DISTRIBUTION AND PREVALENCE OF ELAEOPHORA SCHNEIDERI IN MOOSE IN WYOMING

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ABSTRACT: *Elaeophora schneideri* causes disease in aberrant hosts such as moose. Documented *E. schneideri* infections in moose are relatively rare, yet noteworthy enough that individual cases describing morbidity and mortality have been the norm for reporting. Surveillance efforts for *E. schneideri* in Wyoming moose in the 1970s found zero cases, but since 2000 several moose in Wyoming discovered dead or showing clinical signs of elaeophorosis have been found infected with *E. schneideri.* In 2009 we searched for worms in the carotid arteries of 168 hunter-harvested moose from across Wyoming to determine the prevalence and distribution of *E. schneideri* in moose; 82 (48.8%; 95% CI: 41.4-56.3%) were positive for *E. schneideri*. Prevalence did not differ between sexes or among age classes but there was difference in prevalence among herd units (range = 5-82.6%). Intensity of infection (range = 1-26 worms) did not differ between sexes, among age classes, or among herd units. Our findings indicate that moose do not succumb to the parasite to the extent previously thought. Prevalence and intensity were constant across age classes, suggesting that infected moose are surviving and an acquired, immunological resistance to further infection develops. In addition, moose might sometimes act as natural hosts to the parasite, as indicated by 1) high prevalence of infection in moose in areas where sympatric mule deer had much lower prevalence of infection, and 2) preliminary necropsy findings that revealed microfilariae in skin samples from 3 moose. However, negative impacts to moose and moose populations cannot be ruled out entirely, as this study was limited to apparently healthy hunter-harvested animals. While moose appear to often survive infection with *E. schneideri*, prevalence of ~50% is still cause for concern because it is unknown to what extent this parasite causes subclinical effects in moose that might impact recruitment or productivity. Subsequent research on moose herds where *E. schneideri* occurs should consider the effects of elaeophorosis and attempt to clarify its role.

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Elaeophora schneideri is a filarioid nematode that lives in the cephalic arteries of mule deer (*Odocoileus hemionus*) (Hibler et al. 1970) and black-tailed deer (*O. hemionus columbianus*) (Weinmann et al. 1973). Adult nematodes in the arteries of deer give birth to live young - microfilariae (Hibler and Adcock 1971). Microfilariae then migrate to the capillaries in the dermis of the host's face and forehead where they are taken up in the blood meal of the intermediate host. Horse flies (Family Tabanidae) of the genera *Hybomitra*, *Silvius*, and *Tabanus* (Hibler et al. 1970, Clark and Hibler 1973, Espinosa

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1983) are intermediate hosts of the parasite. Transmission of the third stage infective to the vertebrate host occurs after *E. schneideri* larvae develop in the horse fly vector for 2-3 weeks (Hibler and Adcock 1971, Hibler and Metzger 1974, Davies 1979).

Development of *E. schneideri* in the definitive host has been described previously (Weinmann et al. 1973, Hibler and Metzger 1974). Less is known about development of *E. schneideri* in aberrant hosts but pathogenesis usually stems from the parasite's delayed migration through the host body or complications from circulatory impairment (i.e., elaeophorosis; Adcock and Hibler 1969, Hibler and Metzger 1974, Anderson 2001). Gross clinical signs of infection among aberrant hosts range from dry gangrene of nose and ear tips and antler malformations, to blindness, central nervous system damage, and death. Documented aberrant hosts of *E. schneideri* are moose (*Alces alces),* elk (*Cervus elaphus),* white-tailed deer (*O. virginianus),* bighorn (*Ovis canadensis*), and domestic sheep (Boyce et al. 1999, Anderson 2001).

E. schneideri is widespread across North America occurring in mule deer in Nebraska (McKown et al. 2007), South Dakota (Jacques et al. 2004), Utah (Pederson et al. 1985), Texas (Pence and Gray 1981), Colorado, and New Mexico (Davies 1979). It has been documented in white-tailed deer in Arizona (Hibler 1982), Texas (Waid et al. 1984), and several southeastern states (Prestwood and Ridgeway 1972). Infected elk have been reported from Oklahoma, New Mexico, Arizona, Colorado, and Wyoming (Hibler 1982).

