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EVALUATING THE THRESHOLD DENSITY HYPOTHESIS FOR MOOSE (*ALCES ALCES*), WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*), AND *PARELAPHOSTRONGYLUS TENUIS*

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ABSTRACT: Despite the importance of the *Parelaphostrongylus tenuis* infection for moose (*Alces alces*) and white-tailed deer (*Odocoileus virginianus*) management, only one peer-reviewed study has evaluated the relationship between deer and moose densities and the potential for parasite-mediated competition between the species. Using polynomial-regression modeling, that study identified a deer-density threshold above which moose populations declined; however, the nature of the data and apparent outliers suggests the approach used to develop that threshold may not have been appropriate. We used the data from the original study to test whether alternative models, including linear models and negative binomial models would be less sensitive to outliers and could better explain that relationship. We found no evidence that moose density decreases as deer density increases. We concluded that, although the proposed moose-deer–*P. tenuis* relationship could be partially density dependent, additional factors, such as frequency dependence of disease transmission, gastropod abundance, and shared use of resources by moose and deer should also be considered.

Key words: *Alces alces*, moose, *Odocoileus virginianus*, *Parelaphostrongylus tenuis*, threshold density, white-tailed deer.

INTRODUCTION

Parasite-mediated competition happens when a parasite differentially affects multiple host species that share significant resources on overlapping ranges (Price et al. 1988). Theoretically, an unstable equilibrium and eventual extirpation of the more negatively affected host species will occur when a wide distribution and high prevalence of a parasite is maintained by the less-affected host (Schmitz and Nudds 1994). A warming climate could support range expansions of less-affected host species into native ranges of more-susceptible species, which could increase parasite-mediated competition through the introduction of novel parasites or increased parasite prevalence (Harvell et al. 2002; Pickles et al. 2013; Dawe and Boutin 2016). Therefore, at the margin of a species range, population fluctuations of the most heavily affected host could be severe.

However, parasite-mediated competition at range margins is difficult to detect because a multitude of natural and anthropogenic disturbances also contribute to host-parasite interactions (Harvell et al. 2002). A classic example is the potential effect of white-tailed deer (*Odocoileus virginianus*) parasites on other ungulates, such as moose (*Alces alces*), elk (*Cervus elaphus*), mule deer (*Odocoileus hemionus*), and woodland caribou (*Rangifer tarandus caribou*). Deer expanded northward into the southern edge of the historic moose range in the 1800s and early 1900s, likely because of European settlement and subsequent habitat alterations (Anderson 1972). Consequently, moose and caribou were exposed to a novel set of parasites carried asymptomatically by deer. One such parasite,

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TABLE 1. *Parelaphostrongylus tenuis* infection rates reported in deer (*Odocoileus virginianus*) where they are sympatric with moose (*Alces alces*) in North America. *Parelaphostrongylus tenuis* infection rates in deer have been reported since at least 1967. Some states and provinces report only infection rates, whereas others report infection rates and deer densities (deer/km^2). Deer densities do not appear to correlate with *P. tenuis* infection rates. With the exception of Saskatchewan, Canada, and western North Dakota, USA, deer infection rates were high, regardless of reported deer densities.

Region	Deer infection rate or range (%)	Deer/km^2	Reference
New Brunswick	60	Not reported	Upshall et al. 1987
Maine	73	<1–2	Bogaczyk et al. 1993
Maine	84	Not reported	Behrend and Witter 1968
Maine	63–80	1–6	Gilbert and Frederick 1974
Quebec, Canada	63	Not reported	Bindernagel and Anderson 1972
Upper peninsula, Michigan	44	3–7	Nankervis et al. 2000
Ontario, Canada	63	Not reported	Bindernagel and Anderson 1972
Northwest Ontario, Canada	47	4–5	Saunders 1973
Northwest Ontario, Canada	58	0–9	Whitlaw and Lankester 1994
Northeast Minnesota	82	12	Slomke et al. 1995
Northeast Minnesota	69	2	Vanderwaal et al. 2015
Central Minnesota	69	12	Karns 1967
Northern Minnesota	39	4–6	Karns 1967
Manitoba, Canada	49	Not reported	Bindernagel and Anderson 1972
North Dakota	14.5 (range: 1–35)	Not reported	Maskey et al. 2015
Saskatchewan, Canada	9	Not reported	Bindernagel and Anderson 1972

which differentially affects deer and moose, is the meningeal worm *Parelaphostrongylus tenuis* (Anderson 1972). Deer are the definitive host of *P. tenuis* and, typically, have infection rates of >50% where deer, moose, and gastropod intermediate hosts are sympatric (Table 1). *Parelaphostrongylus tenuis* infection does not appear to have any negative health effects on deer. However, as an incidental host for *P. tenuis*, infected moose present with several neurologic symptoms, such as lack of fear, remaining in one area for extended periods of time, circling, partial paralysis, tilted head, and nystagmus (Lankester et al. 2007), which can eventually lead to death.

