



Death of a Wild Wolf from Canine Parvoviral Enteritis

Authors: Mech, L. David, Kurtz, Harold J., and Goyal, Sagar

Source: Journal of Wildlife Diseases, 33(2) : 321-322

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-33.2.321>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

Death of a Wild Wolf from Canine Parvoviral Enteritis

L. David Mech,^{1,3} Harold J. Kurtz,² and Sagar Goyal,^{2,1} Patuxent Wildlife Research Center, Biological Resources Division, U.S. Geological Survey, Laurel, Maryland 20708, USA; ² College of Veterinary Medicine, University of Minnesota, St. Paul, MN 55108, USA; ³ Current address: North Central Forest Experiment Station, 1992 Folwell Ave., St. Paul, Minnesota 55108, USA

ABSTRACT: A 9-mo-old female wolf (*Canis lupus*) in the Superior National Forest of Minnesota (USA) died from a canine parvovirus (CPV) infection. This is the first direct evidence that this infection effects free-ranging wild wolves.

Key words: Canine parvovirus, wolf, *Canis lupus*, disease, serology, mortality.

In the U.S., serologic evidence of canine parvovirus (CPV) infection has been found among wild populations of wolves (*Canis lupus*) in Minnesota (Mech et al., 1986), Wisconsin (Wydeven et al., 1995), Alaska (Zarnke and Ballard, 1987), and Montana (Johnson et al., 1994). Evidence also was found that CPV infection may be influencing population changes in wolves in the central Superior National Forest (SNF) of Minnesota through early pup mortality (Mech and Goyal, 1995). However, the only documentation of wolves dying from CPV infection involved 10 captive, 2 to 14 mo-old wolves (Mech et al., 1986). In the present note, we document death of a wild wolf due to CPV infection in the SNF (48°N, 92°W).

Female wolf 409 was captured as a 6-mo-old, 22-kg pup on 29 October 1992, blood-sampled, outfitted with a radio-controlled capture collar (Mech and Gese, 1992), and radio-tracked; her serum tested <1:256 for CPV antibodies (hemagglutination inhibition test); we considered this negative because samples with lower titers were negative by the ELISA test whereas samples with 1:256 were positive (Mech et al., 1986). The animal was recaptured via the capture collar on 8 December 1992, weighed 27 kg, and it again was negative for CPV antibodies.

This wolf was last found alive on 21 January 1993. On 2 February 1993 it was dead and weighed 19.5 kg. The carcass

was kept cool, necropsied on 11 February 1993, and found to be dehydrated and in poor condition. The carcass was autolytic. Grossly, the enteric lesions were characterized by a granular consistency over the serosal surface of the terminal portion of the small intestine. There was hyperemia and hemorrhage in the wall of the ileum. The ileal wall was thin, the mucosal surface had a yellow tinge, and contents of the dilated lumen were serosanguinous, bile-stained, and had a putrid odor. The colon contained blood-stained fluid contents. The lungs appeared edematous. No other gross lesions were observed.

The brain, liver, lungs, heart, kidneys, stomach, jejunum, ileum, and colon were fixed in 10% buffered formalin, embedded in paraffin, sectioned at 5 µm and stained with hematoxylin and eosin.

Microscopic lesions were confined to

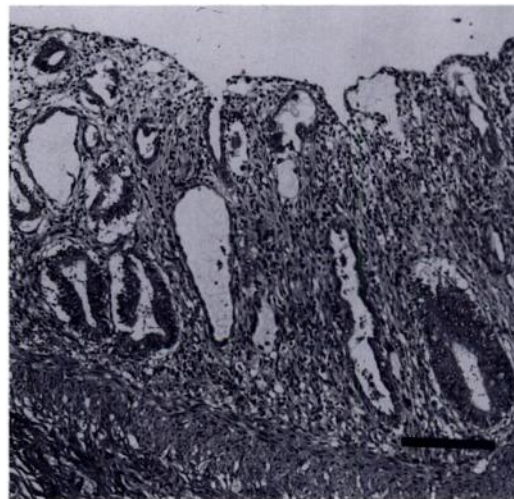


FIGURE 1. Ileal mucosa with loss of villi and superficial enterocytes. The lamina propria is collapsed and contains dilated crypts which are lined by attenuated or hyperplastic enterocytes. H & E. Bar = 100 µm.

