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MENINGEAL WORM (*PARELAPHOSTRONGYLUS TENUIS*) AS A CAUSE OF MORTALITY IN THE RESTORED ELK (*CERVUS CANADENSIS*) POPULATION IN MISSOURI, USA

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ABSTRACT: Meningeal worm (*Parelaphostrongylus tenuis*) is an important cause of mortality of elk (*Cervus canadensis*) in populations in the eastern US and has been implicated in the failure of several restoration attempts. From 2011 to 2013, the Missouri Department of Conservation translocated 108 adult and yearling elk from Kentucky (US) to southern Missouri (US) to establish a free-ranging population. From release in spring 2011 through August 2015, we monitored 167 elk (adult, yearling, and calf) to determine causes of mortality. Of 78 mortalities, 26 (33%) were linked to meningeal worm based on necropsy results and/or observed behavior; this group included 19 elk with confirmed or suspected cases of meningeal worm infection that died of other proximate causes. Other important mortality sources included euthanasia (n=11, 14%), emaciation (n=7, 9%), and predation (n=5, 6%). Eleven of the 26 (42%) meningeal worm-related mortalities were adults, and 22 (85%) were female. Meningeal worm was an important cause of mortality during the restoration of Missouri elk, potentially contributing to the loss of 16% of the monitored individuals. Greater mortality in adult female elk could reduce initial population growth by limiting reproductive output in the restored herd, especially given that females were disproportionately affected in Missouri. Because translocated Missouri elk undoubtedly were exposed to meningeal worm in Kentucky, our results could be explained by exposure to a different genetic strain of meningeal worm once in Missouri, loss of immune response due to translocation, increased dose of larval worms, or some unquantified factor.

Key words: *Cervus canadensis*, elk, meningeal worm, *Parelaphostrongylus tenuis*, restoration, white-tailed deer.

INTRODUCTION

Meningeal worm (*Parelaphostrongylus tenuis*) causes the most common disease in restored elk (*Cervus canadensis*) in the eastern US (Eveland et al. 1979; Raskevitz et al. 1991) and has potentially contributed to the failure of several elk restoration attempts in the region (Carpenter et al. 1973; Severinghaus and Darrow 1976). The parasitic nematode inhabits the central nervous system of white-tailed deer (*Odocoileus virginianus*), which is the primary host, and which rarely experiences adverse effects (Anderson 1972; Maze and Johnstone 1986). Ontario, Canada, and numerous states in the US have already conducted elk restorations, and other states are considering doing so in the future. Thus, assessment of the role of meningeal worm as a cause of mortality in the early years of elk restorations in areas containing white-tailed deer is important for understanding limiting factors and potential population growth (Larkin et al. 2003).

In 2011, the Missouri Department of Conservation (MDC) began restoring a free-ranging elk herd in southern Missouri (Dent 2014). Elk once ranged across the entire state but had been absent for over 150 yr (MDC 2000). Over a 3 yr period, MDC translocated

adult and yearling elk from Kentucky's free-ranging herd, which is located in a 16-county area in the southeastern part of the state. Radiotags were used to monitor survival, resource selection, movement, and dispersal of released elk (Bleisch 2014; Smith 2015). Additionally, some females were pregnant when they arrived, so expandable radiotags were deployed on calves born in the acclimation pens before release or those discovered by field crews during searches after release. Using 167 radiotagged elk, we documented the causes of mortality in the Missouri population and determined the prevalence of meningeal worm during the first 4 yr of restoration.

MATERIALS AND METHODS

The elk restoration zone in the Missouri Ozarks included Carter (36°59′44″N, 91°0′53″W), Reynolds (37°26′6″N, 90°57′31″W), and Shannon (37°9′2″N, 91°21′27″W) counties and was selected due to its high percentage of public land, minimal row crop agriculture, low human population density, and low road density (MDC 2010). The counties covered 602,690 ha in the Ozark Highlands region of Missouri, which is dominated by oak (*Quercus* spp.), hickory (*Carya* spp.), and pine (*Pinus* spp.) forests. The restoration zone was 93% forested, 5% open, and 0.1% cropland (MDC 2010). Open lands were a combination of glades dominated by warm-season grasses and forbs, food plots planted with cool- and warm-season grasses and legumes, and pastures (MDC 2010). The majority of the restoration zone (79%) was open for public access, with 49% publicly owned (and managed by MDC, the US Forest Service, and the National Park Service) and another 30% owned and managed by The Nature Conservancy and the L-A-D Foundation (a local private organization; MDC 2010).