Parelaphostrongylus tenuis cannot exist without the intermediate gastropod hosts and is most common in regions in which deer densities are relatively high (Table 1) and forest cover is between 25% and 75% (Wasel et al. 2003). *Parelaphostrongylus tenuis* prevalence also is negatively correlated with spring and fall temperatures (Wasel et al. 2003). As such, *P. tenuis* infection in deer is low, and infection in moose is rare in western North America, where deer densities are lower, open grassland habitats are abundant, and weather conditions are hotter and dryer than those found in moose ranges in the Great Lakes region and in the northeastern US (Wasel et al. 2003).

In North America, there are typically fewer than 0.5 moose/km^2 across the southern edge of the moose range where moose coexist with deer (Lankester 2010; DelGiudice 2020). Deer populations in these latitudes fluctuate primarily because of severe-winter conditions (Potvin et al. 1981; DelGiudice et al. 2002; Patterson and Power 2002). In these regions of geographic range overlap, *P. tenuis* infection rates in deer are relatively high, ranging from 35–85%, decreasing along a westward longitudinal gradient (Table 1).

There are several examples of moose populations declining along southern range margins during a series of years in which deer populations were increasing (Lankester 2010). In northwestern Ontario, for example, moose populations declined to the extent that the

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moose hunting season was closed during the 1940s, coinciding with an increase in deer populations (Lankester 2010). During the 1990s, the moose population in western North Dakota declined after deer population sizes increased rapidly and the deer range expanded northward (Lankester 2010). During the 1990s and early 2000s, the moose population in northwest Minnesota declined from about 4,000 to fewer than 100 (Murray et al. 2006). A historic peak in deer density was reached in northeast Minnesota in 2003, followed by a period of about a decade in which deer densities were stable or slightly declining (Minnesota Department of Natural Resources 2011). The wolf (*Canis lupus*) population of Minnesota also reached peak levels around 2003–04, when the population was estimated to be 3,020 animals. Wolf numbers remained somewhat stable in the following decade, ranging from estimates of 2,211 to 2,921 animals (Erb et al. 2018). During that time, the northeast Minnesota moose population declined from an estimated 8,000 individuals in 2006 to about 4,000 in 2014 (DelGiudice 2020).

Moose populations also increased after deer populations declined (Lankester 2010). In Nova Scotia, three severe winters in the 1950s depressed deer numbers, whereas moose numbers increased (Pulsifer and Nette 1995). A similar increase in the moose population also occurred during a period of reduced deer densities during the 1990s (Pulsifer and Nette 1995). Although moose populations declined after deer range expansion into northwestern Ontario in the 1920s, they rebounded after deer populations were reduced 50–80% by a series of severe winters in the 1970s (Lankester 2010). In northeast Minnesota, point estimates of moose have remained stable since 2014, which seems to correspond with reduced deer densities after the severe winters of 2012–13 and 2013–14 (DelGiudice 2020).

When either deer or gastropod secondary hosts are absent along the southern edge of moose range, moose density can be an order of magnitude greater than in areas of their range in which deer and gastropods are present. Two examples of moose reaching high population densities in the absence of deer are Isle Royale National Park (NP) and Newfoundland, Canada. Density of moose on Isle Royale NP has fluctuated from <1 to >4 moose/km^2 since the early 1900s, with and without the presence of wolves (Vucetich and Peterson 2004) and without hunting mortality. Newfoundland had densities of <1 to >5 moose/km^2 in the absence of deer and wolves (McLaren and Mercer 2005). Unlike in Isle Royale NP, wolves were extirpated from Newfoundland in the early 1900s, and hunting is the primary cause of mortality for adult moose (McLaren and Mercer 2005). Higher densities of moose in regions in which deer and their associated parasites are absent is consistent with the hypothesis that parasite-mediated competition prevents moose from reaching high densities when deer and *P. tenuis* are present. Despite evidence for parasite-mediated competition based on several observations of an inverse relationship between deer densities and moose population declines, it has been difficult to define the underlying mechanism of disease transmission between deer and moose, specifically whether the moose-deer–*P. tenuis* system is frequency or density dependent.

