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Explicate segment impacts of canine parvovirus on a Minnesota wolf populace

Brann KN[1,2]* and Holifield Nelson[3]

[1]U.S. Geological Survey, Northern Prairie Wildlife Research Center, 8711 – 37th St. SE, Jamestown, ND 58401-7317, USA.
[2]The Raptor Center, 1920 Fitch Ave., University of Minnesota, St. Paul, Minnesota 55108, USA.
[2]College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota 55108, USA.

Abstract

We examined 35 years of relationships among wolf (*Canis lupus*) pup survival, population change and canine parvovirus (CPV) seroprevalence in northeastern Minnesota to determine when CPV exerted its strongest effects. Using correlation analysis of data from five periods of 7-years each from 1973 through 2007, we learned that the strongest effect of CPV on pup survival (r = -0.73) and on wolf population change (*r* = -0.92) was during 1987 to 1993. After that, little effect was documented despite a mean CPV seroprevalence from 1994 of 2007 of 70.8% compared with 52.6% during 1987 to 1993. We conclude that after CPV became endemic and produced its peak effect on the study population, that population developed enough immunity to withstand the disease.

Keywords: Canine parvovirus, *Canis lupus,* CPV, demography, population, survival, wolf

INTRODUCTION

Based on correlations between canine parvovirus (CPV) seroprevalence and wolf-population data since 1973, Mech et al. (2008) found that CPV affected wolf-population change in the central Superior National Forest (SNF) of Minnesota and reduced pup survival there. The study provided a 30-year overview of the relationships between CPV and the wolf population. The implication of the study and of earlier phases (Mech and Goyal, 1995) was that CPV had affected the rate of population change ever since becoming endemic in the early 1980s. However, in an attempt to parse out more specific information on CPV influence, we learned that the population effects of the disease changed throughout the period of infection. The present study seeks to determine the precise period when CPV effects were greatest and relate that to pup survival and population change throughout the (now) 35 years of the CPV-wolf relationship.

STUDY AREA

Our study area was a 2,060 km^2 part of the SNF northeast of Ely, Minnesota centered around 48° N latitude and 91° 15' W longitude and was described in more detail by Mech and Goyal (1995). The wolf population there is part of the much larger surrounding Minnesota wolf population and is the southernmost extension of the Canadian wolf population, and it has never been exterminated. The main prey of wolves there is white-tailed deer (*Odocoileus virginianus*).

METHODS

We live-trapped wolves (Mech, 1974) yearly from May to October or November 1973 through 2004 in the study area and the immediately adjacent area, distinguished wolf pups from adults by the presence of deciduous canine teeth (Van Ballenberghe and Mech, 1975), and collected blood from each wolf. We used the percent pups in our annual capture as an index to pup survival; that is, we assumed that proportion of pups in our annual capture was reasonably representative of the proportion surviving. We radio-collared most of the wolves (Mech, 1974) and located them from

aircraft. We aerially observed radio-tagged wolves and their packmates throughout each winter and counted all members of each pack as detailed by Mech (2009).

We determined antibodies to CPV by the hemagglutination inhibition test (Carmichael et al., 1980) and considered titers of ≥ 1:256 as being positive (Mech et al., 2008). We considered the percentage of wolves positive on their first capture as the seroprevalence for that year (Goyal et al., 1986; Mech et al., 1986). Very few animals were recaptured each year.

We used correlation analyses to relate trends in annual seroprevalence with percentage of pups caught and percent annual changes in the wolf population, but in the present study, we sought to better define different phases of the relationship between CPV and the wolf population. We conducted tests on the data (Table 1) separated into five 7-year periods: 1973 to 1979, 1980 to 1986, 1987 to 1993, 1994 to 2000, 2001 to 2007, based on the 7-year, CPV-seroprevalence periodicity found by Mech et al. (2008). Although the data could have been parsed into any number of arbitrary periods, we felt that basing the parsing on the biological periodicity of CPV seroprevalence was an objective approach. We used a t test to compare means.

RESULTS AND DISCUSSION

The evidence that CPV affected the percentage of pups or caused a change in the SNF population during 1973 to 1979 was slight (Table 2). From 1980 through 1986, the relationship between CPV seroprevalence and percent change in the population remained low, but the relationship with percent pups became substantial (Table 2). By 1987 to 1993, the relationship (r) between CPV seroprevalence and percent pups was -0.73 ($P = 0.06$) and between CPV seroprevalence and percent population change was -0.92 ($P < 0.01$) (Table 2), similar to the findings of Mech and Goyal (1995). Correlation coefficients were also much stronger for this period than before or after (Table 2). After 1993 there was no evidence of any relationship between CPV seroprevalence and percent wolf-population change (Table 2). Evidence for CPV seroprevalence and pup survival after 1993 was slight (Table 2).

With pup survival and percent population change, most of the periods showed some positive, although not statistically significant, relationships. However, when the sample was larger for a longer period (1984 to 2004), the relationship although weak ($r = 0.47$) was significant ($P = 0.03$) (Mech et al., 2008). Nevertheless from 1994 to 2007, there was no relationship ($r = 0.2$; $P = 0.49$).

Wolf-population dynamics are affected by births, recruitment, survival, dispersal and immigration (Fuller et al., 2003). Pup recruitment is especially important because wolf litter sizes are high, averaging six (Mech, 1970). CPV affects pup survival which affects recruitment and later dispersal. The interaction of the demographic variables adjusts annual wolf population size to the local food supply.

