PREVALENCE AND EFFECTS OF Parelaphostrongylus tenuis IN A CAPTIVE WAPITI POPULATION

Authors: WOOLF, A., MASON, C. A., and KRADEL, D.

Source: Journal of Wildlife Diseases, 13(2) : 149-154

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-13.2.149

PREVALENCE AND EFFECTS OF *Parelaphostrongylus tenuis* IN A CAPTIVE WAPITI POPULATION

A. WOOLF, Research Director, Rachelwood Wildlife Research Preserve
R.D. #1, New Florence, Pennsylvania 15944, USA

C. A. MASON, Department of Biology, West Virginia University,
Morgantown, West Virginia 26506, USA

D. KRADEL, Animal Diagnostic Laboratory, Department of Veterinary Science,
Pennsylvania State University, University Park, Pennsylvania 16802, USA

Abstract: Prevalence of neurologic disease attributed to *Parelaphostrongylus tenuis* was investigated in a population of wapiti (*Cervus elaphus canadensis*) enclosed in a 2080 ha. preserve. Brains of 44 animals harvested in 1973, 1974 and 1975 were examined grossly and microscopically. Prevalence of infection increased each year from 26.6% of the sample in 1973 to 64.3% in 1975. These data suggest that wapiti living close to white-tailed deer (*Odocoileus virginianus*) may have a high prevalence of infection, but many individuals may not show signs. Accordingly, routine histologic examination of natural mortality cases without supporting observations of clinical signs would be inadequate to define cause of death. While the effects of the parasite in the population studied did not produce a high herd mortality or a sudden die-off situation, the apparent greater susceptibility of the younger age-classes to the parasite would have a detrimental effect on population recruitment and might limit the growth of wapiti herds living in proximity to white-tailed deer that harbor *P. tenuis*.

INTRODUCTION

Neurologic disease caused by nematodes frequently has been reported in ruminent species.[1,5,8,10] One nematode in particular has been implicated in the disease of several cervid species. It has been identified by several different names, but a consensus has developed that these may all be one nematode, *Parelaphostrongylus tenuis*.[7]

Wapiti (*Cervus elaphus canadensis*) have been infected experimentally with this nematode and the disease has been reported occurring naturally in two populations.[6,7] Evidence of an asymptomatic natural infection was reported for wapiti in Minnesota.[9] *P. tenuis* is present and may be a mortality factor in the introduced wapiti of northcentral Pennsylvania (Unpublished pathology reports, Animal Diagnostic Laboratory, Pennsylvania State University).

A study was begun in 1973 at the Rachelwood Wildlife Research Preserve in Pennsylvania to survey the wapiti population for prevalence of meningeal worm infection; to determine the histopathologic and clinical effects of the disease; and to evaluate the parasite's impact on herd population dynamics. This report presents the results of this study.

HABITAT DESCRIPTION AND HERD HISTORY

The wapiti herd has been enclosed in Rachelwood Wildlife Research Preserve, a 2080 ha. area located on Laurel Ridge in western Pennsylvania. The habitat is primarily a mixed-oak and northern hardwoods forest. About 74 ha. of open fields seeded to perennial grasses and some legumes are scattered throughout

the Preserve to provide grazing opportunities for the high density ungulate population. Wapiti and red deer (*Cervus elaphus elaphus*) shared the habitat with about 1500 white-tailed deer (*Odocoileus virginianus*) and 100 mouflon sheep (*Ovis mousimon*) during the course of the study. The high density populations were supported by supplemental grain provided on a year-round basis in about 125 feeders distributed throughout the area.

Red deer were introduced into the Preserve in the early 1950's. The animals were obtained from game farms and their origin is uncertain. No data are available on the numbers introduced, but the peak population size of 50-75 animals was reached in the mid-1960's. As late as September, 1969, at least 30 red deer were known to inhabit the Preserve. The herd began to decline after 1964 (from intentional overharvest), and by 1976 only 7-10 remained.