The first documented *E. schneideri* infections in moose were in Montana (Worley et al. (1972). Subsequently, its presence in small numbers of moose was documented in Utah (Jensen et al. 1982), Colorado (Madden et al. 1991),Washington (Pessier et al. 1998), and Wyoming in 2000 (W. E. Cook, Wyoming Game and Fish Department [WGFD], unpublished report), and Oregon in 2010 (Matthews, 2012).

In Wyoming moose both the prevalence of infection and the parasite's geographic extent appear to have undergone a recent, notable increase. In 1973-74 Worley (1975) examined 74 apparently healthy, hunter-harvested moose: 69 from Teton and Fremont Counties in northwestern Wyoming, and 5 from Park and Gallatin Counties in southwestern Montana. No Wyoming moose and only 3 of 5 Montana moose were infected with worms in the carotid arteries. Presumably, low prevalence of *E. schneideri* in Wyoming led Hibler (1982) to believe elaeophorosis was of minimal importance to Wyoming elk and moose.

Whereas *E. schneideri* has been documented consistently in small numbers of mule deer and elk in Wyoming since 1967 (H. E. Edwards, WGFD, unpublished data), the first infected moose was not identified until much later. Within 2 weeks in January 2000, 2 moose were euthanized by WGFD field personnel in Fremont County (central Wyoming) because they were lethargic or walking in circles and showed signs of impaired vision; illness in each of those cases was attributed to elaeophorosis (W. E. Cook, unpublished report). In 2008 a 3-yr-old cow moose in western Wyoming was euthanized because of its abnormal behavior (lack of fear, blindness, and loss of motor skills). Upon gross examination, a heavy load of worms (30-50) was found in the carotid arteries and its clinical signs were attributed to elaeophorosis (C. M. Tate, WGFD, unpublished report).

The WGFD and the Wyoming State Veterinary Lab (WSVL) increased opportunistic surveillance of moose in 2008. Animals found dead, euthanized due to illness, and road-kills were examined for *E. schneideri*; several were found infected with *E. schneideri*. Most positive cases were from animals discovered dead or showing clinical signs of illness, and pathology associated with *E. schneideri* was implicated in several cases.

In order to survey moose for *E. schneideri*

more uniformly across Wyoming, a rigorous plan was developed to establish baseline data on prevalence and distribution of *E. schneideri* by surveying hunter-harvested moose during the 2009 hunting season. To our knowledge, this was the most comprehensive and widespread effort to date for surveillance of *E. schneideri* in moose.

STUDY AREA

Brimeyer and Thomas (2004) described the history and status of moose in Wyoming through the early 2000s. Moose in Wyoming occupy 3 distinct ranges: 1) Bighorn Mountain Range in north-central Wyoming, 2) the Snowy Range and Sierra Madre Ranges of southeast and south-central Wyoming, and 3) western Wyoming among comparatively connected mountain ranges from the Utah border north through Yellowstone National Park (Fig. 1a). Moose in Wyoming are managed as 11 herd units (herds) comprising discrete populations for which migration among adjacent herds is thought to account for <10% of a herd population. These herds are further divided into 43 hunt areas to provide flexibility for hunting seasons; begin and end dates of hunting seasons vary among areas. The WGFD has a statewide population objective of 14,630 moose (post-hunt), yet population estimates are considered relatively unreliable or completely lacking in most herds (Thomas 2008).

