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EXPERIMENTAL INFECTION OF MULE DEER WITH *Parelaphostrongylus tenuis*

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Abstract: Six adult and three fawn mule deer *(Odocoileus hemionus)* were experimentally infected with a range of 75-100 infective larvae of *Parelaphostrongylus tenuis*. Five of the six adult deer developed clinical signs of neurologic disease that terminated in paralysis between 35 and 80 days. The sixth deer developed slight signs of neurologic disease for 10 days, but recovered. All three mule deer fawns developed neurologic disease.

Adult meningeal worms were recovered from the subdural space of the spinal cord of two fawns. Eggs were observed on the cranial dura mater of one of these fawns, indicating that *P. tenuis* can complete its life cycle provided mule deer can survive the damage resulting from the infection. Neither eggs nor larvae of *P. tenuis* were recovered from the feces or lungs of infected mule deer.

Clinical signs and histologic lesions observed in experimentally infected mule deer resembled those reported in infected moose *(Alces alces americana)*. Two critical periods were apparent in mule deer infected with *P. tenuis:* nematode migration through the spinal neural parenchyma, and penetration of the adult nematodes into the cranial neural parenchyma. While most adult deer were unable to survive the first critical period, fawns survived the first but succumbed to infection during the second critical period.

INTRODUCTION

Parelaphostrongylus tenuis, a neurotropic nematode, is a common parasite of white-tailed deer in eastern North America.[2,7,9,10,16,21,24] While natural infections of *P. tenuis* usually do not cause neurologic disease in white-tailed deer *(Odocoileus virginianus)*,[2,4] natural infections in moose *(Alces alces)*, wapiti *(Cervus canadensis)*, red deer *(Cervus elaphus elaphus)*, caribou *(Rangifer tarandus terraeonae)*, reindeer *(Rangifer tarandus tarandus)*, fallow deer *(Dama dama)*, and black-tailed deer *(Odocoileus hemionus columbianus)* may cause a fatal neurologic disease.[5,8,11,12,16,17,19,22,25]

Although there are no reports of naturally-occurring neurologic disease in mule deer, experimental evidence suggests that *P. tenuis* is pathogenic to mule deer.[8,19] This report presents the results of experimental infections of mule deer with *P. tenuis* and discusses the pathogenesis and pathologic lesions of the parasite in mule deer.

MATERIALS AND METHODS

Seventy-five to 100 infective larvae recovered from snails *(Triodopsis albolabris)* were administered *per os* to each of six adults and three fawns. All adult deer were females two to five years

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of age when infected with *P. tenuis* and all fawns were males, three to 13 weeks old when infected.

Fecal samples from experimental deer were taken prior to infection and examined by the Baermann technique for the presence of *P. tenuis* larvae.[20] Subsequent fecal samples were monitored daily beginning 50 days post-infection to determine the earliest appearance of first-stage larvae in the feces. Infected deer also were observed daily and abnormal behavior or clinical signs of parelaphostrongylosis were recorded. Movies were taken of deer showing neurologic disorders.

Deer were euthanized by electrocution when they were unable to rise from a prone position without assistance. Cerebrospinal fluid was analyzed for total protein content and number and type of cells present. Fecal samples were examined using the Baermann technique for first-stage larvae of *P. tenuis*. Necropsies were performed and tissues collected for histologic interpretation. Spinal cord, brain, and meninges were examined using the method described by Nettles *et al.*[19] The left lobe of the lung was minced in a Universal Number One food and meat grinder, placed in 3.8 l of water for 24 h, and then strained through a 2 mm sieve. Material passing the sieve was set aside for 30 min to allow larvae to settle. The fluid was repeatedly decanted and replaced with tap water until relatively clear. Most of the fluid then was decanted and the remainder with sediment was poured into a 1.1l jar, allowed to settle for 30 min. and decanted. The remaining fluid and sediment were poured into petri dishes and examined with a dissecting microscope for larvae and eggs of *P. tenuis*. One deer (W553) that did not become paralyzed was euthanized at 193 days post-infection and studied similarly.

RESULTS

A summary of the results is given in Table 1.

TABLE 1. Summary of results of experimental infections of mule deer with *P. tenuis*.