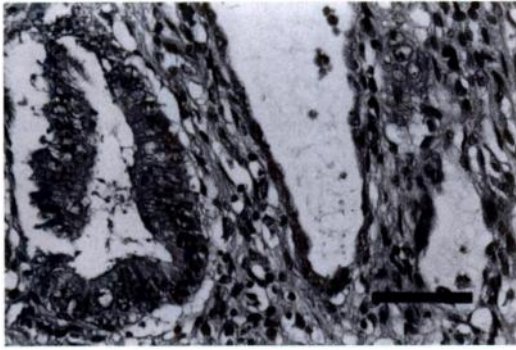


FIGURE 2. Higher magnification of Figure 1. There are hyperplastic enterocytes lining dilated crypts and other dilated crypts lined by attenuated epithelial cells. H & E. Bar = 50 μ m.

the terminal portion of the small intestine and were partially obscured by autolytic changes. Sections of ileum had chronic change consisting of dilated intestinal crypts which were lined by enterocytes which were attenuated and flattened (Figs. 1 and 2). Many dilated glandular crypts contained degenerated inflammatory cells which were principally polymorphonuclear leukocytes. Some crypts were lined by proliferating regenerative enterocytes (Figs. 1, 2). The lamina propria was collapsed and had a fibrous appearance due to loss of enterocytes lining the villi. The intestinal surface epithelium was attenuated, and in some areas there was a complete lack of surface enterocytes. Peyer's patches were almost completely devoid of germinal centers and lymphocytes. The ileal lesions were considered characteristic of parvoviral enteritis. Parvoviruses in low numbers were found in the feces by electron microscopy (Muneer et al., 1988).

From this case we conclude that CPV infection can kill wild wolves older than newborn pups, and wild wolves can be-

come infected with CPV during winter. These findings are of value because they represent the first direct documentation of CPV effects on wild wolves.

We thank the U.S. Fish and Wildlife Service, the U.S. National Biological Service, North Central Forest Experiment Station, and the Veterinary Diagnostic Lab at the University of Minnesota for supporting this study and Michael E. Nelson and numerous volunteers for assisting with the data collection in the field. Linda Munson and Jeff Zuba suggested improvements for the manuscript.

LITERATURE CITED

- JOHNSON, M. R., D. K. BOYD, AND D. H. PLETSCHER. 1994. Serology of canine parvovirus and canine distemper in relation to wolf (*Canis lupus*) pup mortalities. *Journal of Wildlife Diseases* 30: 270-273.
- MECH, L. D., AND E. M. GESE. 1992. Field testing the Wildlink Capture Collar on wolves. *Wildlife Society Bulletin* 20: 249-256.
- , AND S. M. GOYAL. 1995. Effects of canine parvovirus on gray wolves in Minnesota. *The Journal of Wildlife Management* 59: 565-570.
- , ———, C. N. BOTA, AND U. S. SEAL. 1986. Canine parvovirus infection in wolves (*Canis lupus*) from Minnesota. *Journal of Wildlife Diseases* 22: 104-106.
- MUNEER, M. E., I. O. FARAH, K. A. POMEROY, S. M. GOYAL, AND L. D. MECH. 1988. Detection of parvoviruses in wolf feces by electron microscopy. *Journal of Wildlife Diseases* 24: 170-172.
- WYDEVEN, A. P., R. N. SCHULTZ, AND R. P. THIEL. 1995. Monitoring of a gray wolf population in Wisconsin 1979-1991. In *Ecology and conservation of wolves in a changing world*, L. D. Carbyn, S. H. Fritts, and D. R. Seip (eds.), Canadian Circumpolar Institute, Edmonton, Alberta, Canada, pp. 147-156.
- ZARNKE, R. L., AND W. B. BALLARD. 1987. Serologic survey for selected microbial pathogens of wolves in Alaska. *Journal of Wildlife Diseases* 23: 77-85.

Received for publication 13 May 1996.