During January 2011–13, we captured elk in Kentucky using corral traps and held them in pens for 102–129 d to complete health testing (Bleisch 2014), which included screening for diseases (e.g., bovine tuberculosis, brucellosis, blue tongue, epizootic hemorrhagic disease), evaluating body condition, and treatments with antibiotics and anthelmintics (Dent 2014). We fitted elk with GPS radiotags (RASSL 3D cell collar, North Star Science and Technology, King George, Virginia, USA; G2110E Iridium/GPS series model, Advanced Telemetry Systems, Isanti, Minnesota, USA) that contained motion-sensitive mortality sensors programmed to activate after 4 h without movement. We translocated the elk overnight to Peck Ranch Conservation Area (37°2′33″N, 91°11′12″W; Carter County) in the Missouri elk restoration zone and held them in acclimation pens for 19–34 d until release in June (Bleisch 2014). We fitted calves born in the pens or discovered by field crews after being born in the wild with expandable, break-away very high-frequency radiotags (Model M2230B, Advanced Telemetry Systems).

When radiotags indicated mortality, we recovered them as soon as possible to examine carcasses for cause of mortality. Trained personnel conducted necropsies, either in the field or at MDC's Central Regional Office; fresh carcasses were also taken to the University of Missouri's College of Veterinary Medicine for examination by veterinarians and veterinary students. If carcasses could not be retrieved from the field, we collected the head and tissue samples (i.e., brain, heart, lung, liver, spleen, and kidney); tissue samples were either stored fresh or preserved in 10% neutral buffered formalin. We submitted tissue samples to the Southeastern Cooperative Wildlife Disease Study at the College of Veterinary Medicine, University of Georgia, for additional diagnostics. If cause of death was undetermined after the necropsy results and field evidence were examined, the mortality was identified as unassigned. When necropsy and field evidence provided enough data to diagnose cause of mortality, we assigned the proximate cause as the final cause of mortality. We confirmed meningeal worm infection at necropsy based on the presence of adult nematodes, larvae, or eggs in neural tissue, and we suspected meningeal worm infection when necropsy revealed lesions in neural tissue consistent with parasite migration. In the absence of any other proximate cause of mortality for these cases, the cause of mortality was considered to be meningeal worm infection. Thus, we assigned final cause of mortality on a case-by-case basis in the context of field and necropsy findings, and we noted when available evidence indicating that meningeal worm could have been a contributing factor in the mortality. If any elk exhibited clinical signs of meningeal worm infection such as ataxia, circling, and vision impairment (Carpenter et al. 1973) prior to death by any cause, they were considered a meningeal worm–linked mortality, even if there was a proximate cause of death or necropsy was not possible or was inconclusive. For example, following these methods, if an elk was euthanized, its final cause of death was euthanasia, even if it was euthanized because it showed clinical signs of meningeal worm, and subsequent necropsy confirmed the infection; thus, these elk were assigned to the euthanasia category but were also considered meningeal worm–linked mortalities. We

TABLE 1. Causes of mortality following the elk (*Cervus canadensis*) translocation from Kentucky to Missouri, USA, June 2011–August 2015. For each cause of mortality and sex/age class, the linkage to meningeal worm infection was based on behavior and/or necropsy.

		Causes of mortality (no. linked with meningeal worm)									
Sex	Age[a]	Unassigned[b]	Unknown[c]	Other	Euthanasia	Meningeal worm	Emaciation	Predation	Trauma	Vehicle collision	Total
Female	Calf	2	1	4 (1)	4 (3)	1 (1)	4	2 (1)	0	0	18 (6)
	Yearling	3	0	2 (1)	3 (3)	1 (1)	0	1 (1)	0	0	10 (6)
	Adult	11 (3)	8	0	4 (2)	2 (2)	0	0	4 (2)	1 (1)	30 (10)
Male	Calf	2	4	5	0	0	3	2 (1)	0	0	16 (1)
	Yearling	0	0	1	0	2 (2)	0	0	0	0	3 (2)
	Adult	0	0	0	0	1 (1)	0	0	0	0	1 (1)
Total		18 (3)	13	12 (2)	11 (8)	7 (7)	7	5 (3)	4 (2)	1 (1)	78 (26)

[a] Calf (≤1 yr old), yearling (between 1 and 2 yr old), adult (≥2 yr old).

[b] Elk with necropsy, field, or behavioral evidence that was inconclusive for cause of mortality.

[c] Elk without any necropsy, field, or behavioral evidence to support cause of mortality.

classified all elk that were not necropsied and for which there was no behavioral data or obvious cause of death as unknown.

