

UNDERSTANDING THE IMPACT OF MENINGEAL WORM, *PARELAPHOSTRONGYLUS TENUIS*, ON MOOSE POPULATIONS

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ABSTRACT: Declines in moose (*Alces alces*) populations have occurred repeatedly during the past century on the southern fringe of moose range in central and eastern North America, generally in the same geo-climatic regions. These prolonged declines, occurring over a number of years, are associated with higher than usual numbers of co-habiting white-tailed deer (*Odocoileus virginianus*). Numerous proximate causes have been hypothesized but none has gained widespread acceptance among cervid managers. However, current knowledge of the nature of moose declines and the biology of meningeal worm (*Parelaphostrongylus tenuis*) makes this parasite the most credible explanation. Other suggested disease-related causes are rejected, including infection with liver flukes (*Fascioloides magna*). There is no clinical evidence that flukes kill moose. As well, this parasite occurs at only moderate prevalence and intensity in some jurisdictions and is completely absent in others where moose declines are known. Winter ticks (*Dermacentor albipictus*), on the other hand, do kill moose but usually have a distinctly different and more immediate impact on populations. It is recognized that moose, albeit at lowered density, can persist for extended periods in the presence of *P. tenuis*-infected deer at moderate densities. However, it is argued here that parelaphostrongylosis can, when conditions favour sustained high deer densities and enhanced gastropod transmission, cause moose numbers to decline to low numbers or to become locally extinct. Short, mild winters favour deer population growth in areas previously best suited for moose. Wetter and longer snow-free periods increase the numbers and availability of terrestrial gastropod intermediate hosts and the period for parasite transmission. It is hypothesized that these climatic conditions increase rates of meningeal worm transmission to moose and of disease, primarily among younger cohorts. Reports of overtly sick moose are common during declines but may not account for the total mortality and morbidity caused by meningeal worm. Other means by which the parasite may further lower recruitment and productivity causing slow declines still needs clarification. Managers in areas prone to declines should monitor weather trends, deer numbers, and the prevalence of meningeal worm in deer. Moose recovery will occur only after deer numbers are decidedly reduced, either by appropriate management or a series of severe winters.

ALCES VOL. 46: 53-70 (2010)

Key words: *Alces alces*, climate, *Dermacentor albipictus*, *Fascioloides magna*, moose die-offs, moose sickness, *Odocoileus*, parelaphostrongylosis, white-tailed deer.

The neurological disease of moose (*Alces alces*) known as “moose sickness” has been reported in eastern and central North America for almost 100 years. Its cause, the meningeal or brain worm (*Parelaphostrongylus tenuis*) from white-tailed deer (*Odocoileus virginianus*), has been known for almost 50 years (Anderson 1964). But the suggestion made long ago by Anderson (1965, 1972) that the resulting disease (parelaphostrongylosis) can cause moose populations to decline is still not generally accepted (Lankester and

Samuel 2007).

Unequivocal, direct evidence identifying *P. tenuis* as a main cause of moose declines, admittedly, is limited. This probably has encouraged the continued search for alternative explanations including ticks with an attendant bacterium (*Klebsiella paralyticum*), competition with deer, trace element deficiencies, a proposed virus, declining habitat quality, direct and indirect effects of warming climate and heat stress, liver flukes (*Fascioloides magna*), and a variety of other proposed, human-

induced stressors (see review by Lankester and Samuel 2007). None, however, can be as strongly argued as the meningeal worm hypothesis, albeit relying heavily on indirect evidence including knowledge of the parasite's biology in gastropods, deer, and moose, its known pathogenicity, and the reoccurring association of sustained high numbers of infected deer with moose declines. The purpose of this paper is to characterize past and recent moose declines and to highlight the biology of the meningeal worm, *P. tenuis*, considered here to be the most likely cause of periodic, prolonged moose declines.

MOOSE DECLINES

Declines in moose numbers have occurred repeatedly in several eastern North American jurisdictions during the past century (Anderson 1972, Whitlaw and Lankester 1994a, Lankester and Samuel 2007). Declines typically occur, almost imperceptibly, over a number of years and only in the relatively narrow band of mixed coniferous-deciduous forest ecotone extending from the Atlantic, around the Great Lakes Basin, and westward toward the edge of the central Great Plains. Pre-1900, much of this area was covered with mature forests and was the southern extent of recent moose and caribou (*Rangifer tarandus*) range. Since then, extensive habitat rejuvenation and/or extended periods of shorter, less severe winters have periodically created exceptional conditions allowing sustained high deer densities. Otherwise, deer numbers in moose range are kept relatively low (approximately $\leq 5/\text{km}^2$) by periodic harsh winters, regulated hunting, and predators (Whitlaw and Lankester 1994b).

Historical moose declines had certain characteristics; they occurred when moose sickness was being reported and deer numbers were unusually high. Examining long-term (80 years) historical data beginning in 1912, Whitlaw and Lankester (1994a) found an inverse relationship between moose and deer numbers with moose declining when deer

densities exceeded $5/\text{km}^2$. Noted declines were gradual, with moose population estimates going from high to low values over periods of 7-10 years. Analyses showed that high deer numbers, high reporting rates of sick moose (# of cases/# of years with reported cases), and declining moose numbers were coincident in at least 5 of 13 identified declines, despite concerns about the precision of historical densities estimates and doubts that reporting rates were representative. As well, a possible time shift may confound such analyses. Increased rates of infection in gastropods may lag behind a buildup in deer numbers and because some snails live 2-3 years (Lankester and Anderson 1968), the impact on moose could continue after deer decline.

