°30′W), and to determine whether CPV or CD antigenic exposures correlated with pup mortalities.

The Glacier National Park (GNP), Montana, wolf population has been intensively studied since the arrival of a lone wolf in 1979 to the 1991 population of approximately 30 wolves (Ream et al., 1991). Wolves were captured, chemically immobilized, radio-collared, and pup survival was studied as described by Ream et al. (1991). Blood samples were collected from 18 wolves (21 captures) and between 1985 and 1990, which represented 23% of the population. Samples were cooled and serum was extracted within 24 hr of collection and frozen at −20 C until serologically analyzed.

Serum samples from wolves trapped from 1985 to 1989 were analyzed for CPV and LD at the Department of Diagnostic Investigations, College of Veterinary Medicine, St. Paul, Minnesota. Serum was tested for CPV antibodies using the hemagglutination inhibition test (Carmichael et al., 1980). Titers ≥256 were considered positive for CPV. Sera were tested for LD with the indirect fluorescent antibody test (IFA) (Thieking et al., 1992) and were considered positive if titers were ≥100.

Wolf serum samples from 1985 to 1989

TABLE 1. Serum antibody titers for canine parvovirus (CPV) and canine distemper (CD) in relation to pup losses from unknown causes by pack and year from a wolf (*Canis lupus*) population of Glacier National Park, Montana, and southeastern British Columbia, Canada, 1985 to 1990.

			CPV			CD		
Year studied	Pack	Pup losses[a]	Number of wolves sampled	Number positive	Range of positive titers	Number of wolves sampled	Number positive	Range of positive titers
1985	Magic	No	3	0	N/A[b]	3	1	25
1986	Magic	Yes	1	1	2,048	0	N/A	N/A
1987	Camas	No	4	2	256–1,024	4	1	25
1989	Camas	Yes	3	3	2,048–4,096	3	3	1,250–6,250
1989	Headwaters	No	2	1	512	2	0	N/A
1990	Camas—south[c]	No	1	1	100	0	N/A	N/A
1990	Camas—north[c]	No	2	1	400	1	0	N/A
1990	Headwaters	Yes	3	3	600–1,600	3	0	N/A

[a] Pup losses due to unknown causes.
[b] N/A = Not applicable.
[c] In 1990, Camas pack divided into two packs: Camas—north and Camas—south.

were tested for CD and ICH antibodies; and 1990 samples were tested for CPV, LD, CD, and ICH antibodies at Specialized Assays (Nashville, Tennessee, 37202 USA) by IFA (Rose et al., 1992). Titers ≥25 were considered positive for CPV, CD, and ICH. Titers ≥256 were considered positive for LD. Differences in prevalence between sexes were analyzed with the Fisher Exact Test (Sokal and Rohlf, 1981).

The prevalence of CPV antibodies in GNP wolves was 13 (65%) of 20 compared to 75% in Minnesota wolves (Goyal et al., 1986) and 31% in Alaska wolves (Zarnke and Ballard, 1987). Titers for CD were positive in five (29%) of 17 samples compared to 12% among Alaska wolves (Zarnke and Ballard, 1987) and 2% among Canadian wolves (Choquette and Kuyt, 1974). Antibodies against ICH occurred in seven (36%) of 19 samples compared to 95% (Stephenson et al., 1982) and 81% (Zarnke and Ballard, 1987) in Alaska wolf populations, and 13% in Canadian wolf populations (Choquette and Kuyt, 1974). No evidence of LD was found, compared to a prevalence of 3% in wolves from Minnesota and Wisconsin (Thieking et al., 1992). Thus CPV, CD, and ICH appeared to be enzootic to the GNP wolf population, as in most other wolf populations studied. No sex-specific differences in antibody prevalences of any of the diseases tested were present ($P > 0.05$).

Only one of six wolf pups tested positive for CPV, and a different pup tested positive for CD. Presumably, 5-mo-old wolf pups have immunologic capabilities similar to domestic dogs which develop a protective humoral response against CPV as early as 8-wk of age (Swango, 1983). Wolf pups generally are weaned by 8 to 10 wk of age (Mech, 1970). Therefore, maternal antibodies should be negligible at 5 mo of age. We assumed that because no measurable antibodies were detected in most 5-mo-old pups in our study that they had not been exposed to CPV, CD, ICH, or LD. Unfortunately, we were unable to capture pups from packs that lost pups.

In 1986, two of five pups from the Magic Pack disappeared by October; the causes of their disappearance was unknown. Canine parvovirus titer from an adult member of the pack sampled in 1986 was relatively high (2,048) (Table 1). In 1989, the Camas Pack was unsuccessful in raising any young. Two carcasses of 2-wk-old pups were recovered at the den site, but both were too decomposed to determine cause of death. Adult members of the pack sampled in 1989 had CPV titers ≥2,048 or CD titers ≥1,250 (Table 1). In 1990, a maximum of two Headwaters Pack pups sur-

vived to 6 mo. Canine parvovirus titers in two wolves sampled from this pack in 1990 also were high (1,600) (Table 1). Overall, adult pack members had CPV titers ≥1,600 or CD titers ≥1,250 in the three packs which experienced pup mortalities. In wolf packs that successfully raised wolf pups, CPV and CD titers were low. We considered CPV titers ≥1,600 and CD titers ≥1,250 as high titers.

Close physical contact, characteristic of wolf behavior (Mech, 1970), greatly facilitates disease transmission among pack members. Therefore, even though only a few adults from each pack were captured and sampled, the small sample sizes may adequately represent antigenic exposure of the entire pack. Seroconversions of Wolf 8551 to parvovirus and Wolf 8756 to canine distemper provide temporal evidence of antigenic exposure. Pup losses occurred within periods when both wolves developed antibody titers. Based on these high titers and seroconversions, we propose that CPV or CD may have played a role in pup mortalities in the 1986 Magic Pack, 1989 s Pack, and 1990 Headwaters Pack. We did not find any relationship between ICH titers and pup mortalities.

Because serology does not prove the antigen is actually present, stronger evidence is necessary to prove CPV as a mortality factor for wolf pups. Stronger evidence could be acquired by evaluating fecal samples from dens for antigen with negative contrast electron microscopy (Muneer et al., 1988) or hemagglutination (Carmichael et al., 1980). Fecal samples testing positive for parvovirus provide additional evidence; however, necropsies of pups are necessary to prove actual disease is present.

If CPV or CD infections contribute to pup mortality, such infections could significantly hinder natural recolonization for this endangered species. Therefore, it would be prudent for agencies monitoring recovering wolf populations to monitor for CPV and CD exposures or infections in the wolves and other potential carriers. Since canine parvovirus vaccines have been used safely in captive gray wolves and experimental challenges suggest modified-live vaccines against parvovirus are effective as well as safe in gray wolves (J. R. Zuba, pers. commun.), vaccinations could be an additional preventative measure for translocated wolves, providing basic immunoprophylaxis principles are considered.

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