RESULTS

Removing mortalities that occurred before release and radio tag failures (n=2), we monitored 106 (81 females, 25 males) elk translocated from Kentucky to Missouri. Additionally, we monitored 56 calves and five other elk (three females, two males) born, captured, and radiotagged in Missouri during the restoration. In total, we monitored 167 elk in the wild, and of those, we documented 78 mortalities during the study period (Table 1). Cause of mortality could not be determined, and was thus categorized as unknown, for 13 of the 78 (17%) individuals. Additionally, we labeled 18 (23%) as unassigned cause of mortality because available data (i.e., necropsy findings, behavior) were inconclusive. Seven (9%) died directly as a result of meningeal worm infection that was confirmed by necropsy. Additional important mortality sources included various systemic maladies (all included in the "other" category), for example, pneumonia or septicemia (n=12, 15%), euthanasia (n=11, 14%), and emaciation (n=7, 9%; Table 1). In addition to the seven that died of meningeal worm, we confirmed or suspected meningeal worm infection in 19 other elk. For example, of 11 elk that were euthanized by MDC, eight were confirmed or suspected to have meningeal worm infection based on necropsy results and/or behaviors observed in the field (Table 1). Thus, 26 (33%) elk had evidence of meningeal worm infection regardless of their proximate cause of death. Using those 26 elk as a conservative estimate of meningeal worm infection, prevalence of meningeal worm in our radiotagged sample of 167 elk during the restoration was 16%.

Of the 26 mortalities where meningeal worm infection was involved, females comprised the majority (n=22, 85%); also, 11 (42%) were adult, and 8 (31%) were yearling animals. Considering the total number of mortalities by sex, 22 of 58 (38%) female mortalities and four of 20 (20%) male mortalities were confirmed or suspected to be linked to meningeal worm infections. Of the 56 calves monitored during the study period, 34 (61%) died (Table 1); of those, seven (21%) were confirmed or suspected to be linked to meningeal worm infections.

DISCUSSION

Our most conservative assessment of meningeal worm–related mortality during the Missouri elk restoration suggests that 9% of elk deaths were attributable directly to the

parasite; however, other necropsy and behavioral evidence indicates that meningeal worm could have been contributing to a greater proportion of mortalities (up to 33%). Additionally, notable proportions (17%) of the mortalities were from unknown causes, with no necropsy or behavioral data to support a determination, so meningeal worm cannot be eliminated as a potential cause in those deaths. Thus, it is likely that the meningeal worm–related mortality we report is conservative. Indeed, in a review of eastern elk vital rates and cause-specific mortality, Keller et al. (2015) concluded that meningeal worm infections are likely to be underrepresented in the literature because behavioral changes in infected elk make them more likely to succumb to secondary infections, such as pneumonia, and more vulnerable to predation, vehicle collisions, and targeted removal (i.e., euthanasia). Though some evidence shows meningeal worm can be a minor mortality source and causes few population-level effects (Bender et al. 2005), impacts to a recently reintroduced elk population could be problematic when relatively few elk are used to establish the population. Thus, for newly established herds with high levels of meningeal worm mortality, the parasite could be a limiting factor.

The age and sex of elk that die of meningeal worm infection could have profound effects on population dynamics in the years following restoration. In Missouri, older translocated elk (i.e., elk born in Kentucky) comprised a large proportion of the meningeal worm–related mortalities. This result contrasts with results from Kentucky and Pennsylvania (Olsen and Woolf 1979; Larkin et al. 2003) that suggested a higher vulnerability to meningeal worm infection in juveniles and subadults. Additionally, meningeal worm mortality in the Missouri herd comprised a larger proportion of mortalities in females (38%) than males (20%), which is consistent with higher prevalence seen in young female elk in Pennsylvania (Olsen and Woolf 1979). Variation in demographic rates of long-lived herbivores is characterized by high and weakly variable adult survival, with highly variable juvenile survival (Gaillard et al. 1998). Though juvenile survival may drive large herbivore population dynamics due to high variability, it tends to have a lower relative effect on population growth rate compared to adult survival (Gaillard et al. 2000). Hence, high levels of meningeal worm mortality in adult females could play a major role in limiting the population growth rate during the establishment of any translocated elk herd.

