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Source: Journal of Wildlife Diseases, 39(3) : 588-592

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-39.3.588

Journal of Wildlife Diseases, 39(3), 2003, pp. 588–592

MENINGEAL WORM IN A REINTRODUCED ELK POPULATION IN KENTUCKY

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ABSTRACT: Meningeal worm (*Parelaphostrongylus tenuis*) has been implicated in the failure of several elk (*Cervus elaphus*) restoration attempts in the eastern United States. However, limited post-release monitoring and a paucity of published literature prevents a clear understanding of this parasite's role in past failures. During winters of 1997–2001, the Kentucky Department of Fish and Wildlife Resources translocated 1,044 elk from western states to eastern Kentucky (USA) in an effort to restore a free-ranging population. We monitored 521 radio-collared elk over 4 yr to determine the impact meningeal worm had on population establishment. Thirty (23%) of 129 non-capture related mortalities were attributed to meningeal worm. Twenty-two (73%) of these meningeal worm-caused mortalities were animals <3 yr old. If younger elk born in Kentucky suffer higher mortality rates than older translocated elk, the population growth observed during the initial years of restoration may be temporary. Additional research is necessary to determine the influence meningeal worm will have on elk population growth in Kentucky.

Key words: *Cervus elaphus,* elk, meningeal worm, *Parelaphostrongylus tenuis,* reintroduction, translocation.

INTRODUCTION

The most common disease among reintroduced elk (*Cervus elaphus*) in the eastern United States is caused by meningeal worm (*Parelaphostrongylus tenuis*) (Severinghaus and Darrow, 1976; Eveland et al., 1979; Raskevitz et al., 1991). This parasitic nematode inhabits the central nervous system of its primary host the white-tailed deer (*Odocoileus virginianus*), usually without adverse impact (Anderson, 1972; Maze and Johnstone, 1986). Although rarely affected by the parasite, most white-tailed deer become infected by the time they are 1.5 yr of age (Slomke et al., 1995). Meningeal worm can cause fatal neurologic disease in elk and has been implicated in the failure of several attempts to restore the species to eastern states (Carpenter et al., 1973; Severinghaus and Darrow, 1976; Raskevitz et al., 1991). There is a paucity of empirical evidence relating the meningeal worm to these failures so clear cause and effect relationships between the parasite and elk cannot be claimed (Nudds, 1990; Lankester, 2001).

In 1997, the Kentucky Department of Fish and Wildlife Resources (KDFWR) began the reintroduction of free-ranging elk to eastern Kentucky (USA) (Maehr et al., 1999). Prior to this, elk were absent from the state for over 150 yr. The Kentucky (USA) elk restoration project targeted release of 1,800 elk over a 9 yr period and utilized stock from Arizona, Kansas, North Dakota, Oregon, New Mexico, and Utah (USA). The first 500 translocated elk were radio-instrumented and monitored to determine survival, population growth, habitat use, and movement. We documented mortality in the Kentucky elk population to determine factors that may inhibit restoration success. This paper examines occurrence of meningeal worm in elk in Kentucky during the first 4 yr of restoration and provides insight into its effects on elk population establishment and long-term viability.

STUDY AREA AND METHODS

Southeastern Kentucky was selected as the restoration zone because of its low human population and distance from agriculture and major

urban centers (Larkin et al., 2001). The restoration zone comprises 14 counties and covers 1.04 million ha in the Cumberland Plateau physiographic region (Maehr et al., 1999). Active and reclaimed coal surface mines, the Daniel Boone National Forest, and private forests dominate the landscape. Narrow, winding ridges, steep slopes, and narrow valleys characterize much of the restoration zone (McFarlan, 1943). Elsewhere, surface-mining for coal and subsequent reclamation has converted large areas (up to 5,000-ha) of rugged topography into flat to gently sloping grasslands. The restoration zone is 93% forested, 6% reclaimed surface mines, and 1% agriculture (Phillips, 1997). Further details regarding the study area and release sites can be found in Larkin et al. (2001).

