MORTALITY OF ROCKY MOUNTAIN ELK IN MICHIGAN DUE TO MENINGEAL WORM

Authors: Bender, Louis C., Schmitt, Stephen M., Carlson, Elaine, Haufler, Jonathan B., and Beyer, Dean E.

Source: Journal of Wildlife Diseases, 41(1) : 134-140

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-41.1.134

Journal of Wildlife Diseases, 41(1), 2005, pp. 134–140

MORTALITY OF ROCKY MOUNTAIN ELK IN MICHIGAN DUE TO MENINGEAL WORM

Louis C. Bender,[1,6] Stephen M. Schmitt,[2] Elaine Carlson,[3] Jonathan B. Haufler,[4] and Dean E. Beyer, Jr.[5]

[1] U.S. Geological Survey, New Mexico Cooperative Fish and Wildlife Research Unit, PO Box 30003 MSC 4901, Las Cruces, New Mexico 88003, USA
[2] Michigan Department of Natural Resources, Rose Lake Wildlife Disease Laboratory, 8562 East Stoll Road, East Lansing, Michigan 48823, USA
[3] Michigan Department of Natural Resources, 191 S. Mt. Tom Road, Mio, Michigan 48647, USA
[4] Environmental Management Research Institute, 210 Borderlands PO Box 717, Seeley Lake, Montana 59868, USA
[5] Michigan Department of Natural Resources, Northern Michigan University, Department of Geography, 213 West Science Building, Marquette, Michigan 49855, USA
[6] Corresponding author (email: lbender@nmsu.edu)

ABSTRACT: Mortality from cerebrospinal parelaphostrongylosis caused by the meningeal worm (*Parelaphostrongylus tenuis*) has been hypothesized to limit elk (*Cervus elaphus nelsoni*) populations in areas where elk are conspecific with white-tailed deer (*Odocoileus virginianus*). Elk were reintroduced into Michigan (USA) in the early 1900s and subsequently greatly increased population size and distribution despite sympatric high-density (≥12/km^2) white-tailed deer populations. We monitored 100 radio-collared elk of all age and sex classes from 1981–94, during which time we documented 76 mortalities. Meningeal worm was a minor mortality factor for elk in Michigan and accounted for only 3% of mortalities, fewer than legal harvest (58%), illegal kills (22%), other diseases (7%), and malnutrition (4%). Across years, annual cause-specific mortality rates due to cerebrospinal parelaphostrongylosis were 0.033 (SE=0.006), 0.029 (SE=0.005), 0.000 (SE=0.000), and 0.000 (SE=0.000) for calves, 1-yr-old, 2-yr-old, and ≥3-yr-old, respectively. The overall population-level mortality rate due to cerebrospinal parelaphostrongylosis was 0.009 (SE=0.001). Thus, meningeal worm had little impact on elk in Michigan during our study despite greater than normal precipitation (favoring gastropods) and record (≥14 km^2) deer densities. Further, elk in Michigan have shown sustained population rates-of-increase of ≥18%/yr and among the highest levels of juvenile production and survival recorded for elk in North America, indicating that elk can persist in areas with meningeal worm at high levels of population productivity. It is likely that local ecologic characteristics among elk, white-tailed deer, and gastropods, and degree of exposure, age of elk, individual and population experience with meningeal worm, overall population vigor, and moisture determine the effects of meningeal worm on elk populations.

Key words: Cerebrospinal parelaphostrongylosis, *Cervus elaphus nelsoni*, elk, meningeal worm, mortality, *Parelaphostrongylus tenuis*.

INTRODUCTION

Cerebrospinal parelaphostrongylosis caused by the meningeal worm (*Parelaphostrongylus tenuis*) has been implicated in several failures to reestablish cervid populations in the eastern USA (Carpenter et al., 1973; Severinghaus and Darrow, 1976; Bergerud and Mercer, 1989; Raskevitz et al., 1991) or for poor productivity, low population rates of increase, and/or population declines where susceptible cervids are sympatric with white-tailed deer (*Odocoileus virginianus*) (Anderson, 1972; Eveland et al., 1979; Raskevitz et al., 1991; Samuel et al., 1992; Lankester and Samuel, 1998). Elk (*Cervus elaphus*) are known to be vulnerable to meningeal worm infection (Anderson et al., 1966). Consequently, it has been hypothesized that meningeal worm could limit successful elk reintroductions into the eastern USA (Carpenter et al., 1973; Severinghaus and Darrow, 1976; Raskevitz et al., 1991; Larkin et al., 2003) or result in low population productivity where elk persist (Samuel et al., 1992). Reintroduced elk in Pennsylvania apparently suffered high mortality from meningeal worm (Eveland et al., 1979) as did elk translocated to eastern Oklahoma (Raskevitz et al., 1991). Mortality due to cerebrospinal parelaphostrongylosis in reintroduced elk in Ken-

tucky led researchers to conclude that meningeal worm might act to significantly retard elk population growth (Larkin et al., 2003). These examples, however, involved elk populations that were either present in low numbers (Pennsylvania, Oklahoma) or were recent transplants (Kentucky, Oklahoma).