Despite periodic moose declines, it has long been evident that moose can persist with infected deer for extended periods. Karns (1967) suspected that moose could be managed in Minnesota at acceptable numbers provided deer did not exceed $12/\text{mi}^2$ ($4.6/\text{km}^2$). In Ontario, Whitlaw and Lankester (1994b) documented a 10-year period (up to 1992) of relative deer-moose population stability. Managers surveyed in 45 game management units indicated that deer and moose were co-habiting, despite occasional reports of sick moose. Moose numbers were thought to be increasing in 36 and declining slightly in only 5 management units. Nonetheless, moose densities were inversely related to deer densities and were greatest when deer were $<4/\text{km}^2$. Moose density was also inversely related to the intensity of *P. tenuis* larvae in deer feces.

Other jurisdictions similarly reported moose and deer apparently co-existing in close proximity during the 1970s and 1980s, including Maine (Dunn and Morris 1981), northern New Brunswick (Boer 1992), southern Quebec (Dumont and Crete 1996), northern New York (Garner and Porter 1991), and Voyageurs National Park in Minnesota (Gogan et al. 1997). During this period, however, deer in

these locations were not noted to be unusually abundant and were likely constrained over the longer term by weather, habitat, predators, and/or hunting.

It is clear, however, that moose co-habiting with infected white-tailed deer are less productive than elsewhere, although comparisons among identical circumstances are seldom possible. For example, on Isle Royale where moose are constrained primarily by habitat and wolves, density ranges from approximately 1-2/km² (Vucetich and Peterson 2008). Mean density approaching 3/km², and often exceeding 4/km², is realized in Newfoundland where moose exist with bears (*Ursus americanus*) and hunting (McLaren and Mercer 2005). In comparison, over much of mainland eastern North America, where moose exist with infected white-tailed deer as well as with hunters and predators, moose densities typically are <0.4/km² (Timmermann et al. 2002). Apparently, a strong interplay of limiting factors, effects of meningeal worm included, is already reflected in lower moose densities where they persist in habitats with infected deer.

Moose sickness has been reported historically in the Canadian provinces of New Brunswick, Nova Scotia, Quebec, Ontario, Manitoba, and the northern states of Maine, Vermont, New Hampshire, Michigan, and Minnesota (Lankester 2001). Moose essentially disappeared in Wisconsin by the early 1900s (Parker 1990) and have re-colonized only the highland areas of northern New York (Hickey 2008); both states have had relatively high deer densities with regularity since the early 1900s (Culhane 2006). Moose continue to struggle in the Upper Peninsula of Michigan despite 2 reintroductions (Hickie 1944, Dodge et al. 2004).

Of nearly 500 reports of sick moose (literature to 2001), most occurred in Nova Scotia (28%) during the 1940-1950s, and northern Minnesota (20%) during the 1930-1940s (Lankester 2001). More recently, during a prolonged period of climate warming beginning in

the late 1980s, these same 2 jurisdictions have again experienced moose declines. In Nova Scotia, moose were declared an endangered species in 2003 (Beazley et al. 2006), and moose in northwestern Minnesota have all but disappeared (Murray et al. 2006).

These recent studies in Nova Scotia and Minnesota, as well as in North Dakota (Maskey 2008), serve to further characterize the nature of moose declines and confirm their occurrence during prolonged periods of high deer densities. They also provide new information lacking in historical accounts, including estimates of moose mortality rates, necropsy findings, and better estimates of concurrent deer densities. Further insight into the plight of moose facing rising deer numbers is provided by Dodge et al. (2004) in upper Michigan and by anecdotal observations on the health of moose populations in northwestern Ontario and neighbouring southeastern Manitoba.

Nova Scotia

The history of deer is well known (Patton 1991); after an introduction in the 1890s and likely immigration from neighboring New Brunswick, deer numbers increased across the province and a “boom” occurred in the 1940s. By the early 1950s deer were more plentiful than ever, but 3 hard winters in the late 1950s depressed deer numbers and moose responded positively (Benson and Dodds 1977). For the next 15 years deer numbers were fairly stable, producing an annual harvest of about 20,000 deer. Three recognizable moose declines were identified in the period 1930-1975 (Whitlaw and Lankester 1994a). During these declines, a total of 137 cases of moose neurological disease (6.5-10/yr) were noted; a record number of cases for any single jurisdiction. By 1985 a series of relatively mild winters had produced another increase in deer numbers and a harvest of 63,000 deer. Deer declined to one-quarter that number in 1990 and moose numbers may again have responded positively (Pulsifer and Nette 1995).

Parker (2003), in an excellent review of the status of moose in mainland Nova Scotia, suggested that numbers generally showed a somewhat continuous decline beginning as early as the late 1920s, despite a partial hunting closure in 1937 and a total ban in 1981 (excluding Cape Breton Island). Agreeing with earlier authors (Dodds 1963, Telfer 1967, Benson and Dodds 1977), he noted that a decline in the numbers of moose harvested clearly showed an inverse trend with increasing deer harvests.

Presently, moose remaining in mainland Nova Scotia are largely confined to 5 remnant, localized groups, each with 50-600 animals (Beazley et al. 2008). They are mostly in elevated areas with early, deep-snow winters and separated from deer, at least during winter. These areas are thought to serve as partial refugia from paratuberculosis (Telfer 1967, Pulsifer and Nette 1995, Lankester 2001, Beazley et al. 2006). Although data are scant, calf survival in some of these groups was low, and paratuberculosis was observed in the population (Beazley et al. 2006). These authors, citing Benson and Dodds (1977), concluded that sufficient circumstantial evidence exists to suggest that a decline of the mainland moose population began following marked increases in deer numbers in the late 1920s-early 1950s, and continued in association with periodic high deer densities.

