Pathogens, Nutritional Deficiency, and Climate Influences on a Declining Moose Population

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ABSTRACT

Several potential proximate causes may be implicated in a recent (post-1984) decline in moose (*Alces alces andersoni*) numbers at their southern range periphery in northwest Minnesota, USA. These causes include deleterious effects of infectious pathogens, some of which are associated with white-tailed deer (*Odocoileus virginianus*), negative effects of climate change, increased food competition with deer or moose, legal or illegal hunting, and increased predation by gray wolves (*Canis lupus*) and black bears (*Ursus americanus*). Long-standing factors that may have contributed to the moose decline include those typically associated with marginal habitat such as nutritional deficiencies. We examined survival and productivity among radiocollared ($n = 152$) adult female and juvenile moose in northwest Minnesota during 1995–2000, and assessed cause of death and pathology through carcass necropsy of radiocollared and non-radiocollared animals.

Aerial moose surveys suggested that hunting was an unlikely source of the numerical decline because the level of harvest was relatively low (i.e., approx. 15%/2 yr) and the population usually grew in years following a hunt. The majority of moose mortalities (up to 87% of radiocollared moose [$n = 76$] and up to 65% of non-radiocollared moose [$n = 84$]) were proximally related to pathology associated with parasites and infectious disease. Liver fluke (*Fascioloides magna*) infections apparently constituted the greatest single source of mortality and caused significant pathology in the liver, thoracic and peritoneal cavities, pericardial sac, and lungs. Mortality due to meningeal worm (*Parelaphostrongylus tenuis*) was less prevalent and was manifested through characteristic neurological disease. Several mortalities apparently were associated with unidentified infectious disease, probably acting in close association with malnutrition. Bone-marrow fat was lower for moose dying of natural causes than those dying of anthropogenic factors or accidents, implying that acute malnutrition contributed to moose mortality. Blood profiles from live-captured animals indicated that those dying in the subsequent 18 months were chronically malnourished.

Relative to other populations, average annual survival rates for adult females (0.79 [0.74–0.84; 95% CI]) and yearlings (0.64 [0.48–0.86]) were low, whereas those for calves (0.66 [0.53–081]) were high. Pregnancy (48%) and twinning (19%) rates were among the lowest reported for moose, with reproductive senescence among females being apparent as early as 8 years. Pregnancy status was related to indices of acute (i.e., bone-marrow fat) and chronic (i.e., blood condition indices) malnutrition. Opportunistic carcass recovery indicated that there likely were few prime-aged males (>5 yr old) in the population.

Analysis of protein content in moose browse and fecal samples indicated that food quality was probably adequate to support moose over winter, but the higher fecal protein among animals that died in the subsequent 18 months could be indicative of protein catabolism associated with malnutrition. Trace element analysis from moose livers revealed apparent deficiencies in copper and selenium, but there was limited evidence of direct association between trace element concentrations and moose disease, pathology, or mortality. Time-series analysis of regional moose counts (1961–2000) indicated that annual population growth rate was related negatively to mean summer temperature, with winter and summer temperatures increasing by an average of 6.8 and 2.1 C, respectively, during the 40-year period. This change may have increased moose thermoregulatory costs and disrupted their energy balance, and thereby reduced their fitness. Time-series analysis failed to show a relationship between annual population growth rate and moose or deer abundance, indicating that food limitation via resource competition was unlikely. Population viability analyses, using count data (1961–2000) and demographic data collected during this study, suggested that the northwest Minnesota moose population likely would not persist over the next 50 years. More broadly, we conclude that the southern distribution of moose may become restricted in areas where climate and habitat conditions are marginal, especially where deer are abundant and act as reservoir hosts for parasites.

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KEY WORDS

Alces alces, *climate, disease,* Fascioloides magna*, liver fluke, Minnesota, moose, nutrition, parasitism, population viability.*

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Influencia De Los Patógenos, Deficiencias Nutricionales Y Clima En Una Población De Alces En Decrecimiento

RESUMEN

Una reciente disminución (1984) en el número de alces en los límites sur de su distribución en el noroeste de Minnesota pudo haberse debido a diversas causas entre las que se incluye el efecto de patógenos infecciosos algunos de los cuales están asociados con el venado cola blanca (*Odocoileus virginianus*), los efectos negativos del cambio climático, el aumento en la competencia por comida con venados o alces, cacería legal e ilegal y un aumento en la depredación por lobo (*Canis lupus*) y oso negro (*Ursus americanus*). Otros factores de largo plazo que pueden haber contribuido a la disminución de la población de alce incluye a aquellos factores típicamente asociados con un hábitat marginal como deficiencias nutricionales. Evaluamos la supervivencia y productividad entre hembras adultas y alces juveniles con radiocollares ($n = 152$) en el noroeste de Minnesota entre 1995–2000 y determinamos causas de muerte y patología por medio de necropsias en animales con radiocollar y de animales sin collar cuando fuera oportuno. Recorridos aéreos sugieren que la cacería no fue una causa importante de la disminución debido a que el nivel de animales cosechados fue relativamente bajo (aproximadamente 15% en 2 años) y en general la población creció en años posteriores a la cacería. La mayoría de las muertes de alces (hasta un 87% de alces con radiocollar [$n = 76$]; y un 65% de alces sin collar [$n = 84$]) se debió a patologías asociadas a parásitos y enfermedades infecciosas. La infección por el trematodo hepático (*Fascioloides magna*) aparentemente constituyó la causa de mortalidad más importante y provocó patologías significativas en hígado, cavidades del tórax y peritoneo, saco pericárdico, y pulmones. La mortalidad por verme meníngeo (*Parelaphostrongylus tenius*) fue menos prevalente y menos manifiesta como enfermedad neurológica característica. Algunas mortalidades aparentemente estuvieron asociadas a enfermedades infecciosas no identificadas, probablemente interactuando con una malnutrición. Los alces que murieron de causas naturales presentaron menos grasa medular que los que murieron por factores antropogénicos o accidentes, lo que sugiere que la malnutrición aguda contribuyo a las muertes. Los perfiles sanguíneos de animales vivos capturados indicaron que aquellos que murieron en los 18 meses subsecuentes estaban crónicamente malnutridos.

La tasa de supervivencia promedio anual en hembras adultas y hembras de sobre-año fue baja (0.79 [0.74–0.84; 95% IC] y 0.64 [0.48–0.86] respectivamente), mientras que entre las crías la tasa de supervivencia fue alta (0.66 [0.53–0.81]) en comparación con otras poblaciones de alces. Los porcentajes de preñes y de crías cuates fueron de las mas bajas reportadas para alce (48 y 19% respectivamente), presentando envejecimiento reproductivo entre hembras de hasta tan solo 8 años. El estado de preñez estuvo relacionado con índices de malnutrición agudos (grasa de medula ósea) y malnutrición crónica (índices sanguíneos de condición corporal). La evaluación de cadáveres encontrados de manera oportunista reveló que lo mas seguro es que existieran muy pocos machos maduros ($>$ 5 años de edad) en la población.

