EFFECTS OF CANINE PARVOVIRUS ON GRAY WOLVES IN MINNESOTA

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Abstract: Long-term effects of disease on wild animal population demography is not well documented. We studied a gray wolf (*Canis lupus*) population in a 2,060-km² area of Minnesota for 15 years to determine its response to canine parvovirus (CPV). The CPV had little effect ($P > 0.05$) on wolf population size while epizootic during 1979–83. However, after CPV became enzootic, percentage of pups captured during summer–fall 1984–93 and changes in subsequent winter wolf numbers were each inversely related to the serological prevalence of CPV in wolves captured during July–November ($r^2 = 0.39$ and 0.72, $P = 0.05$ and < 0.01, respectively). The CPV antibody prevalence in adult wolves increased to 87% in 1993 ($r^2 = 0.28$, $P = 0.05$). However, because population level remained stable, CPV-induced mortality appeared to compensate for other mortality factors such as starvation. We predict that the winter wolf population will decline when CPV prevalence in adults consistently exceeds 76%. The CPV may become important in limiting wolf populations.

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Wolf densities reflect the densities of their primary prey (Keith 1983, Fuller 1989, Dale et al. 1994), whereas changes in wolf populations tend to parallel changes in numbers of their vulnerable prey (Packard and Mech 1980, Peterson and Page 1988). During 1968–79, wolf population changes in our Minnesota study area generally followed changes in white-tailed deer (*Odocoileus virginianus*) numbers (Mech 1986, Nelson and Mech 1986). However, we documented serological evidence of CPV, a disease of domestic dogs and coyotes (*C. latrans*) (Tho-

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Table 1. Wolf population characteristics in the central Superior National Forest, Minnesota, and results of serologic tests of wolves for canine parvovirus (CPV).

Year	Proportion[a] of pups captured, May–Nov	Total winter wolf no.[b]	% change[c] in wolf no.	Proportion positive for CPV[d] Total	Nonpups[e]
1979	7/16	54	+17	3/15	3/9
1980	11/23	48	−11	15/22	7/12
1981	9/21	47	−2	10/19	7/12
1982	10/19	50	+6	8/18	5/8
1983	8/16	35	−30	5/14	5/7
1984	16/23	54	+54	3/13	1/3
1985	10/22	47	−13	8/18	8/8
1986	3/12	48	+2	5/9	5/7
1987	9/32	59	+23	9/25	7/18
1988	11/26	79	+34	3/20	3/9
1989	2/19	51	−35	18/19	17/17
1990	8/41	56	+10	18/30	18/22
1991	12/26	53	−5	12/26	12/14
1992	9/19	55	+3	8/17	8/10
1993	5/20	55	0	13/20	13/15

[a] No. of pups caught divided by no. adults and pups caught/yr.
[b] Mech (1986, unpubl. data).
[c] From previous yr.
[d] Summer and fall populations.
[e] Wolves ≥1 yr old.

mas et al. 1984), in a wolf population and found it to be fatal (Mech et al. 1986, unpubl. data). In Wisconsin (Wydeven et al. 1994) and Montana (Johnson et al. 1994), CPV also appeared to cause wolf pup mortality. During 1979–90, changes in the wolf population we studied were related to CPV prevalence (Mech and Goyal 1993, 1994). Evidence suggests that the effect of CPV on wolves is increasing.

The availability of 3 additional years of data for the wolf population we studied, the discovery of a 10-month-old wild wolf that succumbed to CPV in 1992, and the development of a new hypothesis about the changing influence of CPV prompted us to reanalyze our data. This appears to be the first study that traces the long-term course, from inception, of a disease on a wild animal population.

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STUDY AREA

We conducted the study during 1979–93 in the east-central Superior National Forest of northeastern Minnesota (48°N, 92°W). Topography of the area varied from large stretches of swamps to rocky ridges; altitudes ranged from 325 to 700 m above sea level. Winter temperatures <−35 C were not unusual, and snow depths (usually from mid-Nov through mid-Apr) generally ranged from 50 to 75 cm exclusive of drifts. Summer temperatures rarely exceeded 35 C. Descriptions of forest vegetation were presented by Ohmann and Ream (1969).

The Minnesota wolf population is contiguous with the Canadian wolf population and has not been extirpated. The main prey of the wolf was white-tailed deer, but moose (*Alces alces*) and beaver (*Castor canadensis*) also were taken (Mech, unpubl. data). Highways and towns occurred within the study area, and domestic dogs were common around residences. Dogs were sometimes killed by wolves (Fritts and Paul 1989).

