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A REVIEW OF THE ECOLOGY OF PARELAPHOSTRONGYLUS TENUIS IN RELATION TO DEER AND MOOSE IN NORTH AMERICA.

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SUMMARY OF FINDINGS

It is well established that white-tailed deer are the normal host for *P. tenuis* and that this parasite either kills moose directly or predisposes them to other causes of mortality. Despite the historical record of moose dying from this parasite, there is little evidence, that *P. tenuis* is a major cause of mortality in moose or that it was responsible for historic declines in moose populations. When white-tailed deer expanded their range northward into moose range following logging, they undoubtedly introduced *P. tenuis* to moose. While it may seem intuitive that higher deer numbers should translate into higher moose mortality, research has not corroborated this relationship. Rather, it has discovered that the transmission of *P. tenuis* between deer and moose is a complex relationship and after almost 45 years, this relationship is still poorly understood. Based on our current knowledge, reductions in deer density on moose range will likely have little effect on the population status of moose in Minnesota.

INTRODUCTION

As early as 1912, a “moose sickness” was identified in Minnesota moose (*Alces alces*; Fenstermacher and Olson 1942). The disease was characterized by apparent blindness, lack of fear, aimless wandering, and ataxia (Karns 1967), which either killed moose directly or predisposed them to other causes of mortality. Histopathological analysis of diseased animals revealed irreversible damage to the central nervous system (Kurtz et al. 1966). Although moose sickness was associated with the presence of white-tailed deer (*Odocoileus virginianus*) as early as the late 1950s (Benson 1958), it was the experimental work by Anderson (1964), who demonstrated that the nematode lungworm (*Parelaphostrongylus tenuis*) caused moose sickness. Since this discovery, several hypotheses have been proposed regarding the relationship between deer, moose, and *P. tenuis*. It is the objective of this review to examine these hypotheses and subsequent research that either corroborate or refute them.

LITERATURE REVIEW

The life cycle of *P. tenuis* normally incorporates a definitive host, white-tailed deer, and an intermediate host, which includes several species of gastropods (slugs and snails). Once a deer is infected, *P. tenuis* larvae develop into adults and live in association with nervous tissue in the spinal cord and in the subdural spaces and venous sinuses of the cranium (Lankester and Samuel 1998). After a complex journey through the deer's body, first stage larvae are shed in the mucosal coating on feces and may survive as long as 10 months outside the host (Lankester and Anderson 1968). Gastropods that live in the litter on the forest floor crawl over the deer feces and become infected with the first stage larvae. Within the gastropod, the larvae ultimately molt into 3rd stage larvae that are infective to cervids if accidentally ingested (Lankester and Samuel 1998). White-tailed deer apparently do not succumb to the neurologic disorders caused by *P. tenuis* in other cervids (Alibasogulu et al. 1961, Anderson 1963).

Shortly after Anderson identified *P. tenuis* as the cause of moose sickness, several authors hypothesized that *P. tenuis* was a major cause of mortality in moose and responsible for historic declines in moose populations (Karns 1967, Telfer 1967). By the mid-1970s, there was general agreement on this supposition, especially in areas of high deer densities in eastern North America (Gilbert 1974, Prescott 1974, Kearney and Gilbert 1976, Lankester 1987). In the 1980s, however, some scientists began to question this conjecture based on the low number of moose deaths attributed to "moose sickness" at a time when both moose and deer populations were increasing on shared range (Brown 1983, Upshall et al. 1987, Lenarz and Kerr unpubl.). Subsequently, Nudds (1990) questioned the hypothesis that *P. tenuis* was a major cause of mortality in moose and suggested that circular logic was used in making this inference. After reviewing all available data from Maine, Minnesota, New Brunswick, and Nova Scotia, Whitlaw and Lankester (1994a) indicated that the historical information available did not corroborate the hypothesis that *P. tenuis* had caused declines in moose populations.