El análisis de contenido de proteína en plantas consumidas por el alce y en muestras de heces fecales indicaron que la calidad de la dieta era adecuada para mantener a los alces durante el invierno, pero el elevado contenido de proteína encontrado en muestras fecales de animales capturados que murieron en los siguientes 18 meses pudo ser indicativo de un catabolismo proteico asociado a la malnutrición. El análisis de elementos traza en hígados de alce revelaron una aparente deficiencia en cobre y selenio, pero hubo poca asociación entre las concentraciones de elementos traza y las enfermedades de los alces, su patología o su mortalidad.

El análisis de series temporales de los conteos de alces (1961–2000) indicó que la tasa de crecimiento anual estuvo relacionada negativamente con la temperatura promedio en verano, al haberse incrementado las temperaturas promedio en el invierno y verano en alrededor de 6.8 y 2.1 C, respectivamente durante el periodo de 40 años. Estos cambios pudieron haber incrementado los costos termo-regulatorios de los alces afectando su balance energético. El análisis de series temporales falló en mostrar una relación entre la tasa de crecimiento estimada de la población de alces y la abundancia de alces y/o de venados, indicando que la población no estuvo limitada por la cantidad de alimento a través de la competencia por el mismo. El análisis de viabilidad de población utilizando los datos de los conteos (1961–2000) así como también datos demográficos colectados en este estudio sugiere que la población de alces del noroeste de Minnesota no persistirá más allá de los próximos 50 años. De manera más general concluimos que la distribución sureña del alce se vera restringida en áreas donde las condiciones de hábitat sean marginales y los venados sean abundantes y actúen como hospederos reserva de parásitos.

L'Effet Des Pathogènes, Des Déficiences Nutritionelles Et Du Climat Sur Le Déclin D'Une Population D'Orignaux

RÉSUMÉ

Plusieurs facteurs pourraient être impliqués dans le récent (depuis 1984) déclin du nombre d'orignaux (*Alces alces andersoni*) dans une population au sud de leur aire de distribution au nord-ouest du Minnesota. Ces facteurs pourraient inclure les effets délétères des infections de pathogènes, dont certains sont associés au cerf de virginie (*Odocoileus virginianus*), les effets négatifs des changements climatiques, l'augmentation dans la compétition pour la nourriture avec les cerfs ou les orignaux; la chasse—légal ou illégal, l'augmentation de la prédation par le loup (*Canis lupus*) et par l'ours noir (*Ursus americanus*). Certains facteurs de longue date pourraient avoir contribué au déclin observé chez l'orignal, incluant ceux associés aux habitats précaires et aux déficiences nutritionnelles. La survie et la productivité de femelles et de jeunes orignaux ($n = 152$) ont été évaluées à l'aide de colliers émetteurs au nord-ouest du Minnesota durant les année 1995–2000. La cause de la mort et la présence de pathogènes étaient aussi évaluées en effectuant une nécropsie sur la carcasse des animaux équipés d'un collier émetteur et sur d'autres carcasses trouvées par hasard.

Les inventaires aériens d'orignaux ne corroboraient pas l'hypothèse que la chasse était la cause du déclin des populations étant donné que le niveau de récolte était relativement bas (i.e. environ 15 % tous les deux ans) et que la population augmentait à la suite d'une année de chasse. La majorité des morts observées (jusqu'à 87 % des orignaux avec collier émetteur [$n = 76$]; jusqu'à 65 % des orignaux sans collier émetteur [$n = 84$]) étaient liées à des parasites et maladies infectieuses. Les infections causées par la grande douve américaine (*Fascioloides magna*) semblent entraîner la plus grande proportion des décès et sont aussi une cause importante de maladies du foie, des cavités thoraciques et péritonéales, du sac péricardique et des poumons. Les décès causés par le ver des méninges (*Parelaphostrongylus tenuis*) étaient moins prévalent et se manifestaient par des maladies neurologiques. Plusieurs morts ont été associées à des maladies infectieuses non identifiées, probablement

liées à la malnutrition. La graisse de moelle osseuse était moins présente chez les orignaux morts de cause naturelle que chez ceux morts dans un accident ou d'une cause anthropogénique, corroborant l'hypothèse qu'une malnutrition aiguë contribue à la mortalité des orignaux. Les profils sanguins d'animaux capturés vivants indiquent que ceux morts dans les 18 mois suivant la capture étaient à un niveau de malnutrition chronique.

Le taux de survie annuel moyen pour les femelles adultes (0.79 [0.74–0.84; 95% IC]) et les jeunes de l'année (0.64 [0.48–0.86]) était bas, considérant que le taux de survie était élevé pour les veaux (0.66 [0.53–0.81]), par comparaison avec d'autres populations d'orignaux. Le taux de gestation (48%) et de gémellité (19%) était l'un des plus bas rapportés chez l'orignal, et la sénescence reproductive chez les femelles a été observée dès l'âge de 8 ans. La prédisposition à la gestation a été liée aux index de malnutrition aiguë (graisse de moelle osseuse) et chronique (indice sanguin). Le peu de carcasses de mâles adultes (>5 ans) retrouvés par hasard indique qu'ils sont très peu nombreux dans la population.

L'analyse du contenu protéique des brouts d'orignaux et des échantillons fécaux indique que la qualité de la nourriture disponible était probablement adéquate pour supporter l'hiver, mais le taux plus élevé de protéines fécales retrouvé chez les animaux morts au cours des 18 mois suivants pourrait être indicatif du catabolisme des protéines associé à la malnutrition. L'analyse d'oligoéléments dans le foie révèle des déficiences en cuivre et en sélénium, mais l'association directe entre la concentration des oligoéléments et la santé/mortalité des orignaux était faible.

Des analyses chronologiques sur les décomptes régionaux d'orignaux (1961–2000) ont démontré que la croissance annuelle de la population était négativement liée à la température d'été moyenne, avec des températures l'hiver et l'été augmentant respectivement de 6.8 et 2.1 C pour la période de 40 ans. Ce changement pourrait avoir provoqué une augmentation du coût de thermorégulation et une rupture dans la balance énergétique de l'orignal. L'analyse chronologique n'a cependant pas démontré de relation entre le taux de croissance estimé de la population d'orignaux et l'abondance du cerf de virginie ou de l'orignal, révélant que la limitation de la nourriture par la compétition était peu probable. L'analyse de la viabilité de la population d'orignaux du nord-ouest du Minnesota, utilisant à la fois des données d'inventaire (1961–2000) et des données démographiques amassées au cours de l'étude, suggère que la population devrait s'éteindre au cours de 50 prochaines années. En conclusion, il est fort probable que la portion méridionale de l'aire de distribution de l'orignal soit restreinte dans les habitats limités et où le cerf de virginie est abondant et source réservoir de parasites.

Contents

INTRODUCTION

Parasites and infectious diseases (i.e., pathogens) are increasingly recognized as influencing the distribution, abundance, and dynamics of many animal species and populations. This increased recognition is largely consequent to the recent and extensive degradation of otherwise natural habitats and the resulting disruption of long-term host–pathogen dynamics, as well as the advent of emergent pathogens in new environments (Scott 1988, Schrag and Wiener 1995, Daszak et al. 2000). For example, most models of host–pathogen coevolution suggest that coexistence is achieved by a complex arms race between pathogen exploitation of the host versus host resistance to the pathogen (Price 1980, Ewald 1994). Disruption of this dynamic balance, such as through

Table 1. Potential hypotheses for the recent moose population decline in northwest Minnesota, and evidence for their support. For most hypotheses, we considered that several key predictions needed to be supported for the hypothesis to be upheld. Individual predictions are not necessarily exclusive to a single hypothesis.