Elk were reintroduced into Michigan (USA) in 1918. Subsequently, elk increased from <20 to >2,000 individuals by the early 1960s (a mean rate-of-increase >11%/year) despite extensive illegal killing, poorer quality habitat compared with that from the 1970s to present, and high (up to 12/km^2) white-tailed deer densities (Moran, 1973; Bender, 1992). During this period, diseased elk with signs compatible with cerebrospinal parelaphostrongylosis were occasionally observed, but meningeal worm was not isolated from any of the elk (Moran, 1973). Elk numbers were dramatically reduced in the 1960s by harvesting and illegal killing to a low of approximately 200 individuals by the early 1970s (Moran, 1973; Bender, 1992). Subsequently, elk in Michigan again increased to >1,200 individuals, where numbers are maintained by an aggressive harvest that removes approximately 13% of both the adult cow and bull populations annually (Bender, 1992; Bender et al., 1999). Since the latest population increase began in the early 1970s, elk in Michigan have maintained a population rate-of-increase of ≥18%, near the highest recorded (Bender, 1992; Eberhardt et al., 1996). Moreover, production and survival of calves in Michigan were among the highest documented for any North American elk population (Bender et al., 2002).

We conducted a long-term (>10 yr) study of elk demographics in Michigan. Herein, our goal is to report mortality factors with an emphasis on meningeal worm mortality on elk. Reintroduced elk in Michigan have the longest history and thus longest exposure to meningeal worm of all eastern populations. Thus, their relations with meningeal worm can provide potentially strong inference into the effects of meningeal worm on mortality and productivity of elk.

MATERIALS AND METHODS

Michigan's primary elk range covers about 1,500 km^2 in the northern lower peninsula (approximately 45.15°N, 84.20°W), centered on the Pigeon River Country State Forest, Vanderbilt, Michigan, and the adjacent Camp 30 Hills area of Black River State Forest, Atlanta, Michigan. Adjacent private forested and agricultural lands comprise the remainder of the elk range. Vegetative cover in the elk range is mostly forest, with scattered agricultural land and wildlife openings. Approximately 79% is in forest cover types, primarily northern hardwoods, aspen (*Populus tremuloides* and *P. grandidentata*), pines (*Pinus* spp.), and coniferous swamps (Moran, 1973). During our study, total annual precipitation averaged 17.8 cm (SD=1.4) in the elk range, 8% greater than the long-term average (16.5 cm), and was greater than normal in 9 of 11 years that had complete precipitation data available. White-tailed deer densities in Michigan peaked during our study (late 1980s) at ≥14 deer/km^2.

From 1981–94 we monitored 100 radio-collared elk of all age and sex classes. We used a staggered-entry design, with 30, 17, 15, and 38 elk initially collared as calves, and at 1, 2, and ≥3 years old, respectively, with many individuals monitored for multiple years. We monitored elk approximately weekly throughout the year and either recovered mortalities in the field or at hunter check stations in the case of most (>95%) legal harvest. Mortalities found in the field that were not either hunter kills or illegal kills were collected and taken intact to the Michigan Department of Natural Resources (MDNR) Wildlife Veterinary Diagnostic Laboratory at Rose Lake Wildlife Research Station, East Lansing, Michigan, where complete necropsies were conducted by MDNR veterinary pathologists.

Mortalities were attributed to cerebrospinal parelaphostrongylosis based on clinical signs (Olsen and Wolf, 1979; Lankester, 2001) and presence of adult or larval *P. tenuis* in neural tissue associated with lesions consistent with nematode migration, including cavitations with inflammation, hemorrhage, and scarring. Other microscopic findings were meningitis with focal disseminated aggregates of lymphocytes, eosinophils, and macrophages. Further, no evidence of other significant disease processes was present.