Where deer and moose ranges overlap and *P. tenuis* is present, a density threshold of <5 deer/km^2 has been recommended for moose persistence, which was based largely on anecdotal evidence (Karns 1967; Whitlaw and Lankester 1994; Lankester 2010) and one attempt to quantitatively test whether there is a significant negative relationship between deer and moose densities (Whitlaw and Lankester 1994). The threshold hypothesis was tested by Whitlaw and Lankester (1994), who analyzed data on moose densities and deer densities from Wildlife Management Units (WMUs) in Ontario, Canada. The authors solicited moose and deer density data from local wildlife managers, who used a range of objective and subjective methods to estimate populations. Moose densities spanned the typical range for populations along their southern range margin in North America (Ontario Ministry of Natural Resources 2018; New Hampshire Fish and

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Game 2019; DelGiudice 2020), whereas most deer densities were low to moderate relative to other parts of their North American range (Quality Deer Management Association 2009; Norton and Giudice 2017). Their conclusion was that deer had a negative effect on moose populations when deer densities exceeded 4 deer/km^2. Their conclusion was based on fitting a third-order polynomial to data on moose and deer population densities. However, moose density was high in some WMUs when deer were present and above the threshold, and low in some WMUs in which deer were absent. This result was partially explained by deer only being present on the southern edges of WMUs because of winter severity and forage productivity limiting moose densities to the north (Whitlaw and Lankester 1994).

The Whitlaw and Lankester (1994) initial analysis of the potential relationship between moose and deer densities illustrates the complexity of the question. Although their third-order polynomial relationship was statistically significant, it only explained about 15% of the variation (Whitlaw and Lankester 1994). The dataset also contained apparent outliers and influential points that could lead to misinterpretation of the results and incorrect inferences about the threshold density of deer that might negatively affect moose populations.

In addition, density-dependent disease transmission typically results in linearly increasing prevalence, so we would expect the relationship between deer densities and moose densities when gastropods are also present to be linear as well (Begon et al. 2002). If transmission is frequency dependent, then, we would not expect a relationship between deer densities and moose densities (Begon et al. 2002). If transmission is, to some degree both density dependent and frequency dependent, then there may be a nonlinear relationship, although the trend should still be increasing prevalence and declining moose populations with increasing deer densities or increasing contact rates between moose and deer (Fenton et al. 2002). None of these potential disease-transmission relationships would be best represented by a third-order polynomial.

These questions about the expected relationships, given a density-dependent or frequency-dependent mode of transmission, led us to reanalyze the dataset using additional regression types. Our objective was to determine whether we could improve upon the model from Whitlaw and Lankester (1994) to gain a better understanding of moose-deer population dynamics in which *P. tenuis* is a disease risk to moose. We identified outliers in the Whitlaw and Lankester (1994) dataset and refit models with outliers removed to test whether model fit could be improved and whether inferential power could be increased.

MATERIALS AND METHODS

We first digitized figure 2 from the Whitlaw and Lankester (1994) article to recreate the moose and deer density dataset. The dataset included estimates of deer and moose densities from 53 WMUs across northwest and southern Ontario. The initial model relating moose density (moose/km^2) to deer density (deer/km^2) used by Whitlaw and Lankester (1994) was a third-order polynomial regression model. We evaluated several alternative candidate models, including simple linear regression, second-order polynomial regression, and negative binomial regression, with and without statistical outliers.

We used the chi-square test in program R (R Core Team 2016) with a package for outliers (Komsta 2011) to first test for the statistical significance of the outliers. We sequentially removed groups of outliers from the analyses, starting with statistically significant outliers and then removing visually identified influential points to test whether predictive power could be improved with a simple linear regression and second-order polynomial regression. Because several WMUs had deer densities near zero, we also performed a negative binomial regression along with a likelihood-ratio test to assess the value of including a deer-density parameter in the model. Models were compared using coefficient of determination (R^2) values and the corrected Akaike information criterion (AICc) for small-sample sizes (Hurvich and Tsai 1989).