It is possible that after a population becomes established, it might be less susceptible to meningeal worm mortality, or susceptibility could shift to younger age cohorts. In Kentucky, younger elk born in the state appeared to be more susceptible to meningeal worm infection than older translocated individuals (Larkin et al. 2003). Larkin et al. (2003) suggested that younger Kentucky-born elk might ingest enough parasites prior to 8 mo of age to develop clinical signs of infection. By contrast, neonates may receive maternal antibodies to meningeal worm, giving young elk the capability to fight off infections (Bowling 2009). Severity of infection may be age dependent (Olsen and Woolf 1979), and older elk may have a stronger immune response to meningeal worm infection (Larkin et al. 2003). Age-specific and dose-dependent (Samuel et al. 1992) responses of elk to meningeal worms are important factors for understanding elk population dynamics and warrant future attention.

Given the high number of unknown mortalities in Missouri and the fact that subclinical meningeal worm infections could have existed in elk that did not die during the study, the true rate of meningeal worm infection in the Missouri elk herd is unknown. Larkin et al. (2003) reported a 29% infection rate among Kentucky elk that did not show clinical signs of meningeal worm infection, and their results are comparable with reports from Pennsylvania, which showed a 34% infection rate, with only 14% of cases exhibiting clinical signs (Olsen and Wolfe 1979). As acknowledged by Larkin et al. (2003), such background levels of infection could predispose elk to other mortality sources (e.g., vehicle collision, hunting), obscuring the overall effect of meningeal

worm on eastern elk population dynamics. In Missouri, concern for animal welfare resulted in euthanasia of elk that showed clinical signs of meningeal worm infection. Missouri currently does not have an elk hunting season, but if hunting seasons are instituted, then future mortality from hunting could further mask background infection rates of meningeal worm.

Larkin et al. (2003) questioned how elk populations could be susceptible to meningeal worm, given that elk inhabited much of eastern North America prior to the arrival of Europeans. Indeed, numerous possibilities exist to potentially explain the population-level effects of meningeal worm mortality that we reported from Missouri. First, elk translocated from Kentucky to Missouri could have been naïve to the parasite. However, available evidence indicates that *P. tenuis* is widespread in Kentucky and that elk are exposed to it (Comer et al. 1991; Alexy 2004), making it unlikely that infection rates upon arrival in Missouri were simply due to naïve elk. That said, use of anthelmintics as part of the translocation could have affected just how ''naïve'' elk were upon their arrival in Missouri. Second, variations in deer density between Kentucky and Missouri could explain differences in elk exposure to larval parasites or doses they ingested. Deer density in the elk restoration zone of Kentucky is approximately four deer per square kilometer (McDermott 2017), while deer density in the elk restoration zone of Missouri is greater (M.C.C. pers. obs.). However, this possible explanation appears unlikely given that elk in Kentucky are exposed to and die from *P. tenuis* even at low deer densities (Larkin et al. 2003); further, Alexy (2004) reported significant infection rates in intermediate hosts across the Kentucky elk restoration zone. Third, individuals may develop an immune response to meningeal worm infection, potentially protecting them from subsequent exposure (Bienek et al. 1998; Bowling 2009). Perhaps the stress of translocation could have resulted in reduced immune function once elk arrived in Missouri, making meningeal worm or other infections more probable (Waas et al. 1999; Teixeira et al. 2007) in the early years of the reintroduction. Fourth, resource selection could predispose elk in Missouri to greater risk of infection. However, diet analysis of elk in Kentucky showed they forage in large openings, like on reclaimed surface mines (Schneider et al. 2006), which is consistent with elk in Missouri foraging in managed openings (Smith 2015). Thus, unless gastropod (i.e., intermediate host) availability, abundance, and infectivity are different across cover types and across states, it seems unlikely that elk foraging behavior explains differences in meningeal worm–related mortality. Finally, it is possible that variations in pathogenicity of *P. tenuis* exist. If genetic differences exist within *P. tenuis* across its range, exposure to novel populations could render previous immunity ineffective. Such a scenario could explain why the elk we moved from Kentucky to Missouri appeared to demonstrate significant morbidity and mortality in their new habitat. Investigating this scenario might help elucidate patterns of innate or acquired immunity, especially if related to dose (i.e., number of larvae ingested or number of times encountered; Samuel et al. 1992). Davidson et al. (1985) suggested that acquired immunity may occur after low-dose infections, but genetic differences in *P. tenuis* across its range may influence dosage effects. Regardless of which of these scenarios best explains our results, understanding how and why local characteristics appear to affect the level of mortality resulting from meningeal worm infection in elk populations, for example, the very low rate of mortality of elk in Michigan (Bender et al. 2005), has important implications for new and established elk herds in eastern North America.

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