CONSIDERING WEATHER-ENHANCED TRANSMISSION OF MENINGEAL WORM, *PARELAPHOSTRONGYLUS TENUIS,* AND MOOSE DECLINES

Murray W. Lankester

101-2001 Blue Jay Place, Courtenay, British Columbia, Canada V9N 4A8; Retired

ABSTRACT: The risk of meningeal worm (*Parelaphostrongylus tenuis*) infection in white-tailed deer (*Odocoileus virginianus*) and neurologic disease in moose (*Alces alces*) in eastern North America is influenced largely by the effects of weather on deer density and gastropod intermediate hosts. Frequent, easy winters result in high survival and density of deer with a large proportion of young animals that shed up to 3 x more *P. tenuis* larvae; both greatly increase the production of first-stage larvae. An early spring increases survival of shed larvae by reducing the timing mismatch between the parasite's "spring rise" and snow melt; larvae deposited into snow experience high mortality. A wetter and longer growing season with moderate temperatures increases the survival of first-stage larvae dispersed in soil, and the density, mobility, and frequency of infected gastropods, including the abundance of infective larvae in them. This weather-enhanced transmission further increases larval output by reducing the proportion of unproductive unisexual infections in deer. High production of larvae and optimal conditions for gastropods increase rates of transmission to co-habiting moose and the occurrence of neurologic disease which is dose-dependent. The density of infected deer at the northern limit of their range is typically limited by winter severity allowing coexistence of deer, moose, and parasite. However, as in Nova Scotia and northwestern Minnesota and adjoining regions, pronounced and prolonged moose declines associated with sustained high deer densities and meningeal worm infection have occurred twice in the past 95 years. These two regions may be prone to extended periods of mild winters and longer, wetter growing seasons that ultimately enhance abundance and transmission of the meningeal worm implicated in moose population declines.

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Parelaphostrongylus tenuis is a common, but innocuous parasite of white-tailed deer (*Odocoileus virginianus*) throughout the eastern half of North America. It is important because it causes neurologic disease in moose (*Alces alces*) in northern forest habitat where the ranges of deer and moose overlap. Transmission involves a complex life cycle in which first-stage larvae are released to the external environment on deer faeces, and infect and develop in terrestrial gastropods which are subsequently ingested by cervids. The potential for weather to influence transmission rates of *P. tenuis* among deer and its importance to the health of moose have been increasingly documented (Peterson et al. 1996, Wasel et al. 2003, Lankester 2010, Maskey et al. 2015).

Deer, moose, and the parasite can coexist for extended periods (Whitlaw and Lankester 1994b, Dumont and Crete 1996) which partially explains why the hypothesis

that *P. tenuis* may be a primary cause of pronounced and prolonged moose declines remains controversial (Lankester 2010). Such declines occurred in northern Minnesota in the late 1920s and 1930s, in Nova Scotia in the 1940s and 1950s, and again in these jurisdictions in the late 1980s – early 1990s (Benson 1958, Karns 1967, Anderson 1972, Whitlaw and Lankester 1994a, Lankester 2001, Parker 2003, Beazley et al. 2006, Murray et al. 2006, Lankester 2010). During the latter period, moose were also declining in upper Michigan (Dodge et al. 2004) and in areas adjacent to northwestern Minnesota including northeastern North Dakota, northwestern Ontario, and southeastern Manitoba (Thompson 2000, Murray et al. 2006, Maskey 2008, Ranta and Lankester 2017, V. Crichton, Manitoba Conservation [retired], pers. comm.). In all instances, moose faced increasing densities of deer with meningeal worm and cases of moose sickness were routinely documented (Lankester et al. 2007, Wünschmann et al. 2015, Ranta and Lankester 2017).

This paper examines how weather likely influences the parasite's rate of transmission and increases its importance as a disease agent for moose. The overall hypothesis is that geographical regions experiencing reoccurring, pronounced, and prolonged moose declines may be prone to lengthy periods of weather-enhanced *P. tenuis* transmission that greatly increase the parasite's role in moose morbidity and mortality.

WEATHER AND FIRST-STAGE WORM LARVAE

First-stage larvae passed by deer are located in a thin film of mucus that covers the surface of each faecal pellet (Lankester 2001). If pellets are deposited in an open area, larvae may be exposed to rapid drying and potentially harmful solar radiation; however, rain washes larvae off pellets into the underlying litter and soil. Laboratory experiments indicate that larvae on pellets or in water can withstand constant sub-zero temperatures for several months, but repeated freezing and thawing greatly reduces survival, as does repeated wetting and drying at room temperature (Shostak and Samuel 1984). Further, 70% of larvae frozen for up to 182 days survived, but only 16% were still alive after 306 days with only one undergoing some development in a snail (Lankester and Anderson 1968).