Hypothesis	Explanation	Prediction	Support
Pathogens	White-tailed deer parasites and/or disease cause high mortality	Negative relation between moose population growth and deer harvest	No
		Mortality due to pathogens	Yes
		Extensive pathogen-related pathology	Yes
Climate change	Upper thermoneutral limits are frequently exceeded	Temporal changes in climate indices	Yes
		Negative relation between climate indices and moose population growth	Yes
		Mortality due to heat stress	No
Habitat loss and intraspecific competition	Food quantity/quality is inadequate to support population	Evidence for density dependence in population abundance data	No
		Mortality due to starvation	Yes
		Low calf production	Yes
		Indicators of chronic malnutrition	Yes
		Poor food quality	No
Competition with deer	Food quantity/quality is inadequate to support coexistence	Negative relationship between moose population growth and deer harvest	No
		Mortality rate due to starvation	Yes
		Low calf production	Yes
		Indicators of chronic malnutrition	Yes
		Poor food quality	No
Harvest	Hunting causes high mortality	Negative relationship between harvest rate and moose population change	No
		Mortality due to hunting	Yes (low)
		Population recovery following hunting limitation/cessation	No
Predation	Predation causes high mortality	Mortality due to predation	Yes (low)

anthropogenic disturbance affecting pathogen transmission or host immunity, can have marked consequences on natural patterns of host population regulation (Wasserberg et al. 2003, Gillespie et al. 2005). Furthermore, habitat degradation may promote the spread of exotic pathogens that can infect immunologically naïve hosts and potentially cause significant changes to their abundance and population dynamics (Lindström et al. 1994, Roelke-Parker et al. 1996). In fact, in the next decades emerging infectious pathogens are likely to alter the distribution and abundance of numerous animal species and play a major role in shaping patterns of biodiversity and ecosystem health (Daszak et al. 2000, Dobson and Foufopoulos 2001, Williams et al. 2002). Ecologists are challenged to understand host–pathogen dynamics in altered environments to better predict, and perhaps mitigate, potentially devastating changes to the distribution and abundance of animals.

Increased awareness of the role of pathogens in animal populations also is related to prior difficulty in documenting pathogen effects in natural systems (Minchella and Scott 1991, Holmes 1995). Indeed, pathogens can affect host survival or productivity in subtle ways, including through interactions with other limiting factors; this implies that their role in determining host fitness may not always be clearly evident. For example, hosts may experience accelerated onset of malnutrition when exposed to various nematodes or ectoparasites, leading to higher rates of starvation or sublethal nutrition-related conditions (Blackburn et al. 1991, Murray et al. 1997, Merino and Potti 1998, Koski and Scott 2001). It follows that because pathogen effects may be muddled by other limiting factors, quantifying such effects in natural systems can be challenging. Accordingly, to the fullest extent possible researchers should consider both proximate and ultimate causation when assessing the role of pathogens on host populations.

Wild ungulates are influenced by a variety of pathogens known to affect their distribution, abundance, and population dynamics (Gulland 1992, Dobson and Meagher 1996, Jorgensen et al. 1997, Stien et al. 1999, Joly and Messier 2005), yet our understanding of ungulate–pathogen dynamics remains deficient for many ungulate species and systems. By way of example, consider that the recent expansion of white-tailed deer into moose range in North America allegedly contributes to moose mortality and population decline through transmission of deer parasites to moose, an aberrant host species (Whitlaw and Lankester 1994*a*,*b*; Table 1). However, moose occupying habitats colonized by deer also are faced with several other potentially limiting constraints and rigorous evidence supporting the presumed effect of deer parasites on moose populations is scant (Nudds 1990, Schmitz and Nudds 1994, Lankester and Samuel 1998).

Moose occur across the boreal and mixed coniferous–deciduous forest and range into the mixed forest–prairie habitat in central North America (Berg 1971, Karns 1998). Many southern moose populations have declined or gone extinct during the last century (Peterson 1955, Boer 1992*a*, Alexander 1993**,** Vecellio et al. 1993), and although some populations have recovered in recent years, many have remained depressed. It seems likely that although not all moose population declines are necessarily related proximally to the same factor(s), ultimately the influence of anthropogenic disturbance is a common underlying agent. Indeed, although the leading hypothesis explaining southern moose population decline and range constriction remains apparent competition with white-tailed deer, mediated through parasitism,

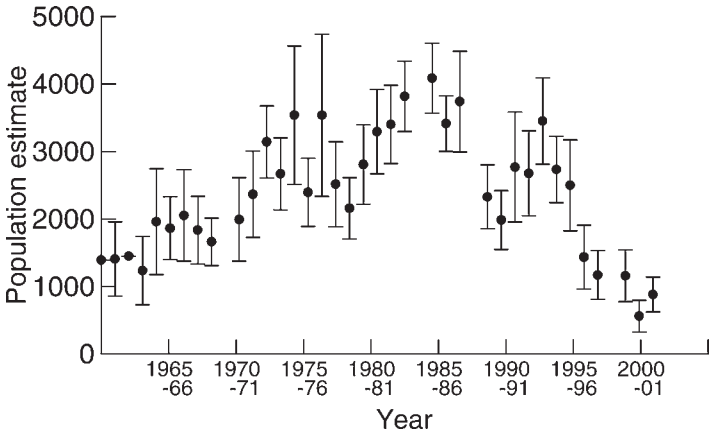

Figure 1. Moose population estimates and 90% confidence intervals for northwest Minnesota, USA, 1960–1961 through 2000–2001.

a number of alternate hypotheses warrant consideration (Table 1). For instance, the alleged primary factor limiting moose distribution at low latitudes is climate (Renecker and Hudson 1986, 1990; Karns 1998), and it is conceivable that recent climate change in North America has shifted northward the thermoneutral zone for this species. Alternatively, habitat loss in areas of high human density may have reduced the carrying capacity for moose and thereby aggravated intraspecific competition for resources. Because deer may compete with moose for food or habitat (Telfer 1970, Peek et al. 1976, Ludewig and Bowyer 1985), deer population expansion could potentially result in high interspecific competition for resources. Also, moose mortality may outweigh productivity in the southern range due to direct human exploitation (e.g., Boer 1992*a,b*; Alexander 1993), or from increased predation through the recovery and expansion of large carnivore populations in areas where prey are highly vulnerable (Berger et al. 2001). Several of the above hypotheses may act concurrently upon a given moose population, either additively or synergistically, and thereby elicit population decline.

In response to a moose population decline in northwestern Minnesota that began in the 1980s and apparently continues to this day (Fig. 1, Minnesota Department of Natural Resources [MNDNR], unpublished data), we initiated a study assessing the potential role of the above factors in the observed numerical decline. After a preliminary assessment, and when several of the hypotheses appeared to be false, we focused our efforts on quantifying the direct and potentially interactive role of the more salient factors on moose population demography. Although this study was largely descriptive, our analyses constitute the most detailed examination of a southern moose population decline to date, and the wide range of perspectives covered herein should be relevant to other declining populations. Yet, we offer the caution that our observational approach precluded fully differentiating between causation and correlation, or between proximate versus ultimate factors acting on the population.