We determined proportions of mortalities associated with meningeal worm infection and all

TABLE 1. Summary of mortality causes by age class (calves=0–1, yearlings=1–2, and adults=≥2-yr-old) for 76 Rocky Mountain elk in Michigan, 1981–94.

Cause	Calves	Yearlings	Adults	Total
Legal harvest	1	3	40	44
Illegal kill	0	1	16	17
Disease[a]	0	0	5	5
Malnutrition	1	0	2	3
Cerebrospinal parelaphostrongylosis	1	1	0	2
Unknown	0	1	4	5

[a] Includes enteritis/enterotoxemia (3), eosinophilic metritis (1), and corn toxicity (1).

other mortality factors by age classes (calves, yearlings, 2-yr-old, and ≥3-yr-old), because younger elk are thought to be more susceptible (Lankester, 2001). We also determined annual cause-specific mortality rates attributable to meningeal worm by age classes using the method of Heisey and Fuller (1985). We pooled calf, 1-yr-old, 2-yr-old, and ≥3-yr-old elk over years to determine a representative annual effect attributable to cerebrospinal parelaphostrongylosis. We also determined the overall population-level effect of meningeal worm by calculating a mortality rate due to cerebrospinal parelaphostrongylosis for all elk combined. We compared age-specific mortality rates due to cerebrospinal parelaphostrongylosis using Z-tests (Heisey and Fuller, 1985). All comparisons were conducted at $\alpha=0.05$. For comparisons of age-specific mortality rates, we partitioned the overall experiment-wise error rate ($\alpha_{exp}=0.05$) by the number of paired comparisons (k=6; calves vs. yearlings, 2-yr-old, and ≥3-yr-old elk; yearlings vs. 2-yr-old and ≥3-yr-old elk; and 2-yr-old vs. ≥3-yr-old elk) to determine a pair-wise comparison rate: $\alpha_{com}=1-[(1-\alpha_{exp})\ (1/k)=0.009$ (Zar, 1996). We then use α_{com} for all paired comparisons to maintain $\alpha_{exp}=0.05$ for the overall test.

RESULTS

We documented 76 mortalities from 100 radio-collared elk, 1981–94. In addition, 10 elk lost their radio collars and contact with an additional six elk was lost. Legal harvest (44/76; 58%) was the leading cause of elk mortality in Michigan, followed by illegal killing (17/76; 22%), disease other than meningeal worm (5/76; 7%), malnutrition (3/76; 4%), cerebrospinal parelaphostrongylosis (2/76; 3%), and unknown (5/76; 7%; Table 1). Cerebrospinal parelaphostrongylosis was diagnosed in one 8-mo-old bull calf and one 22-mo-old cow. In both cases, the elk were observed in the field with neurologic impairment prior to death. Other mortalities classified as disease included one case of corn toxicity, one case of eosinophilic metritis, and three cases of enteritis, likely enterotoxemia. Of the unknown losses, four of five elk were adults (Table 1). Despite intensive behavioral observations (Bender, 1992) no other radio-collared elk with signs of neurologic impairment were observed.

Annual cause-specific mortality rates due to cerebrospinal parelaphostrongylosis were 0.033 (SE=0.006; $n=30$ elk years), 0.029 (SE=0.005; $n=35$ elk years), 0.000 (SE=0.000; $n=47$ elk years), and 0.000 (SE=0.000; $n=196$ elk years) for calf, yearling, 2-yr-old, and ≥3-yr-old elk, respectively. Overall, the annual population-level mortality rate due to cerebrospinal parelaphostrongylosis was 0.009 (SE=0.001; $n=308$ elk years) for elk in Michigan. Annual mortality rates due to cerebrospinal parelaphostrongylosis did not differ between calves and yearlings ($P_{com}>0.500$). Mortality rates for calves and yearlings were both greater ($P_{com}<0.009$) than for 2-yr-old and ≥3-yr-old elk, which did not differ ($P_{com}=1.000$).

DISCUSSION

The population-level mortality rate due to cerebrospinal parelaphostrongylosis was 0.009 in Michigan, indicating that meningeal worm had little impact on elk during our study. This was true despite periodic population reductions that decreased over-

all population size to low levels (<200 individuals) that could have facilitated limitation by density-independent processes such as meningeal worm (Lankester, 2001) despite the presence of high-density white-tailed deer populations (≥14/km^2), and annual precipitation averaging 8% greater than the long-term average and thereby favoring gastropod populations. Additionally, some researchers have hypothesized that susceptible cervids might be able to coexist with white-tailed deer due to habitat segregation (Telfer, 1967; Gilbert, 1974; Kearney and Gilbert, 1976; Raskevitz et al., 1991). However, both elk and deer use similar habitats in Michigan, especially forage plantings, aspen, northern hardwoods, northern white-cedar (*Thuja occidentalis*) and mixed conifer swamps, and agricultural fields in and adjacent to the elk range (Moran, 1973; Bender et al., 1997). Thus, population persistence of elk in Michigan was not due to habitat segregation. Similarly, recent information indicates that moose (*Alces alces*) persistence was not due to habitat segregation (Whitlaw and Lankester, 1994; Dumont and Crête, 1996; Gogan et al., 1997; Lankester, 2001).