RESULTS

We were unable to attribute points in figure 2 from Whitlaw and Lankester (1994) with WMU location and other spatial metadata in

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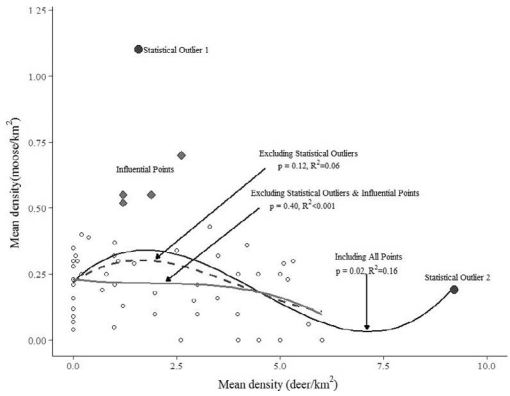

FIGURE 1. Model results from the original third-order polynomial regression presented by Whitlaw and Lankester (1994) and from our subsequent models after removing groups of outliers from their data. The solid black line is the original third-order polynomial regression line as presented in Whitlaw and Lankester (1994). The dashed gray line indicates our refitting of the model by excluding two statistical outliers. An additional four points were identified as influential, and so we removed those points and refit the model again, indicated by the solid light-gray line. Data points included in all model iterations are open circles. Statistical outliers included only in the first model are closed gray circles, and influential points removed during the third-model iteration are gray diamonds. In each iteration, model fit and statistical significance was reduced, and no relationship was found between moose and deer densities in this dataset.

table 1 of their article and, thus, could not examine the spatial distribution of density estimates and how deer densities might relate to latitude using WMUs. Apparent influential points were initially identified visually in figure 1 in Whitlaw and Lankester (1994). The outlier initially identified was a moose density of 0.2 moose/km^2 and a deer density of 9 deer/km^2. An additional set of outliers visually identified were moose densities of >0.50 moose/km^2. The data point at 1.1 moose/km^2 and 2.25 deer/km^2 was a statistically significant outlier (χ^2=19.13, $P<0.001$), as was the moose density of 0.2 moose/km^2 at a deer density of 9.2 deer/km^2 (χ^2=10.87, $P<0.001$). The third group of potential influential points included four points with moose densities of >0.5 moose/km^2. Values in the second and third group visually identified as potential influential points were not statistically significant (χ^2=1.22, P=0.262).

TABLE 2. Relationship between moose density (moose/km^2) and deer density (deer/km^2) as presented in Whitlaw and Lankester (1994) and additional candidate models we tested using data from their study. The regression column denotes the type of regression model tested, the outliers column indicates when outliers were included or excluded from the models, df is the degrees of freedom for each model, and adjusted R^2 indicates the variance explained by the model. The relationship between deer and moose densities was only significant when using a second- (this study) or third-order (Whitlaw and Lankester 1994) polynomial model. For the former, this was true only when two statistical outliers were removed.

Regression	Outliers (included/ excluded no.)	df	Adjusted R^2	P value
Third-order polynomial	Included	3	0.16	0.021
	Excluded 2	3	0.06	0.119
	Excluded 6	3	<0.001	0.404
Second-order polynomial	Included	2	0.01	0.330
	Excluded 2	2	0.08	0.050
	Excluded 6	2	0.03	0.348
Linear regression	Included	1	0.01	0.197
	Excluded 2	1	0.02	0.162
	Excluded 6	1	0.02	0.153

Polynomial-regression models did not improve with removal of statistical outliers or influential points, with nonsignificant P values and decreasing R^2 values (Table 1 and Fig. 1). Two of the models, the replication of the Whitlaw and Lankester (1994) regression and the second-order polynomial with statistical outliers removed, were significant at P=0.050 (Table 2). There was no significant relationship when influential points (n=2 and n=6) were removed for any other models (Table 2 and Fig. 1). The AICc values indicated that the best models were a second-order polynomial regression that included statistical outliers (AICc=–18.73), and a linear regression, excluding statistical outliers (AICc=–18.74; Table 2); <20% of variability was explained by any model, regardless of inclusion or removal of potential outliers. The negative binomial-regression model was not significant

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(P=0.192). Including deer density as a parameter in the negative binomial-regression model was also not significant (P=0.209).