One of the most effective buffering mechanisms is dispersal. The rate and age at which maturing wolves disperse greatly influences the population size for a given year (Fuller et al., 2003). If food supply increases, potential dispersers remain longer, and the population increases (Mech et al., 1998). The number of dispersers depends partly on the survival rate of pups born 1 to 3 years earlier. High pup survival means potentially high future dispersal and vice versa. As with our previous studies, we were unable to monitor actual neonate pup survival, so our conclusions rely on correlations and thus are subject to the limitations of that approach, similar to the studies of Wydeven et al. (1995) and Peterson et al. (1998).

Thus, when CPV affects pup survival, it can variably influence population size. CPV does not necessarily kill all members of a litter or cohort (Eugster et al., 1978; Pollock and Carmichael, 1982). Low pup survival leads to low recruitment, but if food supply is adequate, the population can remain stable or increase merely through lowered dispersal plus recruitment of any surviving pups. Therefore, CPV could kill pups without necessarily changing a population level. Conversely even if pup survival is high, increased dispersal and high mortality could reduce a population.

In our study, the statistical link between CPV seroprevalence and both annual pup survival and annual population change during 1987 to 1993 was strong, although the net population change was only -7%. However, after 1993, the relationship with survival was less, and there was no evidence of the disease affecting annual population changes. At least partly because of this fact, the population increased 47% between 1994 and 2007 (Mech, 2009). This increase took place despite a mean CPV seroprevalence from 1994 to 2007 (71%) that was higher ($P = 0.02$) than during 1987 to 1993 (53%).

Therefore, it appears that CPV strongly affected our pup survival negatively from the early 1980s until about 1993 and then waned. From 1987 through 1993, this strong pup-survival effect then affected annual population change. Later (1994 to 2007) the CPV effect on pup survival was less or non-existent, and the population increased.

The unanswered question is why CPV affected the population after becoming endemic but not for longer than about 7 years despite high exposure. This phenomenon may be related to immunity developing in the first cohorts of wolves exposed to the disease and then persisting as “herd immunity.” Although wolves can live longer than 7 years most die by that age (Mech, 2006).

Another hypothesis would be that while CPV was affecting population change, wolf food supply was substandard. Later, that improved and may have helped CPV-infected wolves to better overcome the disease. However, the opposite appears to be the case. Our main wolf prey, white-tailed deer, was more abundant from about 1985 through 1992 than before or after (Mech, 2009.

Table 1. Data related to canine parvovirus (CPV) effect on measures of wolf population change in the central superior national forest of Northeastern Minnesota[1].

Year	N[2]	Percent CPV positive	Percent pups in capture	Wolf population	Percent change in wolf population
1973	9	56	67	65	-7
1974	11	45	44	44	-32
1975	16	35	41	56	27
1976	15	40	50	45	-20
1977	18	11	67	50	11
1978	12	8	10	46	-8
1979	15	20	44	54	17
1980	23	73	48	48	-11
1981	19	53	43	47	-2
1982	18	44	53	50	6
1983	14	36	50	35	-30
1984	14	21	70	54	54
1985	18	44	45	47	-13
1986	11	64	25	48	2
1987	25	36	28	59	23
1988	21	14	42	79	34
1989	17	100	11	51	-35
1990	30	60	20	56	10
1991	26	46	46	53	-5
1992	17	47	47	55	3
1993	20	65	25	55	0
1994	14	57	7	55	0
1995	16	56	44	69	25
1996	11	73	6	56	-19
1997	28	64	40	55	-2
1998	11	55	40	50	-9
1999	9	67	25	44	-12
2000	18	72	28	52	18
2001	9	78	0	53	2
2002	8	80	10	58	9
2003	14	64	7	62	7
2004	11	82	21	74	19
2005	15	83	14	81	9
2006	13	60	15	81	0
2007	17	100	0	84	4

[1] Data from 1973 to 2004 are from Mech et al. (2008). [2] N = number of wolves sampled.

The population effects of CPV in our study parallel those in Wisconsin and on Isle Royale, Michigan. Early in the disease's history, it apparently attenuated Wisconsin's developing wolf population but after a few years had little effect (Wydeven et al., 1995). Similarly, on Isle Royale, CPV apparently suppressed the small population just during the 1980s (Peterson et al., 1998). Both these populations were small and isolated. Hence the finding that CPV only affected them for short periods does not necessarily mean that in a larger population it would have the same effect. Nevertheless, our results indicate that CPV also had only a temporary population effect despite our study population being part of the entire wolf range from Minnesota through most of Canada. By parsing our 35 years of CPV data, this study greatly details the changing population effect of the disease over time in a free-ranging wolf population and sheds new light on the natural history of this relatively new disease.

Table 2. Correlations (r) and probability (parentheses) among annual CPV seroprevalence, annual percent-pup index and annual percent wolf population change within each period in the central Superior National Forest, 1973 to 2007.

Period	CPV vs percent pups	Percent pups vs population change[1]	Percent CPV vs. population change	Net population change[2]
1973-1979	-0.45 (0.31)	0.10 (0.87)	-0.40 (0.37)	-0.25[3]
1980-1986	-0.73 (0.06)	0.53 (0.20)	-0.48 (0.27)	0.0
1987-1993	-0.73 (0.06)	0.45 (0.29)	-0.92 (< 0.01)	-0.07
1994-2000	-0.42 (0.35)[4]	0.48 (0.28)	-0.25 (0.59)	+0.05
2001-2007	-0.33 (0.47)[4]	0.62 (0.13)	+0.25 (0.60)	+0.58

[1]Although none of these relationships was significant for the given periods, *r* for 1984-2004 was 0.47, and P was 0.03 (Mech et al., 2008). [2] Net population changes from beginning of period to end of period; this shows the net difference for each period, but because it was a 7-year integrated figure, it could not be used in the correlation analyses.[3] Decline attributable to drastic deer decline.[4] When data for these periods were combined, r = -0.57 (P = 0.04).

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