Infected deer pass up to 3 × more larvae during spring than at other times of the year (Peterson and Lankester 1991, Slomke et al. 1995). Larval production is believed lower in late-starting springs as larvae on pellets deposited in snow survive poorly despite moderated temperatures beneath snow cover; presumably, actions by subnivean invertebrates and molds reduce survival (Forrester and Lankester 1998). In northeastern Minnesota, the mean number of larvae produced by deer of all ages increased from a low of 289/gdf (grams dried faeces) in December to a peak of 1127/gdf in early March. Although larval production peaked in early March while snow remained on the ground, ~75% of larvae deposited from January until snowmelt in mid-April died (Forrester and Lankester 1998). This "spring rise" may be an adaptation maximizing progeny output at a time best suited for their survival and transmission. The meningeal worm likely evolved in southern climes with its normal white-tailed deer host and may remain ill-adapted to long northern winters. Earlier springs, however, will presumably increase larval survival during the peak production period.

WEATHER AND TERRESTRIAL GASTROPODS

First-stage larvae must penetrate and develop to the third infective stage in the terrestrial gastropod intermediate host in which

the rate of larval development is determined by ambient temperatures. Cool, moist woodland habitats are preferred by gastropods (Lankester and Anderson 1968, Hawkins et al. 1997, Maskey et al. 2015), whereas the litter of predominantly coniferous forests is believed less favourable for snails (Gleich and Gilbert 1976, Boag and Wishart 1982). Mobility varies among species, with slugs more mobile than snails, particularly in wet conditions. The greater mobility of slugs allows avoidance of dry conditions, whereas snails withdraw into their shell and aestivate.

Several gastropod species are capable intermediate hosts of *P. tenuis* (Lankester 2001, Nankervis et al. 2000, Maskey et al. 2015), but 3 species are most numerous and frequently infected: the marsh slug (*Deroceras laeve*) and 2 woodland snails (*Zonitoides* spp. and *Discus cronkhitei*) (Lankester 2001, Cyr et al. 2014). The marsh slug thrives in wet conditions but is adaptive to resist dehydration (Luchtel and Deyrup-Olsen 2001); the snails tolerate slightly drier sites.

Deroceras leave provides an example of how changes in weather may influence the role of intermediate hosts. This ubiquitous Nearctic slug has spread throughout the world attesting to its versatility (Pilsbry 1946, Faberi et al. 2004), and is the only land gastropod known that deliberately enters water, surviving for days while submerged in inundated areas. It has a clear watery slime that might be easier for *P. tenuis* larvae to penetrate compared to the viscous slime of some other species. It is mobile, gliding quickly over vegetation and covering relatively large distances. In rainy or foggy weather, *D. laeve* climbs low vegetation where it is better positioned to be consumed by cervids. This slug is also adapted to a wide range of temperature, surviving to at least -8 ºC (Getz 1959, Faberi et al. 2004). Live specimens were found under cardboard sheets during over-night temperatures close to freezing (Lankester and Peterson 1996). It is one of the first gastropods active in spring and the last active in autumn.

Deroceras laeve lives for only one year in the temperate regions of North America (Lankester and Anderson 1968, Boag and Wishart 1982, Lankester and Peterson 1996). The prevalence of *P. tenuis* peaks in adult *D. laeve* before their death in mid-summer and again in maturing slugs in autumn; infective larvae survive in this slug over winter (Lankester and Anderson 1968). In northeastern Minnesota, large *D. laeve* were moderately numerous in June and absent in July and August, with maturing slugs most numerous in September and October and remaining active until mid-November. In contrast, the availability of the longer-lived snails *Zonitoides arboreus* and *Discus cronkhitei* was less bimodal during the growing season (Lankester and Peterson 1996).

Gastropod abundance correlates with precipitation (Burch 1962, Whitlaw et al. 1996, Hawkins et al. 1997). Gastropods are most active on forest floor litter and low vegetation during the wet seasons of spring and autumn, and less active in summer (Lankester and Peterson 1996). Many more gastropods are found in the upper layer of soil than are active on the surface. Cardboard sheets placed on the forest floor had ~2% of the number of gastropods estimated in soil cores from the upper 10 cm of soil beneath the sheets (Hawkins et al. 1998). As well, collections dominated by *D. laeve* peaked when temperature beneath the boards was ~15 ºC; abundance declined at lower and higher temperatures.