Moose were almost extirpated on Cape Breton Island by the late 1800s, but an introduction of animals from Alberta in the late 1940s led to a hunted population of about 5000 (Beazley et al. 2006, Bridgland et al. 2007). Moose continue to prosper on the highlands of Cape Breton Island where deer do not winter, but are absent in southeastern lowland areas. Beazley et al. (2008) concluded that moose were not excluded from the lowlands by lack of suitable habitat, but possibly by climatic and geological factors, including a role played by meningeal worm in white-tailed deer.

Much of Nova Scotia (excluding the

highlands of Cape Breton) has a climate moderated by proximity to the sea and is warmer and wetter than much of southern, continental moose habitat. Although periodic hard winters along with coyote predation are known to impact deer numbers (Patterson et al. 2002), extended periods of deer population growth and a wetter climate conducive to gastropod transmission may explain why moose numbers outside of refugia have not recovered. It may be significant that much higher prevalence of *P. tenuis* infection has been found in gastropods in Nova Scotia and southern New Brunswick (2.6% and 2.3%, respectively) than is found in northern New Brunswick and more continental parts of eastern Canada (<0.5%; see review by Lankester 2001).

Northwestern Minnesota

A recent moose decline commencing about 1985 in an area including the Agassiz National Forest Wildlife Refuge, the Red Lake Wildlife Management Area, and adjacent agricultural land was studied by Eric Cox (deceased). Data from related aerial moose surveys, and necropsy of radio-collared (females and neonates) and accidentally killed moose from 1995-2000 were later analyzed by Murray et al. (2006).

The geography of the general area is rather atypical of moose habitat with much within the Northern Minnesota Wetlands Ecoregion that is characterized by standing water and permanent wetlands (Murray et al. 2006). It comprises a mosaic of private farmlands and protected areas, with marshes dominating natural areas along with lowland areas of willow (*Salix spp.*), aspen (*Populus spp.*), and black spruce (*Picea mariana*), and uplands with aspen and oaks (*Quercus spp.*).

Following an earlier decline in the 1940s, moose numbers in northwestern Minnesota grew from an estimated 1300 animals in 1960-61 to about 4000 by 1984-85 (Murray et al. 2006). In 1971 the population was deemed high enough to support the first hunt in 49 years

(Karns 1972). Numbers declined slightly after 1985 only to rise again by the early 1990s. Thereafter, moose began a step-wise decline that continued to 2000-01 when numbers may have been as low as 500 (Murray et al. 2006, Peterson and Moen 2009); the limited hunting was stopped after 1996. After declining for almost 2 decades, an aerial survey in 2007 estimated the moose population at <100 (Lenarz 2007b).

Deer numbers began to increase in the 1970s, declined somewhat in the mid-1980s, but peaked again in the mid-1990s. Peak aerial estimates of about 9/km² in 1980 and 8/km² in 1994 were recorded in the Agassiz National Forest Wildlife Refuge (Peterson and Moen 2009). Two severe winters in 1995-97 reduced deer numbers dramatically, but ensuing mild winters and restricted hunting allowed recovery by the early 2000s. Simulation models in 2007 estimated pre-fawning deer density at 5-14/km² in the northwest forest hunting zones (Lenarz 2007a).

Initially during the moose decline (1984-1997), mid-winter calf survival was relatively high (54-94 calves:100 cows; Murray et al. 2006). Only in subsequent years (1997-2001) did mid-winter calf:cow ratios fall below 50:100; the annual survival rate of male calves was half that of females. Pregnancy and twinning rates in the declining population were low (<50%) for most female age classes and correlated with nutritional state (bone marrow fat), and female reproductive senescence was apparent as early as 8 years old. Excessive mortality generally was thought to begin among the yearling age class and worsen progressively with age. Population age structure was skewed toward younger age classes and relatively few prime breeding-aged animals (4-7 years old) were present (Murray et al. 2006). The population experienced a pooled mean annual mortality rate of 24% of which 87% was attributed to pathology associated with parasitic disease and related malnutrition (Murray et al. 2006). The prevalence of liver

fluke (*Fascioloides magna*) in the population was 89%, and the authors concluded that up to 32% of parasite-related death was due to this parasite. Meningeal worm was believed responsible for 5% of the deaths in a radio-collared sub-sample, and 20% in a second submitted by the public. The cause of up to 25% of deaths was classified as “unknown” but thought to be parasite-related.

Murray et al. (2006) classified moose as having died from liver flukes if severe organ and tissue damage was evident with no other overt cause of death; those without damage but with numerous flukes were considered “probable liver fluke deaths”; 89% percent were infected. Given the absence of published evidence linking the death of moose to fluke infection, their analyses may overstate the importance of this parasite in the decline. It should also be noted that Karns (1972) sampled much the same area and found a similar prevalence of fluke infection (87%) 28 years earlier when the population had grown to huntable numbers and continued to do so, thereafter. Murray et al. (2006) considered death was due to meningeal worm if at least one *P. tenuis* was found in the cranium and no other suspected cause of death was evident. Known difficulties in locating *P. tenuis* in moose heads (Lankester et al. 2007) suggest that deaths during the decline due to this parasite were likely underestimated. Overall, Murray et al. (2006) concluded that the recent moose decline was primarily the result of climate change (increasing summer and winter temperatures), possibly directly through summer heat stress or indirectly through ecosystem changes including higher deer numbers and parasite-related disease and malnutrition. Inappetence and/or inanition may have caused reduced condition since food was not thought lacking and the growing season over the study period had increased by up to 39 days (Murray et al. 2006).

