Prevalence and Mortality of Moose (Alces alces) Infected with Elaeophora schneideri in Montana, USA

Authors: DeCesare, Nicholas J., Harris, Richard B., Peterson, Collin J., and Ramsey, Jennifer M.

Source: Journal of Wildlife Diseases, 59(4) : 748-752

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/JWD-D-22-00075

DOI: 10.7589/JWD-D-22-00075

Journal of Wildlife Diseases, 59(4), 2023, pp. 748–752

Prevalence and Mortality of Moose (*Alces alces*) Infected with *Elaeophora schneideri* in Montana, USA

Nicholas J. DeCesare,[1,5] Richard B. Harris,[2] Collin J. Peterson,[3] and Jennifer M. Ramsey[4] [1]Montana Fish, Wildlife and Parks, 3201 Spurgin Road, Missoula, Montana 59804, USA; [2]Montana Fish, Wildlife and Parks, 1420 East Sixth Avenue, Helena, Montana 59620, USA; [3]Montana Fish, Wildlife and Parks, 490 North Meridian Road, Kalispell, Montana 59901, USA; [4]Montana Fish, Wildlife and Parks, 1400 South 19th Avenue, Bozeman, Montana 59718, USA; [5]Corresponding author (email: ndecesare@mt.gov)

ABSTRACT: *Elaeophora schneideri* is a filarial nematode of North America that occasionally infects aberrant ruminant hosts such as moose (*Alces alces*). The role *E. schneideri* plays in clinical morbidity or mortality of moose remains uncertain. We sampled predominantly hunter-killed adult moose (n=127) to characterize the spatial patterns of prevalence and intensity of worms in carotid arteries of moose in Montana. We compared prevalence and intensity of *E. schneideri* within these moose to a separate sample of adult moose that died of health-related causes (n=34). We found lower prevalence in northwest Montana (0.06) than in the remainder of the state (0.42). We also found both higher prevalence of *E. schneideri* and higher intensity to be correlated with increased probability of health-related mortality. Our results suggest presence and intensity of *E. schneideri* correlate with mortality of moose, although the mechanisms of mortality remain uncertain.

Key words: Arterial worm, carotid artery worm, *Elaeophora schneideri*, Montana, moose, mortality, parasites, survival.

Elaeophora schneideri, a filarial nematode (family Onchocercidae), lives in its adult stage for 3–4 yr in the carotid and other arteries of ruminants (Fig. 1) and is transmitted by intermediate host species of horse fly within the Tabanidae (Hibler and Metzger 1974; Anderson 2001). Initially described in domestic sheep (*Ovis aries*), *E. schneideri* has since been detected in multiple ruminant species across the contiguous US. The most common definitive hosts are mule deer and black-tailed deer (*Odocoileus hemionus*; Anderson 2001), which are typically not clinically affected. Presence has also been documented in other wild ruminants including white-tailed deer (*Odocoileus virginianus*; Couvillion et al. 1986), elk (*Cervus canadensis*; Adcock and Hibler 1969), moose (*Alces alces*; Henningsen et al. 2012; LeVan et al. 2013), and bighorn sheep (*Ovis canadensis*; Boyce et al. 1999), as well as within introduced populations of sika deer (*Cervus nippon*; Robinson et al. 1978), Barbary sheep (*Ammotragus lervia*; Pence and Gray 1981), and a captive Malayan sambar (*Rasa unicolor equina*; Bernard et al. 2016).

Moose are an aberrant host that are susceptible to pathogenic effects from ischemic necrosis resulting in elaeophorosis, a condition associated with malformation of ears and muzzle, damage to the optic nerve, food impaction, and other maladies (Anderson 2001). However, the relationship between *E. schneideri* and clinical morbidity or mortality of moose is unclear (Henningsen et al. 2012; LeVan et al. 2013). Elaeophorosis has been previously detected in moose from Montana (Worley et al. 1972). We provide further information regarding the geographic patterns of prevalence and intensity of *E. schneideri* in moose in Montana. We also assessed whether patterns of presence and intensity were correlated with health-related mortality among moose.