Moreover, both historic (mean rate of population increase = 11%/year) and recent (≥18%/year) demographic performance of elk in Michigan indicates that elk can persist in areas with meningeal worm at extremely high levels of population productivity. Annual rates of mortality to cerebrospinal parelaphostrongylosis were low (0.033 for calves, 0.029 for yearlings, 0.000 for ≥2.5-yr-old elk, and 0.009 for the population in total) in Michigan, whereas calf production was among the highest reported (calf:cow ratios = 51–56/100 in November; Bender et al., 2002), annual calf survival was the highest documented for elk (0.80–0.94; Bender et al., 2002), and the population has maintained annual growth rates of ≥18% (Bender, 1992). Thus, elk have thrived in Michigan despite the presence of high white-tailed deer densities and meningeal worm, perhaps due to superior nutrition and consequently population vigor (Bender et al., 2002, 2003). Although our data might underestimate total mortality associated with meningeal worm because of subclinical infections predisposing elk to other mortality such as harvest (Lankester, 2001), the demographic performance of elk in Michigan indicate that any such subclinical effect would have to be far greater than observed levels of meningeal worm mortality to have any significant effect on elk population performance in Michigan.

In contrast to the situation in Michigan, meningeal worm was implicated in a 50% decline in elk numbers in Pennsylvania in the early 1970s, and was considered the primary limiting factor on elk populations in Pennsylvania (Eveland et al., 1979) and eastern Oklahoma (Raskevitz et al., 1991). Larkin et al. (2003) felt that mortality associated with meningeal worm could significantly lower elk population growth rates in Kentucky. Eveland et al. (1979) hypothesized that elk in Pennsylvania were limited by meningeal worm during wet years, when ample moisture favored populations of intermediate hosts (gastropods). Conversely, during dry years elk populations expanded, presumably due to moisture-mediated impacts on gastropod population size and/or distribution. However, we saw no significant effect of meningeal worm despite wet years predominating during our study. It has also been hypothesized that differential habitat use between white-tailed deer and susceptible cervids might allow coexistence (Kearney and Gilbert, 1976), although this hypothesis has recently been discounted (Whitlaw and Lankester, 1994; Dumont and Crête, 1996; Gogan et al., 1997; Lankester, 2001). Samuel et al. (1992) demonstrated that the effect of meningeal worm on elk was dose-dependent and that elk can tolerate and might be able to serve as definitive hosts of meningeal worm if the infective dose was low. Very likely, the impact of meningeal worm on elk populations could be a function of numbers of larvae

consumed, age at first exposure, and prior experience with the parasite, as hypothesized for moose by Lankester (2001).

The latter point could be significant for elk. Elk were reintroduced into Pennsylvania about the same time as Michigan (1913–26; Eveland et al., 1979) but have always been present at very low numbers (<100 historically and <400 recently; Eveland et al., 1979; Cogan and Dieffenbach, 1998) that would make them demographically susceptible to any mortality factor. Elk in Kentucky have only recently been reintroduced (1997) with reintroductions coming from the western United States where meningeal worm is absent; thus the elk are inexperienced with the parasite (Lankester, 2001). In Michigan, elk have a long history of meningeal worm exposure, including historical reports of mortality likely (but not definitively) due to cerebrospinal parelaphostrongylosis (i.e., "elk disease"; Moran, 1973). Thus, most adults probably have been exposed to meningeal worm throughout their lives and the population has had a long history of exposure while rapidly increasing and/or while present in high population densities. This contrasts with elk in Pennsylvania (low population size), Kentucky (no past history of exposure), and eastern Oklahoma (low population numbers and a relatively short [since 1969–71] history of exposure; Raskevitz et al., 1991). In Kentucky, mortality occurred in adult elk when first exposed to meningeal worm, although <3-yr-old elk experienced the greatest mortality (Larkin et al., 2003). Similarly, in Michigan mortality was seen only in calves and yearlings, corroborating the hypothesis that younger age classes are more susceptible to cerebrospinal parelaphostrongylosis (Olsen and Wolf, 1979; Lankester, 2001; Larkin et al., 2003). However, mean annual mortality was exceedingly low even for calves, yearlings, and 2-yr-old elk in Michigan, suggesting that elk populations might develop tolerance or some degree of immunity following long-term exposure to meningeal worm (Lankester, 2001). As a minimum, high survival of juveniles despite exposure to meningeal worm results in adults experienced with the parasite and thus perhaps unaffected by meningeal worm infection.