Deer*			First neurologic	Paralysis or	CSF Analysis		
Number	Age	No. of infective larvae given	signs (days post-infection)	death (days post-infection)	cell/mm^3	Total/ protein (mg%)	Locality of histopatholgic lesions in CNS
W451	Adult	75	31	36	5	33	Lumbar region of spinal cord
W452	Adult	75	27	38	1	28	Spinal cord
W454	Adult	75	39	44	1	29	Cervical & thoracic regions of the spinal cord
W461	Adult	75	44	54	15	50	Spinal cord
W469	Adult	75	34	79	44	41	Brain stem & spinal cord
W553	Adult	75	66	193	5	28	No significant lesion
W480	Fawn	100	43	66	44	45	Brain
W483	Fawn	75	27	60	64	90	Brain & spinal cord
W487	Fawn	75	18	87	302	164	Brain

*Tissues on file at the Wild Animal Disease Center under case number given.

The time between the onset of clinical signs of parelaphostrongylosis and paralysis was only five to 11 days post-infection (PI) in four adult mule deer (W451, W452, W454, W461). Lameness in a hind leg was the first sign in these deer. They were reluctant to rise and walked gingerly with arched backs and stiff legs. Posterior weakness and ataxia developed within the next day or two. Coordination decreased rapidly and deer could run only a short distance before falling. Soon after posterior weakness and ataxia was noted, deer were found in lateral recumbency unable to lift their heads. Nystagmus was evident in two of these deer.

The remaining mule deer had a less acute reaction to *P. tenuis* and had a remission of clinical signs for varying periods of time. In all but one deer (W553) neurologic signs returned and ended in paralysis. Deer W553 developed lameness in her right hind leg (66 days PI), but 10 days later the lameness disappeared. Clinical signs of neurologic disease were not again noted in this deer.

The earliest appearance of clinical signs was 18 days PI in fawn W487. This fawn showed slight posterior weakness and lameness in his right front leg. Posterior weakness and lameness gradually diminished and disappeared on the 28th day PI. The fawn appeared normal for one week, after which time posterior weakness returned. Posterior weakness gradually increased and the fawn became less and less coordinated. Fifty-two days after the onset of the second set of signs (87 days PI) fawn W487 could not rise without assistance and was euthanized. Although clinical signs appeared earlier in the infection in this fawn than in other infected deer, he survived the infection longer than any deer which became paralyzed.

The first clinical sign in adult deer W469 and fawns W480 and W483 was lameness in one leg (See Table 1 for days PI). Lameness was noted for only one day in fawn W480, for four days in fawn W483, and for 15 days in deer W469. Both fawns and deer W469 had a remission of neurologic signs which lasted five to seven days. However posterior weakness then became apparent and progressive neurologic debilitation ended in paralysis 11 to 19 days after posterior weakness was noted.

While the onset of clinical signs and the time between the onset of signs and paralysis was variable among deer, the sequence of clinical signs was similiar. Clinical signs of parelaphostrongylosis in deer which became paralyzed developed from 18 to 44 days (av. 33 days) PI. Although all deer appeared to be eating, three adult deer (W451, W452, and W469) were quite emaciated at the time of necropsy. All deer responded to audio and visual stimuli, even when paralyzed.

The bladder of one adult deer (W454) was distended. Several petechial hemorrhages were evident on the mucosal surface of the abomasums of two deer (W451 and W469).

Gross lesions were not observed in the central nervous system (CNS) of adult deer. However, cranial subdural hemorrhages were present in all the fawns. Eight immature adult *P. tenuis* were recovered from the subdural space at the C-7 and T-1 levels of the spinal cord of fawn W483. One mature female *P. tenuis* was recovered from the subdural space of the ventrolateral region of C-6, near the dorsal nerve root of fawn W487.

Numerous eggs of *P. tenuis* were recovered from the cranial dura mater of fawn W487, but no egg or larva was recovered from the lungs or feces, respectively, of this deer. Neither eggs nor larvae of *P. tenuis* were recovered from feces, dura mater, or lungs of the other deer. The results of the cerebrospinal fluid (CSF) analysis are listed in Table 1.

