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Source: Journal of Wildlife Diseases, 8(4) : 304-310

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-8.4.304

THE ECOLOGICAL RELATIONSHIPS OF MENINGEAL WORM AND NATIVE CERVIDS IN NORTH AMERICA*

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Abstract: The author reviews the relationship of meningeal worm *(Parelapostrongylus tenuis)* and its usual host, the white-tailed deer *(Odocoileus virginianus)*. Important alterations in the environment in the past 100 years have greatly expanded the northern range of white-tailed deer and brought host and parasite into contact with other native cervids such as moose *(Alces americana)*, mule deer *(Odocoileus hemionus)*, and woodland caribou *(Rangifer tarandus)* in which meningeal worm is highly pathogenic. There is evidence the parasite is spreading westward with deer in the aspen-parklands of Canada. Meningeal worm can apparently have considerable impact on moose populations in endemic areas. Possibly the existence of clinical disease in moose in an area should be regarded as evidence of a much more widespread disease problem which may have eventually a serious impact on the population.

INTRODUCTION

Pryadko and Boev[21] have now transferred meningeal worm to the genus *Parelaphostrongylus* and thereby settled a long-standing controversy concerning the generic position of this helminth. Therefore, the correct name for this worm is now *Parelaphostrongylus tenuis* (Dougherty, 1945) Pryadko and Boev, 1971. The following names are synonyms: *Pneumostrongylus tenuis, Odocoileostrongylus tenuis, Elaphostrongylus tenuis* and *Neurofilaria cornellensis.*

This review emphasizes the problem of the epizootiology of meningeal worm and how this relates to members of the deer family in North America.

The Parasite and White-tailed Deer

The usual host for meningeal worm is the white-tailed deer *(Odocoileus virginianus)* which tolerates the parasite well. Indeed, there are apparently only two published cases of clinical disease associated with the presence of the parasite in this deer.[12,19] Experimentally, it has not been possible to produce important clinical disease by giving fawns large numbers of infective larvae.[1,3]

The adult parasite inhabits the cranial venous sinuses and the subdural space. Eggs are generally deposited into the venous blood and are carried to the lungs where they embryonate into first-stage larvae which pass up the respiratory tract, are swallowed, and eliminated with the feces.[1] Larvae occur only in the mucous coat of the fecal pellet and they are resistant to freezing temperatures and desiccation. The larvae penetrate into the foot of terrestrial molluscs which generally abound on deer range, and reach the infective stage in 3-4 weeks at summer temperatures. In southern Ontario the main intermediate hosts are apparently *Deroceras laeve*, the small, annual, ubiquitous native slug, and *Zonitoides nitidus*, a small, common native snail. In northern Ontario, the latter species is probably replaced by *Z. arboreus* as a suitable host.[18]

* Presented at the 2nd International Conference on Wildlife Disease, University of Sussex, England, July 18-22, 1971.

There is no evidence aquatic snails play a role in field transmission although experimentally some species can be infected. First-stage larvae quickly fall off fecal pellets dropped in water and since they are rapidly dispersed would not readily be available for ingestion by aquatic snails. Also, penetration of the foot of gastropods is dependent upon the presence of a film of moisture and larvae cannot progress when totally immersed. For these and other reasons, transmission in the field is presently believed dependent upon terrestrial gastropods, especially *Deroceras laeve* and *Zonitoides* spp. It is interesting that larvae fail to develop satisfactorily in certain common introduced gastropods such as *Arion circumscriptus* and *Deroceras reticulatus*.[18]

Studies have shown infective larvae can survive winter in gastropods in both the southern deciduous forests and the more northerly hardwood-coniferous forests of Ontario. Also, development is greatly retarded in estivating and hibernating gastropods but continues when the latter become active again. Presumably, development ceases during dry periods and with the approach of winter and only continues with the arrival of warm wet weather. Snails do not acquire an immunity to infection and it is possible to superimpose one infection on the other.[18]

Deer become infected by accidentally ingesting gastropods containing small numbers of infective larvae. An average of only 2.9 larvae was found in 426 wild infected gastropods in an endemic area in southern Ontario (Navy Island). Suitable sampling techniques are required to appreciate the abundance and ubiquity of terrestrial gastropods on deer range. They prefer low, cool, moist places and are generally not obvious to the untrained observer. At night, and during moist, cool weather, they emerge from leaf litter, and hiding places near the bases of plants and crawl over the vegetation, often at considerable heights above the ground, where they are readily available to feeding deer.