Northeastern Minnesota

This area differs from northwestern Minnesota in it is largely mixed boreal forest with longer colder winters, and has yet to show clear signs of a prolonged decline in moose numbers, although it may be imminent (Lenarz et al. 2009, Peterson and Moen 2009). Aerial survey data (1983-2008; M. Lenarz, unpublished in Peterson and Moen 2009) indicate that the moose population has fluctuated between 4,000-7,500 with no evident long-term trends. Over the last 12 years of this interval, calf:cow ratios and hunter success rates have both declined (Lenarz 2009). Limited hunting has occurred annually since 1971 except in 1991. Deer densities are lower than in northwestern Minnesota; increasing numbers in the late 1980s and 1990s were severely reduced during 2 harsh winters (1995-97). Estimates of pre-fawning densities averaged 2.2/km² in 1996, rose to about 4/ km² by 2003, and have remained fairly stable since (Lenarz 2007b).

An ongoing 6-year study of 116 radio-collared adult bulls and cows indicates that pregnancy and calf survival rates are nearly normal although twinning rates and mid-winter calf:cow ratios trend downward; the calf:cow ratio was 0.32 in January 2009 (Lenarz et al. 2009). The estimated annual, non-hunter mortality rate was 21% over the 6-year period, or about twice that expected, most in the southern portion of the study area. Point estimates for the finite rate of increase averaged 0.86 indicative of a long-term declining population. About 60% of observed adult mortality was classified as “unknown causes”, a good portion believed to be related to the effects of parasites and disease worsened by warming climate and heat stress (Lenarz et al. 2009); most deaths occurred in spring and fall. Liver fluke infection is much less common in moose in northeastern Minnesota than the northwest and was not thought important, whereas meningeal worm infections were detected in some dead moose.

Annual mortality estimates of radio-

collared moose led Lenarz (2009) to suggest that a decline in the northeastern moose population was occurring despite a lack of clear evidence from annual aerial surveys. Using data from the radio-collared animals, Lenarz et al. (2009) found that warming January temperatures above estimated physiological thresholds for moose were inversely correlated with subsequent annual survival. Temperatures exceeding thresholds were considered to constitute heat stress by increasing energy expended to stay cool. They hypothesized that parasitic disease was likely a proximate cause of mortality while lowered productivity and increased mortality due to heat stress best explained what was occurring in this population (Lenarz et al. 2009).

North Dakota

Maskey (2008) completed a hallmark dissertation study (University of North Dakota) of factors likely to have been important in a recent moose population decline in the Pembina Hills area of northeastern North Dakota. His findings are of particular interest because of this population's close proximity to one in neighboring northwestern Minnesota believed by Murray et al. (2006) to have declined due to heat stress and liver flukes. Moose populations here experienced a period of growth beginning in the 1960s. By the mid-1990s, however, moose in the northeastern Pembina Hills area began a steady, decade-long decline to very low numbers (Johnson 2007). In a sample of 32 moose dying, 19% had *F. magna* infection while 75% of the moose exhibited signs consistent with meningeal worm infection (Maskey 2008). As in Minnesota, the decline in the Pembina Hills coincided with an unprecedented increase in the range and abundance of white-tailed deer in northern North Dakota (Smith et al. 2007), and with a long-term, wet climate cycle beginning around 1993 (Todhunter and Rundquist 2004 cited in Maskey 2008). Interestingly, moose numbers in 2 other areas west of the Pembina Hills

were either steady or increasing, despite local increases in deer numbers. Significantly, deer in these areas had lower rates of meningeal worm infection, presumably because of drier conditions less suitable for parasite transmission (Maskey 2008).

Michigan

Moose were extirpated from lower Michigan by the late 1800s and most were gone from the Upper Peninsula by 1900 (Dodge et al. 2004). An attempt to reintroduce them in the 1930s failed. A second establishment of 61 animals brought from Ontario in 1985 and 1987 has persisted but grown more slowly than expected. Despite meningeal worm initially causing 38% of observed mortality, this protected population, free of most predators, showed some growth when deer were estimated at 4.3/km² (Aho and Hendrickson 1989). However, rather than reaching a predicted population of 1000 by the year 2000, aerial surveys conducted in 1996-1997 estimated <150 moose; radio-collaring subsequently occurred in 1995-2001 to investigate the decline (Dodge et al. 2004). Of 17 deaths of marked moose, more than half were attributed to meningeal worm and liver flukes. Survival rates of adults, yearlings, and calves were all similar to those found in other non-hunted, lightly predated populations. They concluded that poor population growth was due to low pregnancy and twinning rates, and no yearling reproduction caused by less than optimal food quality and supply (Dodge et al. 2004).

State-wide, deer increased dramatically after about 1985 (Frawley 2008) and by 2007 the harvest approached 484,000 animals. In the Upper Peninsula (UP), peak populations of the early 1990s were reduced briefly by 2 severe winters (1995-97) but resumed their increase until another severe winter in 2007-08. Nankervis et al. (2000) found 44% of deer heads sampled in the UP had meningeal worm and 0.7% of gastropods contained infective larvae.