Recent research in Minnesota also suggests that while present, *P. tenuis* is not a major cause of mortality. In northwestern Minnesota, for example, mortality of only 5% of radiocollared moose was attributed to *P. tenuis* (Murray et al. 2006). In northeastern Minnesota, 17% (18/108) of moose had positive titers for *P. tenuis* (Lenarz et al., unpublished data). Assuming that this parasite was responsible for the subsequent death of moose testing positive (except for 1 capture mortality and 1 hunter kill), annual cause specific mortality from *P. tenuis* averaged 4% (0 to 10%) and represented an average of 19% (0 to 32%) of the total mortality the population experienced each year (Lenarz et al., unpublished data). Considering the relatively low proportion of moose mortality attributed to *P. tenuis* in both northeastern and northwestern Minnesota, it is questionable whether this parasite represents a major threat to the moose populations.

Early researchers also suggested that the infection rate in moose increased as a direct response to increasing deer density (Anderson 1965, Karns 1967, Kelsall and Prescott 1971, Gilbert 1974). These early researchers reasoned that as deer density increased, more deer were infected, and more larvae would be shed. As a consequence of the increased number of larvae, more gastropods would be infected, and the probability that moose would consume an infected gastropod and die would

increase. Subsequent research, however, has indicated that the relationships between deer density, *P. tenuis* infection rates, and moose mortality are complex and poorly understood (Anderson and Prestwood 1981, Whitlaw and Lankester 1994a, b; Lankester and Samuel 1998).

The hypothesis that more larvae are shed as deer density increases assumes that the prevalence of *P. tenuis* in deer is constant or increases as deer numbers increase. Based on meager evidence, Karns (1967) and Behrend and Witter (1968) suggested that the prevalence of *P. tenuis* increased as deer numbers increased. Gilbert (1973), however, found a lower prevalence at higher deer density after comparing 2 areas in Maine. Thomas and Dodds (1988) found no relationship between deer infection rates and deer density (2 levels) or moose density (3 levels) in Nova Scotia. Based on deer sampled from 17 Deer Management Districts, Bogaczyk et al. (1993) found that neither prevalence nor intensity of infection in white-tailed deer was associated with deer density over a range of 1.4 to 5.8 deer/km². Hence, there are few data to suggest a relationship between prevalence and deer density. It is logical to assume, however, that more deer will deposit more feces on the landscape and unless prevalence declines in response to deer density, there will be more infected feces.

Even if the density of infected feces is high, it doesn't imply that higher numbers of gastropods are infected. Lankester and Peterson (1996) found a prevalence rate of only 0.16% (7 out of 4,401) in a deeryard that seasonally supported 50 deer/km². Other surveys in Minnesota and Ontario have generally found a prevalence rate less than 1% (Lankester 1967, Kearney and Gilbert 1978, Pitt and Jordan 1995, Lankester and Peterson 1996). Research that reported both infection rates of gastropods and local deer density is limited (Lankester and Anderson 1968, Maze and Johnstone 1986, Platt 1989, Lankester and Peterson 1989, Pitt and Jordan 1994). Pooling these data, there was no correlation ($r = 0.09$, $P = 0.86$) between prevalence and deer density. The infection rate of gastropods is likely dependent on the density, residence time, and defecation rates of infected deer; the survivorship of first-stage larvae on the feces or in the soil; and the abundance of and mobility of suitable gastropods (Lankester and Peterson 1996). Even in a situation with 120 deer living year around on a 1.3 km² island (240 deer/mi²), the prevalence of *P. tenuis* in gastropods was only 4.2% (Lankester and Anderson 1968).

Considering the extremely low infection rate of gastropods, it is unclear how large numbers of deer become infected. Based on the prevalence documented by Lankester and Peterson (1996) in northeastern Minnesota and assuming that a deer or moose could be infected by consuming a single infected gastropod, each deer or moose on summer range would need to consume 2,500 gastropods to become infected. Anderson and Prestwood (1981) proposed that infected gastropods might live in small concentrations that are not adequately sampled by researchers but encountered by foraging cervids. In a study of gastropod climbing behavior, McCoy and Nudds (1997) found that species were highly variable in the degree that they climbed; some species climbed infrequently while other were primarily arboreal. They suggested that data from studies which restricted sampling to the use of damp cardboard (e.g. Gleich and Gilbert 1976, Kearney and Gilbert 1978, Upshall et al. 1986, Lankester and Peterson 1996), resulted in estimates biased to the less arboreal species, which are more likely to

encounter *P. tenuis* larvae. If correct, prevalence rates in gastropods would be even lower than currently estimated.