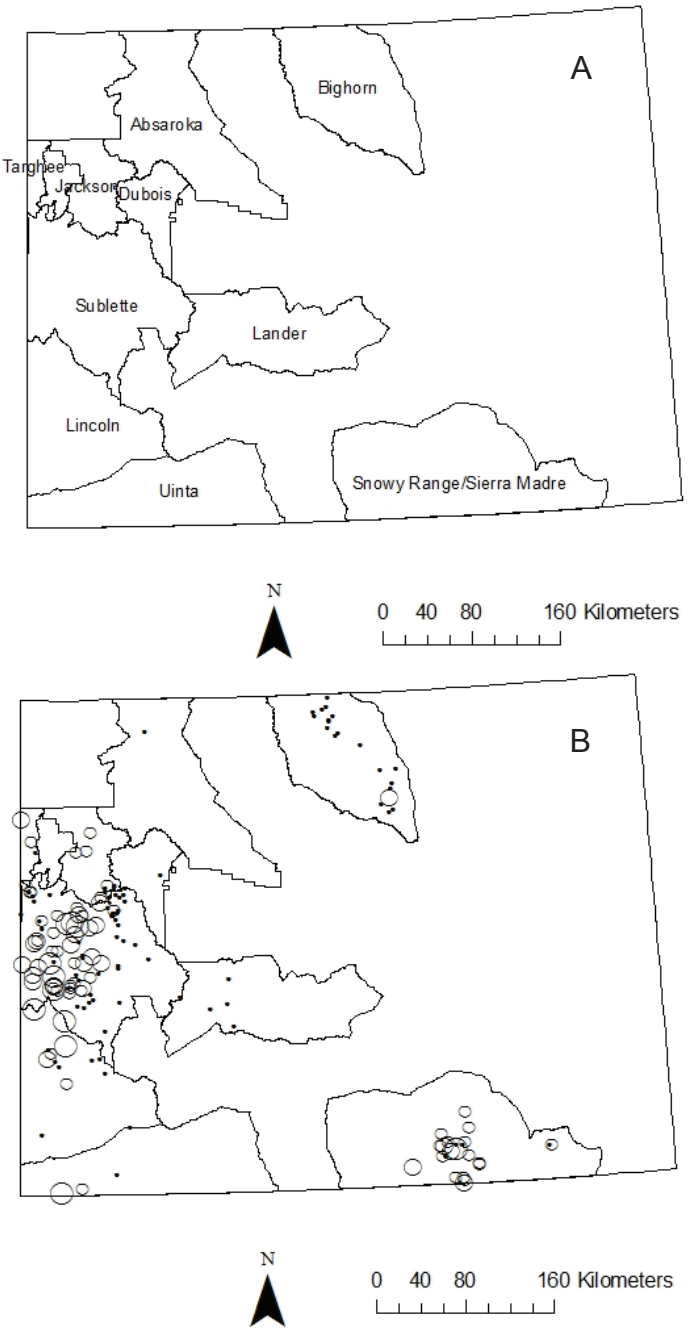

Fig. 1. A) Moose herd units that hunter-harvested moose were collected and sampled for *Elaeophora schneideri* in 2009 in Wyoming, USA. B) Intensity of *Elaeophora schneideri* found in carotid arteries categorized as none (•), low (○), moderate (○), and high (○) in hunter-harvested moose by herd unit in 2009 in Wyoming, USA.

METHODS

We examined hunter-harvested moose for the presence of immature or adult worms in the terminal portion of the common carotid arteries and in some instances the proximal portion of the internal maxillary arteries (hereafter field examinations). Many field examinations were conducted at hunter check stations and during opportunistic field checks of successful hunters. Field examinations also took place when hunters brought heads of harvested moose to WGFD regional offices, taxidermists, or meat processors.

Incisor teeth were collected from harvested moose for aging by cementum annuli. The specific ages obtained via cementum annuli are reported in whole years (i.e., yearling is 1). Successful development and migration of *E. schneideri* to the carotid arteries of moose would be expected to take 5-6 months (Hibler and Metzger 1974), thus nematodes would not be expected in the carotids until typically December. Thus field examinations as conducted in this study could not adequately diagnose infections in calves, and surveillance of calves was not included in this study.

Intensity of infection with worms (Bush et al. 1987) was recorded as 1 of 3 categories: 1-6, 7-13, and ≥14. These categories were based on intensities observed previously in moose from Wyoming and other states (Worley et al. 1972, Worley 1975, Madden et al. 1991). In addition to examining for the presence of *E. schneideri*, visual signs of elaeophorosis (e.g., cropped ears, necrotized tissues, lesions, or malformed antlers) were recorded.

Statistical Analysis

Prevalence was based on the total number of positive individuals; 95% confidence intervals (CI) around these proportions were calculated based on the binomial distribution (Rózsa et al. 2000). Fisher's exact test was used to test for homogeneity in prevalence between sexes. Chi-square was used to test for homogeneity in prevalence among age classes and herds; Fisher's exact test was used to test for differences between pairs of herds when contingency tables had ≤5 observations in ≥1 cell. Because of the small number of examined animals from individual age classes, 4 combined age classes were created (1, 2-4, 5-7, ≥ 8) for statistical comparisons. Likewise, some adjacent herds (Jackson and Targhee, Lincoln and Uinta) were combined to obtain adequate sample sizes for analyses. Some herds were dropped from analyses because they had both small sample sizes and were too geographically separate to justify merging. Because intensity was recorded as a categorical variable, chi-square was used to test for differences in intensity among sexes, age classes, and herds. Statistical significance was set at $P \leq 0.05$ for all tests. Calculations were accomplished using SIGMAPLOT 11 (Systat Software, San Jose, CA.).