Management implications

Despite widespread belief that meningeal worm can limit susceptible cervids, few studies have shown a conclusive cause-and-effect relationship between mortality due to meningeal worm and limitation of cervid populations (Whitlaw and Lankester, 1994; Dumont and Crête, 1996; Gogan et al., 1997; Lankester, 2001; however, caribou [*Rangifer tarandus*] are an exception [Bergerud and Mercer, 1989]). Meningeal worm can apparently have varying impacts on elk populations, likely due to a complex interaction of local ecological conditions involving white-tailed deer and elk densities, gastropod population dynamics, infective doses, age of elk, experience of individuals and populations with the parasite, and perhaps precipitation as a minimum number of contributing factors. As evidenced by historic rates of population increase (>11%/yr) and recent elk survival and population demographics in Michigan, a generic hypothesis of meningeal worm affecting elk populations by limiting population productivity or rate-of-increase is clearly inadequate. Much more detailed information on host relations, population dynamics, and factors that influence these issues are needed to understand and thereby predict effects of meningeal worm on elk or other cervid populations. Moreover, these factors are likely to be highly site specific; assessments of meningeal worm as a potential limiting factor on susceptible cervids need to be site specific as well. Further, whereas some populations of susceptible cervids initially suffered high mortality from meningeal worm (i.e., moose in Michigan; Aho and Hendrickson, 1989), these same populations have subsequently expanded their sizes and distributions (Aho et al., 1995). As evidenced by these and the case of elk

in Michigan, elk and other susceptible cervids (excluding caribou) might be able to acquire a high degree of tolerance or immunity to meningeal worm (Lankester, 2001). Thus, elk management needs to proceed on a case-by-case basis with regard to meningeal worm, rather than by a blanket prescription.

ACKNOWLEDGMENTS

We thank all Michigan State University and Michigan Department of Natural Resources, Wildlife and Forestry Divisions, employees who assisted in collection or evaluation of this data. Funding was provided by the MDNR, Rocky Mountain Elk Foundation, and U.S. Geological Survey. The Agricultural Experiment Station at New Mexico State University provided additional financial support. We thank S. Smallidge, J. Boren, E. Williams, and two anonymous referees for helpful reviews of this manuscript.

LITERATURE CITED

AHO, R. W., AND J. HENDRICKSON. 1989. Reproduction and mortality of moose translocated from Ontario to Michigan. Alces 25: 75–80.

———, S. S. SCHMITT, J. HENDRICKSON, AND T. R. MINZEY. 1995. Michigan's translocated moose population: 10 years later. Wildlife Division Report Number 3245, Michigan Department of Natural Resources, Lansing, Michigan, 18 pp.

ANDERSON, R. C. 1972. The ecological relationships of meningeal worm and native cervids in North America. Journal of Wildlife Diseases 8: 304–310.

———, M. W. LANKESTER, AND U. R. STRELIVE. 1966. Further experimental studies of *Pneumostrongylus tenuis* in cervids. Canadian Journal of Zoology 44: 851–861.

BENDER, L. C. 1992. The Michigan elk herd: Ecology of a heavily exploited population. PhD Dissertation, Michigan State University, East Lansing, Michigan, 192 pp.

———, D. L. MINNIS, AND J. B. HAUFLER. 1997. Wildlife responses to thinning red pine. Northern Journal of Applied Forestry 14: 141–146.

———, D. E. BEYER, JR., AND J. B. HAUFLER. 1999. The effects of short duration high intensity hunting on elk wariness in Michigan. Wildlife Society Bulletin 27: 441–445.

———, E. CARLSON, S. M. SCHMITT, AND J. B. HAUFLER. 2002. Production and survival of elk (*Cervus elaphus*) calves in Michigan. American Midland Naturalist 148: 163–171.

———, ———, ———, AND ———. 2003. Body mass and antler development patterns of Rocky Mountain elk (*Cervus elaphus nelsoni*) in Michigan. American Midland Naturalist 150: 169–180.