The frequency of *P. tenuis* infection is generally low (i.e., < 0.1%) in gastropods in boreal areas, as is the mean number of larvae

recovered from each gastropod (2-3). This low recovery suggests that gastropods become infected by crawling over dried faeces or litter and soil, rather than fresh faeces (Lankester and Peterson 1996). Gastropods are more readily infected by larvae on moist than dry soil, and can be infected repeatedly (Lankester and Anderson 1968). Although some gastropods show a degree of attraction to fresh deer faeces (Garvon and Bird 2005), this behaviour or interaction may be uncommon. Whether infective larvae of *P. tenuis* leave gastropods and survive on vegetation is unknown.

Annual infection rates in gastropods vary relative to temperature, moisture, and the duration of conditions suitable for activity. Terrestrial gastropods survive over winter in the boreal region as do developing *P. tenuis* larvae in them (Lankester and Anderson 1968, Lankester and Peterson 1996). Larval development is arrested at low temperatures and during dry periods, but resumes with the return of suitable conditions.

There is a direct linear relationship between ambient temperature selected by the gastropod host and the rate of larval development (Jenkins et al. 2006). This relationship has not been well studied for *P. tenuis*, but closely related *P. odocoilei* shows little or no development below 8.5 °C and requires a minimum of 163 accumulated degree-days of heat to reach the infective stage in *D. laeve* (Jenkins et al. 2006). Accurate field estimates of developmental rates in *P. tenuis* will require conducting experiments of the type described by Kutz et al. (2002) who held infected slugs in enclosures over summer while monitoring weather parameters. The rate of larval development also varies among host species. For example, in laboratory studies, 95% of *P. tenuis* larvae in the snail *Mesodon thyroidus* reached the infective stage after 35 days at 21 °C; only 34% completed development in the slug *Deroceras reticulatum* (Lankester and Anderson 1968).

The hypothesis that the prevalence of *P. tenuis* infection in gastropods is positively correlated with increased deer density has not been adequately tested. However, in northeastern Minnesota, the frequency of infection (0.16%) was 4 x higher in gastropods where deer wintered at density of 50 animals/km^2 compared to summer habitat with 4 animals/km^2 (Lankester and Peterson 1996). Prevalence in gastropods can also be much higher (4-9%) on more southerly range where deer usually exist at higher densities year-round (Lankester 2001); however, data from these regions also reflect the differences and effects of climate, weather, and growing seasons.

INFLUENCE OF WEATHER ON DEER AND LARVAL OUTPUT

Severe winters typically limit the density of deer on northern range often shared with moose (Karns 1980, Nelson and Mech 1986, Mech et al. 1987, Dumont et al. 2000, DelGiudice et al. 2002, Patterson and Power 2002, Nelson and Mech 2005). A series of successive easy winters can markedly increase deer density, particularly the proportion of fawns and yearlings. The overall output of first-stage *P. tenuis* larvae increases proportionately with increased deer density and is also influenced by herd demographics (Fig. 1). Young, newly infected deer pass 2-3 x more larvae than older deer, and because output diminishes with age, fawn and yearling deer are disproportionately influential in a growing deer population (Slomke et al. 1995, Peterson et al. 1996). Higher deer density also increases habitat overlap between deer and moose, thereby increasing the risk of infection to moose.

Favourable weather increases larval output by deer not only by increasing deer density and altering demographics, but also by