RESULTS

Prevalence

The reported harvest in fall 2009 was 548 moose (394 adult males, 135 adult females, and 19 calves); 126 males and 42 females were examined for *E. schneideri* from 1 September-14 November (Table 1), or 31% of adult females and 32% of adult males in the harvest. *E. schneideri* was present in the carotid arteries of 48.8% of all moose ≥1 yr of age (95% CI: 41.4-56.3). Exactly 50% of males (95% CI: 41.4-58.6%) and 45.2% of females were infected (95% CI: 31.2-60.1%); prevalence did not differ by sex ($\chi^2 = 0.127$; $P = 0.72$). The number of females checked in each herd was small, but prevalence did not appear to differ between sexes within any individual herd. Thus, we combined sexes for statistical comparisons of prevalence among age classes and herds.

Ages were obtained from 151 of 168 moose: 9 were yearlings, 54 were 2-4 years old, 71 were 5-7 years old, and 17 were ≥8 years. The infection rate was 56% in yearlings (95% CI = 26.6-81.2%), 43% in 2-4 year olds

Table 1. Prevalence of *Elaeophora schneideri* in hunter-harvested moose, Wyoming, USA, 2009.

Herd	No. examined	No. infected	% infected (prevalence)	No. moose with differing intensities of infection			
				No worms	Low (1-6)	Moderate (7-13)	High (≥14)
Absaroka	1	0	0	1	0	0	0
Big Horns	20	1	5	19	0	1	0
Dubois	1	0	0	1	0	0	0
Jackson	10	6	60	4	6	0	0
Lander	4	0	0	4	0	0	0
Lincoln	13	5	38.5	8	2	2	1
Snowy Range[1]	23	19	82.6	4	13	4	1
Sublette	90	47	52.2	43	21	18	8
Targhee	3	2	66.7	1	1	1	0
Uinta	3	2	66.7	1	1	0	1
Total	168	82	48.8	86	44	26	11

[1]Includes 1 animal found positive for E. schneideri for which number of worms was not recorded.

(95% CI = 30.3-55.8%), 56% in 5-7 year olds (95% CI = 44.8-67.3%), and 41% in those ≥8 years (95% CI = 21.6-64.0%). There were no statistical differences in prevalence of *E. schneideri* among age classes (χ^2 = 2.420; df = 3; P = 0.490).

Moose from 10 herds were checked for *E. schneideri* (Table 1). To obtain adequate sample size for statistical comparison, the adjacent Jackson and Targhee herds, and the Lincoln and Uinta herds were combined; the Absaroka, Dubois, and Lander herds were dropped because of low sample sizes and geographic separation (Table 1). Prevalence was different geographically (χ^2 = 27.082, df = 4, P < 0.001). The lowest prevalence occurred in the Bighorn herd (5%; 95% CI = 0-25.4%) and was lower than that in the other 4 herds in the analysis. The Snowy Range herd had the highest prevalence (82.6%; 95% CI = 62.3-93.6%) which was higher than in the Bighorns, Lincoln-Uinta (43.8%; 95% CI = 23.1-66.8%), and Sublette herds (52.2%; 95% CI = 42.0-62.2%), but not different than in the Jackson-Targhee herds (61.5%; 95% CI = 35.4-82.4%).

Intensity

Of the 82 positive cases, we found 44, 26, and 11 moose with low, moderate, and high *E. Schneideri* intensity, respectively (Table 1); intensity was not recorded for 1 positive individual. The greatest number of worms counted in any moose was 26. Parasite intensity was similar between sexes (χ^2 = 0.564; df = 2; P = 0.754) and among age classes (χ^2 = 4.177; df = 6; P = 0.653). A low-intensity worm burden was most common in all age classes, ranging from 40-71%. None of the age classes had a large number of high-intensity worm loads. High-intensity infections were not observed in the 7 infected moose in the oldest age class (≥8 years).

