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MENINGEAL WORM-INDUCED NEUROLOGIC DISEASE IN BLACK-TAILED DEER[†]

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Abstract: Neurologic disease attributable to *Parelaphostrongylus tenuis* was diagnosed in five black-tailed deer (*Odocoileus hemionus columbianus*) relocated from Oregon to Tennessee. Mortality occurred in the pre-release enclosure and in the release area. Infection with *P. tenuis* was considered the cause of an unsuccessful stocking attempt. In addition, neurologic disease was produced by experimental infection of a black-tailed x white-tailed deer hybrid. Clinical and pathologic findings were described for black-tailed and hybrid deer, and epizootiologic aspects of *P. tenuis* infections were discussed.

INTRODUCTION

Neurologic disease caused by infection with meningeal worm, *Parelaphostrongylus tenuis*, has been reported from a variety of wild and domestic ruminants. The naturally-occurring disease has been well documented in moose (*Alces alces*),^{2,13} caribou (*Rangifer tarandus terranova*),¹⁴ reindeer (*R. t. tarandus*),³ wapiti (*Cervus canadensis*),⁷ domestic goats (*Capra hircus*)⁹ and sheep (*Ovis aries*).^{1,10,16} Although neurologic disease was induced in a mule deer (*Odocoileus hemionus*) by administration of infective *P. tenuis* larvae,⁶ naturally-occurring neurologic disease in mule deer is unknown. The present report describes naturally-occurring cerebrospinal nematodiasis caused by *P. tenuis* in black-tailed deer (*Odocoileus hemionus columbianus*) introduced into the state of Tennessee and neurologic disease experimentally induced by *P. tenuis* in a hybrid black-tailed x white-tailed deer.

HISTORY

In 1966 and 1967, the Tennessee Wildlife Resources Agency obtained 68 black-tailed deer fawns from Oregon for use in an experimental relocation program. It was thought that black-tailed deer would be desirable big game animals and could thrive in the urbanized areas of east Tennessee where restocking white-tailed deer had failed.

After importation, the black-tailed deer were released into a 1,860 ha enclosure on the Volunteer Ordnance Depot near Chattanooga, Tennessee. They adapted well to the habitat and reproduced successfully. Although the enclosure initially was devoid of white-tailed deer, a few white-tailed deer came over the 2.5 m chain-link fence and entered the enclosure. During this period, hybridization of black-tailed and white-tailed deer was investigated in small enclosures located on the Cheatham Wildlife Area near Nashville and the Bufalo Springs Game Farm near Rutledge,

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Tennessee. Black-tailed deer readily hybridized with the white-tailed deer and produced fertile offspring that were not easily recognized as hybrids.¹⁵

By fall of 1973, the population in the Volunteer Ordnance Depot had increased to over 400 black-tailed deer, the cross-breeding experiments had resolved the possible question of sterile hybrids, and problems with diseases had not been encountered. Capture operations were initiated on 28 February, 1973, to stock 46 of the pen-raised black-tailed deer into Hamblen County, Tennessee, an area with extremely few white-tailed deer.

On 2 March, 1973, the first instance of neurologic disease was observed in the Volunteer Ordnance Depot. An adult female black-tailed deer was found in moribund condition. It was evident that the doe had been violently thrashing about in right lateral recumbency for several days. She died shortly after discovery. No further instances of neurologic disease have been observed in the Ordnance Depot. The population presently has stabilized at approximately 400 black-tailed deer.

The 46 black-tailed deer released in Hamblen County were monitored closely by observations and radio telemetry. They settled in the general vicinity of the release site, and reproduction during the first year was good. An ingress of white-tailed deer into Hamblen County also occurred that year. In April of the following year (1974), a male black-tailed deer showing signs of incoordination was captured by hand. The deer's health rapidly deteriorated, with paralysis preceding death. During February and May of 1975, three more affected animals were observed. Incoordination was evident in each animal. One of these deer drowned while attempting to swim a lake, a second died shortly after it was attacked by a large dog, and a third deer was captured by hand. The latter developed tetraplegia and nystagmus within 24 h. Several other dead black-tailed deer were found that were too badly decomposed for necropsy. In spite of good reproduction during 1973, 1974, and 1975, the black-tailed deer population has steadily declined while the

white-tailed deer population has increased. Only a few black-tailed does and hybrid deer are known to be alive at present.