We monitored presence and intensity of *E. schneideri* within hunter-killed adult moose and other opportunistically encountered moose carcasses across their statewide distribution during 2009–2022, which was within five of seven administrative regions of Montana (northwest, west-central, southwest, north-central, and south-central; Fig. 2). We also conducted live-capture studies in three focal study areas (Fig. 2) during 2013–2022, with subsequent postmortem examinations of collared adult female moose after mortalities. Additional details regarding study areas and capture and handling procedures are available in Newby and DeCesare (2020).

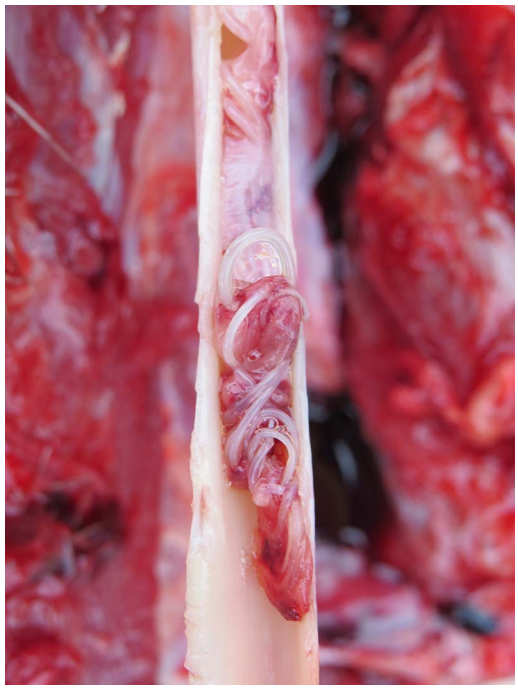

FIGURE 1. Adult *Elaeophora schneideri* within the carotid artery of a moose (*Alces alces*) seen during postmortem examination, western Montana, USA, 2017.

We assigned proximate causes of death for each moose and further consolidated these into broad categories of "control" and "health-related." Mortalities in the control group were those for which the proximate causes of death were judged to be unrelated to underlying health conditions, including hunter kills (n=109), illegal human kills (n=11), and those succumbing to accidents but with no other underlying morbidity (n=7). We assumed that any underlying disease within control group moose was independent of their death. Mortalities categorized as health-related included animals found dead opportunistically (n=3) or by telemetry collars (n=26) but without evidence of predation, plus those opportunistically encountered in poor condition and euthanized by field staff (n=5). We censored samples for which cause of death could not be confidently assigned to either category (e.g., road kill, n=11, entrapped in fences, n=3, blind by keratoconjunctivitis, n=3, and caused by live-capture efforts, n=5). We examined all moose for the presence and intensity (i.e., total count) of *E. schneideri* through dissection and gross examination of the terminal portions of both left and right carotid arteries of each animal (Fig. 1). Samples included in analyses generally contained 15–20 cm of each carotid artery leading to the terminal bifurcation. For the subset of collared moose, we also recorded the presence or absence of ear-tip necrosis, a symptom of ischemic necrosis previously associated with *E. schneideri* infection (Henningsen et al. 2012), at the time of postmortem examination.

We tested for spatial differences in *E. schneideri* prevalence among geographic regions by logistic regression. We used only control group animals in this analysis (n=127), and we further tested by negative binomial regression for regional differences in intensity among animals that were infected (n=40). In both cases, we tested for significant differences among regions, adjusting for multiple comparisons with estimated marginal means (Searle et al. 1980).

We then compared control (n=127) to health-related (n=34) mortalities with respect to *E. schneideri* presence-absence (binary) and intensity (count) to assess whether presence or intensity were correlated with health-related mortalities. We evaluated both metrics of infection by mixed-effects logistic regression (with control [0] and health-related [1] as a binary response variable) including random intercepts for geographic regions, and we assessed intensity with a restricted data set of infected animals. Lastly, we ensured that differences in seasonality of sampling did not confound our results, by confirming that results did not strongly differ when restricting analyses to the fall season when most control samples were collected. We conducted all statistical tests in R version 4.04 with additional packages lme4 and emmeans (R Core Team 2021).