STUDY AREA

The study site was located in northwest Minnesota, which constitutes the southern range limit for moose in central North America (Boer 1998). The region was composed of a mosaic of private farmlands and federal- and state-owned natural areas, hence moose habitat was patchy. We classified our site into 3 study areas based upon site occupancy and movements of radiocollared moose: (1) Agassiz National Wildlife Refuge (ANWR), (2) Red Lake Wildlife Management Area (RLWMA), and (3) agricultural areas (AGR), which included the Thief Lake Wildlife Management Area and the Viking agricultural area (Fig. 2). Radiocollared moose showed minimal interarea migration during the study.

The 249-km^2 ANWR site was covered by marshes (~56%) comprised of cattails (*Typha* spp.), sedges (*Carex* spp.), and the common reed (*Phragmites australis*); lowlands (~22%) with willow (*Salix* spp.), aspen (*Populus* spp.), black spruce (*Picea mariana*), and eastern larch (*Larix laricina*); open water (~10%); and upland forests (~12%, *Quercus* spp.; United States Fish and Wildlife Service [USFWS], ANWR files). Both RLWMA and portions of AGR were located within the Beltrami Island State Forest (BISF). The 2,700-km^2 BISF was covered by lowlands (44%), open marsh (17%), and uplands (39%; Fritts and Mech 1981). The RLWMA comprised 593 km^2 of the BISF; radiocollared moose inhabited this area, as well as tracts of lowland to the west and north and bog to the south and east. The AGR was comparable in size to ANWR and composed primarily of farm fields interspersed with aspen uplands and willow and aspen lowlands. Farm fields were planted mainly with wheat, barley, sugar beets, and bluegrass.

All 3 study areas were inhabited by black bears and gray wolves, potential predators of moose. Black bears appeared to be relatively abundant across the study area (G. Mehmel, MNDNR, personal communication), whereas wolf populations recently increased throughout the region and were common in the ANWR and RLWMA study areas (Fritts and Mech 1981, Chavez 2002). The areas also were occupied by a high density of white-tailed deer and fewer elk (*Cervus elaphus*), both of which are hosts to liver flukes. White-tailed deer are the only natural hosts to meningeal worm. The intermediate hosts of both *F. magna* and *P. tenuis* are gastropod snails (Lankester 2001, Pybus 2001), and our assessment of snail abundance and *P. tenuis* prevalence in the snail population revealed intermediate–high prevalence in the study site (E. W. Cox, University of Idaho, unpublished data). Winter ticks (*Dermacentor albipictus*), which may reduce moose fitness (Samuel and Baker 1979, Glines and Samuel 1989), also were present in this region (MNDNR, unpublished data), but failed to present a prevalent cause of death during our study. Mammalian species that potentially could compete for food with moose included snowshoe hare (*Lepus americanus*) and beaver (*Castor canadensis*).

METHODS

Population Estimation

Aerial Moose Population Estimation.—The moose population in northwest Minnesota has been surveyed annually since 1960–1961 from fixed-wing aircraft. The MNDNR conducts surveys throughout the northwest region, whereas USFWS conducts surveys on ANWR. Sightability correction factors (Gasaway et al. 1986) were estimated with the use of survey techniques by both agencies beginning in 1983. Annual moose

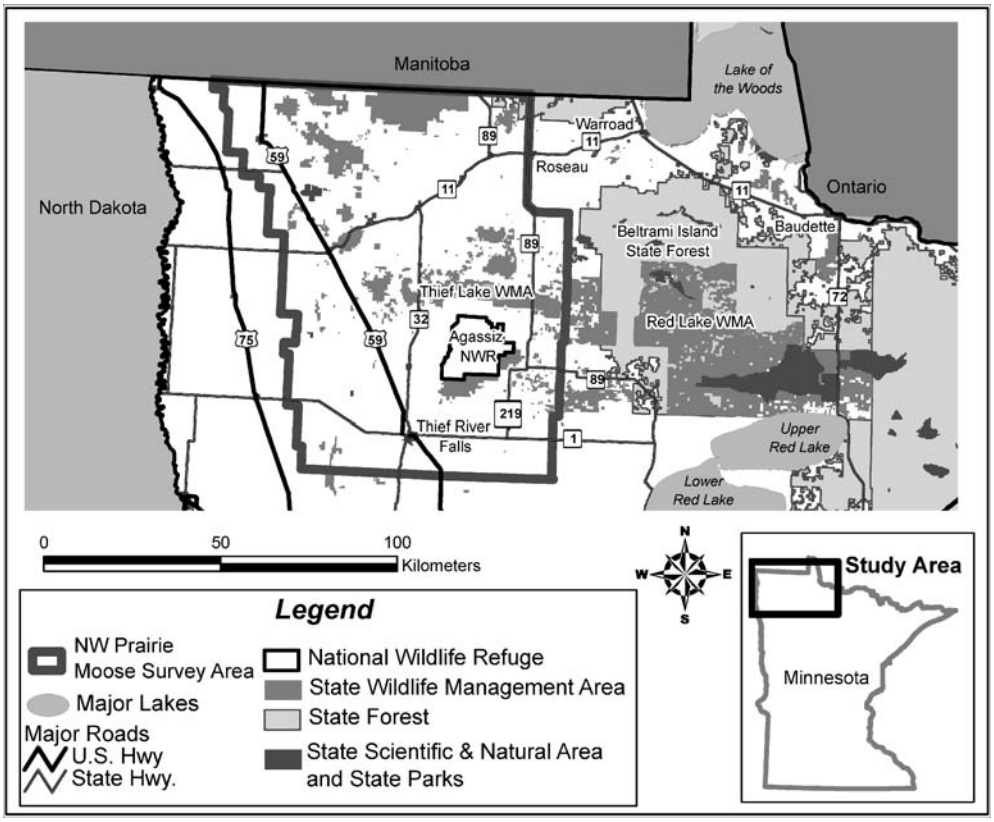

Figure 2. Study areas in northwest Minnesota, USA, where causes of moose population decline were investigated during 1995–2000.

population estimates were calculated using 90% confidence intervals for the northwest prairie region. Moose were classified as females, calves, males, or unidentified beginning in 1979 (MNDNR, unpublished data). Classifications of females with twin or single calves were initiated in 1983.

Beginning with the winter of 1982–1983, the MNDNR used 2 different survey areas: Northwest Prairie (NWP) and Northwest Forest (NWF). The NWP included most of northwest Minnesota east of Highway 54; the ANWR and AGR study areas represented a subset of this MNDNR survey area. The NWF includes the area east of Highway 54 and included the RLWMA study area. We use the NWP survey data for analyses of count data because this survey area had a longer history of data, and surveys were conducted more frequently than those in NWF. Either-sex moose hunting seasons were closed in 1922 and reopened in 1971 by permit only. Seasons were held in alternate years through 1993, and then annually through 1996, when the season was closed.