The histopathology seen in infected mule deer was similiar to that reported by Anderson[2] in two experimentally infected moose calves. For an excellent and more detailed description of histological

lesions noted in *P. tenuis* infections see Anderson.[4]

The lesions seen in adult deer were less marked than those seen in fawns and were found primarily in the spinal cord. The major lesions apparent in adult deer were cellular infiltration, axonal degeneration, and focal myelomalacia (Figs. 1 & 2). Mononuclear cells, primarily plasma cells and lymphocytes, were noted commonly in the leptomeninges, around dorsal root ganglion, and in the choroid plexus (Fig. 3). Eosinophils were interspersed among lymphocytes and plasma cells in some areas, but were not a prominent feature.

Scattered areas of degenerate axons were present throughout the white matter of the spinal cord in adult deer (Fig. 2). Massive areas of degenerate axons were associated with small hemorrhages, perivascular cuffing, and glial proliferation in both adults and fawns. Immature nematodes were found primarily in the cervical and thoracic regions of the spinal cord of adult deer, and were not associated with a host response. The nematodes were coiled upon themselves displacing neural tissue. Male and female nematodes were found within the neural parenchyma of the brain in fawns, but in only one case was an area of hemorrhage associated with a nematode. Generally little or no inflammatory response was evident in the immediate vicinity of nematodes.

Hemorrhage, focal malacia, and glial scarring were seen more commonly in fawns than in adult deer. In fawns, numerous tracts of degenerate axons, hemorrhage, and gliosis, presumably tracts left by migrating nematodes, were seen in the neural parenchyma of the brain and appeared to parallel blood vessels.

DISCUSSION

The neurologic syndrome seen in mule deer infected with *P. tenuis* was similiar

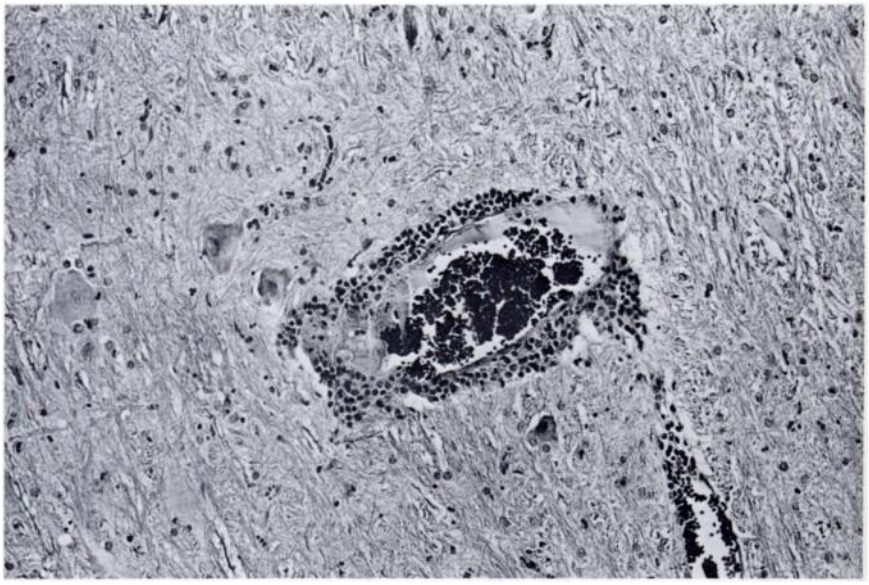

Figure 1. Lymphocytic infiltration in the perivascular region of a blood vessel in the medulla oblongata, deer W454. H&E ×63.