Prevalence of *E. schneideri* was lower in Montana's northwest region (0.06) than elsewhere in the state (0.418; Table 1). Among infected moose, intensity was higher in the southwest region than in other regions of Montana (Table 1). Presence of *E. schneideri* was positively correlated with health-related mortality ($\beta_{presence}$=1.453, SE=0.463, z=3.1,

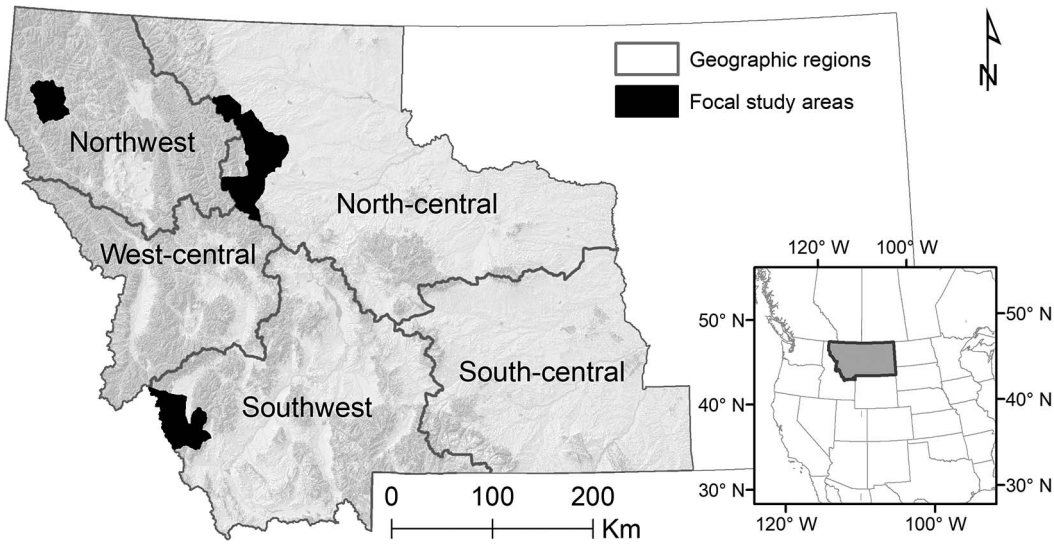

FIGURE 2. Geographic regions of western Montana, USA, 2009–2021, sampled for *Elaeophora schneideri* prevalence and intensity in moose (*Alces alces*), including three focal study areas where telemetry collar–based monitoring of adult females was used to supplement regional data. Inset map shows the location of Montana within North America.

P=0.002; Fig. 3) and prevalence was higher among health-related mortalities (0.65; 95% confidence level (CL) 0.46, 0.80; n=34) than the control group (0.31; 95% CL 0.24, 0.40; n=127). Among infected animals, increased intensity of infection was further correlated with health-related mortality ($\beta_{intensity}$=0.089, SE=0.026, z=3.4, P<0.001; Fig. 3). The odds of mortality being health-related increased by 4.3 times when infected with *E. schneideri*. Furthermore, within infected moose the odds of health-related mortality increased by 2.4 times for each 10 worms found (Fig. 3). Lastly, 10/18 (56%) infected moose in the collared subset also presented evidence of ear-tip necrosis. In 9/10 moose showing ear necrosis and infection, the necrosis had also been previously observed at the time of capture, up to 2 yr before.

Our finding that *E. schneideri* was rare among moose living in the relatively mesic and densely forested northwestern Montana is consistent with its rarity in adjacent British Columbia, Canada (H. Schwantje pers. comm.) and northeastern Washington, USA (Harris et al. 2021). Similarly, our findings that prevalence and

TABLE 1. Prevalence and intensity of *E. schneideri* among the control group of samples by geographic region in Montana, USA, 2009–2022.

	Prevalence[a]		Intensity[b]	
Region	n	Proportion infected	n	Mean count (SD) when infected
Northwest	36	0.06 A	2	6.00 (7.07)
West-central	4	0.50 AB	2	4.50 (3.54)
Southwest	80	0.38 B	30	10.00 (11.26) A
North-central	4	0.75 B	3	4.67 (3.21)
South-central	3	1.00 BC	3	3.33 (2.31)

[a] Prevalence among regions with different letters differ at P<0.03.
[b] Intensity among positive samples with different letters differ at P<0.01.