DISCUSSION

The variability in moose population density explained by deer density in the regression models we fit was within the margin of error of any moose population estimate (DelGiudice 2020). In our reanalysis, using additional regression types that are more appropriate for density-dependent transmission mechanisms and removing highly influential outlier points, it was clear that, despite qualitative and semiquantitative observations (Lankester 2010), the hypothesis of declining moose densities caused by correspondingly high deer densities is not supported by the data presented in Whitlaw and Lankester (1994).

Since the 1900s, moose population declines have been documented from Nova Scotia and New Brunswick to Minnesota and northwestern Ontario, Canada (Murray et al. 2006; Lankester 2010). High levels of *P. tenuis* infections in moose caused by high deer densities have often been implicated as a significant factor in these declines. A qualitative analysis of these declines identifies *P. tenuis* infection, as a result of high deer densities, as the most probable cause (Lankester 2010). However, evidence for a statistically significant relationship between high deer densities and lower moose populations remains equivocal, and whether transmission of *P. tenuis* is driven by density dependence, frequency dependence, or both is uncertain.

Lack of statistical significance in tests attempting to link moose population declines to *P. tenuis* and deer may be an example of a type II error, where the probability of falsely inferring the absence of a relationship may have been exacerbated because of uncertainty in estimating population densities, small sample sizes of moose with *P. tenuis*, and moose population declines that extended over a decade. Additional complicating factors vary by location and include differences in weather, changes in habitat, the presence and abundance of predators, and the presence and abundance of other parasites that can affect moose health, for example, liver fluke (*Fascioloides magna*) and winter tick (*Dermacentor albipictus*), as described by Samuel (2004) and Murray et al. (2006). Parasites such as liver flukes and winter ticks, however, vary temporally in their life cycles and in their effects on moose health. Liver flukes, for example, are trematode parasites of deer and moose and have been implicated in moose population declines (Murray et al. 2006). However, despite marked infections found in some moose livers, evidence that liver fluke–induced hepatitis can cause mortality in moose is limited (Lankester 2010; Wünschmann et al. 2015). Winter ticks are an external parasite of moose that can also cause mortality (Addison 2007; Carstensen et al. 2017; Jones et al. 2019). In contrast to *P. tenuis* and liver fluke infections, which may induce persistent and prolonged health effects on moose, winter tick infestations are cyclic and highly seasonal (Samuel 2004). During years with high infestation rates, moose mortality can be significant and more acute than mortality from *P. tenuis* or liver flukes (Samuel 2004, 2007).

The prevalence of internal and external parasites is likely to increase in moose range as a consequence of climate change, either by range expansion and increased deer densities or by increasingly hospitable environmental conditions for parasites. For instance, in northeastern North America, climate change projections indicate a 3–5 C increase in annual average temperature and 20–30% increase in spring precipitation by the end of the century (Hayhoe et al. 2008). Increasing annual average temperatures are projected to cause losses in early spring snow cover and longer growing seasons (Demaria et al. 2016). In those regions in which deer populations fluctuate mainly in response to winter weather (Potvin et al. 1981; DelGiudice et al. 2002; Patterson and Power 2002), increasingly mild winters with less snow or early loss of snow should increase deer survival. Additionally, deer excretion of larvae increases threefold in

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spring (Peterson and Lankester 1991; Slomke et al. 1995). External *P. tenuis* larval survival and transmission is increased with early springs, increased precipitation during the growing season, and longer growing seasons (Ranta and Lankester 2017; Lankester 2018). Therefore, less-severe winters and, consequently, more stable or increasing deer populations, combined with increased *P. tenuis* larval survival in the external environment, is likely to increase infection risk for moose where they co-occur with deer.

Although the third-order polynomial regression model used by Whitlaw and Lankester (1994) predicted a significant moose density decline as deer densities increase above 4 deer/km^2, the authors recognized the limitations of the dataset. Despite their acknowledgment of weak inferential power, their specific threshold is used as a benchmark for managing deer in sympatric range (Minnesota Department of Natural Resources 2011).

The threshold hypothesis states there is a population density of deer below which the disease cannot persist at high enough levels in the environment to be a significant threat to moose (Schmidt and Ostfeld 2001). Although Whitlaw and Lankester (1994) suggest that the threshold is 4 deer/km^2, recorded deer densities within moose range in Minnesota have never exceeded 4 deer/km^2 (D'Angelo and Giudice 2016). However, since the beginning of a project initiated by the Minnesota Department of Natural Resources in 2013 investigating cause-specific mortality of moose, the annual adult mortality rate was around 15%, and approximately 30% of moose deaths were attributed to parasites, primarily *P. tenuis* (Carstensen et al. 2017). During that same period of time the moose population was relatively stable (DelGiudice 2020), whereas deer densities in the region were the lowest observed in more than a decade (D'Angelo and Giudice 2016).