The moose population on deer-free Isle Royale has behaved quite differently from that in the UP in the past 30 years. Reduced wolf numbers, following a canine parvovirus outbreak about 1980, allowed moose numbers to increase to about 2500 (3/km²) by the winter of 1995-96 (Vucetich and Peterson 2008); however, that severe winter and a delayed spring reduced the population of poorly nourished animals to about 500. The population subsequently doubled by 2002 but declined again despite adequate food. Since 2002 moose have been subjected to high numbers of ticks and hair loss in spring, possibly due to a decade-long trend of warmer than average summers; however, late-winter mortality of infested moose was not prominent. Instead, much mortality since 2002 is attributed to high predation on calves by a disproportionately high wolf population (Vucetich and Peterson 2008). Changes in the numbers of moose on Isle Royale over the past 50 years fluctuated, sometime abruptly, largely in relation to winter severity, food constraints, and changes in wolf predation. By comparison, numbers of moose confined in 2 other parks (Sleeping Giant and Algonquin Provincial Parks, Ontario) changed slowly but dramatically when co-habiting with increasing numbers of unhunted deer with meningeal worm (Whitlaw and Lankester 1994b).

Northwestern Ontario and Southeastern Manitoba

The ranges of moose and deer first overlapped in northwestern Ontario in 1900-1920. Thereafter, deer increased provincially until the late 1950s (Whitlaw and Lankester 1994b) when they were as far north as Sioux Lookout. Moose declined during the 1940s and the hunting season was closed briefly in 1949. A series of deep snow winters began in the early 1960s, but deer numbers remained robust until 2-3 extremely severe winters in the mid-1970s reduced their populations by at least 50% (probably closer to 80%; B. Ranta,

personal communication). Moose increased during the early part of this period but apparently declined somewhat by the mid-1970s. From 1980-2007, winters were increasingly warmer and shorter, interrupted by only a few hard winters (1995-97 and 2007-09). Surveys in 1980-1992 of management units with both moose and deer indicated that moose numbers were stable to slightly increasing over much of the region, and were highest where deer density was estimated at $<4/\text{km}^2$ (Whitlaw and Lankester 1994b).

By the mid-1990s, the Kenora District had some of the highest moose populations in the province at about $2/\text{km}^2$ on the Aulneau Peninsula in Lake of the Woods where only a black powder and archery hunt was allowed (B. Ranta, personal communication.). But by the early 2000s, deer were becoming noticeably more abundant throughout much of northwestern Ontario. This period of deer increase was characterized by several years of shorter, milder winters and large tracts of forests in the region subject to blow-down and insect damage (spruce budworm [*Choristoneura fumiferana*] produced a bonanza of readily available forage when the dead and dying balsam was colonized with arboreal lichens, principally *Usnea* sp.). Deer peaked at high numbers in the Kenora area in the winter of 2006-07 and were again as far north as Sioux Lookout. By 2007, moose were virtually absent on the Aulneau Peninsula. By the end of the 1990s, moose numbers in parts of northwestern Ontario were in decline, especially south of Highway 17 between Kenora and Thunder Bay. Little information is available on the demography of the current moose population but the trend in numbers was downward and poor calf productivity and recruitment was notable (A. Rodgers, Ontario Ministry of Natural Resources, personal communication).

Moose in southeastern Manitoba, east of Winnipeg and south of Lac du Bonnet, have historically shared habitat with an infected and

widely fluctuating deer population primarily regulated by winter severity. In the early 1970s, deer were numerous in much of the area and sightings of sick moose with meningeal worm (and *F. magna*) were common (Lankester 1974). More recently (1995-2008), deer increased in number and northern distribution after several easy winters. Meanwhile, moose declined and virtually disappeared from the extreme southeast corner of the Province, south of Highway 1, and licensed hunting was discontinued in 2000 (V. Crichton, Manitoba Wildlife & Ecosystem Protection Branch, personal communication).

MENINGEAL WORM TRANSMISSION

The importance of climate in understanding rates of transmission of meningeal worm to deer and the risk of infection to moose is under appreciated. Firstly, winter length and severity are important determinants of deer numbers at the northern limits of their range. Secondly, climate in summer (amount of precipitation and length of summer) determines 1) the survival of the parasite outside its host (as first-stage larvae), 2) the survival, abundance, and mobility of gastropods (the intermediate host), and 3) the suitability and length of the snow-free period when transmission is possible (Lankester 2001). Thusly, climate determines the density of deer and gastropods, and in turn, the rates at which each becomes infected. As emphasized by Wasel et al. (2003), the odds of encounter between an infected gastropod and a white-tailed deer (or moose) depend on the density and degree of spatial overlap of both.

Meningeal Worm in Gastropods

Terrestrial gastropods (both snails and slugs) are required intermediate hosts of meningeal worm. They become infected with *P. tenuis* by encountering first-stage larvae on deer feces or in soil where they are readily washed by rain and melting snow (Lankester

2001). Although the prevalence of infection in gastropods can be quite low (e.g., 1/1000), transmission is, nonetheless, very efficient. Because of the large amounts of vegetation consumed daily by deer, a low prevalence of infection in gastropods can still result in almost all deer becoming infected and at a young age (Slomke et al. 1995, Lankester and Peterson 1996).

A variety of gastropod species can serve as sources of infection but one in particular is most important, the small, ubiquitous dark slug *Deroceras laeve* (Lankester and Anderson 1968, Lankester 2001). It is often the most frequently infected species and the one with the most meningeal worm larvae, probably because it is very mobile. It is one of the first land gastropods to become active in spring and one of the last to cease movement in autumn (Lankester and Peterson 1996). Terrestrial gastropod populations respond to wet climate; moisture (precipitation and dew) determines their reproductive success and survival as well as mobility in ground litter and on low vegetation. Hawkins et al. (1997, 1998) demonstrated their potential to become more numerous on surface vegetation during wet periods. For every snail sampled on the surface during moderately dry periods, almost 50 more were present in the first 5 cm of underlying duff and soil (Hawkins et al. 1998). Further, models describing the potential impact of meningeal worm on moose were most sensitive to changes in the intrinsic rate of increase in gastropods (Schmitz and Nudds 1994).