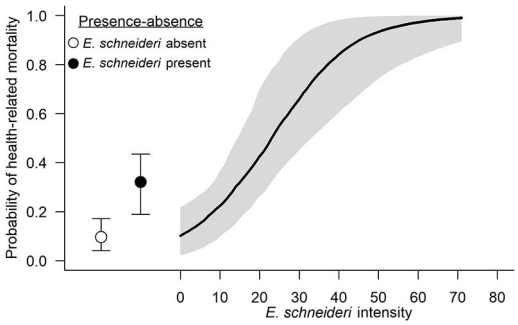

FIGURE 3. Predicted probability of moose (*Alces alces*) mortality in western Montana, USA, 2009–2021, being health-related as a function of *Elaeophora schneideri* presence-absence and of intensity (number of *E. schneideri* per moose).

intensity were higher in drier portions of central and southwestern Montana were consistent with findings in Wyoming, USA (Henningsen et al. 2012), and Utah, USA, where prevalence averaged 0.64 among hunter-killed moose during 2009–2012 (n=97; K. Hersey pers. comm.). Elaeophorosis has not been documented to date (May 2022) among moose in Alberta, Canada (M. Pybus pers. comm.). Geographic patterns of elaeophorosis in the western US may relate to abundance of the definitive host (*O. hemionus*), tabanid intermediate hosts, or both, aligning with drier habitats in this region (Boyce et al. 1999; Anderson 2001). However, detection of *E. schneideri* in the eastern US indicates that reservoirs of the parasite are sustained in white-tailed deer populations, as well (Couvillion et al. 1986; Grunenwald et al. 2018).

Henningsen et al. (2012) noted that *E. schneideri* presence among Wyoming moose increased during the decades preceding their 2009 survey. We observed no evidence of an increase in prevalence during 2009–2020 (N. DeCesare pers. comm.), although sample sizes in some years were small. Systematic differences in the time between death and post-mortem examination could impose confounding biases when comparing control and health-related samples. Given that our control sample was drawn predominantly from hunter-killed animals where such time was short, we do not expect our results of lower prevalence and intensity in such animals to be the result of biased methodology. LeVan et al. (2013) showed that histologic examination detected more elaeophorosis than gross inspection, suggesting that our data may underestimate prevalence. We also observed ear necrosis in two individuals that were not found to be infected, which may suggest either that the animals had since cleared a previous infection or that we failed to detect infection with our methods.

Despite correlation between *E. schneideri* observations and mortality of adult moose, specific causes of death remain uncertain. Hibler and Metzger (1974) suggested three critical periods during infection with implications for the health of aberrant hosts: 1) 7–14 d after infection when larvae develop in small arteries of the aberrant host instead of migrating to larger arteries; 2) 3–5 wk after infection, when immature adults traverse the cerebral retia; and 3) more than 7 wk after infection, when adults may return to cerebral arteries after development within the carotids. Although based on infection in elk, these phenomena may offer mechanisms for acute mortality of moose associated with thrombosis of cerebral arteries. Additionally, chronic and possibly subclinical effects of infection may increase with the number of worms within carotid arteries.

Our data show correlation but not causation between *E. schneideri* and mortality of moose. The significance of parasitism may be driven by other underlying factors such as nutritional condition or coinfections (Holmes 1995; Telfer et al. 2010). Thus, *E. schneideri* infection may instead reflect immunosuppression caused by unmeasured factors with greater health implications. For a subset of health-related mortalities (n=26), we further categorized 12 (46%) as acute mortality of animals in good nutritional condition according to fat deposits in the rump and femur marrow. Conversely, we categorized 14 (54%) as mortality potentially because of chronic disease of animals in poor condition. *Elaeophora schneideri* was common in both acute (58%) and chronic (79%) groups, suggesting that the correlation between parasite and mortality was robust to variation in nutritional condition, although coinfections could not be specifically addressed. Future studies of high

priority in this parasite-host system include the development of an antemortem test to detect and study subclinical effects of infection, as well as research aimed to identify the specific parasite life stages, host tissues, and physiologic mechanism(s) behind both chronic and acute effects of infection on host health.