MATERIALS AND METHODS

Natural Infections

Each of the five aforementioned black-tailed deer were submitted for examination; two were live, two were frozen, and one had died 24 h. previously. Living deer were observed for a short period and then killed. At necropsy, brains were removed with the dura intact. After removal, the dura was incised and gently reflected. Spinal cord with meninges intact, brain, and cranial dura were placed in a large quantity of 10% buffered formalin and allowed to fix for a minimum of 2 weeks. After fixation, nervous tissue was examined for helminths with the aid of a dissecting microscope (3-30X). Tissue samples were taken from the cerebral cortex, cerebellum, brain stem, spinal cord, cranial meninges and visceral organs. In three cases, spinal cord was sampled at each spinal nerve root to study lesion distribution. Samples were embedded in paraffin, sectioned at 7 μ m, and stained with hematoxylin and eosin.

Experimental Infection

On 6 June 1974, a 5-day old female hybrid fawn ($\frac{3}{4}$ black-tailed x $\frac{1}{4}$ white-tailed deer) was transferred from the Volunteer Ordnance Depot enclosure to the Laboratory Animal Medicine facility, College of Veterinary Medicine, University of Georgia. The fawn was reared and maintained helminth-free on concrete, and was infected when 6 months old.

Origin of infective material was first-stage larvae collected by the Baermann technique from white-tailed deer feces obtained at Berry College, Floyd County, Georgia. A previous parasitologic survey of five deer from that area had shown the Berry College deer herd to be heavily infected with meningeal worm, *Parelaphostrongylus andersoni*, the deer musclemoorm which produces first-stage

larvae indistinguishable from *P. tenuis*, was not found in any of the deer necropsied. Laboratory-reared *Mesodon perigraptus* were used to obtain infective larvae by methods similar to those described previously.¹¹ One hundred and fifty third-stage larvae were given to the hybrid fawn and a similarly aged helminth-free white-tailed deer. Feces were monitored weekly by the Baermann technique, and whole blood in EDTA was collected from the hybrid fawn at weekly intervals. The hybrid deer was killed 52 days after infection and examined in a manner similar to that described for the black-tailed deer.

RESULTS

Natural Infections

The two living deer were unable to rise from lateral recumbency. Periods of violent thrashing, extension of the neck, and nystagmus were observed in each animal. Flexor (withdrawal) reflexes were present in all limbs and each deer reacted to visual stimuli. Blood values for these deer are presented in Table 1.

At necropsy, lacerations and abrasions of the legs and skin overlying bony prominences of the head and body were found routinely. Distention of the urinary bladder, constipation, and localized bronchopneumonia due to *Dictyocaulus viviparus* were frequently observed. The deer that drowned had a diffuse pulmonary emphysema attributed to laryngospasm. Various methods for detecting protostrongylid eggs and first-stage lar-

vae, viz., direct lung smears, Baermann examinations of lungs and feces, and histologic examination of lung tissue were all negative.

The most prominent gross lesions in the central nervous system were multiple plaques of pale green caseous exudate on the inner surface of cerebral and spinal dura mater and an apparent excess of cerebrospinal fluid. Similar exudate often was present in the perineurium surrounding the spinal nerve roots. Diffuse subdural hemorrhage overlying the frontal areas of the cerebrum was evident in one deer.

Immature female *P. tenuis* were recovered intact from three deer. In addition, three degenerated nematode larvae were found in the fixative solution which had been used to preserve the spinal cord of a fourth deer. Most helminths were located in the meninges of the brain or cervical spinal cord, but one was found in the lateral ventricle and another within the optic nerve (Fig. 1). A maximum of three worms was recovered from a single deer.

Microscopically, each deer had subacute to chronic granulomatous eosinophilic leptomeningitis and spinal root perineuritis (Fig. 2). Lesions generally were more diffuse and severe in the cervical and cranial thoracic spinal cord, with more focal distribution in the cerebral and lumbar meninges. Inflammatory cells consisted of a mixture of lymphocytes, plasma cells, macrophages, and eosinophils. The proportions of these cell types varied widely, with collections of

TABLE 1. Blood values for two male black-tailed deer and one female blacktail-whitetail hybrid deer afflicted with terminal neurologic disease caused by *Parelaphostrongylus tenuis*.