BERGERUD, A. T., AND W. E. MERCER. 1989. Caribou introductions in eastern North America. Wildlife Society Bulletin 17: 111–120.

CARPENTER, J. W., H. E. JORDAN, AND B. C. WARD. 1973. Neurologic disease in wapiti naturally infected with meningeal worms. Journal of Wildlife Diseases 9: 148–153.

COGAN, R. D., AND D. R. DIEFFENBACH. 1998. Effect of undercounting and model selection on a sightability-adjustment estimator for elk. Journal of Wildlife Management 62: 269–279.

DUMONT, A., AND M. CRÊTE. 1996. The meningeal worm, *Parelaphostrongylus tenuis*, a marginal limiting factor for moose, *Alces alces*, in southern Quebec. Canadian Field-Naturalist 110: 413–418.

EBERHARDT, L. E., L. L. EBERHARDT, B. L. TILLER, AND L. L. CALDWELL. 1996. Growth of an isolated elk population. Journal of Wildlife Management 60: 369–373.

EVELAND, J. F., J. L. GEORGE, N. B. HUNTER, D. M. FORNEY, AND R. L. HARRISON. 1979. A preliminary evaluation of the ecology of the elk in Pennsylvania. *In* North American elk: Ecology, behavior, and management, M. S. Boyce and L. D. Hayden-Wing (eds.). University of Wyoming, Laramie, Wyoming, pp. 145–151.

GILBERT, F. F. 1974. *Parelaphostrongylus tenuis* in Maine: II—Prevalence in moose. Journal of Wildlife Management 38: 42–46.

GOGAN, P. J. P., K. D. KOZIE, E. M. OLEXIA, AND N. S. DUNCAN. 1997. Ecological status of moose and white-tailed deer at Voyageurs National Park, Minnesota. Alces 33: 187–201.

HEISEY, D. M., AND T. K. FULLER. 1985. Evaluation of survival and cause-specific mortality rates using telemetry data. Journal of Wildlife Management 49: 668–674.

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.

LANKESTER, M. W. 2001. Extrapulmonary lungworms of cervids. *In* Parasitic diseases of wild mammals, W. M. Samuel, M. J. Pybus, and A. A. Kocan (eds.). Iowa State University Press, Ames, Iowa, pp. 228–278.

LANKESTER, M. W., AND W. M. SAMUEL. 1998. Pests, parasites, and diseases. *In* Ecology and management of the North American moose, A. W. Franzmann and C. C. Schwartz (eds.). Smithsonian Institution Press, Washington D.C., pp. 479–517.

LARKIN, J. L., K. J. ALEXY, D. C. BOLIN, D. S. MAEHR, J. J. COX, M. W. WICHROWSKI, AND N. W. SEWARD. 2003. Meningeal worm in a reintroduced elk population in Kentucky. Journal of Wildlife Diseases 39: 588–592.

MORAN, R. W. 1973. The Rocky Mountain elk in Michigan. Research and development report Number 267, Michigan Department of Natural Resources, Wildlife Division, Lansing, Michigan, 93 pp.

OLSEN, A., AND A. WOLF. 1979. A summary of the prevalence of *Parelaphostrongylus tenuis* in a captive wapiti population. Journal of Wildlife Diseases 15: 33–35.

RASKEVITZ, R. F., A. A. KOCAN, AND J. H. SHAW. 1991. Gastropod availability and habitat utilization by wapiti and white-tailed deer sympatric on range enzootic for meningeal worm. Journal of Wildlife Diseases 27: 92–101.

SAMUEL, W. M., M. J. PYBUS, D. A. WELCH, AND C. J. WILKE. 1992. Elk as a potential host for meningeal worm: Implications for translocation. Journal of Wildlife Management 56: 629–639.

SEVERINGHAUS, C. W., AND R. W. DARROW. 1976. Failure of elk to survive in the Adirondacks. New York Fish and Game Journal 23: 98–99.

TELFER, E. S. 1967. Comparison of moose and deer winter range in Nova Scotia. Journal of Wildlife Management 31: 418–425.

WHITLAW, H. A., AND M. W. LANKESTER. 1994. A retrospective evaluation of the effects of parelaphostrongylosis on moose populations. Canadian Journal of Zoology 72: 1–7.

ZAR, J. H. 1996. Biostatistical analysis. Third edition. Prentice Hall, Upper Saddle River, New Jersey, 662 pp. and appendices.

Received for publication 24 October 2003.