During the winters of 1997–2001, elk were captured in western source states. If an elk passed a series of diagnostic tests and was in good health, it was translocated non-stop to Kentucky and released upon arrival. Prior to translocation, elk were fitted with VHF transmitter collars that contained mortality sensors programmed to activate after 4 hr of non-movement.

Monitoring of radio-collared elk began within 24 hr post-release. Elk were monitored on a weekly basis from December 1997 through December 2001 unless weather conditions precluded telemetry flights. Transmitters in mortality mode were recovered as soon as possible and dead animals were examined for cause of death (Unsworth et al., 1993). We also investigated deaths of uncollared elk reported by local citizens and conservation officers. Fresh carcasses were brought to the University of Kentucky Livestock Disease Diagnostic Center (LDDC; Lexington, Kentucky) where a complete necropsy was conducted to determine cause of death. If carcasses could not be retrieved from the field, the head and blood samples were collected and submitted to the LDDC. Elk submitted for necropsy were examined for gross and microscopic lesions and tissue and fluid samples, when suitable, were collected for routine bacterial culture and virus isolation. Tissues for histologic examination were immersed in 10% neutral buffered formalin, processed for sectioning, embedded in paraffin, sectioned at 5 μm, and stained with Harris hematoxylin and eosin.

We used the following protocol to exam brains for meningeal worm infection. The brain was removed intact and the outer surface examined for the presence of adult nematodes. The cerebral hemispheres were then separated along the longitudinal fissure and removed from the brainstem and again re-examined for adult nematodes. Sections through the anterior, middle, and posterior portions of the cerebrum, cerebellum, thalamus, pons, and medulla oblongata at the level of the obex were taken for histologic evaluation. Histologically, the diagnosis of meningeal worm infection was based on the presence of adult nematodes and or larvae in neural tissue and lesions consistent with parasite migration (longitudinal tracts or cavitations with or without hemorrhage, glial scarring, eosinophilic perivascular inflammation, and eosinophilic or nonsuppurative meningitis with hemosiderin laden macrophages). Deaths were attributed to meningeal worm infection if the above evidence was observed and there was an absence of concurrent histologic, microbiologic, or virologic disease processes. Dead elk that were not necropsied and for which there was no obvious cause of death were categorized as unknown. Death locations were recorded as Universal Transverse Mercator (UTM) coordinates with accompanying habitat descriptions.

RESULTS

A total of 1,044 elk were translocated from five western source states during the first 4 yr of restoration efforts. Five-hundred twenty-one (50%) translocated elk were equipped with radio-collars. At least 650 calves were born during the first four calving seasons (Larkin, 2001; Larkin et al., 2002). The population numbered as many as 1500 on 31 December 2001.

We documented 214 deaths from December 1997 through December 2001 (Table 1). Of 436 radio-collared elk that survived translocation, 8 (2%) died as a result of meningeal worm infection. Meningeal worm was responsible for 30 of 129 (23%) non-capture related mortalities. Seventeen (57%) of these mortalities were calves (<1 yr old) and yearlings. Twelve (70%) of these 17 meningeal worm deaths were of Kentucky-born elk, whereas, five occurred among translocated calves from western states. Meningeal worm killed five elk that were 2 yr old (three Kentucky-born and two translocated). Eight meningeal worm deaths occurred among elk that were ≥3 yr old (four translocated as calves and four as adults). Cause of death was not positively determined for 37 animals. Among 34 cases, wherein the parasite did not appear to directly contribute to mortality (poaching,

TABLE 1. Elk mortality in a restored elk population in eastern Kentucky from December 1997 through December 2001. Numbers to the left of parenthesis represent mortalities of translocated, radio-collared elk. Numbers inside of parenthesis represent mortalities of animals born in Kentucky. Numbers to the right of parenthesis represent mortalities of translocated, uncollared elk.