Deer Number	Hematocrit (Percent)	Leucocytes (10 ³ /mm ³)	L	N	M	E	B
			Differential (%)				
Black-tailed Deer							
2	69	3.1	43	55	1	1	0
5	50	2.9	33	59	6	2	0
Blacktail-Whitetail Hybrid							
910	45	4.4	60	39	0	1	0

lymphocytes, plasma cells, and a scattering of eosinophils being most frequent. Massive numbers of eosinophils also were common, either interspersed among other cell types or in pure populations. The latter finding routinely was

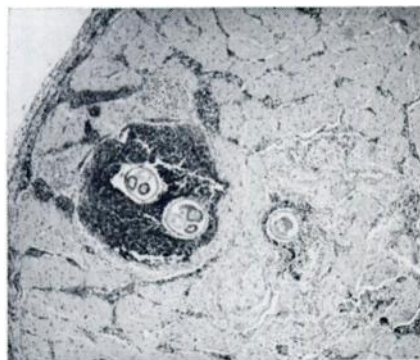


FIGURE 1. *Parelaphostrongylus tenuis* in a cross-section of optic nerve from a black-tailed deer. Leukomalacia and a heavy infiltration of eosinophils are present. H & E stain X 37.

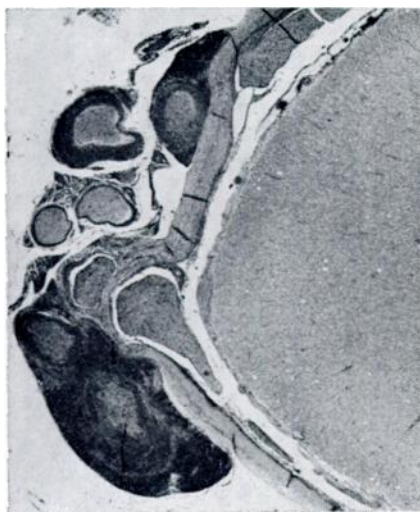


FIGURE 2. Cross-section of spinal cord and spinal nerve roots (T-2) from a black-tailed deer in which severe perineuritis and mild leptomeningitis are present. H & E stain X 15.

associated with degenerated nematodes or with brightly staining amorphous eosinophilic debris. Fibroplasia and focal foreign body granulomas frequently were observed. Thin subdural plaques of mineralization were seen in many areas.

Lesions of brain and spinal cord parenchyma were multiple foci of malacia, gliosis, and microhemorrhage. Associated with these areas were perivascular cuffs of lymphocytes and eosinophils. Malacic areas often appeared linear, which was suggestive of helminth migration. Phagocytic cells containing hemosiderin occasionally were found in these tracts. Parenchymatous lesions in spinal cord commonly were in the cervical and cranial thoracic regions and were less severe than those of the brain stem and cerebrum. In one animal, however, the entire center of a cervical spinal cord segment was malacic. In the brain stem and cerebrum, lesions were most common in white matter, although a few granulomatous nodules were seen in gray matter of the cerebrum. Large accumulations of eosinophils were present in the choroid plexus of two deer.

Experimental Infection

Forty-seven days after infection the hybrid deer developed kyphosis and weakness in the hindquarters. The deer became progressively less coordinated, with complete loss of hindlimb control and partial paralysis of the forelimbs occurring within 5 days. A mild eosinophilia of 357 to 670 cells/mm³ (5 to 11%) occurred 2 to 7 weeks post-infection, but eosinophilia was absent prior to necropsy (Table 1). Cerebrospinal fluid collected 18 h. before necropsy was clear and contained 57 mg% protein. Erythrocytes and leukocytes were 215 and 327/mm³, respectively. A fecal sample taken 47 days post-infection was negative for protostrongylid larvae.