Although the management goal cited by Whitlaw and Lankester (1994) has served as a general rule for moose management, additional biologic support for managing deer densities for the benefit of moose is needed. Given our findings of poor support for a deer-density threshold, we are unable to assess whether management efforts that depress deer densities will benefit moose. In addition, relying on a poorly supported threshold may also result in management agencies overlooking other factors that could influence moose population declines. For example, the fact that we found no evidence of a density-dependent relationship between moose and deer populations in which *P. tenuis* infection is a risk factor may imply a frequency-dependent disease transmission mechanism. Frequency dependence is posited as a factor in chronic wasting disease transmission (Jennelle et al. 2014) and could apply in this complex system as well. If the transmission mechanism in the moose-deer–*P. tenuis* system is frequency dependent, contact rates between deer, moose, and gastropod hosts are more-important drivers of disease transmission than deer density. Therefore, if this is a frequency-dependent system, management efforts that aim only at keeping deer densities low within a moose range would be insufficient to prevent *P. tenuis* transmission from deer to moose.

Management decisions could benefit from research that elucidates more completely the dynamics of the moose-deer–snail-parasite–habitat relationship. Focusing solely on the density-dependence requirement for disease transmission from deer to moose does not provide information about whether habitat partitioning exists between moose and deer on sympatric range. Understanding similarities in the use of resources between moose and deer could shed light on how the contact rates between species might be influenced by parts of the landscape that are either mutually selected or mutually avoided by moose and deer. For example, on sympatric range in northeast Minnesota, moose were more likely to become infected with *P. tenuis* if their fall home ranges contained high proportions of upland shrub and conifer land-cover types and if their home ranges contained higher proportions of lowland cover types, regardless of season. However, moose infection risk did not increase with higher deer densities

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(Ditmer et al. 2020). Moose density in that area averaged 0.2/km^2 (DelGiudice et al. 2020), and deer densities ranged from 2–4/km^2 (Norton and Guidice 2017). These relationships indicate the potential for a frequency-dependent transmission mode.

In Minnesota, ≥70% of deer are infected with *P. tenuis* (Peterson et al. 1996; Gogan et al. 1997; Vanderwaal et al. 2015). *Parelaphostrongylus tenuis* larvae are generally present in <0.5% of gastropods (Lankester and Peterson 1996). To date, the likelihood of moose and deer ingesting infected gastropods has been explained by the fact that deer and moose eat large quantities of vegetation, and, despite the low incidence of infection in gastropods, some moose and most deer will still become infected through incidental ingestion of many snails (Lankester 1967).

Moose population declines, if caused by *P. tenuis*, would necessarily occur only if moose forage in areas in which deer defecate and where gastropods occur. Therefore, biologic questions remain about how and when resources are used by moose and deer and in what biologically significant ways moose are at high risk of becoming infected. Specifically, does moose distribution overlap with deer presence at a time of year when *P. tenuis* larvae are most prevalent and developed to infective stages in gastropods? How often and when does the overlapping distribution of moose and deer occur in areas that provide forage and, therefore, a potential route of infection? This can be tested using fine-scale location data of moose and deer to determine resource use for each species and to identify riskier portions of landscape for moose. Analyzing interactions between deer and moose in terms of resource selection on sympatric ranges may provide novel insights into questions related to moose-deer–parasite dynamics and into whether parasite-mediated competition may contribute to declining moose populations (McGraw 2019; Ditmer et al. 2020). Additional research is necessary to determine to what degree moose populations can be negatively affected by deer and whether *P. tenuis* infection rates in moose populations are best explained by density-dependent or frequency-dependent transmission.

That mechanism of disease transmission might be further elucidated by pairing resource selection studies with biologic tests (i.e., enzyme-linked immunosorbent assay blood test; Ogunremi et al. 2002) to directly measure levels of exposure and infection to *P. tenuis* on free-ranging, monitored moose at capture and at death. Concurrent captivity trials, to determine dose-dependent effects on adult moose health and mortality, similar to Lankester (2002), would provide additional insights into how moose respond in a controlled environment to various levels of infection challenges by *P. tenuis*.

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