Cause of death	Adult female (>2 yr)	Adult male (>2 yr)	Yearling female	Yearling male	Calf female (<1 yr)	Calf male (<1 yr)	Total
Capture-related	43 (0) 0	2 (0) 0	16 (0) 0	5 (0) 0	9 (0) 0	10 (0) 0	85 (0) 0
Auto collision	8 (2) 4	2 (1) 0	2 (3) 0	1 (0) 0	0 (1) 0	0 (1) 0	13 (8) 4
Meningeal worm	4 (3) 5	1 (0) 0	3 (3) 2	0 (3) 0	0 (2) 0	0 (4) 0	8 (15) 7
Poached	2 (1) 0	0 (0) 0	1 (0) 0	1 (1) 0	0 (2) 0	0 (0) 0	4 (4) 0
Removed	4 (0) 0	1 (0) 0	0 (0) 0	0 (0) 0	0 (0) 0	0 (1) 0	5 (1) 0
Unknown	22 (0) 0	1 (0) 0	8 (0) 0	4 (0) 1	0 (0) 0	1 (0) 0	36 (0) 1
Hunting	0 (1) 4	1 (1) 4	0 (0) 0	0 (0) 0	0 (0) 0	0 (1) 0	1 (3) 8
Other	6 (0) 0	3 (0) 0	0 (0) 0	0 (0) 0	1 (0) 0	0 (1) 0	10 (1) 0
Total	89 (7) 13	11 (2) 4	30 (6) 2	11 (4) 1	10 (5) 0	11 (8) 0	162 (32) 20

out-of-zone removals, hunting, and automobile trauma), 10 (29%) were infected with meningeal worm.

DISCUSSION

Our observations indicate meningeal worm has the potential to significantly retard elk population growth in Kentucky. While only 2% of radio-collared elk that survived translocation died from meningeal worm, the parasite may have affected up to 10% as there were 36 mortalities for which the cause of death could not be determined. Further, an even greater proportion of the total elk population (translocated plus Kentucky-born elk) may be affected by meningeal worm because elk that were born in Kentucky were not monitored for mortality; our data suggests this group is the most susceptible to meningeal worm-caused mortality.

Elk <3 yr of age accounted for 73% of all meningeal worm deaths suggesting a higher vulnerability of juvenile and subadults. A similar pattern was reported in a captive elk herd in Pennsylvania (USA) (Olsen and Wolfe, 1979). Additionally, our findings suggest younger elk born in Kentucky may be more susceptible to meningeal worm infection than older translocated elk. Of 47 radio-collared calves that survived translocation to Kentucky in 1998, none died from meningeal worm prior to 1.5 yr of age. Conversely, five (15%) of the calves born in Kentucky during 1998 died from meningeal worm within 1.5 yr of birth. The actual rate of calf mortality due to meningeal worm may have been higher because calves born in Kentucky were not radio-collared during the first 4 yr of study. Translocated calves were >8 mo of age when released and rarely developed the disease (Larkin, 2001). This suggests that younger Kentucky-born elk ingest enough parasites prior to 8 mo of age to develop signs of infection. Severity of infection may be age dependent (Olsen and Woolf, 1979), and elk >8 mo of age could have a stronger immune response to meningeal worm infection.

The 29% infection rate among Kentucky elk without clinical signs of meningeal worm infection is comparable to findings in a Pennsylvania herd (Olsen and Wolfe, 1979). Although Pennsylvania elk had 34% infection, only 14% of these cases exhibited clinical signs (Olsen and Wolfe, 1979). While meningeal worm infection was not the primary cause of death for some infected elk in Kentucky, it is possible that the parasite could have made them more susceptible to other mortality factors (i.e., auto collision or sport hunting). Elk that inhabit areas with high white-tailed deer densities may have a greater risk of meningeal worm-related death due to increased exposure to infected gastropods (Karns, 1967; Raskevitz et

al., 1991). Habitat segregation and divergent foraging behaviors are believed to protect elk from white-tailed deer-induced meningeal worm infection elsewhere (Raskevitz et al., 1991). If Kentucky-born elk suffer higher mortality rates than translocated elk, the population growth that occurred during the early years of restoration may be unsustainable. Samuel et al. (1992) suggested that self-sustaining elk populations in the east rely on individuals that survive with low amounts of meningeal worm infestation.