Infection of gastropods is affected by climate; for example, snails and slugs in a wet forested area on Navy Island, Ontario, were >6 x (5.1 versus 0.8%) more likely to be infected with *P. tenuis* than those in a dry upland forest habitat (Lankester and Anderson 1968). A number of studies report overall prevalence in gastropods in many deer/moose areas to be much less than 1%, yet higher mean values of 2.6-9.0% occur in the Canadian maritime provinces and in deer-only areas

of the southeastern United States (Lankester 2001). These higher values probably reflect a warmer, moister climate with longer periods of gastropod activity, and probably higher deer densities as well (Lankester 2001). Presumably, climatic conditions that favor gastropod numbers and mobility not only increase their rates of encountering larvae on feces or in soil, but also the likelihood that they will be ingested by cervids.

Infection of gastropods also reflects the density of infected deer. There was a 4-fold difference (0.04% versus 0.16%) in the prevalence of meningeal worm larvae between summer range in northern Minnesota (4 deer/km²) and winter range where they aggregate (50 deer/km²) for a few months (Lankester and Peterson 1996). Likewise, gastropods on Navy Island where deer exist year-round at density of about 90/km², were 100 times more frequently infected (4.2%) than gastropods on summer range in Minnesota. Unusually high prevalence in gastropods is realized only where deer density is exceptionally high for long periods (Lankester 2001).

Meningeal Worm in Deer

The importance of winter to deer survival is well known (Karns 1980) but there is a lack of reliable data needed to test suspected relationships between climate, deer density, and *P. tenuis*-infection rate. Measuring deer density with acceptable precision is notoriously difficult and continues to be attempted by only a few jurisdictions. As well, accurately measuring the prevalence and intensity of meningeal worm in deer requires proper techniques and an understanding of the parasite's developmental biology (Slomke et al. 1995, Forrester and Lankester 1997).

Of exclusive interest here is the biology of meningeal worm in deer near the northern limits of their distribution where they periodically share habitat in large numbers with moose. Valuable information comes from a study of heads and feces from road-kill deer collected in

mid- to late winter within a deer wintering area near Grand Marais, Minnesota (Slomke et al. 1995). Deer density (pre-fawning) at that time was estimated conservatively at about 2/km² (Lenarz 1993) and the moose population was deemed fairly stable. Maturing worms were detected in the heads of a large percentage of fawns in early winter (December-February) indicating that deer are exposed to the parasite early in life (Slomke et al. 1995); >90% had encountered the parasite by their second autumn. Infected deer retain the same live worms in their cranium for life, young animals pass twice as many larvae as older animals, and larval output is highest in spring.

In deer, the parasite requires 3-4 months before first-stage larvae occur in feces. Traditionally, deer heads used for assessing infection rates are collected during the autumn deer hunting season, but this is not the ideal time to obtain an accurate estimate of infection rates because a large portion of the harvest could be fawns and yearlings with recent infections. Because some worms will still be developing inside the spinal cord and be difficult to detect, heads of fawns in hunter-killed samples should either be excluded or analyzed separately. Deer feces to be examined for larvae are best collected in late winter when most viable infections will be patent, and feces can be collected off snow and not be contaminated with soil nematodes.

Only low numbers of adult worms are found in the heads of deer living in moose range (e.g., $\bar{x} = 3.2$, range = 1-13; Slomke et al. 1995) because infection rates in gastropods are low and many have only a single larva. And, shortly after becoming infected, deer develop an immune protection against further infection even if exposure is to only a single infective larva (Slomke et al. 1995). Only those worms acquired within a few months of the first exposure are able to reach the cranium and mature before protection develops against further infection. Hence, fawns infected in late summer or autumn are likely to be im-

mune to further infection by the time snow melts the following spring when gastropods resume activity. This so-called concomitant immunity no doubt protects deer from acquiring too many worms that will eventually reside close to the brain.

Many deer initially pick up only a single infective larva (or 2 of the same sex) and do not encounter another before the immune response becomes protective. As a result, up to one-third of all infected deer may never pass larvae because a mature worm of the opposite gender cannot gain foothold (Slomke et al. 1995). Hence, the rates of infection in deer, mean numbers of mature worms, and the proportion of sterile infections in a population are determined by the rate of initial acquisition of infective larvae by fawns and yearlings. Therefore, year-to-year changes in infection rates and the factors responsible can only be detected by examining successive fawn cohorts (Peterson et al. 1996). Or, if the protection against re-infection is as strong as believed, past differences in annual transmission rates might be revealed using cohort studies of worm numbers in adult deer.

Considerable data support the prediction that the prevalence of meningeal worm in deer increases with increased deer density (Karns 1967, Behrend and Witter 1968, Slomke et al. 1995, Peterson et al. 1996, Wasel et al. 2003, Maskey 2008). The mean number of larvae in feces of fawns after their first winter (intensity of infection) is also positively correlated with deer density (Whitlaw and Lankester 1994b, Slomke et al. 1995, Peterson et al. 1996).

The importance of climate in meningeal worm transmission deserves more emphasis. The prevalence in deer is positively correlated with summer and autumn precipitation that presumably favours gastropod infection rates and availability (Gilbert 1973, Brown 1983, Bogaczyk 1990, Bogaczyk et al. 1993, Peterson et al. 1996, Wasel et al. 2003, Maskey 2008). Using 9 years of fecal data, Peterson et al. (1996) found that the prevalence in

10-month-old fawns correlated positively with the number of days in autumn when deer could still access ground vegetation.