Transmission of meningeal worm takes place during feeding and it is important to know the feeding habits of deer in relation to the presence or absence of meningeal worm and the availability of molluscs in any particular habitat. Studies in Ontario[15] have shown that certain parts of a deer range may be more suitable than others for molluscs and for transmission of meningeal worm as judged from populations of molluscs present and the numbers in the population harbouring larvae of meningeal worm. Thus a low damp forest had many more molluscs and a much higher prevalence of meningeal worm larvae than a dry, elevated forest. A grassy field had relatively few molluscs but the prevalence of larvae in them was high and, since deer fed a great deal in this field, it was probably an important focus of transmission to the herd. Probably any particular range of a deer herd has its own special peculiarities as it relates to feeding and movements of the herd and transmission of meningeal worm. Much more field work is necessary. The importance of yarding areas in transmission is still unknown but there is ample evidence many fawns become infected within the first 6 months of their lives.[5] The prevalence of infection in herds is sometimes astonishingly high in many parts of eastern North America where deer have been examined. It is estimated, for example, that in Algonquin Park, Ontario, 80% of yearling and adult animals pass larvae in their feces. Karns[14] has provided evidence that prevalence of the parasite in Minnesota is dependent on deer density.

Little is known about immunity in deer infected with meningeal worm. There is limited experimental evidence from deer given large numbers of larvae that this host has a good resistance to infection and that chronicity is reached in a few months.[3] It is sometimes possible to increase the output of first-stage larvae in feces by giving heavy doses of infective larvae to chronically infected deer. The effects of (a) small numbers of infective larvae (2-3) on the production of first-stage larvae and (b) re-infection, have not been investigated.

The white-tailed deer is a highly adaptable animal most typical of the deciduous forest biome (Fig. 1). Its northern range has expanded remarkably during the present century largely as a result of human activities which have in many regions broken up the mature deciduous-coniferous forest and the adjacent coniferous