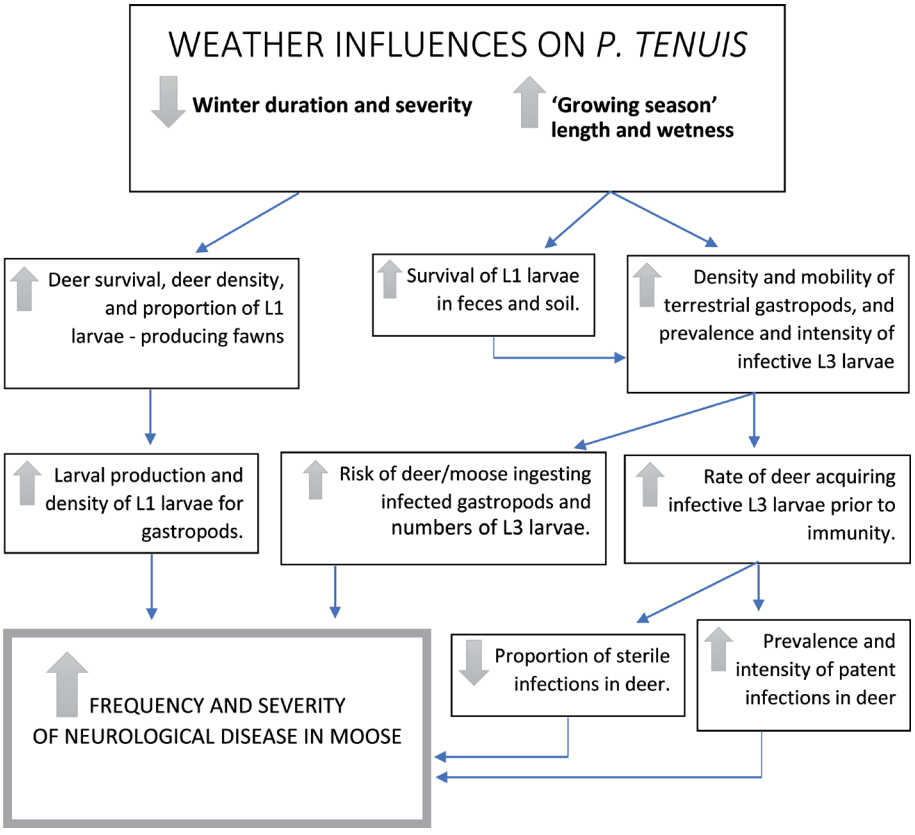

Fig. 1. Schematic illustrating the hypothesized influences of weather on deer and gastropod abundance that lead to increased transmission of meningeal worm to deer and moose.

increasing the rate at which naïve deer acquire their first infective larvae. This rate of transmission determines whether a deer develops a patent infection and produces first-stage larvae in its faeces, or instead has a sterile infection. If conditions for transmission are sub-optimal, only 1-2 infective larvae may become established before a fawn's first winter. This parasite is bisexual and infection with only a single worm, or of 2 or more worms of the same gender, will produce no first-stage larvae. Within about 6 months of ingesting infective larvae, the fawn develops an immune response that prevents further infection. Established worms are thought to be long-lived and to maintain this protection against supra-infection for the life of the deer.

These biological characteristics of *P. tenuis* have been confirmed in both field and laboratory studies (Slomke et al. 1995, Duffy et al. 2002, 2004). Up to one-third of infected deer examined in northeastern Minnesota had unisexual, sterile infections (Slomke et al. 1995), and 58% of deer examined in northern Michigan had single worm infections (Nankervis et al. 2000). Favourable weather will, by increasing the rate at which infective larvae initially are acquired, reduce the proportion of unisexual, sterile infections and thereby increase larval output by the fawn cohort. At the parasite's

western limits, high proportions of sterile infections and low prevalence of infection are thought to reflect rates of transmission that are limited by low precipitation and marginal conditions for gastropods (Wasel et al. 2003, Jacques et al. 2015, Maskey et al. 2015).

Some temperate northeastern forests provide conditions favorable for the meningeal worm to reach its final host. For example, despite low levels of *P. tenuis* infection in gastropods, almost all deer become infected by 2 years of age. Lankester and Peterson (1996) argued that this can be explained by the large volume of vegetation eaten close to the ground, particularly in spring and autumn. In a Minnesota study area with a stable deer population estimated at 2 animals/km^2, 79% of fawns became infected within their first year of life despite only a 0.08% rate of gastropod infection; eventually, 96% of deer became infected (Slomke et al. 1995).

MEASURING TRANSMISSION RATES

It would be advantageous to monitor changes in transmission rates of *P. tenuis* in deer, but peculiarities of the parasite's biology make this difficult. Metrics such as the prevalence and intensity of adult worms in deer heads are not particularly useful because almost all deer in the northeastern forests of Minnesota have at least one worm (sustained prevalence ~100%, Slomke et al. 1995). Likewise, the mean intensity of worms in the head varies little other than minor changes in the fawn cohort. Deer acquire only a small number of worms during their first year or two of life, and none thereafter. Higher deer densities that increase the number of larvae dispersed in the environment might be expected to increase the abundance of adult worms in the heads of deer, but field evidence is unsupportive. Slomke et al. (1995) measured similar abundance (3.5 ± 1.8 worms) in the heads of deer confined at a year-round density of 30 deer/km^2, as in a nearby, free-ranging population (3.0 ± 2.0 worms) at 2 deer/km^2.