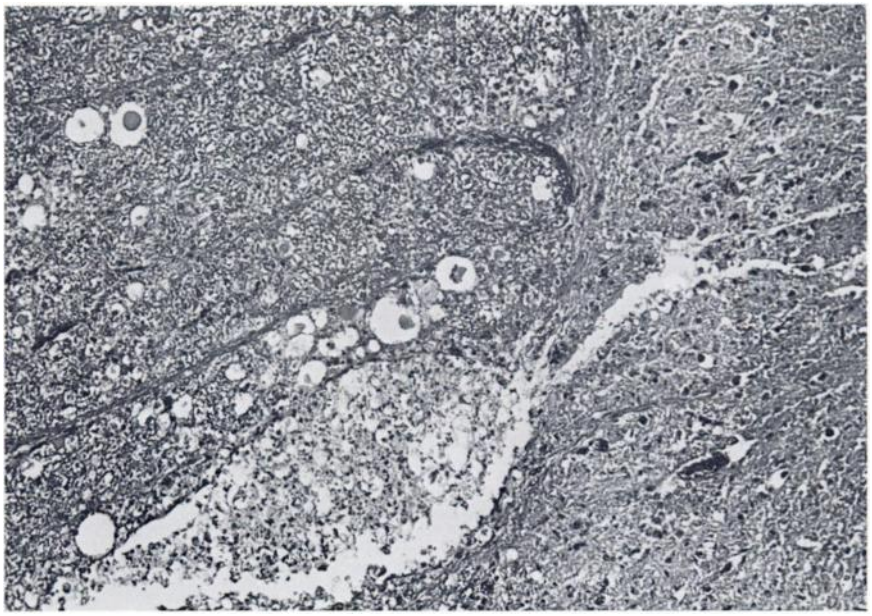

Figure 2. Myelomalacia, degenerate axons, and proliferated glial cells adjacent to one dorsal horn of C-7, deer W461. H&E ×63.

to that reported in other abnormal hosts and characterized by lameness, posterior weakness and ataxia, and progressive neurological deterioration.[2,5,10,17,18,24] Periods of remission noted in infected mule deer are a common feature of *P. tenuis* infection and have been reported in elk, sheep, goats, and one mule deer fawn.[1,7,18]

The onset of clinical signs in mule deer was caused most likely by larval migration through the spinal parenchyma. According to Anderson, immature adults migrate from the neural parenchyma into the subdural space between 25 and 40 days PI in white-tailed deer. This migration is the first critical period that abnormal hosts must overcome.[4,6] Four adult deer (W451, W452, W45r, and W461) succumbed to infection during this first critical period, while the other deer survived it and their clinical signs disappeared. The disappearance of clinical signs probably was due to the entrance of subadults into the subdural space. Nematodes then migrated anteriorly into the craniumn as indicated by the distribution of histologic lesions in deer which survived longer periods of time.

The reappearance of clinical signs in three fawns (W480, W483, and W487) and one adult (W469) possibly was due to the entrance of both male and female nematodes back into the neural parenchyma. The second critical period in mule deer began at an average of 54 days and terminated in paralysis at an average of 71 days PI. Anderson[6] suggested that the intensification of clinical signs leading to paralysis in abnormal hosts is due to the increasing size of the nematodes as well as to their increasing activity.

Deer that became paralyzed after 60 days post-infection had an abnormally high number of mononuclear cells in the cerebrospinal fluid. Since an inflammatory lesion in the meninges generally

produces more abnormalities in the cerebrospinal fluid than an equally severe lesion in the neural tissue, the increased number of cells in the cerebrospinal fluid may be related more to an inflammatory process in the meninges than to one within the neural tissue.[22] This assumption is supported further by Anderson's[4] research with white-tailed deer and *P. tenuis*. He noted that the dura mater became progressively more infiltrated with eosinophils, lymphocytes, and plasma cells as the nematodes moved into the subdural space.

Histologic lesions observed in experimentally infected mule deer were more similar to those previously described in moose and wapiti than to those described in naturally-infected black-tailed deer.[2,7,18] Unlike infected black-tailed deer, neither granulomatous eosinophilic leptomeningitis nor subdural plaques of mineralization were observed. Anderson and co-workers reported a striking lack of cellular infiltrates in the leptomeninges of an experimentally infected mule deer fawn.[7] However, in the present study, infiltrates of mononuclear cells were noted commonly in the leptomeninges of both adult and fawn mule deer. Nettles *et al.*[18] reported aggregates of eosinophils in the spinal cord of naturally-infected black-tailed deer was a common occurrence, while in mule deer, eosinophils were only occasionally interspersed with lymphocytes and plasma cells. Differences between histologic lesions in black-tailed deer and mule deer indicate that black-tailed deer naturally-infected with *P. tenuis* develop a stronger cellular response than mule deer experimentally infected with *P. tenuis*.