Low rainfall, and possibly lower deer density, probably determine the westernmost limit of meningeal worm and have prevented its spread to vulnerable cervid communities in western Canada (Wasel et al. 2003). Unfavourable conditions for transmission in western Manitoba and central North Dakota resulted in low rates of infection (<20% with worms in the head). Of equal importance was the remarkably high proportion (44%) of deer that had only a single worm in the cranium; this is an expected consequence of low transmission rates. A large number of deer with only a single worm are effectively immunized and thought never to develop patent infections (Slomke et al. 1995).

Meningeal Worm in Moose

The meningeal worm rarely matures to produce larvae in moose, hence, infection depends on the presence of infected deer. Infected moose show behavioral and neuro-motor disease of varying severity (Lankester et al. 2007). Some animals show almost imperceptible or intermittent motor deficiencies with slight toe-dragging or stumbling. Severely affected animals can exhibit profound weakness of the hind quarters and may be unable to rise. Others that become laterally recumbent and flail their legs probably die. Some animals exhibit chronic debility including loss of fear of humans and weight loss, and may remain within a restricted area for an extended period; those escaping predation may recover eventually.

Overtly sick moose show a typical suite of neurological signs, but the severity of signs manifest is not necessarily reflective of the number of worms found on examination. Moose usually acquire <10 worms ($x = 2.5 \pm 0.6$; Lankester et al. 2007). Those with >3-4 in the cranium invariably show severe neurological impairment, but severe signs are

observed in moose with only a single worm, and sometimes none. Failure to locate worms may relate to the body site and/or killed worms, or host inflammatory response (i.e., meningitis and perineuritis). In addition, it is suspected that some infected animals may experience unobservable, physiological or behavioural abnormalities. The difficulty of finding adult worms at necropsy often makes a definitive diagnosis elusive. For example, in a sample of 34 moose showing typical clinical signs of *parelaphostrongylosis*, adult meningeal worms were found in only 44% (Lankester et al. 2007).

Moose of all ages are affected but younger animals certainly predominate. The mean age of animals showing signs in the above study was 3.6 years (range = 0.6-14). Those with worms detectable in the cranium were younger (1.8 ± 0.5 years) than those with signs but without worms in the head (5.2 ± 1.2 years). Females made up 76% of sick animals ≥ 3 years old. The sexes were more balanced (10 male:7 female) among younger animals (<3 years). There was no indication that females acquire fewer worms than males, but results suggest that over time, worms are overcome by moose and that females may survive infection longer. Results were similar in a smaller sample of 10 sick moose from southeastern Manitoba (Lankester 1974).

Only one experimental study has investigated the response of moose to low numbers of infective larvae, similar to that encountered in nature (Lankester 2002). All 5 moose (5-9.5 months) given 3-10 larvae showed abnormal locomotory signs after 20-28 days. Symptoms became progressively more pronounced with front limb lameness and hindquarter weakness. However, after 77-130 days, marked signs persisted in only one animal, were diminished markedly in 2, and disappeared in 2. Two animals were challenged with 15 larvae (199 days after initial infection) with no noticeable effects; at necropsy, one had a single worm believed to be from the challenge. Results

indicate that all young moose ingesting a few larvae show some impairment, even if intermittent and temporary. Worms in the cranium were overcome and some animals recovered, at least for the short-term, and such animals appeared to have a degree of protection that may reduce the impact of subsequent infections. How long-lasting such protection might be in naturally infected moose is unknown.

An enzyme-linked immunosorbent assay (ELISA) developed using excretory products of *P. tenuis* larvae was positive for all of the animals infected above (Ogunremi et al. 2002). The level of antibody response was strongly correlated with the inoculating dose, and levels remained high in all animals that still had worms in the cranium when euthanized. But, titers diminished in 2 and were undetectable in a third animal that had overcome worms by the time of necropsy (186 days after infection). Antibodies became elevated after challenge infection. More serological study and accompanying, competent necropsy of sick moose is needed to fully appreciate the utility of the ELISA in measuring and monitoring the impact of meningeal worm on moose populations.

OTHER POTENTIAL DISEASE-CAUSING PARASITES

Of several parasites known in moose (Lankester and Samuel 2007), only 2 (liver fluke and winter tick [*Dermacentor albipictus*]) have come to be associated with dead or sick animals during moose population declines. For several reasons, neither is likely to be the cause of gradual moose declines discussed here.

Liver Fluke

The giant liver fluke, or deer fluke, has a spotty and limited distribution across moose range. It occurs in the Great Lakes Basin including central and northwestern Ontario, southeastern Manitoba, northern Minnesota, the Upper Peninsula of Michigan, Quebec,

and a few locations in western Canada (Pybus 2001). The parasite has a water-based life cycle and is found only where aquatic snails of a particular genus (*Lymnaeus* spp.) occur. White-tailed deer and elk (*Cervus elaphus*) are its principal hosts and the source of infection to moose. It is acquired by eating an intermediate larval stage that emerges from snails and encysts on aquatic vegetation. Moose are a dead-end host and do not propagate the parasite. Their risk of infection presumably is directly related to the density of co-habiting infected deer and to the densities of suitable aquatic snails.

There is no clinical evidence that flukes kill or debilitate moose. However, the considerable tissue pathology seen in some heavily infected livers has led to the suggestion that flukes may cause mortality when moose are stressed (see reviews by Pybus 2001, Lankester and Samuel 2007). In moose, like in domestic cattle, migrating flukes cause bloody tracts, extensive fibrosis and compensatory liver tissue hypertrophy; infected livers may be more than double normal size.