Wapiti introductions began in February, 1959, with four pregnant cows obtained from Yellowstone National Park. Two additional cows and four bulls from the same source were introduced between 1959 and 1964. The wapiti herd increased to a peak population of 70-80 by fall, 1973, when the study was started. Forty-two wapiti were known to be in the Preserve at the termination of the study in 1976.

Hybridization between wapiti and red deer occurred in the Preserve since the late 1960's and appeared to coincide with the decline of the red deer herd and the increasing numbers of wapiti. The herds were reportedly separated most of the time prior to this period.

Clinical signs believed to be caused by meningeal worms were reported in red deer from the earliest introductions and in wapiti beginning in the late 1960's. A wapiti necropsied in 1968 was diagnosed as suffering from cerebral nematodiasis. Since 1968, 15 necropsies were performed on *Cervus* sp. found dead in the Preserve and cerebral nematodiasis was demonstrated in 12.

METHODS

Wapiti, red deer, and hybrids were assumed to be physiologically similar for the purpose of this study. No distinctions were made in evaluating findings although individual animals were classified as to subspecies.

Harvested animals were considered to be a random sample of the population except when individuals showed unusual behavioral signs. Internal organs of all animals were examined grossly. In 1973, brains were examined and sections of cerebrum, cerebellum, and medulla were preserved in 10% formalin. Histological sections were cut at 6 μm and stained with hematoxylin and eosin. Brains of animals harvested in 1974 and 1975 were examined after formalin fixation by transverse slicing at about 10 mm intervals to search for gross lesions. Six sections were routinely taken from each brain for histological examination: a section from the first third of each cerebral hemisphere mid-way into the parenchyma; a section adjacent to the posterior part of the third ventricle from each hemisphere; a section from the midportion of the cerebellum; and a cross section from the medulla.

Three animals with clinical signs of meningeal worm infection were captured with immobilizing drugs and maintained in captivity for observation. At the death of each animal, all organs and tissues were examined grossly and histopathologically. Brains were examined as in the harvested wapiti.

A concurrent study on wapiti behavior was in progress at the Preserve since June, 1975 to the termination of this study. Individual animals with clinical signs were observed over a period of months before they were killed. Behavioral signs attributable to meningeal worm infection were recorded and when recognizable individuals were included in the random harvest, they were used to correlate the presence or absence of clinical signs with histologic findings.

In 1973, 1974 and 1975, white-tailed deer harvested at the Preserve were examined grossly for the presence of

meningeal worms in the cranial cavity. A sample of 45, 61, and 69 was examined in each respective year.

RESULTS

Presence of Meningeal Worms

Adult nematodes were found in the cranial cavity of 8 of 44 animals examined from 1973 to 1975. These were identified as *P. tenuis* by Dr. W. M. Samuel and specimens have been deposited in the U.S. National Museum Helminthological Collection (Acc. Nos. 75-102, 75-179, 356977, and 361325).

Meningeal worms are commonly found in the white-tailed deer living in the Preserve. In 1973, 42.2% of the sample examined had adult nematodes in the cranial cavity; 72.1% in 1974 and 68.1% in 1975.

Prevalence of Infection

The results of histologic study of brains collected from harvested animals are shown in Table 1. The prevalence of lesions attributable to *P. tenuis* infection increased from 1973 to 1975. The overall prevalence of infection, based on histologic evidence, was 45.4% for the three-year period. Yearlings and 2.5 year-old animals had the highest prevalence, 59.2%. Animals 3.5 years and older had a 21.4% prevalence of infection.

Six harvested animals were recognizable as individuals and were observed during the period June to November 1975. Three of six had histologic lesions attributable to *P. tenuis* infection. Two other harvested animals showed abnormal behavior during field observations and both had brain lesions.

Clinical Signs

Two harvested animals showing abnormal behavior both remained apart from other wapiti and had an unusual tolerance to close human presence. In one case, mild ataxia was also noted.

The three clinical cases maintained in captivity had common signs. All were alone when captured and were rarely seen with other wapiti or red deer prior to capture. All also acted "tame" at various times. Ataxia in varying degrees was evident in all clinical cases as was a transient stiffness of gait.