Finally, if moose mortality was a simple function of deer density, there should be an inverse correlation between changes in deer and moose density. In the 1980s and 1990s, several authors documented simultaneous increases in sympatric moose and deer populations in some eastern states and provinces (Clark and Boyer 1986, Upshall et al. 1987, Thomas and Dodds 1988, Boer 1992, Bogaczyk et al. 1993). Several hypotheses were proposed to explain this conundrum. Working in Maine, Clark and Bowyer (1986) found a high prevalence of *P. tenuis* larvae in moose feces and suggested that co-evolution favoring a reduction in the debilitating effect of *P. tenuis* upon moose may have occurred. McCullough and Pollard (1993), however, suggested that faulty lab procedures might have been responsible for the high prevalence of *P. tenuis* in moose feces found by Clark and Bowyer (1986). Upshall et al. (1987) found no larvae in New Brunswick moose and suggested that moose were feeding in different areas than deer, an argument first proposed by Telfer (1967) and subsequently challenged by Nudds (1990).

LITERATURE CITED

- Alibasogulu, M. D., C. Kradel, and H. W. Dunne. 1961. Cerebral nematodosis in Pennsylvania deer (*Odocoileus virginianus*). *Cornell Veterinarian* 54:431-441.
- Anderson, R. C. 1963. The incidence, development, and experimental transmission of *Pneumostonylys tenuis* Dougherty (Metastrongyloidea: Protostrongyloidea) of the meninges of white-tailed deer (*Odocoileus virginianus borealis*) in Ontario. *Canadian Journal of Zoology* 41:775-792.
- _____. 1964. Neurologic disease in moose infected experimentally with *Pneumostongylus tenuis* from white-tailed deer. *Pathologica Veterinaria*. 1:289-322.
- _____. 1965. An examination of wild moose exhibiting neurological signs in Ontario. *Canadian Journal of Zoology* 43:635-639.
- Anderson, R. C. and A. K. Prestwood. 1981. Lungworms. Tall Timbers Research Station Miscellaneous Publication. 7:266-317.
- Behrend, D. F. and J. F. Witter. 1968. *Pneumostongylus tenuis* in white-tailed deer in Maine. *Journal of Wildlife Management* 32:963-966.
- Benson, D. A. 1958. "Moose sickness" in Nova Scotia. *Canadian Journal of Comparative Medicine* 22:244-248.
- Boer, A. H. 1992. History of moose in New Brunswick. *Alces* (Suppliment) 1:16-21.

- Bogaczyk, B. A., W. B. Krohn, and H. C. Gibbs. 1993. Factors affecting *Parelaphostrongylus tenuis* in white-tailed deer (*Odocoileus virginianus*) from Maine. *Journal of Wildlife Diseases* 29:266-272.
- Brown, J. E. 1983. *Parelaphostrongylus tenuis* Dougherty in Nova Scotian moose and deer. Honors Thesis, Acadia University, Wolfville, Nova Scotia.
- Clark, R. A. and R. T. Bowyer. 1986. Occurrence of protostrongylid nematodes in sympatric populations of moose and white-tailed deer in Maine. *Alces* 22:313-321.
- Fenstermacher, R. and O. W. Olson. 1942. Further studies of diseases affecting moose III. *Cornell Veterinarian* 32:241-254.
- Gilbert, F. F. 1973. *Parelaphostrongylus tenuis* in Maine: I - The parasite in white-tailed deer (*Odocoileus virginianus*, Zimmerman). *Journal of Wildlife Disease* 9:136-143.
- _____. 1974. *Parelaphostrongylus tenuis* in Maine: II - prevalence in moose. *Journal of Wildlife Management* 38:42-46.
- Gleich, J. G. and F. F. Gilbert. 1976. A survey of terrestrial gastropods from central Maine. *Canadian Journal of Zoology* 54:620-627.
- Karns, P. D. 1967. *Pneumostongylus tenuis* in deer in Minnesota and implications for moose. *Journal of Wildlife Management* 31:299-303.
- Kearney, S. R. and F. F. Gilbert. 1976. Habitat use by white-tailed deer and moose on sympatric range. *Journal of Wildlife Management* 40:645-657.
- _____. 1978. Terrestrial gastropods from the Himsforth Game Preserve, Ontario, and their significance in *Parelaphostrongylus tenuis* transmission. *Canadian Journal of Zoology* 56:688-694.
- Kelsall, J. P. and W. Prescott. 1971. Moose and deer behavior in snow. *Canadian Wildlife Service Report Series* 15. 27pp.
- Kurtz, H. J., K. I. Loken, and J. C. Schlotthauer. 1966. Histopathological studies on cerebrospinal nematodiasis of Moose in Minnesota naturally infected with *Pneumostongylus tenuis*. *American Journal of Veterinary Research* 27:548-557.
- Lankester, M. W. 1967. Gastropods as intermediate hosts of *Pneumostongylus tenuis* Dougherty, University of Guelph, Ontario, Canada.
- _____. 1987. Pests, parasites and diseases of moose (*Alces alces*) in North America. *Swedish Wildlife Research Supplement* 1:461-489.