We thank public and agency staff who assisted with animal sampling. Funding was provided by the general sale of hunting and fishing licenses, the annual auction of moose hunting licenses (MCA 87-2-724), Federal Aid in Wildlife Restoration grants W-157-R through W-157-R8, and the Safari Club International Foundation. We also thank E. Almberg, O. Krone, and two anonymous reviewers for previous reviews.

LITERATURE CITED

Adcock JL, Hibler CP. 1969. Vascular and neuro-ophthalmic pathology of elaeophorosis in elk. *Pathol Vet* 6:185–213.

Anderson RC. 2001. Filarioid nematodes. In: *Parasitic diseases of wild mammals*, Samuel WM, Pybus MJ, Kocan AA, editors. Iowa State University Press, Ames, Iowa, pp. 342–356.

Bernard J, Grunenwald C, Stalis IH, Varney M, Zuba J, Gerhold R. 2016. Elaeophora in the meninges of a Malayan sambar (*Rasa unicolor equina*). *J Vet Diagn Invest* 28:735–738.

Boyce W, Fisher B, Provencio H, Rominger E, Thilsted J, Ahlm M. 1999. Elaeophorosis in bighorn sheep in New Mexico. *J Wildl Dis* 35:786–789.

Couvillion CE, Nettles VF, Rawlings CA, Joyner RL. 1986. Elaeophorosis in white-tailed deer: Pathology of the natural disease and its relation to oral food impactions. *J Wildl Dis* 22:214–223.

Grunenwald CM, Butler E, Wünschmann A, Armien AG, Carstensen M, Hildebrand E, Moon RD, Gerhold RW. 2018. Emergence of the arterial worm *Elaeophora schneideri* in moose (*Alces alces)* and tabanid fly vectors in northeastern Minnesota, USA. *Parasit Vectors* 11:507.

Harris RB, Goerz J, Oyster J, Cook RC, Mansfield K, Atamian M, Lowe C, Prince A, Turnock BY. 2021. Bottom-up and top-down factors contribute to reversing a moose population increase in northeastern Washington. *Alces* 57:47–69.

Henningsen JC, Williams AL, Tate CM, Kilpatrick SA, Walter WD. 2012. Distribution and prevalence of *Elaeophora schneideri* in moose in Wyoming. *Alces* 48:35–44.

Hibler CP, Metzger CJ. 1974. Morphology of the larval stages of *Elaeophora schneideri* in the intermediate and definitive hosts with some observations on their pathogenesis in abnormal definitive hosts. *J Wildl Dis* 10:361–369.

Holmes JC. 1995. Population regulation: A dynamic complex of interactions. *Wildl Res* 22:11–19.

LeVan IK, Fox KA, Miller MW. 2013. High elaeophorosis among harvested Colorado moose. *J Wildl Dis* 49: 666–669.

Newby JR, DeCesare NJ. 2020. Multiple nutritional currencies shape pregnancy in a large herbivore. *Can J Zool* 98:307–315.

Pence DB, Gray GG. 1981. Elaeophorosis in Barbary sheep and mule deer from the Texas panhandle. *J Wildl Dis* 17:49–56.

R Core Team. 2021. *R: A language and environment for statistical computing*. R Foundation for Statistical Computing, Vienna, Austria. https://www.R-project.org/. Accessed February 2021.

Robinson RM, Jones LP, Galvin TJ, Harwell GM. 1978. Elaeophorosis in sika deer in Texas. *J Wildl Dis* 14: 137–141.

Searle SR, Speed FM, Milliken GA. 1980. Population marginal means in the linear model: An alternative to least square means. *Am Stat* 34:216–221.

Telfer S, Lambin X, Birtles R, Beldomenico P, Burthe S, Paterson S, Begon M. 2010. Species interactions in a parasite community drive infection risk in a wildlife population. *Science* 330:243–246.

Worley DE, Anderson CK, Greer KR. 1972. Elaeophorosis in moose from Montana. *J Wildl Dis* 8:242–244.

Submitted for publication 23 June 2022.
Accepted 11 May 2023.