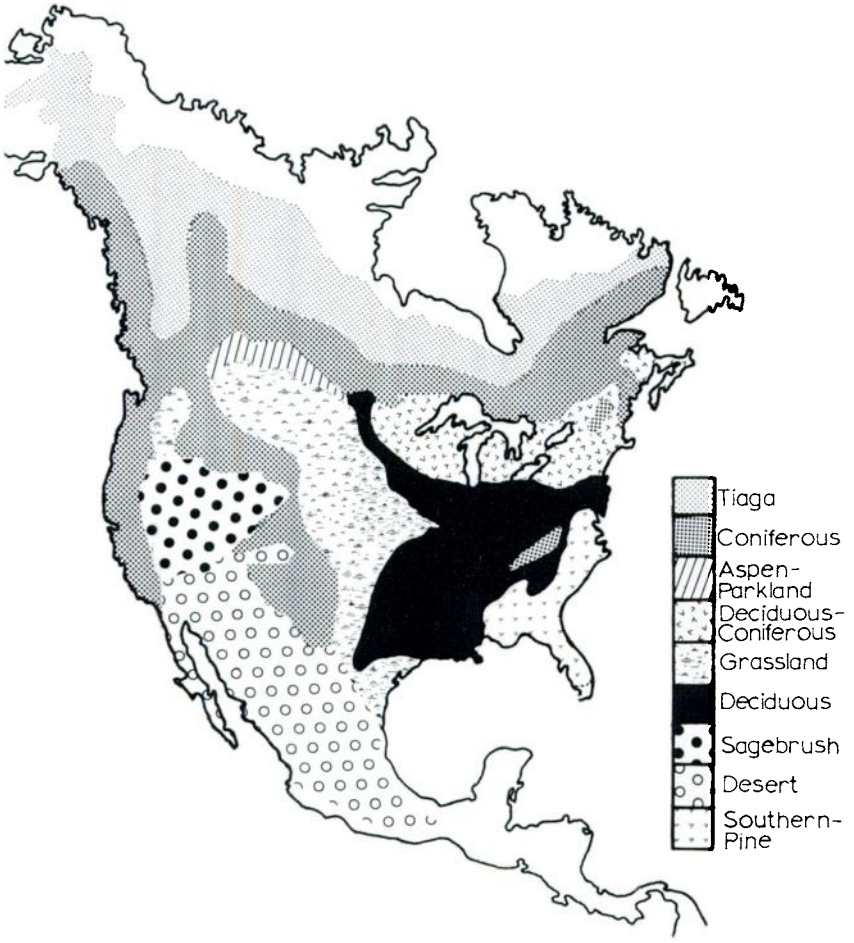

FIGURE 1. A schematic map of the major biomes of North America. Adapted from Ecology and Field Biology by Robert L. Smith, Harper and Row 1966.

forest. In regions of substantial snowfall, whitetails require ample winter browse and cover provided by conifers. They flourish in a decidous-coniferous forest turned to an earlier succession and broken by fire, agriculture and forestry. In the northern parts of its range browse of deciduous trees, shrubs, and conifers such as balsam fir and cedar, form important winter food and cover. At other times of the year deer consume a variety of herbaceous plants, shrubs and grasses. In the southern regions of its range acorn mast may form an important part of the winter food.

Within the past four decades the whitetail has replaced moose as the dominant cervid in Nova Scotia and Maine. Once rare west of Ontario, it now replaces mule deer *(Odocoileus hemionus)* as the dominant deer in many parts of the aspen - parklands. It is apparently not known if this western increase in whitetails is the result of the buildup of small local populations or a gradual spread of populations from the deciduous-coniferous

ecotone in the region of Minnesota and southern Manitoba but the former is most likely.

Meningeal worm is virtually absent in deer of the coastal plain region of southeastern North America, an area which apparently has suitable intermediate hosts.[20] There are no reports of meningeal worm in the grassland biome. The parasite has now been found in white-tailed deer in the aspen parklands of Manitoba and eastern Saskatchewan.[10] Although the grasslands might act as a barrier to the westward spread of the parasite, the aspen parklands could serve as a corridor by which the parasite could reach western North America. Before settlement the climax deciduous-coniferous forest in northern Minnesota and southern Manitoba may have acted as a barrier to the spread of infected deer from the deciduous forest biome into the aspen parklands. Further field work is necessary in this region of North America. There is naturally some concern that meningeal worm might eventually spread to the eastern slopes of the Rocky Mountains with its rich and diverse big game fauna.

In white-tailed deer, infective larvae of meningeal worm migrate, after ingestion, to the spinal cord where they develop to adulthood in the dorsal horns of grey matter. They then migrate out of the neural parenchyma and into the spinal subdural space and from there to the cranium.[3] Most worms enter the venous sinuses by penetrating the dura mater. The entire part of the life cycle in deer (from infective larvae to the appearance of first-stage larvae in the feces) is almost exactly 3 months. Meningeal worm behaves similarly in moose *(Alces americana)*, woodland caribou and European reindeer *(Rangifer tarandus)*, wapiti *(Cervus canadensis)*, and mule deer. However, worms cause excessive trauma to the central nervous system and even small numbers may result in neurologic disease often terminating in paraplegia and death in these animals. Moose, woodland caribou, and reindeer exhibit little resistance to infection and seem particularly susceptible to neural invasion. These animals must become infected in the same way as white-tailed deer, by the ingestion of terrestrial gastropods containing infective larvae. Although moose may take a variety of aquatic plants, and caribou and reindeer lichen, all these species may feed on the same vegetation as deer during certain times of the year.