A seven-month-old female wapiti calf was captured on 7 February when severe ataxia was noted. Ataxia disappeared three days later, but a stiffness in her gait remained evident for one more day. During one month of subsequent observation in captivity, the calf was without apparent clinical signs except for a transient circling that lasted one day. After release in mid-March, the calf was not observed again until mid-June. From then, until killed in early July, the animal became increasingly lethargic and tolerant of human disturbance.

On 16 May, an 11 month-old male hybrid wapiti-red deer was seen circling to the right and moderately ataxic. Six days later, he held his head and neck at about a 60 degree angle to the right and tolerated human approach to about 15 m. He was captured for observation and the following day appeared normal and remained asymptomatic for almost four months. Mild ataxia was noted in mid-September and became progressively more severe. By mid-October, he was in lateral recumbency, unable to rise, and was killed for necropsy.

A yearling red deer stag was the final clinical case. This animal was observed in the field between 5 August and 11 September with clinical signs of circling, ataxia, staggering, and "tameness". Additionally, there were abrasions present on the head and neck and the right antler was broken at the pedicel. Once, while retreating from the observer, the red deer ran into a tree suggesting impaired vision. He was captured on 13 September and his condition deteriorated rapidly until he was killed on 22 September.

Pathology

Gross examination of the brains of harvested animals and clinical cases disclosed lesions in only one animal. An adult female observed with clinical signs prior to harvest had yellowish discoloration of the dura with brown-colored

TABLE 1. Prevalence of histologic lesions attributable to **P. tenuis** infections in the brains of 44 **C. elaphus** harvested at Rachelwood Wildlife Research Preserve, 1973-1975.

Age and Sex		1973 Number Examined	1973 Number Positive	1974 Number Examined	1974 Number Positive	1975 Number Examined	1975 Number Positive	Total Number Examined	Total Number Positive
Calf	—M	0	0	0	0	0	0	0	0
	F	1	0	1	0	1	1	3	1
1½ yrs	—M	4	1	5	3	3	2	12	6
	F	3	1	3	2	3	2	9	5
2½ yrs	—M	0	0	2	1	0	0	2	1
	F	1	1	1	1	2	2*	4	4
3½+ yrs	—M	0	0	0	0	1	0	1	0
	F	6	1	3	0	4	2*	13	3
		15	4	15	7	14	9	44	20
Percent positive		26.6		46.6		64.3		45.4	

* One of two showed clinical signs and/or abnormal behavior when harvested.

debris on the surface. The posterior cerebral hemispheres were softer than normal, suggestive of malacia and the central nervous system fluid was excessive.

Histopathologic lesions found varied in severity, but generally had consistent features. Most cases had focal meningeal infiltration, primarily wtih mononuclear cells and eosinophils. Amorphous, granular, brown pigmented debris was common and present both in macrophages and lying free. Perivascular cuffing, primarily with eosinophils, was a consistent feature. Glial foci, occasional microabscesses, a few histocytic nodules associated with parasitic debris or ova, and microcavitation were frequently observed. Lesions were found in all portions of the brain examined, but were most severe in the white matter and at the junction of gray and white matter.

Histologic lesions in the brains of the three clinical cases maintained in captivity were similar to those found in the harvesetd animals except that the degree of inflammatory response was less, especially the eosinophilic response. Sections of spinal cord examined in the clinical cases all had lesions. These were limited to confluent areas of microcavitation and malacia of the white matter, swelling of axis cylinders, and demyelination. Virtually no inflammatory response was noted in sections of spinal cord examined.

DISCUSSION

The increasing prevalence of cerebral nematodiasis found during this study is unexplained. The percentage of *Cervus* sp. that had adult nematodes present in the cranial cavity increased from 1973 to 1975 as did the percentage that had histological lesions attributable to *P. tenuis*. Fewer areas of brain were histologically examined in 1973 than in 1974 and 1975, but prevalence increased each year and cases considered to be positive invariably had lesions distributed throughout the brain.