Although prevalence was high in Snowy Range moose, most (74%) had low-intensity infections (Fig. 1b). Similarly, most positive individuals in the Jackson-Targhee herd (88%) had low-intensity infections. The only infected moose found in the Bighorns had a moderate-intensity infection (Table 1). The pattern of intensity was reversed in the Lincoln-Uinta and Sublette herds; more individuals had moderate and high intensities than low intensities. However, patterns of intensity were not different among herd units

($\chi^2 = 11.950$; df = 8; $P = 0.153$).

Clinical Signs

When possible, tissues were examined for gross evidence of damage as a result of infection by *E. schneideri*. More thorough examinations only occurred after heads had been prepared for taxidermy or when hunters donated their antlerless specimens. Of the 31 infected moose that were thoroughly examined, 10 showed visual signs of elaeophorosis: 7 displayed cropped or hardened ears, 1 had antler malformation, and 2 had cropped ears and antler malformation. Three of the 10 moose with visual signs had low-intensity infections, 4 had moderate-intensity, and 3 high-intensity infections.

DISCUSSION

Prevalence of *E. schneideri* in Wyoming moose was much higher than anticipated. Documented infections in moose have been fairly rare and noteworthy enough that individual cases have been the norm for reporting (Worley et al. 1972, Jensen et al. 1982, Madden et al. 1991, Pessier et al. 1998). The prevalence reported here is probably biased low because only the main cephalic arteries were examined for worms, yet post-mortem migration of worms occurs (Adcock and Hibler 1969). Furthermore, there was potential for false negatives because the length of the carotid artery was often short and compromised from hunter processing; there was no corresponding risk of false positives.

Prevalence of *E. schneideri* in adult mule deer has been 100% in certain local populations in the southwestern United States (Hibler and Adcock 1971). Prevalence has been as high as 93% in elk (Hibler et al. 1969, Davies 1979); high prevalence in elk occurs only in areas where mule deer also have high prevalence of infection. Our study focused solely on moose so we have no analogous surveillance data from deer and moose for comparison. However, opportunistic sampling indicated ~10% prevalence of *E. schneideri* in mule deer in a portion of the area comprising the Jackson, Targhee, and Sublette moose herds (J. C. Henningsen, unpublished data). It may be that prevalence of *E. schneideri* in mule deer is too low in Wyoming to generate >90% prevalence in moose; however, the Snowy Range had 82.6% prevalence.

It has long been believed that deer are the only competent definitive hosts for *E. schneideri* (Anderson 2001). However, some researchers (Worley et al. 1972, Madden et al. 1991) found gravid adult female worms in moose suggesting that they may be competent hosts. Histopathologic and laboratory evidence from 3 different cases in our study support the idea that moose are a competent host for *E. schneideri*: 1) several microfilariae associated with an adult female worm were in a cross-section of formalin-fixed carotid artery, 2) several microfilariae were in a section of formalin-fixed skin overlying the mandibular artery at the jugular notch of the mandible, and 3) one dead microfilaria was in an overnight saline soak of fresh forehead skin. Although not definitive, our evidence suggests that moose are competent hosts for *E. schneideri* reproduction and transmission. If this is the case, prevalence in mule deer and spatial overlap with infected mule deer could be less influential in determining *E. schneideri* prevalence in moose.

We can only speculate about the increased prevalence of *E. schneideri* in Wyoming moose over recent decades. Because elaeophorosis was perceived to have no effect on Wyoming ungulate populations, there is inconsistent historical data to make inferences. Numerous case reports have expanded our knowledge of the general distribution of *E. schneideri*, but recent reports have not attempted to describe the ecology of *E. schneideri* and explain observed prevalence in wildlife (e.g., Davies 1979). Prevalence of the disease is presumably related to the density of definitive hosts as well as the abundance of tabanid vectors.

Tabanid populations can be highly variable among years depending on weather conditions, because temperature and precipitation influence the timing of fly emergence, seasonal longevity, and total population size (Pence 1991). Thus gradual climate change has been attributed with observed and predicted increases in the effects of vector-borne parasites (Patz et al. 1996, Hoberg et al. 2008, Laaksonen and Oksanen 2009). We might have either conducted our surveillance when stochastic weather conditions were temporarily conducive for high *E. schneideri* transmission and/or prevalence, or changing conditions over decades has lead to higher prevalence of *E. schneideri*. Determining the vectors of *E. schneideri* in Wyoming and subsequently confirming the impacts of temperature and precipitation on those vectors will require further research.

