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SHORT COMMUNICATIONS

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Canine Parvovirus Effect on Wolf Population Change and Pup Survival

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ABSTRACT: Canine parvovirus infected wild canids more than a decade ago, but no population effect has been documented. In wild Minnesota wolves (*Canis lupus*) over a 12-yr period, the annual percent population increase and proportion of pups each were inversely related to the percentage of wolves serologically positive to the disease. Although these effects did not seem to retard this large extant population, similar relationships in more isolated wolf populations might hinder recovery of this endangered and threatened species.

Key words: Canine parvovirus, wolf, *Canis lupus*, disease, survival, mortality, serology, endangered species, conservation biology.

Canine parvovirus (CPV) disease of domestic dogs, first reported in 1977, is generally fatal to pups and older dogs (Eugster and Nairn, 1977). It has been found in numerous wild species including various canids (Mech et al., 1986; Goyal et al., 1986). In coyotes (*Canis latrans*) CPV apparently caused death of pups when the populations first contracted the disease (Thomas et al., 1984). Serological evidence of CPV has been found in wolf (*Canis lupus*) populations in Wisconsin (USA), Minnesota (USA), Isle Royale National Park, Michigan (USA), and Montana (USA) (Goyal et al., 1986; Peterson and Krumenaker, 1989; R. R. Ream, pers. comm.; R. P. Thiel, pers. comm.). This has led to speculation that CPV contributed to a recent decline in the Isle Royale wolf population (Peterson and Krumenaker, 1989) and in the unexpectedly slow increase of wolves in Wisconsin (Wydeven, 1991).

In captive wolves, CPV caused death of nine pups and two yearlings (Mech et al., 1986). However, no documentation exists that CPV has had a long-term influence on wild populations of any species. Our objective was to determine any apparent negative effects of CPV on a wild wolf population in northeastern Minnesota.

The study area was a 2,060-km² area of the central Superior National Forest (47°30′ to 48°00′N, 91°00′ to 92°00′W), part of a much larger wolf range contiguous with the entire Canadian wolf population. Wolves have never been extirpated there.

Wolves were live-trapped in the study area from May through November 1968 through 1991 with modified-steel foot traps; they were anesthetized and had their blood sampled (Mech, 1974). We removed the serum and stored it at −20 C. Pups were born in April and early May but rarely were captured before July. The wolves were also radio-tagged and aerially radio-tracked at least weekly from altitudes at which they were not disturbed (Mech, 1974). Numbers of pack members with each wolf in the study area were counted during winter when visible from aircraft. In years when all possible packs were not radio-tagged, estimates of pack size were based on sightings of nonradioed packs or their tracks or were inferred from pack sizes in previous or later years (Mech, 1986, unpubl.).

For current study, 11 to 42 wolves were caught and blood sampled each year from

1979 through 1991 in and around the wolf census area.

Antibodies to CPV were determined by the hemagglutination inhibition (HI) test (Carmichael et al., 1980). The sera were heat-activated, treated with 25% kaolin, and absorbed with packed porcine erythrocytes to remove non-specific hemagglutinins. Serial dilutions of sera in 96-well microtiter plates were mixed with eight hemagglutination units of CPV followed by incubation at 4 C overnight. A 1% suspension of porcine erythrocytes was then added, and the test was read after a 2-hr incubation period at 4 C. The antibody titer was expressed as the reciprocal of highest serum dilution that completely inhibited hemagglutination. Titers of ≥ 256 were considered evidence of CPV infection. Details about titer levels, ages of wolves sampled, and titer changes with time were provided by Mech et al. (1986) and Goyal et al. (1986).

The prevalence of CPV antibodies among wolves sampled each summer varied from 13 to 95% (Table 1). Winter wolf numbers in the census area during this period ranged from 35 to 79, and percent of pups in the capture each year varied from 8 to 72 (Table 1). A simple linear regression (Statistix 3.5, 1991) was used to determine relationships between percentage of wolves with positive serological titers to CPV per summer from 1979 through 1990 and (a) change in wolf numbers in the study area from December and January of one year to December and January of the next and (b) percentage of pups captured each summer.

We found an inverse relationship between the prevalence of CPV antibodies among summer wolf samples (x) and the percentage increase in the wolf population, from the winter preceding CPV sampling to the winter following the sampling (y) ($r^2 = 0.48$; $P = 0.01$; $y = 39 - 0.8x$). We also found an inverse relationship between the prevalence of CPV antibodies among summer wolf samples (x) and the percent pups composed of the total wolves caught during the same sampling period (y) ($r^2 = 0.56$; $P = <0.01$; $y = 70 - 0.6x$).

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

Year	Percent pups captured July to November	Total winter wolf numbers[a]	Percent change[b] in wolf numbers	Percent positive for CPV antibodies[c]
1979	47	54	+17	20
1980	48	48	−11	64
1981	43	47	−2	51
1982	50	50	+6	45
1983	50	35	−30	35
1984	64	54	+54	20
1985	43	46	−15	38
1986	21	39	−15	62
1987	72	42	+8	37
1988	52	59	+40	13
1989	8	51	−14	95
1990	20	51	0	55

[a] Mech (1986, unpubl.).
[b] From previous year.
[c] Summer and fall populations.