If elk populations are susceptible to meningeal worm induced disease then how did the species inhabit much of eastern North America before the arrival of Europeans? An understanding of the ecologic relationship between white-tailed deer and elk in pre-settlement eastern North America would be requisite in speculating as to what interspecific relationship existed. Moreover, such thought may provide managers with insight into the dynamics of future elk populations that have been, or will be, reintroduced to areas sympatric with meningeal worm. We suggest several scenarios regarding the potential relations that may have existed among pre-European settlement elk, white-tailed deer, and meningeal worm. First, white-tailed deer and elk densities were likely lower in pre-settlement North America compared to today due to less edge habitat and the presence of large predators. Contact between elk and deer may have been limited and, thus, elk infection rates lower than what we observe in elk populations inhabiting fragmented landscapes of modern day eastern North America. An alternative explanation may have been that while both elk and white-tailed deer both occurred in eastern North America, they may have utilized different habitats. Any attempt by elk to utilize white-tailed deer habitats may have resulted in significant elk mortality due to meningeal worm, a phenomenon referred to as parasite-mediated competition (Price et al., 1988). Population declines of other ungulate species such as moose (*Alces alces*) and caribou (*Rangifer tarandus*) have been associated with a range expansion of white-tailed deer, and high mortality due to meningeal worm infection was thought to be the primary cause (Anderson, 1972; Nudds, 1990).

Another scenario is that elk survived low-level infections in areas sympatric with white-tailed deer. Individual animals may have succumbed to infection, however single animals do not dictate the success of a population. Although cervid population size may be inversely related to intensity of meningeal worm infection, populations can still exist, albeit at lower densities (Samuel et al., 1992; Schmitz and Nudds, 1994; Whitlaw and Lankester, 1994). Innate or acquired immunity in eastern elk may have allowed them to survive if they were exposed to low numbers of larvae. Several field studies on various cervids have suggested that an acquired immunity may occur following low-dose infections of meningeal worm (Davidson et al., 1985 [fallow deer; *Dama dama*]; M. W. Lankester, pers. comm. [moose]).

A final possibility is that the eastern elk subspecies may have been subjected to the disease for a long enough period to have become a suitable host or resistant to the disease altogether (Anderson, 1972). Regardless of the relationship between pre-Columbian elk and white-tailed deer, additional research is necessary to determine the influence meningeal worm will have on elk population growth in Kentucky. Future studies should use an experimental approach specifically designed to examine the spatial relationship between and relative densities of elk, white-tailed deer, and meningeal worm in the eastern Kentucky landscape.

ACKNOWLEDGMENTS

We appreciate the financial support of the Rocky Mountain Elk Foundation, Kentucky Department of Fish and Wildlife Resources, Shikar Safari Foundation, and the University of Kentucky. We are thankful to T. Toman, R. Grimes, and J. Gassett for their enthusiastic

support of our research. We thank C. Logsdon, D. Crank, S. Ray, W. Amburgey, and G. Ison for their efforts in the field. We are especially grateful to Division of Air Transport pilots L. Austin, J. Bigham, and M. Clemens, and Friendship Flying pilots J. Hylton, L. Cole, N. Woods, P. Teas, D. Hylton, and J. Taylor. We thank the citizens of eastern Kentucky for their overwhelming support and enthusiasm regarding the restoration effort and for reporting dead or sick animals. This manuscript is number 03-09-048, and is approved for publication by the Director of the Agricultural Experiment Station at the University of Kentucky.

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Received for publication 20 September 2002.