Deer Population Trend.—The MNDNR uses antlered male harvest data to index deer population trends (Lenarz and McAninch 1994). Deer hunting seasons in the study area have consistently been 9 days long since the mid 1970s, with a legal deer being any deer with antlers >7.6 cm long and a bag limit being one animal/person. The moose study area encompassed 8 deer harvest permit areas (i.e., areas 202, 203, 205–208, 211, and 404); we used pooled data from these areas to index deer population trends.

Capture and Radiotelemetry

Moose were captured by net gun from a helicopter and equipped with mortality-sensitive transmitters by Helicopter Wildlife Management (Salt Lake City, Ut., USA) or Outbound Aviation (Calgary, Alberta, Canada), during spring 1995 and winters 1996–1998. Neonate calves were caught by hand or net gunned with the use of airboats or helicopters during springs 1995–1998. Calves were equipped with mortality-sensitive ear-tag transmitters or expandable radiocollars (Ballard et al. 1979). We monitored moose for survival daily or on alternate days from the ground, although some relatively inaccessible animals were checked only once every 5–10 days, usually by fixed-wing aircraft. We also located radiocollared moose during the day 1–4 times monthly by fixed-wing aircraft, helicopter, ground triangulation, and ground homing (White and Garrott 1990).

Productivity and Recruitment

We evaluated moose productivity by measuring pregnancy rates, twinning rates, and calf and yearling survival. Radioimmunoassay for fecal progestagens provided the basis for determining pregnancy status of radiocollared females (Monfort et al. 1993, Schwartz et al. 1995), and these data were verified by radioimmunoassay for serum progesterone (Smithsonian Institution, Conservation and Research Center, Front Royal, Va., USA) and pregnancy-specific protein B (University of Idaho, Moscow, Id., USA, Stephenson et al. 1995; E. W. Cox, unpublished data). Paired fecal and serum samples were assayed according to methods outlined in Monfort et al. (1993) and Schwartz et al. (1995). We

verified presence or absence of calves each spring during parturition.

We collected blood and feces from captured and recaptured adult moose; feces from moose not recaptured were obtained via radiotelemetry homing during winter. During these activities we used changes in signal gain and angle to determine proximity and subsequent departure of targeted moose; we used sightings, tracks in snow, and pellet size to ascertain the identity of the correct moose. Feces were not collected if other moose tracks were found within a 200–500-m radius except when the targeted female was accompanied by offspring and the feces could be identified as belonging to the female.

We checked for the presence of neonate calves with females via fixed-wing or rotary wing aircraft within 1 week to 1 month after birth. We rated reliability of sightings subjectively when determining if a neonate calf was present; females testing positively for pregnancy were checked at least twice if a neonate was not observed during the first sighting. Subsequent effort depended on our subjective assessment of visibility during previous flights for a given animal. A female had to be standing (i.e., not running) in an open area with little cover nearby during several sightings before she was considered as lacking a calf.

Age Structure

We estimated age structure of yearlings and adults using tooth cementum annuli counts obtained through carcass collection (Sergeant and Pimlott 1959). For radiocollared individuals we back-calculated age estimates from necropsied carcasses to estimate age at first capture. We considered this method to be less biased than age at death because it is a better representation of the age structure of the living population. We compared these ages to those for carcasses from nonradioed yearlings and adults that we recovered opportunistically. Because we failed to find differences in the age structure of radioed versus nonradioed groups, or between animals dying of natural versus unnatural causes, we pooled all cementum annuli samples to describe the overall age structure of the population. However, it is important to note that our sample size of males was small, and therefore it is arguable that male age distribution in the population was not representative. Also, it is relevant to consider that because several radiomonitored animals were never recovered and thus could not be aged precisely through tooth cementum analysis, the sample of aged moose could be biased in favor of either those that died or those that were initially marked as calves and monitored thereafter.

Assessment of Mortality Factors

We examined moose death sites for evidence of predators (Ballard et al. 1979, Roffe et al. 1994), signs of behavior indicating meningeal worm infection (Anderson and Lankester 1974), and other indicators of cause of death (Ballard et al. 1979). Gross necropsies followed Nettles' (1981) guidelines whenever time, equipment, and facilities permitted. We examined carcasses for pathology and then collected fresh or formalin-preserved tissues and fecal samples. Findings were tape-recorded as observed during field evaluation and later transcribed. We sent fresh and formalin-preserved tissues to diagnostic laboratories (i.e., National Wildlife Health Center, Madison, Wis., USA; North Dakota State University Veterinary Diagnostic Laboratory, Fargo, N.D., USA; or University of Minnesota Veterinary Diagnostic Laboratory, St. Paul, Minn.) for pathogen examination. Road kills, other accidental deaths, illegally shot animals, and naturally dead or euthanized nonradioed animals received similar treatment.

We classified proximate causes of death into a variety of categories based on evidence at the death site and ancillary information from carcass necropsy. Causes of death included possible or probable liver fluke, probable meningeal worm, probable disease or starvation (usually involved animals that were not recovered immediately after death and that were not clearly killed by other major causes), predation, vehicle collision, poaching, and unknown (no clear evidence of cause of death, but in most cases predation, vehicle collision, and poaching were excluded). We classified deaths as being proximally due to parasitism or disease when the alleged causative pathogen caused abnormal and extensive pathology, and when there was no other obvious cause of death. Non-radiocollared moose also died of starvation and natural accidents (e.g., drowning). We also recategorized carcasses as representing animals that were "dead" versus "live" based on whether the mortality was natural or "accidental" (e.g., drowning, vehicle collision, poaching), respectively. Animals dying of "accidental" causes were presumed to be representative of the live population.

Parasites and Diseases

We tallied the number of flukes present in moose livers by serially sectioning the liver at 3–10-mm intervals, agitating the slices in clean water 3 times, and sieving the washings. We cross-hatched any liver section >5 mm and attempted to recover whole flukes by applying pressure to the liver and extracting flukes with tweezers. After December 1997, we also rinsed the thoracic cavities of most dead moose, collecting flukes on a 1-mm mesh sieve. Before this time, thoracic cavities received cursory examinations for flukes. We classified moose as dying from liver flukes if there were signs of severe pathological damage to tissues and organs and no other overt cause of death was determined. If serious pathological damage was absent but liver flukes were abundant, we assigned those individuals to possible or probable liver fluke death, assuming that no other mortality agents were detected.

We examined moose heads for meningeal worms by removing the dorsal portion of the cranium, carefully slicing the dura mater laterally, extracting the brain, and examining all exposed surfaces, particularly the sagittal venus sinus (Anderson 1965). Death due to meningeal worm was considered if ≥ 1 worm was present and no other cause of death was apparent. Moose brains and usually spinal cords were also examined histologically for meningitis. We also recorded incidence of a variety of pathologies in moose carcasses, although such work was conducted exclusively on radiocollared animals and was conditional on carcass condition at the time of retrieval.

We assayed moose sera for antibodies to *Leptospira interrogans* serovar *bratislava*, *canicola*, *grippotyphosa*, *hardjo*, *icterohaemorrhagiae*, and *pomona* using the microscopic agglutination test (Cole et al. 1973), and for *Brucella abortus* using the slide agglutination test at the Washington State Veterinary Diagnostics Laboratory (Pullman, Wash., USA). Sequential serologic tests on an animal

are needed to distinguish reliably between past and active leptospirosis (Thorne 1982); hence, we considered titers of 1:100 to 1:500 as indicative of seropositivity only (Blood et al. 1983).