Since fawns survived the infection longer than adult deer, the distribution of nematodes and lesions was different. Lesions and nematodes in fawns were

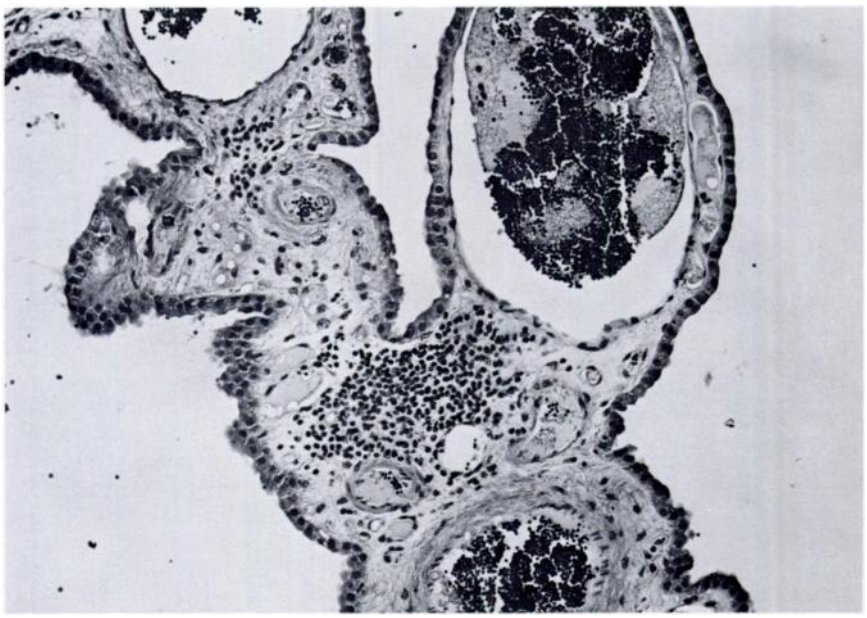

Figure 3. Choroid plexus thickened by fluids, congested blood vessels, and infiltrations of mononuclear cells, deer W461. H&E ×100.

found primarily in the brain. The nematodes found in fawns were larger than those found in adults due to the longer survival time of fawns. Larger nematodes displaced more neural tissue and caused more traumatic damage to the central nervous system than did the smaller, less mature nematodes found in adult deer.

The results of this study indicate that *P. tenuis* could not establish in mule deer populations. The deer (W553) which did not become paralyzed did not shed infective larvae in her feces and no evidence of *P. tenuis* was found in her tissues. An explanation for the recovery of deer W553 might be that she received fewer infective larvae than the other deer and was able to overcome those she did receive. If this were the case it would suggest that mule deer are refractory to low doses of infective larvae. However, mule deer receiving 75 or more infective larvae were killed by the parasite before first stage larvae hatched. Presently, there is no evidence that infective larvae could be passed to the extent that transmission could ever become established in either mule deer or black-tailed deer populations.

The results of this study also indicate that transplants of mule deer into areas inhabited by infected white-tailed deer would be unsuccessful. *P. tenuis* has been found to be a major limiting factor for moose, black-tailed deer, elk, reindeer, and a suspected limiting factor for caribou.[6,7,8,11,16,19,25] The investigation fully agrees with the recommendation of Anderson[5] and Nettles *et al.*[19] that the presence of *P. tenuis* in white-tailed deer be a major consideration before big game species are relocated.