METHODS

We livetrapped wolves (Mech 1974) during May–October or November in a 2,060-km² census area (Mech 1986) and in a few areas immediately surrounding the census area. The study area included the census area as well as the adjacent area in which the wolves were livetrapped. Pups were born in April and early May, but we rarely captured them before July. We drew blood, attached radio collars, and later located wolves from a fixed-wing aircraft (Mech 1974). We observed radio-tagged wolves and their packmates throughout each winter and counted all members of each pack in the census area (Mech 1973, 1986; Mech and Goyal 1993). Numbers presented here represent the maximum observed members per pack during December–January each year (Table 1). We followed an approved U.S. Fish and Wildlife Service animal welfare protocol.

We removed serum from blood samples and stored it at −15 to −20 C until assayed for CPV. We determined antibodies to CPV by the hemagglutination inhibition test (Carmichael et al. 1980). We heat-activated sera, treated it with 25% kaolin, and absorbed it with packed porcine erythrocytes to remove nonspecific hemagglutinins. We mixed serial dilutions of sera in 96-well microtiter plates with 8 hemagglutination units of CPV and incubated them at 4 C overnight. We then added a 1% suspension of porcine erythrocytes and read the test after 2 hours incubation at 4 C. We expressed the antibody

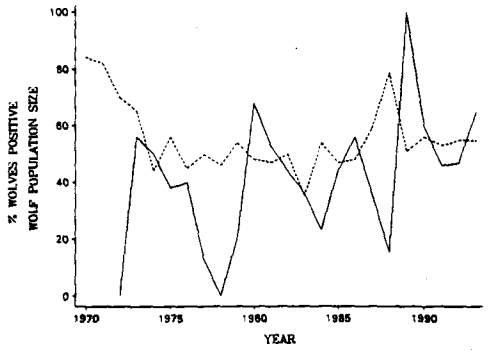

Fig. 1. Percent wolves livetrapped during summer 1970–93 in the central Superior National Forest, Minnesota, that were serologically positive (≥256) to canine parvovirus (solid line) ($r^2 = 0.28$, $P = 0.05$) and wolf population trend (dotted line) in the central Superior National Forest (Mech 1973, 1986, unpubl. data).

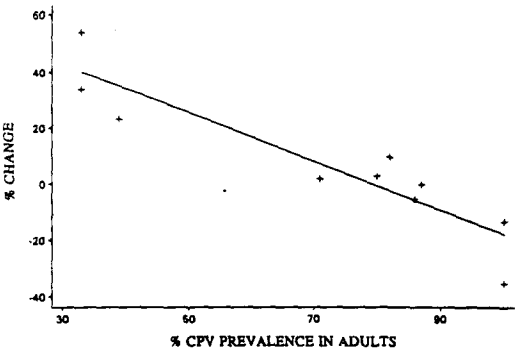

Fig. 2. Relationship during 1984–93 between percent change in the population of wolves in the central Superior National Forest, Minnesota, from 1 winter to the next and the canine parvovirus antibody prevalence of adult wolves livetrapped during the intervening summer ($r^2 = 0.83$, $P < 0.001$; $y = 65.6631 - 0.86305x$).

titer as the reciprocal of highest serum dilution that completely inhibited hemagglutination. We considered titers ≥256 as evidence of CPV exposure (positive). Mech et al. (1986) and Goyal et al. (1986) provided details about titer levels, ages of wolves sampled, and titer changes with time.

The CPV had an unknown history before 1979 (Parrish et al. 1985). Thus, we restricted our analyses to 1979–93, even though there was possible serologic evidence of the virus in the population we studied as early as 1973 (Fig. 1). Data from recaptured wolves were counted in our analyses only during the wolves' first capture year. We hypothesized that as the CPV epizootic had time to become enzootic (Thomas et al. 1984), its effect on the population would tend to increase. Therefore, we examined data from the first 5 years of the study, which we considered the epizootic period, separately from the last 10 years (enzootic period).

We used simple linear regression (Statistix 4.0 1992) to relate trends with years, percentages of pups caught, and percent annual changes in the wolf population. There is disagreement about the suitability of this method applied to percentages without arcsine transformations (R. L. Zarnke, Alaska Dep. Fish and Game, Fairbanks, pers. commun.). However, a test comparing results between arcsine-transformed data and nontransformed data indicated comparable results (Shapiro-Wilk Rankit = 0.93–0.98).

RESULTS

We captured and extracted blood from 9–30 wolves each summer and fall during 1979–93 (Table 1). Total CPV seroprevalence varied from 3 of 20 to 18 of 19/year, and from 1 of 3 to 17 of 17 for adults (Table 1). Prevalence generally increased and showed some possible cyclicity (Fig. 1). Pups represented from 2 of 19 to 16 of 23 of the wolves captured each year. Wolf numbers in winter in the census area ranged from 35 to 79, and winter-to-winter changes in numbers varied from −35 to 54% (Table 1).