For the same reasons prevalence varies over time, it can exhibit high spatial variability. We found higher than expected prevalence among most herds; the Snowy Range and Bighorns stood out as having especially high and low prevalence, respectively. Moose habitat use and behavior could differ across herds in ways that affect sympatry with mule deer or susceptibility to horse flies (Davies 1979). Domestic livestock grazing adds another layer of complexity. Livestock could either increase horse fly populations and exacerbate the transmission potential among wildlife, or dilute the effect because tabanids would prey on domestic animals instead of wildlife (Davies 1979). Further research is needed to fully understand the spatial dynamics of elaeophorosis in moose and other species in Wyoming.

On a more basic level, the effects of elaeophorosis on individual moose remain unknown. The high prevalence of apparently healthy infected moose suggests elaeophorosis is often not debilitating to this host. Yet *E. schneideri* has been implicated in morbidity or mortality in several cases (Worley et al. 1972, Madden et al. 1991, Pessier et al. 1998). As was demonstrated in elk (Adcock and Hibler 1969, Hibler and Adcock 1971, Hibler and Metzger 1974), pathogenic effects of *E. schneideri* on moose are more complex than a simple linear or threshold response by the host to number of worms. Complications from infection could arise at a number of critical stages in the life cycle of the parasite. Even slightly compromised basic functions resulting from impaired blood flow such as vision, hearing, mastication, smell, and brain function could expose individuals to malnutrition, predation, and ultimately lower survival and reproduction. While the maximum number of worms found in a hunter-harvested moose in our study was 26, preliminary necropsies of symptomatic moose have sometimes revealed double that intensity (J. C. Henningsen, unpublished data). Additionally, while none of the ≥8-yr-old moose had high-intensity infections, this may have been an artifact of low sample size. Limiting surveillance to hunter-harvested moose possibly eliminates important cases from consideration. Comprehensive surveillance that includes sick and dead moose with subsequent histopathologic examinations will be valuable in elucidating impacts of this parasite on individual moose.

Prevalence in our study was consistent across age classes. We interpret this to mean that moose of all ages are equally susceptible to infection and that infection does not affect survival differently across ages. Constant intensity of *E. schneideri* across ages of checked moose might additionally indicate a mechanism limiting worm burdens in moose. Perhaps individuals that tolerate initial infection acquire some immunity against further infection; Hibler and Metzger (1974) suggested as much for infected elk. Immune protection has been demonstrated in other ungulate-nematode systems involving long-lived adult worms. *Parelaphostrongylus tenuis* intensities in white-tailed deer are constrained across age classes (Slomke et

al. 1995) and Prestwood and Nettles (1977) demonstrated white-tailed deer acquire immunity to additional *P. andersoni* infections. This hypothesis presumes *E. schneideri* are long-lived; however, it is unknown how long *E. schneideri* can live in moose. Other filarioid nematodes live in their definitive hosts from 2->10 years (review by Gems 2000).

Alternatively, constant *E. schneideri* prevalence and intensities with increasing age of moose might simply reflect new infections occurring at a rate that essentially replace those mature nematodes that die naturally. Under this scenario, immune protection would not be perfect and new infections would continue through life at some rate that is tolerated by the host. On the other hand, pathologies arising from dead nematodes in the vascular system (Adcock and Hibler 1969) would be inconsistent with a hypothesis where moose can survive unaffected beyond the lifespan of the adult parasite. Thus this hypothesis presumes moose can tolerate not only live parasites, but individuals that die within their vascular system. Regardless of the mechanism, constant intensity and prevalence across age classes indicate infected moose are surviving, hence mortality caused by *E. schneideri* is lower than previously suggested.

While moose might not overtly succumb to elaeophorosis to the extent previously thought, prevalence of 50% is still cause for concern. At high prevalence, even a moderate proportion of infected individuals suffering from subclinical effects might impact recruitment or productivity at the population level. Subsequent research on moose herds where *E. schneideri* is present should consider the effects of elaeophorosis and attempt to clarify its role in moose population dynamics.

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