The Parasite and Moose

In many regions of eastern North America, ranges of white-tailed deer and moose overlap, especially in the young mixed deciduous-coniferous forests largely created by human activities. In many of these regions neurologic disease is common in moose. "Moose sickness" has been associated with marked declines in moose populations in Nova Scotia and New Brunswick during the 1940's and 50's and similar experiences have been reported in Maine and Minnesota.[9,14,17] The disease is common in southwestern Ontario[2,4] and its impact on moose populations is now being studied in this area. The moose is peculiarly adapted to the coniferous forest biome and the tiaga ecotone and it can tolerate greater snow depths than deer. Thus, moose neurologic disease may only be a problem in regions where white-tailed deer can do well since there is little evidence the parasite can establish itself in moose populations; further studies are required, however, since Karns and Jordan[15] reported larvae in feces of moose on Isle Royale. Unfortunately, much excellent moose range is also suitable for deer and expansion of forestry practices may well exacerbate the problem of moose disease by opening up new range suitable for deer. Studies of remnant moose populations in Nova Scotia and Fundy National Park, New Brunswick, indicate that moose survive mainly in elevated regions where deer do poorly, mainly because of snow conditions.[16,21] When moose venture from these higher refuges into deer range they succumb to neurologic disease caused by meningeal worm.[16]

The Parasite and Wapiti

We still know little about the effects of meningeal worm on wild wapiti. The eastern race has been extinct for over a century but there have been several introductions of western races of this species

into eastern North America. These attemps cannot be considered highly successful and it is known neurologic disease occurs in these animals which are associated with white-tailed deer.[5,13] It would be useful to know the ecological relationships which existed between deer and wapiti in eastern North America before the arrival of Europeans to the continent. Was the relationship such as to preclude the possibility of wapiti becoming infected with meningeal worm or was the now extinct eastern race resistant to infection or alternately a suitable host? Like the moose, the wapiti is regarded as a recent arrival to the North American continent.

The Parasite and Mule Deer

We still know nothing about the relationship between white-tailed deer, mule deer, and meningeal worm. Whitetails are replacing mule deer as the main species in many regions west of Ontario. This is usually attributed to the higher reproductive rate and greater adaptability of the former species. Experimentally there is evidence meningeal worm is pathogenic to mule deer[6] but there are no reports of neurologic disease in wild animals.

The Parasite and Caribou

There is no satisfactory explanation for declines of woodland caribou over much of its southern range in many parts of continental North America, especially Ontario. Caribou rely heavily in winter on ground and tree lichens but at other times of the year their food requirements may be similar to those of white-tailed deer. There is documented evidence that in some localities declines in woodland caribou were associated with the arrival into caribou range of white-tailed deer.[11] However, wildlife specialists mention range destruction as the prime factor, although in Manitoba and Ontario vast areas of apparently suitable range remain underpopulated today.[23] Meningeal worm is highly pathogenic to caribou.[6,8]

A remarkable attempt to introduce a small herd of reindeer from Norway to a large island in Georgian Bay, Ontario permitted a study of the effects of meningeal worm on a small herd of susceptible animals released onto deer range.[7] This is a region of Ontario which historically had caribou and not white-tailed deer.[11] It is now heavily populated by the latter species and caribou are absent. In May the reindeer were placed on range used by deer. The animals developed neurologic disease caused by meningeal worm and the disease was the direct cause of the failure of this attempted introduction. Several important facts emerged from this study. The reindeer became infected in June from ingesting infected gastropods which had survived winter with infective larvae. Clinical disease was the result of small numbers of meningeal worms developing in the central nervous system. Adult animals began to show clinical signs about 6 weeks after they were exposed to infection by the emergence of gastropods at the onset of warm wet weather in June. Calves became infected soon after they were weaned. Clinical signs varied from listlessness, ataxia, abnormalities in the eyes and in the position of the head, to lumbar paralysis and death but the disease affected all members of the herd. The obvious conclusion was drawn that it will be impossible to reintroduce woodland caribou onto range now occupied by white-tailed deer with a high prevalence of meningeal worm. This disease may have been the reason why introductions over a number of years in certain parts of continental North America have been failures.

In conclusion, we require more detailed field information about the prevalence of meningeal worm and its intermediate hosts throughout the range of white-tailed deer in North America, especially in areas where this cervid seems to share range with related animals. Studies now in progress in southwestern Ontario and in Minnesota and Maine will hopefully provide important information about the relationship of deer, moose, and meningeal worm. Possibly the existence of clinical disease in moose in an area should be regarded as evidence of a much more widespread disease problem which may have a serious impact on the population. Some of the management implications of such studies have already been raised by Severinghaus and Jackson.[22]