During the epizootic period (1978–83), wolf population change was not related to total or adult CPV seroprevalence ($r^2 = 0.12$ and 0.64, $P = 0.57$ and 0.10, respectively). However, during the enzootic period (1984–93), we found an inverse relationship between CPV prevalence in wolves and the percent change in the wolf population the following winter ($r^2 = 0.71$, $P < 0.01$) (for prevalence in adults, Fig. 2).

We also found an inverse relationship between percent pups captured each year in summer–fall 1984–93 and CPV prevalence in the population (Fig. 3). The percent annual change in wolf population during 1984–93 was positively related to the proportion of pups in the total number of wolves captured each summer ($r^2 = 0.39$, $P = 0.05$). There is some evidence of a decline in pup recruitment from 1979 to 1993 ($r^2 = 0.22$, $P = 0.08$).

DISCUSSION

The origin and history of CPV is uncertain (Parrish et al. 1985), but there is evidence that this viral strain was the result of a mutation (Pollock 1984). Typical CPV symptoms were first reported in dogs in Texas in 1977 (Eugster and Nairn 1977). Serologic evidence for earlier

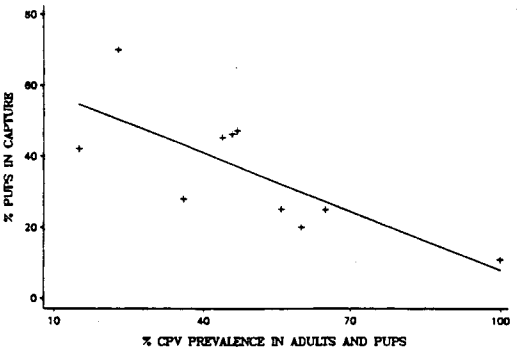

Fig. 3. Relationship between proportion of pups in a sample of wolves livetrapped during summer–fall 1984–93 in the central Superior National Forest, Minnesota, and the canine parvovirus antibody prevalence of all wolves in the sample (r^2 = 0.57, P = 0.01; y = 62.9476 − 0.54975x).

existence of the virus was found in dogs in the Netherlands in 1976 (Schwers et al. 1979), in dogs in Greece in 1974 (Koptopoulos et al. 1986), and in wolves in our study area in 1973 (Goyal et al. 1986; Mech, unpubl. data). Coyotes from Texas, Utah, and Idaho showed serological evidence of CPV in 1979 (Thomas et al. 1984).

Antibodies to CPV in a wild animal indicate exposure to CPV. Exposed dogs (Pollock and Carmichael 1981) or wolves (Mech and Goyal, unpubl. data) may die from exposure, show clinical symptoms and recover, or resist symptoms (McCandlish et al. 1981). Individuals that are nutritionally stressed (Carman and Povey 1982) or parasitized, at least those with *Giardia* (Pollock 1982), may be more severely affected. Active infections that do not kill an animal may incapacitate it, perhaps for ≤3 months in wild wolves (Mech and Goyal, unpubl. data). Thus, wolves caught in our study that had CPV titers probably did not have an active infection when sampled. Dogs that recover are immune to reinfection for ≥18 months (Pollock and Carmichael 1981). Whether the same is true for wolves is unknown, for wolves would more likely be nutritionally stressed and parasitized than would dogs.

Antibodies in dogs persisted ≥2 years after an infection (Carmichael and Binn 1981), and ≥1 year in a wolf pup (Mech et al. 1986), but how long antibodies persist in wolves without active infection or re-exposure is unknown. Dogs and coyotes passed maternal antibodies derived from an infection or vaccination on to some, although not all, of their pups. Antibodies persisted for ≤8 weeks (Green et al. 1984). After that, pups that had maternal antibodies were no longer protected (Meunier et al. 1981).

The CPV generally killed 1–12-week-old dogs (Eugster and Nairn 1977, Meunier et al. 1981). Although CPV can kill wolves ≤15 months old (Mech et al. 1986; Mech and Goyal, unpubl. data), it probably primarily affects 1–12-week-old pups (Johnson et al. 1994, Wydeven et al. 1994) as supported by the inverse relationship between CPV seroprevalence and percent pups trapped each year. These results are conservative because our methods measured seroprevalence in survivors only (Thomas et al. 1984).