Body Condition

Blood Serum Analyses.—We collected blood from live-captured moose (1995–1998) to index the general condition of live animals. Laboratory analyses were performed by Dakota Heartland Health System (Fargo, N.D.) and followed standard protocols (Franzmann and LeResche 1978, Franzmann 1985, Franzmann et al. 1987). Blood analyses provided counts of white blood cells, red blood cells (RBC), percent hemoglobin (HGB), percent hematocrit (HCT), average red blood cell size (MCV), hemoglobin amount relative to size of red blood cell (MCH), hemoglobin amount relative to size of cell/red blood cell (MCHC), red blood cell distribution width, platelet counts, mature neutrophils, lymphocytes, monocytes, and eosinophils. The first 7 assays were conducted on the full complement of serum samples ($n = 80$) whereas the remaining assays were carried out on a subsample ($n = 20$–65); when analysis of the subsampled assays failed to reveal strong patterns, we focused our analysis on the main 7 assays. We evaluated moose blood profiles in relation to pregnancy status and probability of survival during the 18 months following the sampling event. Blood profiles are widely recognized in human and veterinary medicine as providing a general index of condition and physiological functioning (Tizard 1992, Duncan et al. 1994); thus, blood results herein were used as 1 of several indices of moose condition.

Bone-Marrow Fat.—We assayed moose femurs for fat content to estimate nutritional status at time of death (Neiland 1970, Mech and DelGiudice 1985). A plug of marrow was removed from the middle third of each femur, weighed to the nearest milligram, and dried until marrow mass stabilized. We calculated percent marrow fat as weight of dried marrow divided by mass of the wet marrow (Neiland 1970). We estimated the volume of the cross-section of the femur from which the marrow was obtained by filling the upright cross-section with water from a burette, which allowed a correction for dehydration (Peterson et al. 1982). We estimated percent marrow fat from the following equation: marrow fat (%) = 3.7 + 108 (dry mass of marrow volume of cross section). Recognizing that marrow fat is the last fat deposit to be fully mobilized before starvation (Mech and DelGiudice 1985), we considered animals with low (i.e., <30%) bone-marrow fat (BMF) as suffering from acute malnutrition (Franzmann and Arneson 1976, Mech and DelGiudice 1985, Ballard 1995).

Winter Browse and Fecal Pellets.—We assessed basic nutritional quality of winter browse during January–February of winters 1997–1998, 1998–1999, and 1999–2000 by walking to locations of individual radiotagged moose and sampling current annual growth of major moose browse species (i.e., alder, aspen, dogwood [*Cornus* spp.], and willow) along tracks in snow, usually from freshly browsed plants. For comparison, we also collected plant samples opportunistically (i.e., randomly) from areas lacking fresh moose sign. Forage samples were ground with a Wiley mill until particles passed through a 1-mm screen, and then analyzed with the sequential detergent method (Goering and Van Soest 1970, Mould and Robbins 1981). We estimated percent acid detergent fiber (ADF), percent neutral detergent fiber (NDF), and percent crude protein (P) (P = 6.25 × percent nitrogen [N]; Robbins 1993).

Fecal samples collected during January–March 1998 and 1999 also served to assess protein content of ingested food; percent fecal nitrogen was determined by mass spectrometry (University of Idaho Stable Isotope Laboratory, Moscow, Id.) and converted to P. Although our analyses of browse and fecal pellets were inadequate to provide a comprehensive assessment of plant quality in northwest Minnesota, these data serve specifically to evaluate the possibility that plant protein was limited during winter. It follows that our sample sizes and sampling regime also were inadequate to rigorously address questions related to moose browse selection.

Trace Elements.—To evaluate potential nutritional deficiencies in moose habitat in northwest Minnesota, we subjected liver-tissue samples (approx. 25 g) from dead moose to trace element analyses (Custer et al. 2004). Before extracting the sample, all instruments were washed without soap in doubly deionized water, air dried, and immersed repeatedly in other parts of the same liver. Samples were placed in a preweighed chemically clean jar, weighed, and frozen at −20 C. We analyzed moose livers for concentrations of 19 macrominerals and trace elements (i.e., trace elements). We chose specific trace elements for analysis on the basis of reports of pathology associated with their deficiency or toxicity in domestic or wild ungulates (Church and Pond 1978, Church 1980, Robbins 1993). Samples were freeze-dried, weighed, and then homogenized in a blender. Subsamples were digested in stages with heat and nitric-perchloric acid and then analyzed for selenium (Se) and arsenic by graphite furnace atomic absorption spectrophotometry, and for aluminum, barium, beryllium, boron, cadmium, chromium, copper (Cu), iron (Fe), lead, magnesium (Mg), manganese (Mn), molybdenum (Mo), nickel, strontium, vanadium, and zinc by inductively coupled plasma–atomic emission spectrophotometry (Custer et al. 2004). Separate subsamples were digested by nitric acid reflux and analyzed for total mercury (Hg) by cold vapor atomic absorption spectrophotometry. We report concentrations of trace elements on a dry-mass basis; approximate wet-mass values can be calculated by using the average percent moisture of 70.9% in livers from this study. Nine trace elements were detected in sufficiently high concentrations to permit analysis with the use of parametric statistics, and the remainder of trace elements are reported according to detectability thresholds, for descriptive purposes. A subsample ($n = 81$) of livers from this study was analyzed previously to determine baseline concentrations in comparison with those from other moose populations (Custer et al. 2004); our present results include additional samples ($n = 26$) and evaluate specifically the relationship between trace element composition and moose pathology and cause of death.

Data Analysis

Despite the observational foundation underlying this study, we sought to apply a hypothetico-deductive approach in our data analysis and interpretation. Many statistical analyses followed a standard model selection framework (Burnham and Anderson 2002), but in cases where specific hypotheses were tested frequentist methods often were used. We aimed to conduct investigation that emphasized ecology rather than a specific

analytical paradigm, and therefore we availed ourselves to a range of statistical methods to be used as we deemed appropriate (Guthery et al. 2005).

Parasites and Bone-Marrow Fat.—The statistical distribution of liver fluke numbers in moose was skewed, and fluke prevalence and intensity were analyzed separately, with intensity being subject to log transformation prior to analysis. We analyzed prevalence of fluke, meningeal worm, *Echinococcus*, and *Sarcosystis*, via chi-square or logistic regression (Hosmer and Lemeshow 1989), and we analyzed liver fluke intensity via analysis of variance (ANOVA; Sokal and Rohlf 1995). Percent BMF, corrected for desiccation, was transformed by arcsin–square root (Krebs 1999) prior to analysis. We examined age-structure comparisons between various groupings in the study population using paired *t*-tests. We examined factors determining pregnancy status with the use of logistic regression (Hosmer and Lemeshow 1989).