At necropsy the animal was extremely dehydrated. Traces of serous fluid were present in the abdominal and thoracic cavities. The liver was heavily scarred and had large cyst-like bile ducts containing viscous bile. Complete obstruction of the bile duct was not evident

since similar bile was present in the small intestine. Gross lesions were not found in other visceral organs. Examination of the longissimus dorsi and psoas muscles failed to reveal muscleworm.



FIGURE 3. Larval *Parelaphostrongylus tenuis* in the central canal of the spinal cord (T-4) of a black-tailed x white-tailed deer hybrid. H & E stain X 160.

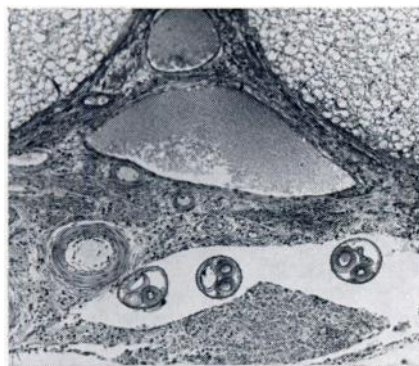


FIGURE 4. *Parelaphostrongylus tenuis* located in a non-suppurative inflammatory reaction on the spinal leptomeninges (L-5) of a black-tailed x white-tailed deer hybrid. H & E stain X 118.

Although gross lesions were not observed in the brain, five immature *P. tenuis* were recovered from the cerebral meninges. Dissection and subsequent histologic examination of the spinal cord revealed an additional 13 worms. Eleven of these were located between the dura and the leptomeninges, but one was found beneath the leptomeninges and another was located in the central canal (Fig. 3).

Histopathologic changes in the brain were mild, consisting of occasional perivascular cuffing with lymphoid cells. More outstanding was a subacute, non-suppurative spinal meningitis, with inflammatory reaction most severe in thoracic and lumbar areas (Fig. 4). The leptomeninges contained numerous histiocytes, lymphocytes, and fibroblasts in a fibrinous matrix. Eosinophils were present but not numerous. In many areas, diffuse or nodular aggregations of lymphoid cells were present in the extradural fat around spinal nerve roots. Inflammation within the spinal nerve roots, as seen in the black-tailed deer, was not observed. Moderate to severe perivascular cuffing, small areas of axonal degeneration and occasional glial nodules were observed in the spinal cord. Focal granulomas were located in the central canal and in white and gray matter.

Diffuse cholangiohepatitis characterized by bile duct hyperplasia, portal fibrosis, and infiltration of numerous eosinophils and neutrophils was evident in the liver. The lungs contained a few protostrongylid eggs and larvae surrounded by mild mononuclear cell inflammatory response.

DISCUSSION

The neurologic disease syndrome in black-tailed deer was similar to that observed in previous reports for other abnormal hosts. Common features were: progressive neurologic debilitation; an apparently non-selective damage to neural parenchyma accompanied by a non-suppurative host reaction; and the presence of *P. tenuis* in low numbers.

It is doubtful that black-tailed deer or black-tailed x white-tailed deer hybrids

are suitable definitive hosts for *P. tenuis* since only immature fifth-stage worms were found when fatalities occurred. Parasite maturation may be more complete in afflicted moose and reindeer, as protostrongylid eggs and larvae occasionally have been recovered from the meninges and lungs of these animals.^{2,3} Recently, unidentified protostrongylid larvae were found in the feces of woodland caribou (*Rangifer tarandus caribou*) which may have originated from a neurotropic *Elaphostrongylus* sp.⁸ Until the identity and host range of this new parasite is resolved, the ability of many wild cervids to act as host for *P. tenuis* will be difficult to establish.

Protostrongylid eggs and larvae observed in the lung sections of the hybrid deer were attributed to *P. andersoni* since the white-tailed deer fawn given the same material became infected with both *P. tenuis* and *P. andersoni*. Despite prior negative muscle examinations in five animals, the presence of *P. andersoni* in the Berry School deer population exemplifies the obscurity of this parasite in the host. Based on this presumptive evidence (eggs and larvae in the lungs), black-tailed x white-tailed deer hybrids seem capable of serving as hosts for *P. andersoni*.