Moose experimentally infected with *F. magna* and observed for >12 months showed no outward signs of disease (M. Lankester and W. Foreyt, unpublished). Two calves and a yearling were given 50-225 metacercariae and observed for up to 16 months. The liver of animals infected as calves were enlarged and contained bloody tracks, extensive fibrosis, and walled capsules; 1 and 11 flukes were recovered. That of the yearling had 3 large, thick-walled cysts but no flukes were found at necropsy. Growth, weight gain, and behaviour of all 3 were similar to uninfected, farm-reared moose.

In most hosts studied (deer, elk, caribou and cattle [*Bovus* spp.]), the prevalence of fluke infection increases with host age and plateaus in older age classes (Pybus 2001); young-of-the-year are rarely infected. Mean intensity generally is similar within each infected age class suggesting that an acquired,

immunological resistance to further infection develops. As well, flukes have a highly aggregated distribution in normal cervid hosts. Most have only a few flukes, while a small number of animals may carry large numbers. In dead end hosts like moose and cattle, long-standing chronic infections are characterized by large paste-filled, thick-walled, closed cysts with few recoverable live flukes (Lankester 1974). Flukes are not considered important to the health of cattle despite infections that resemble those in moose (Wobeser et al. 1985).

Liver fluke infections were noted during earlier moose declines in Minnesota (Fenstermacher and Olsen 1942), and flukes were found in livers of moose taken in the first hunt in many years; 17 and 87% were infected from the northeastern and northwestern regions, respectively (Karns 1972). Moose populations in Minnesota continued to grow even with hunting, and more recently, as moose in northwestern Minnesota experienced a marked decline (from about 1995-2005), the prevalence of fluke infection was essentially unchanged at 89% (Murray et al. 2006). Flukes were common in the wetter habitat of northwestern Minnesota during population growth and decline.

Flukes are less common in northeastern Minnesota and not thought to play a primary role in any decline there (Lenarz et al., unpublished data). In a study in adjacent northeastern North Dakota, flukes were found in only 18% of sick moose and were not considered responsible for declining moose numbers (Maskey 2008). In Nova Scotia, with a history of moose declines and where moose have been declared “endangered”, liver fluke has never been present. In total, these examples and data indicate that the deer liver fluke is not a significant factor in moose declines.

Winter Tick

The winter tick can be found on virtually all moose wherever they occur in North America, with the exception of the island of

Newfoundland and north of approximately 60° N latitude (Samuel 2004). Disease results when moose acquire unusually large numbers and are subjected to a long, severe winter and possibly diminished food quality or availability. Infested animals exhibit increased grooming and restlessness due to the irritation of blood-sucking ticks, lose weight, and experience extensive hair loss in late winter, all of which can contribute to death.

Die-offs attributed to moose ticks have particular characteristics. Relatively large numbers of infested moose, many with premature hair loss, are found dead in late winter/spring. Such conspicuous mortality usually occurs after a prolonged cold winter, and is often reported in unusually high density populations such as those protected in parks. Calves and yearlings are thought to be the most severely affected but older animals may not be spared. Die-offs often are widespread and rapid but short-lived (Samuel et al. 2000), continuing for only 1-2 consecutive springs. Such epizootics are independent of deer density.

Moderate tick infestations presumably have an on-going, sub-clinical impact on moose, but conspicuous die-offs most often occur when moose are dense, tick numbers are high, and nutritionally stressed animals have experienced a severe winter. Warmer, shorter winters result in increased survival of adult females dropping off moose onto litter in March and April and increased tick populations on moose the following winter (Drew and Samuel 1986, Samuel 2007). Warm, snow-free Octobers increase the survival of seed ticks and their likelihood of infesting moose (Samuel and Welch 1991).

Tick numbers generally correlate positively with moose density (Samuel 2007). Over a 12-year study in Elk Island National Park, Alberta, there was a 1-year lag in tick numbers relative to moose numbers. Die-offs occurred when moose approached 3/km² and mean numbers of ticks on moose reached 50-60,000. Since ticks generally have their

greatest impact on individual moose when populations are high, this parasite is unlikely to be the cause of prolonged, relentless declines in moose populations.

CONCLUSIONS

Pronounced declines in moose numbers on the southern limits of their distribution in eastern North America have occurred repeatedly over the past century. The most conspicuous reductions have occurred generally in the same geo-climatic regions, the milder and moister parts of eastern Canada (NS) and a central mixedwood, wetlands area, west of Lake Superior (comprising parts of northwestern Minnesota, northeastern North Dakota, southeastern Manitoba, and northwestern Ontario). The most recent declines were accompanied by a warmer and wetter period. Earlier declines (1930s-1950s) followed large-scale successional renewal of harvested mature forests, but also coincided with a lesser-known warm period (Le Mouel et al. 2008). It is concluded here that extended periods of warmer, and possibly wetter climate provide conditions conducive to moose declines resulting from increased winter survival of white-tailed deer and increased transmissibility of disease-causing meningeal worm. Reports of overtly sick moose are common during declines, but the number of recognizably sick animals may not represent the total mortality and morbidity caused by meningeal worm. Additional means by which the parasite may lower recruitment and productivity causing slow, insidious declines still require clarification. Managers in areas prone to declines can lessen impending harm to moose by monitoring weather trends, deer numbers, and the prevalence of meningeal worm in deer. If retention of high quality moose hunting is desired, deer numbers can be reduced where indicated by adjusting deer harvest and banning supplemental winter feeding that artificially elevates deer populations relative to habitat.

ACKNOWLEDGEMENTS

I gratefully acknowledge the hard work and inspiration of several graduate students and close colleagues in pursuing a better understanding of an important and re-occurring wildlife problem.

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