Changes in transmission rates are potentially reflected in the frequency of infection in gastropods, but measuring the frequency of infection is challenging. Because the prevalence of infection in snails and slugs in northeastern forests is typically very low, extensive, labor-intensive sampling is required to detect significant changes. As well, considerable skill is required to distinguish the larvae of *P. tenuis* from those of several other species of nematodes found in these hosts. Nonetheless, higher frequency of infection in gastropods has been identified in more southerly deer range where infection opportunity is presumably increased by higher deer density, longer growing seasons, or more favourable gastropod habitat (Lankester 2001).

Annual changes in transmission rates can only be monitored by examining deer faeces for first-stage larvae (Peterson et al. 1996, Maskey et al. 2015). Ideally, faecal samples should be collected off snow during late winter after newly acquired worms have matured and produced larvae. Changes in prevalence and intensity of larvae in an opportunistically collected sample of faeces should reflect changes in the proportion of fawns in the population, as well as weather-related transmission rates determining the frequency of sterile unisexual infections. Examining only fawn faeces, Peterson et al. (1996) found that both prevalence and intensity varied annually and correlated best with changing deer density and the duration of the previous autumn transmission period.

Transmission likely occurs exclusively during the snow- and frost-free periods referred to here as the growing season. The annual length of the growing season varies considerably (Murray et al. 2006) which

alters the time period in which transmission is possible any given year (Fig.1). Larval output by deer is maximum in spring, the wettest season, yet autumn presents unique opportunities for *P. tenuis* transmission. The entire fawn cohort is susceptible to infection in late summer and autumn, whereas by snow melt the following spring, almost 80% could be resistant to further infection. Also, gastropod abundance peaks by autumn prior to any over-winter mortality. Any delay in the onset of winter lengthens the period for possible infection (i.e., ingestion by deer and moose) of the new cohort of *D. laeve*. Visibly sick moose are frequently seen in spring, suggesting that infection occurred the previous autumn (Lankester 2001). Autumn is similarly considered the most important season for transmission of related protostrongylid nematodes in sheep (*Ovis* spp.) and mule deer (*Odocoileus heminous*) (Samuel et al. 1985, Jenkins et al. 2006).

WEATHER-ENHANCED TRANSMISSION AND MOOSE

Weather-enhanced transmission of *P. tenuis* will increase the number of infective larvae available in gastropods, and the rate at which deer and moose ingest them over their lifetime. Deer will be unaffected and the prevalence and mean intensity of worms in their heads will change little. Many ingested larvae may be unable to migrate beyond the intestines. Others may die in tissues *en route* to the spinal cord but, nonetheless, be important in boosting immunity to reinfection. Moose, on the other hand, are more susceptible and the rate at which they ingest infective larvae during the growing season may determine the severity of neurological disease (Lankester 2001). Moose given relatively high numbers of *P. tenuis* larvae (15-25) showed severe and unmistakable signs of moose sickness including circling, hind-quarter weakness, and eventually an inability to stand. However, 4 moose given doses of 3-5 larvae, more closely resembling those acquired from a single naturally-infected gastropod, developed only mild neurological signs for periods of 1 to 3 months; one had no detectable signs at termination. Further, other results suggest that a degree of protection against future infection may result from a low-dose exposure (Lankester 2002).

Young moose may be the most susceptible to neurological disease. Disease occurs in animals of all ages, but many sick animals are < 2 years old (Lankester et al. 2007, Carstensen et al. 2015, Wünschmann et al. 2015). Young males that consume more food in early life might be expected to ingest more larvae than young females. Interestingly, in the current long-term decline in Minnesota, Murray et al. (2006) found lower survival of male than female calves. It is reasonable to predict that the infection rate of wild moose will be most influenced by the rate of acquiring infective larvae; however, even low-dose exposure and sub-clinical infection can be important. Rempel (2011) suggested that indirect effects of parasites like *P. tenuis* might reduce recruitment through increased predation, and possibly have greater impact on moose populations than direct mortality.