- Lankester, M. W. and R. C. Anderson. 1968. Gastropods as intermediate host of meningeal worm, *Pneumostongylus tenuis*, Dougherty. Canadian Journal of Zoology 46:373-383.
- Lankester, M. W. and W. J. Peterson. 1996. The possible importance of wintering yards in the transmission of *Parelaphostrongylus tenuis* to white-tailed deer and moose. Journal of Wildlife Diseases 32:31-38.
- Lankester, M. W. and W. M. Samuel. 1998. Pests, parasites and diseases. Pages 479-517 in A. W. Franzmann and C. C. Schwartz, editors. Ecology and management of the North American moose. Smithsonian Institution Press. Washington, D. C., USA.
- Maze, R. J. and C. Johnstone. 1986. Gastropod intermediate hosts of the meningeal worm *Parelaphostrongylus tenuis* in Pennsylvania: observations on their ecology. Canadian Journal of Zoology 64:185-188.
- McCullough, M. A. and K. A. Pollard. 1993. *Parelaphostrongylus tenuis* in Maine moose and the possible influence of faulty Baermann procedures. Journal of Wildlife Diseases 29: 156-158.
- McCoy, K. D. and T. D. Nudds. Interspecific variation in climbing by gastropods: implications for transmission of *Parelaphostrongylus tenuis*. American Midland Naturalist 137:320-328.
- Murray, D. L., E. W. Cox, W. B. Ballard, H. W. Whitlaw, M. S. Lenarz, T. W. Custer, T. Barnett, and T. K. Fuller. Pathogens, nutritional deficiency, and climate influences on a declining moose population. Wildlife Monographs 166.
- Nudds, T. D. 1990. Retroductive logic in retrospect: the ecological effects of meningeal worms. Journal of Wildlife Management 54:396-402.
- Pitt, W. C. and P. A. Jordan. 1995. A survey of the nematode parasite *Parelaphostrongylus tenuis* in the white-tailed deer, *Odocoileus virginianus*, in a region proposed for caribou, *Rangifer tarandus caribou*, re-introduction in Minnesota. Canadian Field-Naturalist 108:341-346.
- Platt, T. R. 1989. Gastropod intermediate hosts of *Parelaphostrongylus tenuis* (Nematoda: Metastronyloidea) from northwestern Indiana. The Journal of Parasitology 75:519-523.
- Prescott, W. H. 1974. Interrelationships of moose and deer of the genus *Odocoileus*. Naturaliste Canadien 101:493-504.
- Telfer, E. S. 1967. Comparison of moose and deer winter range in Nova Scotia. Journal of Wildlife Management 31:418-425.

- Thomas, J. E. and G. D. Dodds. 1988. Brainworm, *Parelaphostrongylus tenuis*, in moose, *Alces alces*, and white-tailed deer, *Odocoileus virginianus*, of Nova Scotia. Canadian Field-Naturalist 102:639-642.
- Upshall, S. M., M. D. B. Burt, and T. G. Dilworth. 1987. *Parelaphostrongylus tenuis* in New Brunswick: the parasite in white-tailed deer (*Odocoileus virginianus*) and moose (*Alces alces*). Journal of Wildlife Diseases 23:683-685.
- Whitlaw, H. A. and M. W. Lankester. 1994a. A retrospective evaluation of the effects of parelaphostrongylosis in moose populations. Canadian Journal of Zoology 72:1-7.
- _____. 1994b. The co-occurrence of moose, white-tailed deer, and *Parelaphostrongylus tenuis* in Ontario. Canadian Journal of Zoology 72:819-825.