LITERATURE CITED

1. 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.
2. ANDERSON, R. C. 1964. Neurologic disease in moose infected experimentally with *Pneumostrongylus tenuis* from white-tailed deer. Path. vet. 1: 289-322.
3. ANDERSON, R. C. 1965. The development of *Pneumostrongylus tenuis* in the central nervous system of white-tailed deer. Path. vet. 2: 360-376.
4. ANDERSON, R. C. 1965. An examination of wild moose exhibiting neurologic signs in Ontario. Can. J. Zool. 43: 635-639.
5. 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.
6. ANDERSON, R. C., and U. R. STRELIVE. 1968. The experimental transmission of *Pneumostrongylus tenuis* to caribou *(Rangifer tarandus terraenovae).* Can. J. Zool. 46: 503-510.
7. ANDERSON, R. C. 1971. Neurologic disease in reindeer *(Rangifer tarandus tarandus)* introduced into Ontario. Can. J. Zool. 49: 159-166.
8. BEHREND, D. F., and J. F. WILLER. 1968. *Pneumostrongylus tenuis* in white-tailed deer in Maine. J. Wildl. Mgmt. 32: 963-966.
9. BENSON, D. A. 1958. "Moose sickness" in Nova Scotia 1-11. Can. J. comp. Med. 22: 244-248, 282-286.
10. BINDERNAGEL, J. A., and R. C. ANDERSON. 1972. Distribution of meningeal worm *(Pneumostrongylus tenuis)* in white-tailed deer in Canada. J. Wildl. Mgmt. in press.
11. CRINGAN, A. T. 1956. Some aspects of the biology of caribou and a study of the woodland caribou range of the Slate Islands, Lake Superior, Ontario. M.A. thesis, Univ. Toronto.
12. ECKROADE, R. J., G. M. ZURHEIN, and W. FORETYT. 1970. Meningeal worm invasion of the brain of a naturally infected white-tailed deer. J. Wildl. Dis. 6: 430-436.
13. KARNS, P. D. 1966. *Pneumostrongylus tenuis* from elk *(Cervus canadensis)* in Minnesota. Bull. Wildl. Dis. Assoc. 2: 79-80.
14. KARNS, P. D. 1967. *Pneumostrongylus tenuis* in deer in Minnesota and implications for moose. J. Wildl. Mgmt. 31: 299-303.
15. KARNS, P. D., and P. A. JORDAN. 1969. *Pneumostrongylus tenuis* in moose on a deer-free island. J. Wildl. Mgmt. 33: 431-433.
16. KELSALL, J. P., and W. PRESCOTT. 1971. Moose and deer behavior in snow. Can. Wildlife Service Rept. Series No. 15, 25 pp.
17. LAMSON, A. L. 1941. Maine moose disease studies. M.S. thesis, Univ. Maine, 61 pp.
18. LANKESTER, M. W., and R. C. ANDERSON. 1968. Gastropods as intermediate hosts of *Pneumostrongylus tenuis* Dougherty of white-tailed deer. Can. J. Zool. 46: 373-383.
19. PRESTWOOD, A. K. 1970. Neurologic disease in a white-tailed deer massively infected with meningeal worm *(Pneumostrongylus tenuis)* J. Wildl. Dis. 6: 84-86.
20. PRESTWOOD, A. K., and J. F. SMITH. 1969. Distribution of meningeal worm *(Pneumostrongylus tenuis)* in deer in the southeastern United States. J. Parasitol. 55: 720-725.

21. PRYADKO, E. J., and S. N. BOEV. 1971. (Systematics, phylogeny, and evolution of elaphostrongyloid nematodes of deer). Izdatel'stvo Akad. Nauk Kazakhskoi SSR, Alma-Ata, 5: 41-48 (in Russian).

22. SEVERINGHAUS, C. W., and W. J. JACKSON. 1971. Feasibility of stocking moose in the Adirondacks. N.Y. Fish Game J. 17: 18-32.

23. SIMKIN, D. W. 1965. A preliminary report of the woodland caribou study in Ontario. Sec. Rept. (Wildlife) No. 59, Ontario Dept. of Lands and Forests, Can. pp. 1-76.

24. TELFER, E. S. 1967. Comparison of moose and deer winter range in Nova Scotia. J. Wildl. Mgmt. 31: 418-425.

Received for publication April 3, 1972