Based on this regression equation, the population would be stable when 51% of the population was CPV-positive. We infer that CPV tended to affect wolf population change in our study area through early pup mortality since the proportion of pups in the total percentage of samples of wolves caught after July each year already was inversely related to the prevalence of CPV antibodies in the total sample. This is consistent with the effect of CPV on domestic dogs and captive wolves (Eugster and Nairn, 1977; Mech et al., 1986). However, we have found no adult wolves dead from CPV even though our study techniques should allow us to detect such deaths.

It is interesting to note that despite the apparent influence of CPV on wolf pup survival and population change, wolf numbers in the study area generally were higher at the end of the study than at the beginning (Mech, 1986; Table 1). In addition based on a two-sample t-test (Statistix 3.5, 1991), there was no significant difference between the mean percent pups

caught before and after 1978 ($P = 0.84$). Although we did find positive titers before 1979 (Goyal et al., 1986), the prevalence of CPV antibodies in the population before 1979 always was low, whereas after 1978 it was clear the virus was established in our population. These findings imply that CPV as a mortality factor may be primarily compensatory for other mortality factors affecting the wolf population. Because wolves have a high reproductive potential, with litter sizes averaging six (Mech, 1981), established populations can sustain considerable mortality. Usual mortality factors in an inaccessible and legally protected population are intraspecific strife and starvation (Mech, 1977). In our study population, CPV may now be taking the place of pup starvation.

The wolves in our study area live close enough to domestic dogs to have contracted from them originally, although it is unknown whether that was the source of infection. However, because wolves can disperse for distances over 800 km (Fritts, 1983), and our wolf population lies adjacent to the Canadian wilderness, our population probably is receiving immigrants that previously have not been exposed to CPV. In years when general infection by CPV is low, offspring of these immigrants may constitute an important proportion of the pups that survive and are recruited into our population.

Although CPV may not be affecting the well-established wolf population in our study area too adversely, the disease constitutes another serious mortality factor. Thus it could limit isolated, disjunct, or colonizing wolf populations such as those in Wisconsin, Michigan, Montana, Idaho, and Washington (USA). In those areas, high productivity and survival is important to allow such populations to increase, The smaller the population, the greater the effect of any loss. Secondly, such disjunct populations have lower potential for immigration. Not only does minimal immigration have a numerical effect, but it also minimizes the chances of an influx of CPV-free wolves that could at least temporarily replenish the population.

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LITERATURE CITED

CARMICHAEL, L. E., J. C. JOUBERT, AND R. V. H. POLLOCK. 1980. Hemagglutination by canine parvovirus: Serologic studies and diagnostic applications. American Journal of Veterinary Research 41: 784–791.

EUGSTER, A. K., AND C. NAIRN. 1977. Diarrhea in puppies: Parvovirus-like particles demonstrated in their feces. Southwest Veterinarian 30: 59.

FRITTS, S. H. 1983. Record dispersal by a wolf from Minnesota. Journal of Mammalogy 64: 166–167.

GOYAL, S. M., L. D. MECH, R. A. RADEMACHER, M. A. KHAN, AND U. S. SEAL. 1986. Antibodies against canine parvovirus in wolves of Minnesota: A serologic study from 1975 through 1985. Journal of the American Veterinary Medical Association 189: 1092–1094.

MECH, L. D. 1974. Current techniques in the study of elusive carnivores. Proceedings of the International Congress of Game Biologists 11: 315–322.

———. 1977. Productivity, mortality and population trend in wolves from northeastern Minnesota. Journal of Mammalogy 58: 559–574.

———. 1981. The wolf. University of Minnesota Press, Minneapolis, Minnesota, 389 pp.

———. 1986. Wolf numbers and population trend in the Superior National Forest. Research Paper NC-270. U.S. Department of Agriculture, North Central Forest Experiment Station, St. Paul, Minnesota, 6 pp.

———, S. M. GOYAL, C. N. BOTA, AND U. S. SEAL. 1986. Canine parvovirus infection in wolves (*Canis lupus*) from Minnesota. Journal of Wildlife Diseases 22: 104–105.

PETERSON, R. O., AND R. J. KRUMENAKER. 1989. Wolves approach extinction on Isle Royale: A biological and policy conundrum. George Wright Forum 6: 10–15.

STATISTIX 3.5. 1991. Statistix 3.5. Analytical Software, St. Paul, Minnesota.

THOMAS, N. J., W. J. K. FOREYT, J. F. EVERMANN, L. A. WINDBERG, AND F. F. KNOWLTON. 1984.

Seroprevalence of canine parvovirus in wild coyotes from Texas, Utah, and Idaho (1972–1983). Journal of the American Veterinary Medical Association 185: 1283–1287.

WYDEVEN, A. P. 1991. Wisconsin's forty wolves. International Wolf 1(3): 40.

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