Survival.—Radiotelemetry allowed for estimation of moose survival and mortality rates, with delayed entry for animals recruited after the study was initiated. Animals that died of capture-related causes ($n = 7$), as well as those whose radiosignal was lost ($n = 31$) or that survived until the end of the study ($n = 36$), were right-censored. We partitioned telemetry data into 6 'years' (1995–2000), and 4 'seasons' related to biological events relevant to adult females: parturition and lactation (summer: 16 May–31 Jul), rut (autumn: 1 Aug–30 Nov), early winter pregnancy (winter: 1 Dec–28 Feb), and late winter pregnancy (spring: 1 Mar–15 May). Year and season were made available for inclusion in mortality risk models as a single variable designed to capture a continuous trend, and as a set of dummy variables representing year or season individually. Other variables under consideration as possible determinants of moose mortality included study area, gender, and age (categorical variable representing calves, yearlings, and adults; dummy variables isolating calves and adults). Relevant variables were entered into the data set in the form of time-dependent covariates (Cleves et al. 2003) and the influence of age was further analyzed for a subsample of animals whose exact age was known (i.e., first captured as calves or aged post-mortem via tooth cementum annuli analysis). This latter analysis enabled assessment of the importance of age-specific survival rates within the adult cohort (i.e., continuous age variable) but could be biased because animals that were never aged were excluded. We also considered pregnancy status and calf presence (as a current-time and a time-delayed [$t - 1$] variable) as potential determinants of moose mortality risk. Annual, seasonal, and cohort-specific survival rates, and cause-specific mortality rates (and their corresponding confidence intervals; Heisey and Fuller 1985) were calculated for descriptive purposes and for parametrizing the population transition matrix. We determined that the assumption of constant survival probability was met during our biological seasons and that Heisey–Fuller survival rates were comparable to those generated using Kaplan–Meier calculations (Hougaard 2000, Murray 2006).

We used Cox proportional hazard (CPH) models (Hougaard 2000, Therneau and Grambsch 2000) to analyze determinants of moose survival. Assuming $h_i(t)$ is the instantaneous probability of death for an individual i at time t (called a hazard function), and that the ith individual is associated with a covariate vector $x_I = (x_{i1}, x_{i2}, \ldots, x_{ip})$, the CPH model is:

$$h_i(t) = h_0 \exp(\beta_1 x_{i1} + \beta_2 x_{i2} + \cdots + \beta_p x_{ip})$$

where $h_0(t)$ represents the baseline hazard and corresponds to the hazard function of an individual with covariate vector $x_I = (0, 0, \ldots, 0)$, and β is an unknown parameter. The CPH model is semiparametric because the distribution of lifetimes and the baseline hazard function are unspecified. It follows that $h_i(t)$ and $h_j(t)$ differ only because $x_{i1} = x_{j1} + 1$, so that the hazard ratio $h_i(t)/h_j(t) = \exp(\beta_1)$ is time-independent. It follows that $h_i(t)$ and $h_j(t)$ are proportional through time and differ only multiplicatively. The assumption of proportional hazards is critical to model fit and was determined by model reestimation to verify that the coefficient on the squared linear predictor was nonsignificant (Cleves et al. 2003), and also by plotting scaled Schonfeld residuals versus survival times and computing a chi-square significance test for the resulting correlation (Hougaard, 2000, Therneau and Grambsch 2000). When the proportional hazards assumption failed to be satisfied, a stratified CPH model was estimated that assumed different baseline hazard functions for individuals across strata. Under the stratified CPH model, proportionality of hazard functions was assumed among individuals in the same stratum only. Although desirable from the standpoint of controlling for different baseline hazard rates, a drawback of the stratified CPH model is that the relationship between hazard and the variable used to define the stratum cannot be estimated directly. In this case, differences between strata were evaluated using log-rank tests (Hosmer and Lemeshow 1999). We compared CPH models using standard model selection and multimodel inferencing procedures (Burnham and Anderson 2002). Specifically, for candidate models we calculated Akaike's Information Criterion corrected for sample size (AIC_c), AIC_c differences (Δ_i), and AIC_c weights (w_i) to guide model selection. Interaction terms were added to later models only (Hosmer and Lemeshow 1999) and variables that were not complete for all individuals (e.g., reproduction and continuous age data) were subject to a restricted survival analysis.

Body Condition and Trace Elements.—Serum and trace element indices may exhibit correlated, synergistic, or interactive effects (Osman and Sykes 1989, Robbins 1993), and we used principal-components analysis (PCA) to reduce the number of variables under consideration. We used multivariate analysis of variance (MANOVA; Hair et al. 1998) to examine the influence of age and gender on serum and trace-element PCA factors. For serum, the 2 PCA factors also were subject to logistic regression against pregnancy status at the time of capture and survival status over the next 18 months. We used logistic regression for trace elements to assess the relationship between PCA factors and moose disease, pregnancy status at time of death, pathology, and cause of death. Logistic regression models were selected via model selection procedures involving Δ_i and w_i (Burnham and Anderson 2002).

Climatic Influence.—Through linear regression, we first assessed the potential temporal change in climate indices in northwest Minnesota during 1960–2001. Indices were selected a priori and included mean monthly winter (Jan–Feb) temperature and snowfall, mean monthly summer (Jul–Aug) temperature and

precipitation, dates of last spring freeze and first autumn freeze, and annual duration of growing season (days between last spring freeze and first autumn freeze). We also included indices for exceedance in the upper critical temperatures for moose during winter (Mar temperature >−5 C; Schwartz and Renecker 1998) and summer (Sep temperature >14 C; Renecker and Hudson 1986, 1990). We developed dummy codes to identify years according to whether the above thresholds had been breached. We excluded an index of snow severity (snow depth >60 cm; Renecker and Schwartz 1998) because this threshold was exceeded during a single year. Daily temperature and precipitation values were obtained from the State Climatology Office for Minnesota for a permanent weather station in ANWR.

The dependent variable in moose population time-series analyses was the annual rate of population change, $(\ln[N_{t+1}/N_t])$. The log-transformed rate of change is an appropriate metric for such analyses because it typically exhibits a relatively symmetric error distribution and can be readily analyzed via linear models (Royama 1992). Although moose survey data across the time series likely included temporal shifts due to sampling bias (Messier 1995, Lenarz 1998), we opted to use original rather than smoothed survey data because the latter transformation could obfuscate inter-annual variability in moose population rate of change (McRoberts et al. 1995). We developed general linear models (Sokal and Rohlf 1995) to test the hypothesis that moose population change was related to unfavorable climate, moose abundance, moose harvest rate, and deer (male) harvest rate. Because of the notable disparity in moose population trends before versus after the 1984–1985 population peak (Fig. 1), and because deer harvest records only were available during the post-1984–1985 time segment, we built 2 sets of models: one for the entire 41-year time series and the second for the post 1984–1985 years only. The post 1984–1985 time series was notably short (17 yr) and therefore was subject to limited statistical power (see Gibbs 2000).

We used the first-order autocorrelation (ACF) statistic to assess independence between successive observations. Although levels of autocorrelation were low for all time series, we adopted a conservative approach by applying a correction factor $(df = N[(1 - a_1a_2)/(1 + a_1a_2)])$, where N is the number of paired samples, and a_1 and a_2 are the degree of first-order autocorrelation in the dependent and independent variables, respectively (Bartlett 1946, Patterson and Power 2002). Cross-correlation function analysis served to identify appropriate lags between dependent and independent variables, and although few significant lags were detected, ungulate responses to weather conditions are potentially delayed by several years (Mech et al. 1987, McRoberts et al. 1995, Post and Stenseth 1998). Accordingly, we made available in models all climatic variables lagged by up to $t - 3$ years, and we also evaluated the potential for cumulative weather effects by using summed seasonal weather data for the previous 2 and 3 years. We evaluated main-effects–only models using model selection (Burnham and Anderson 2002), and we evaluated interaction terms for the better models only.