Mortalities resulting from meningeal

worm infection apparently were a significant factor in the black-tailed deer's inability to establish a population in Hamblen County, Tennessee. The potential of *P. tenuis* as a primary limiting factor has been obvious for moose and suspected for elk and caribou.^{4,5} The results of this investigation indicate that black-tailed deer can be added to that list of species. Anderson^{4,5} viewed the westward encroachment of white-tailed deer into mule deer range in Canada with concern since a mule deer was susceptible to experimental infection. The natural occurrence of *P. tenuis*-induced neurologic disease in a mule deer subspecies, as reported herein, is indicative that more research is needed to determine the disease potential in mule deer.

The presence of meningeal worm in white-tailed deer should be a determining factor in decisions of big game relocation or introduction. Diagnosis of meningeal worm infection can be made only by postmortem examination since infection with the closely related helminth, *P. andersoni*, results in identical larvae being shed in the feces.¹² Particular care should be taken to prevent relocation of infected white-tailed deer into non-infected areas. The introduction of susceptible species into the range of infected white-tailed deer also is not advised.

LITERATURE CITED

1. ALDEN, C., F. WOODSON, R. MOHAN and S. MILLER. 1975. Cerebrospinal nematodiasis in sheep. J. Am. vet. med. Ass. 166: 784-786.
2. ANDERSON, R. C. 1965. An examination of wild moose exhibiting neurologic signs, in Ontario. Can. J. Zool. 43: 635-639.
3. ———. 1971a. Neurologic disease in reindeer (*Rangifer tarandus tarandus*) introduced into Ontario. Can. J. Zool. 49: 159-166.
4. ———. 1971b. Recent advances in our knowledge of neurologic disease caused by helminths. 2nd Intl. Conf. on Wildl. Dis., Univ. of Sussex, England. July 18-22.
5. ———. 1972. The ecological relationships of meningeal worm and native cervids in North America. J. Wildl. Dis. 8: 304-310.
6. ———, M. W. LANKESTER and U. R. STRELIVE. 1966. Further experimental studies of *Pneumostrongylus tenuis* in cervids. Can. J. Zool. 44: 851-861.
7. 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.

8. LANKESTER, M. W., V. J. CRICHTON and H. R. TIMMERMAN. 1976. A protostrongylid nematode (Strongylida: Protostrongylidae) in woodland caribou (*Rangifer tarandus caribou*). Can. J. Zool. 54: 680-684.
9. MAYHEW, I. G., A. deLAHUNTA, J. R. GEORGI and D. G. ASPROS. 1976. Naturally occurring cerebrospinal parelaphostrongylosis. Cornell Vet. 66: 56-72.
10. NIELSEN, S. W. and J. AFTOSMIS. 1964. Spinal nematodiasis in two sheep. J. Am. vet. med. Ass. 144: 155-158.
11. PRESTWOOD, A. K. 1972. *Parelaphostrongylus andersoni* sp. n. (Metastrongyloidea: Protostrongylidae) from the musculature of white-tailed deer (*Odocoileus virginianus*). J. Parasit. 58: 897-902.
12. ———, V. F. NETTLES and F. E. KELLOGG. 1974. Distribution of muscle-worm, *Parelaphostrongylus andersoni*, among white-tailed deer of the southeastern United States. J. Wildl. Dis. 10: 404-409.
13. SMITH, H. J., R. M. ARCHIBALD and A. H. CORNER. 1964. Elaphostrongylosis in maritime moose and deer. Can. Vet. J. 5: 287-296.
14. TRAINER, D. O. 1973. Caribou mortality due to the meningeal worm (*Parelaphostrongylus tenuis*). J. Wildl. Dis. 9: 376-378.
15. WHITEHEAD, C. J. 1972. A preliminary report on white-tailed and black-tailed deer cross breeding studies in Tennessee. Proc. Ann. Conf. Southeastern Assoc. Game and Fish Commissioners 25: 65-69.
16. WHITLOCK, J. H. 1952. Neurofilariosis, a paralytic disease of sheep: II. *Neurofilaria cornellensis*, n. g., n. sp. (Nematoda, Filarioidea), a new nematode parasite from the spinal cord of sheep. Cornell Vet. 42: 125-132.

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