Intuitively, the exposure rate of moose to meningeal worm is directly related to deer density; however, two problems make it difficult to clearly demonstrate this relationship. It is difficult to 1) correctly census clinically ill and minimally compromised moose, and 2) estimate deer density that varies seasonally and annually. Nonetheless, field data (Whitlaw and Lankester 1994a, Maskey 2008) and several anecdotal studies in northeastern forests suggest that when infected deer density increases, moose numbers decline (Karns 1967, Saunders 1973, Gilbert 1974, Dumont and Crete 1996, Gogan et al. 1997, Lankester 2001, Lankester

and Samuel 2007). Yet, if deer density remains < ~5 animals/km^2, moose density remains relatively stable for extended periods (Karns 1967, Whitlaw and Lankester 1994b), albeit at densities lower than where deer are absent (e.g., on the island of Newfoundland and on Isle Royale, Michigan) (Timmermann et al. 2002, Lankester 2010).

Pronounced and prolonged moose declines have occurred repeatedly in particular regions of shared moose and deer habitat (Lankester 2010). These include much of Nova Scotia, northwestern Minnesota, and areas to the west of Lake Superior including northeastern North Dakota, southeastern Manitoba, and northwestern Ontario. The most recent decline in northwestern Minnesota began during a period of milder, shorter winters and has lasted 25 years. In 15 years moose numbers declined to ~100 animals from an estimated 4,000 in the late 1980s (Murray et al. 2006, Lenarz et al. 2009).

Recent pronounced declines have had certain shared characteristics. All were associated with conditions likely to have enhanced transmission of meningeal worm; i.e., extended series of warmer winters, frequent or sustained high deer densities, and wetter and longer than usual growing seasons (Beazley et al. 2006, Maskey 2008, Lenarz et al. 2009, Ranta and Lankester 2017). The annual growing season during the moose decline in northwestern Minnesota was on average 12 days longer, and up to a maximum of 39 days longer than during pre-decline years (Murray et al. 2006). Although precipitation records for northwestern Minnesota revealed no change during the decline, a long-term, wet climate cycle beginning in 1993 was reported in adjacent northeastern North Dakota (Todhunter and Rundquist 2004 in Maskey et al. 2015). Ranta and Lankester (2017) found that the growing season during a pronounced moose decline in northwestern Ontario was only marginally longer than in pre-decline years, but decidedly wetter than average. In northeastern Minnesota, Lenarz et al. (2009) found that warming January temperatures were inversely correlated with subsequent annual survival of moose. And in northwestern Minnesota, disease has played a measurable role in the moose decline; the majority (87%) of the 24% annual mortality rate was attributed to pathology associated with parasitic disease and related malnutrition (Murray et al. 2006).

CONCLUSIONS

It is argued here that transmission rates of *P. tenuis* and the risk of debilitating meningeal worm infection in moose are driven primarily by weather, specifically by winter severity and the length, precipitation, and temperature during the 'growing season'. Warmer, shorter winters permit higher densities of infected deer which increase the density of first-stage larvae on range. Longer, wetter growing seasons increase the density of infected gastropod intermediate hosts and parasite transmission rates. Over much of their shared range in the mixed coniferous-deciduous forests of eastern North America, moose can persist with infected deer where typical winter severity effectively limits or stabilizes deer density. But sustained high deer density and weather-enhanced transmission of *P. tenuis* can potentially cause local moose abundance to decline markedly over time and remain low. Further, these declines do not occur without warning as they are seemingly preceded by a number of successive winters (e.g., >10) favourable to deer survival. In northwestern Ontario, both deer and moose abundance rose in response to habitat rejuvenation and easier winters. Only after 15 years of slow, but steady increases in deer numbers did moose begin to decline (Ranta and Lankester 2017).

After discovering that *P. tenuis* causes moose sickness, Anderson (1972) suggested that this parasite might be capable of seriously impacting moose populations. His assertion was based in large part on historical reports of moose sickness in Nova Scotia and northern Minnesota, and in this restricted sense, it increasingly appears that his conclusion was correct. These two regions have experienced repeated, pronounced and prolonged declines in moose abundance suggesting that they are prone to extended periods of weather that favour winter survival of deer and enhanced transmission of meningeal worm implicated in such declines.

In regions where weather-enhanced transmission of meningeal worm appears to have occurred in the past or is likely in the future, management options to minimize this disease in moose should focus on maintaining deer density at < 5 animals/km^2 as per Karns (1967) and Whitlaw and Lankester (1994a). Strategies might include higher harvests of antlerless deer, possibly less-focused habitat management that prioritizes deer wintering areas, and certainly regulating/prohibiting winter feeding of deer to enhance their populations. Given that long-term climate change will be favourable to deer, maintenance of more southerly moose populations will require concerted management efforts and may prove difficult. Learning to manage the ebb and flow of co-existing deer and moose populations may be a better approach than attempting to maintain each species in a steady state.

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