Recovered CPV-infected dogs can shed fecal viruses for months, especially when re-exposed to CPV, and in cold and shade, feces can retain infective viruses for ≤7 months (Gordon and Agrick 1986). Wolf feces might remain infective ≤5 years (Muneer et al. 1988). Thus, a large population of CPV-seropositive wolves indicates that exposure to CPV has been high and probably will remain so. Even wolf pups with maternal antibodies would be at risk after weaning, especially if parasitized or nutritionally stressed.

Because CPV prevalence in our area has increased gradually over 15 years and shown some possible cyclicity, it differs from the CPV in coyote populations in Texas, Utah, and Idaho. There, seroprevalence of CPV increased from 0 to ≥90% in 2–3 years (Thomas et al. 1984), although whether it remained that high after the study is unknown. The difference might be attributable to timing of sampling. Thomas et al. (1984) sampled in fall and spring, which allowed individuals ≤1 year old to be exposed longer to the virus, whereas we sampled during summer and early fall before pups were very mobile.

Seroprevalence of CPV in our adult wolf population, while variable, increased an average of 4%/year during 1979–93 and reached 87% in 1993. On the basis of the regression equation (y = 65.6631 − 0.86305x), when CPV antibody prevalence in adults consistently exceeds 76%, the wolf population might be expected to decrease. Because wolf litters average 6 pups (Mech 1970), pup recruitment is an important determinant of annual population change. Pup recruitment may be decreasing.

Although we found relationships between CPV prevalence and both wolf population change and percent pups, no net decline was apparent in the wolf population. This suggests that CPV mortality compensated for mortality

factors, such as starvation and intraspecific strife (Mech 1977) that had been restricting this population prior to the CPV epizootic. Whereas before CPV many wolf pups succumbed to starvation by about 6 months of age (Van Ballenberghe and Mech 1975), now they may perish from CPV before 3 months old. The compounding effect of starvation and parasitism on CPV severity probably is also important. Furthermore, with CPV reducing pup recruitment, total food competition might decrease. This then might reduce aggressiveness that results in intraspecific mortality along and outside territorial borders (Mech 1994).

With average liter sizes of 6 (Mech 1970), and winter pack sizes also comprising about 6 (Mech 1973, 1986), this population could potentially double annually. Therefore, the population can afford about 50% mortality without declining. Nevertheless, if CPV prevalence increases, pup recruitment may decrease, ultimately resulting in a population decline. Thomas et al. (1984: 1286) concluded that 5 years after CPV infected 3 coyote populations, the effect of CPV on those populations "remains obscured."

We found no pups ≤9 months old dead from CPV, but the techniques we used did not enable such a finding. We could not observe dens from a distance, and close observation could disturb them enough to risk abandonment. We did not radiocollar pups until they were ≥5 months old, after most presumed mortality would have occurred. However, the radio tracking we have used since 1968 on animals ≥5 months old did result in a confirmed mortality by CPV in 1993.

Our analyses support our conclusions (Mech and Goyal 1993, 1994), with many of the same data, that CPV can affect wolf pup survival and degree of annual wolf population change. However, with 3 more years of data and a separate examination of the epizootic and enzootic periods of CPV history, we have found evidence of a stronger apparent dependence of wolf population change on CPV prevalence, and a greater apparent effect of CPV during the last 10 years of the study than during the first 5 years. These findings support the hypothesis of an increasing effect of CPV on the Superior National Forest wolf population. They also lend credence to the hypothesis that CPV may have hindered wolf recovery in Wisconsin (Wydeven et al. 1994) and Montana (Johnson et al. 1994).

Our findings differ from those of McClandish et al. (1981) who found the greatest effects of CPV on dogs during the epizootic period. However, the fact that our study population involved wild animals that are often nutritionally stressed (Van Ballenberghe and Mech 1975, Mech 1977) might explain why CPV appears to be affecting our population more during the enzootic period.

MANAGEMENT IMPLICATIONS

The wolf is on the endangered species list in the 48 contiguous United States and has been recovering slowly in the Lake Superior and northern Rocky Mountain areas since the early 1970s (Mech et al. 1994). Knowledge about the possible role of CPV in retarding wolf recovery will be useful for resource managers to consider when attempting management measures. There seems to be little that managers can do to prevent CPV in wolves. In small colonizing wolf populations, CPV vaccinations of wolves (Johnson et al. 1994) may be useful. However, to be effective, 3 vaccinations are necessary (McClandish et al. 1981) and would be difficult to accomplish without using a capture collar (Mech and Gese 1992). Awareness of the CPV issue may prevent managers from attempting economically and politically costly management measures such as road closures (Thiel 1985), which may not be effective if CPV is found to be more limiting in certain areas than illegal killing by humans, for example.

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