There is a lack of correlation between the presence of clinical signs and tissue damage as observed with routine histological examination of the brain. While lesions always were found in animals showing signs, of six known animals observed prior to harvest, three had brain lesions but no history of clinical signs. This inconsistency might be attributed to various factors:

1. Our observations and those of Anderson[3] suggest that at least some animals will have periods of apparent normalcy following signs attributable to meningeal worm infection. These periods of normalcy may in part result from the animal learning to compensate for local neurological deficiency.
2. Clinical signs probably depends on the specific areas of brain involved, the extent of the neurologic damage, and the number of invading parasites. Examination of a relatively few small areas of the brain and spinal cord does not provide sufficient information about the total damage present. An understanding of the relationship between pathology and clinical signs may require the systematic examination of the entire brain and spinal cord using specialized staining techniques.
3. Our observations would suggest that clinical cases have a less severe inflammatory cell reaction, but more extensive demyelination and malacia of the brain and/or spinal cord as evidenced by confluent areas of microcavitation. We also have observed this extensive microcavitation with minimal inflammatory cell response in severely affected fallow deer (*Dama dama*). The possibility that the severe central nervous system damage in some individuals of the more susceptible species (wapiti and fallow deer as contrasted with white-tailed deer) may depend more on damage that is immunologically mediated than the inflammatory reaction to the parasite as a foreign body needs to be considered.

Cerebral nematodiasis diagnosed in this herd did not produce a die-off type situation, nor was it a high herd mortality factor. However, the mortality factor may exert an important population

significance because of the apparent greater susceptibility of the younger age-classes to the effects of the parasite. Higher than normal mortality among the younger age-classes would have a detrimental effect on population recruitment and would, at least, limit the growth rate of wapiti herds living in proximity to white-tailed deer herds. Another consideration is that subclinical infections might render animals more susceptible to predation. For example, our sample of "normal" animals may well be biased because animals with subclinical infections may be more exposed to predation in the form of harvest by man.

LITERATURE CITED

1. ALIBASOGLU, M., D. C. KRADEL and H. W. DUNNE. 1961. Cerebral nematodiasis in Pennsylvania deer (*Odocoileus virginianus*). Cornell Vet. 51: 431-441.
2. ANDERSON, R. C., M. W. LANKESTER and U. R. STRELIVE. 1966. Further experimental studies of *Pneumostrongylus tenuis* in cervids. Can. J. Zool. 44: 851-861.
3. ————. 1971. Lungworms. p. 81-126. In: J. W. Davis and R. C. Anderson (eds.). *Parasitic Diseases of Wild Mammals.* Iowa State Univ. Press, Ames, Iowa. 364 p.
4. ————. 1972. The ecological relationships of meningeal worm and native cervids in North America. J. Wildl. Dis. 8: 304-310.
5. CARPENTER, J. W., HELEN E. JORDAN and J. A. MORRISON. 1972. Meningeal worm (*Parelaphostrongylus tenuis*) infection in white-tailed deer in Oklahoma. J. Wildl. Dis. 8: 381-383.
6. ————, ———— and B. C. WARD. 1973. Neurologic disease in wapiti naturally infected with meningeal worms. J. Wildl. Dis. 9: 148-153.
7. FAY, L. O. and J. N. STUHT. 1973. Meningeal worms in association with neurologic disease in Michigan wapiti. 1973 Wildl. Dis. Conf. Papers and Abstracts, No. 24.
8. GILBERT, F. F. 1973. *Parelaphostrongylus tenuis* Dougherty in Maine: 1. The parasite in white-tailed deer (*Odocoileus virginianus,* Zimmerman). J. Wildl. Dis. 9: 136-143.
9. KARNS, P. D. 1966. *Pneumonstrongylus tenuis* from elk (*Cervus canadensis*) in Minnesota. Bull. Wildl. Dis. Ass. 2: 79-80.
10. KRADEL, D. C. and H. W. DUNNE. 1964. Diseases of the brain and spinal cord observed in Pennsylvania deer and other wildlife. N.E. Wildl. Conf., Hartford, Conn. 6 p. (mimeo).

Received for publication 6 August 1976