LITERATURE CITED

1. ALDEN, C., F. WOODSON, R. MOHAN, S. MILLER. 1975. Cerebrospinal Nematodiasis in Sheep. J. Am. vet. med. Ass. 166: 784-786.
2. ANDERSON, R.C. 1963. The incidence, development, and experimental transmission of *Pneumostrongylus tenuis* Dougherty *(Metastrongyloidea: Protostrongylidae)* of the meninges of the white-tailed deer *(Odocoileus virginianus borealis)* in Ontario. Can. J. Zool. 41: 775-792.
3. ———. 1965. Neurologic disease in moose infected experimentally with *Pneumostrongylus tenuis* from white-tailed deer. Path. Vet. 1: 289-322.
4. ———. 1965. The development of *Pneumostrongylus tenuis* in the central nervous system of white-tailed deer. Path. Vet. 2: 360-379.
5. ———. 1971. Lungworms. Pp. 83-81. In: *Parasitic Diseases of Wild Mammals.* J.W. Davis and R.C. Anderson, eds. Iowa State Univ. Press, Ames.
6. ———. 1971a. Neurologic disease in reindeer *(Rangifer tarandus tarandus)* introduced into Ontario. Can J. Zool. 49: 159-166.
7. ———. 1972. The ecological relationship of meningeal worm and native cervids in North America. J. Wildl. Dis. 8: 304-310.
8. ———, M.W. LANKESTER and U.R. STRELIVE. 1966. Further experimental studies of *Pneumostrongylus tenuis* in cervids.
9. BEHREND, D.F. and J.K. WITTER. 1968. *Pneumostrongylus tenuis* in white-tailed deer in Maine. J. Wildl. Manage. 32: 963-966.
10. BINDERNAGEL, J.A. and R.C. ANDERSON. 1972. Distribution of the meningeal worm in white-tailed deer in Canada. J. Wildl. Manage. 36: 1349-1353.
11. CARPENTER, J.W., H.E. JORDAN and B.C. WARD. 1973. Neurologic disease in wapiti naturally infected with meningeal worms. J. Wildl. Dis. 9: 148-153.

12. DAUPHINE, T.C. 1976. The disappearance of caribou reintroduced to Cape Breton Highlands National Park. Can. Field-Naturalist 89: 299-310.

13. FENSTERMACHER, R. 1934. Diseases affecting moose. The Alumni Quarterly (Minnesota) 22: 81-94.

14. ———. 1934a. Further studies of diseases affecting moose. Univ. Minn. Agric. Expt. Sta. Bull. No. 308, 26p.

15. ———. 1937. Further studies of diseases affecting moose. II. Cornell Vet. 27: 25-37.

16. GILBERT, F.F. 1973. *Parelaphostrongylus tenuis* (Dougherty) in Maine: I. The parasite in white-tailed deer *(Odocoileus virginianus,* Zimmerman). J. Wildl. Manage. 9: 136-143.

17. KISTNER, T.P., G.R. JOHNSON, and G.A. RILLING. 1977. Naturally occurring neurologic disease in fallow deer infected with meningeal worms. J. Wildl. Dis. 13: 55-58.

18. MAYHEW, I.G., A. DE LAHUNTA, J.R. GEORGI and D.G. ASPROS. 1976. Naturally occurring cerebrospinal parelaphostrongylosis in goats. Cornell Vet. 66: 56-72.

19. NETTLES, V.F., A.K. PRESTWOOD and R.G. NICHOLS. 1977. Meningeal worm-induced neurologic disease in black-tailed deer. J. Wildl. Dis. 13: 137-143.

20. PRESTWOOD, A.K. 1972. *Parelaphostrongylus tenuis* sp. n. (Metastrongyloidea: Protostrongylidae) from the musculature of white-tailed deer *(Odocoileus virginianus).* J. Parasit. 58: 897-902.

21. ———, and J.K. SMITH. 1969. Distribution of meningeal worm *(Pneumostrongylus tenuis)* in deer in the southeastern United States. J. Parasit. 55: 720-725.

22. SMITH, H.J. and R.M. ARCHIBALD. 1967. Moose sickness, a neurologic disease of moose infected with the common cervine parasite, *Elaphostrongylus tenuis.* Can. Vet. J. 8: 173-177.

23. STEINBERG, S.A. 1969. Pp. 166-180. In: Textbook of Veterinary Clinical Pathology. W. Medway, J.E. Prier and J.S. Eilkinson, eds. The Williams and Wilkinson Co., Baltimore.

24. THURSTON, D.R. and R.G. STROUT. 1978. Prevalence of meningeal worm *(Parelaphostrongylus tenuis)* in white-tailed deer from New Hampshire. J. Wildl. Dis. 14: 89-96.

25. WOOLF, A., C.A. MASON and D. KRADEL. 1977. Prevalence and effects of *Parelaphostrongylus tenuis* in a captive wapiti population. J. Wildl. Dis. 13: 149-154.

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