Population Viability Analyses

Count-Based Projection.—We examined the likelihood of moose population persistence using survey data (1960–1961 to 2000–2001). Initially, we fit density-independent (exponential), linear density-dependent (Ricker equation), nonlinear density-dependent (theta-logistic equation), and inversely density-dependent (Allee effects models) to moose population growth rate with the use of least-squares regression (Morris and Doak 2002). We included population size at t, $t - 1$, and $t - 2$ to examine both current-time and time-delayed population regulation, and we used model selection to evaluate model fit (Burnham and Anderson 2002). Time-delayed theta-logistic models ultimately were excluded from model selection because they produced unrealistic parameter estimates or failed to converge during model fitting (Sibly et al. 2005). Inverse density-dependent effects models were excluded because of unrealistic parameter estimates and low inference due to overparametrization. Model selection revealed that density-independent ($w_i = 0.32$), density-dependent at $t - 2$ ($w_i = 0.25$), and density-dependent at t ($w_i = 0.24$) models were indistinguishable (all $\Delta AIC_c < 0.52$), whereas density-dependent at $t-1$ and theta-logistic at t models provided a poorer fit ($\Delta AIC_c > 2.17$). We chose a density-independent model to project the moose population because of its qualitatively better fit and greater parsimony, and also because separate tests (Dennis and Taper 1994) failed to provide compelling evidence for the occurrence of density dependence at the moose population densities under consideration. Log mean annual rate of change (μ) and its variance (σ^2) were determined with the use of regression-based methods (Dennis et al. 1991). The regression line for $\ln(N_{t+1}/N_t)$ versus the intersurvey interval was forced through the origin, with μ being estimated by the slope and σ^2 by the residual mean square (Dennis et al. 1991). Three missing count data were adjusted (Dennis et al. 1991).

We compared estimated μ during pre- (1960–1961 to 1984–1985) versus post- (1984–1985 to 2000–2001) population peak periods using a 2-tailed t-test; pre- versus postpeak differences in σ^2 were determined with the use of a 2-tailed F-test for homogeneity of variance (Snedecor and Cochran 1980). Notably, neither the complete time series nor the 2 segments exhibited significant first-order autocorrelation. Conservatively, we used μ and σ^2 estimated for the complete time series, and the corresponding cumulative distribution function (CDF), to project the northwest Minnesota moose population. We considered the probability that the population would breach 4 quasiextinction thresholds (400, 200, 100, and 50 moose) in the next 50 years. The CDF also served to determine the conditional time to extinction (Dennis et al. 1991, Morris and Doak 2002).

Understanding that natural populations experience complex dynamics that are difficult to characterize using phenomenological models derived from count-based data (White 2000, Boyce 2001), the present population viability analysis (PVA) serves to provide qualitative insights regarding the predicted future trend in the moose population. Our approach assumes that counts are representative of the surveyed moose population, annual variation in counts is biologically based, and that major catastrophes or bonanzas are lacking during the survey period. We considered that each of these assumptions was largely upheld by the data set for northwest Minnesota, but we also recognize that such long-term count data often are plagued by a general lack of accuracy (Lenarz 1998, Meier and Fagan 2000, Yoccoz et al. 2001). Because we

suspected that annual variability in survey data could be due mostly to measurement error rather than process error, we suggest that the benefits of analyzing a single 40-year time series to estimate population parameters should outweigh any costs of modest assumption violation.

Demographic Projection.—We used vital rates estimated during our demographic study to develop a stage-specific Lefkovich transition matrix for the moose population (Caswell 2001). Because moose from our 3 study areas had comparable vital rates, and limited movement between areas was observed, we considered a single continuous moose population for modeling purposes. We parameterized the transition matrix with survival and productivity estimates for females from 3 stage classes (i.e., calf, yearling, adult); we chose a stage-specific rather than age-specific matrix because roughly 50% of monitored females were not aged precisely and thus vital rates could have been biased. We calculated fecundity rates by the product of pregnancy and fertility rates for the population; because we lacked data on twinning rates among yearling mothers, we used the rate for adults and assumed newborn sex-ratio parity. The dominant eigenvalue of the Lefkovich matrix was the annual finite rate of change (λ) of the population under the above conditions (Caswell 2001). Elasticity analyses served to determine the response of λ to variability in each nonzero element of the transition matrix (Caswell 2001, Morris and Doak 2002).

The assumption that moose vital rates remained stationary over time was implicit in our deterministic analysis (Taylor 1995, Boyce 2001); this assumption was relaxed during stochastic simulations by adding a variance component corresponding to the observed interannual variability in stage-specific survival and pregnancy rates. Simulations assumed density-independent population growth because of the low moose density estimates for northwestern Minnesota (Lenarz 1998) and our inability to detect conclusive evidence of density dependence. Demographic stochasticity was modeled via Monte Carlo simulations (Akçakaya et al. 1999), and results from simulations excluding demographic stochasticity were qualitatively similar to those reported herein. The population was projected forward 50 years with the use of half the 2000–2001 population estimate from aerial survey (442 F) as the starting population size. We used 500 simulations and 4 quasiextinction thresholds (i.e., 200, 100, 50, and 25 F) that corresponded to the 4 thresholds used in the aforementioned count-based population projection, assuming sex-ratio parity. Simulations were conducted with the use of RAMAS Geographic Information System (Applied Biomathematics, Setauket, New York).

We explored the potential moose population response to modest reductions in the demographic costs of pathogens or other limiting factors by calculating the dominant eigenvalue for the transition matrix and the female population size at $t = 50$, after minor changes in the vital rates. Specifically, we reduced the number of mortalities across all stage classes by 5%, 10%, or 20%, we increased pregnancy rates among yearlings and females by 5%, 10%, or 20%, and we reduced mortality and increased pregnancy rates simultaneously by 5%, 10%, or 20% each. This exercise assumed that management-induced changes in vital rates did not lead to compensation by other sources of demographic limitation (e.g., predation, malnutrition), and it sought to determine parameter estimates promoting a stationary or increasing moose population trend. This analysis was restricted to deterministic projections because of the unknown influence of hypothetical changes in vital rates on their interannual variability, and the fact that deterministic sensitivity values typically approach those obtained from stochastic models (Caswell 2001).

RESULTS

Moose Population Trends

Aerial survey results suggested that the northwest Minnesota moose population began a major decline beginning in 1984–1985 (Fig. 1). Although not all portions of the region experienced a synchronous decline, and the magnitude of the decline also varied spatially, moose numbers in most regions generally began to decline within 3–5 years of 1984–1985 (MNDNR, unpublished data). During the span of 10 years, regional moose numbers declined to about 35% of the 1984–1985 peak, and remained low thereafter. Currently, moose populations in northwest Minnesota continue to be depressed, with the most recent estimate (Jan 2003) being particularly low (237 $\pm$ 73 [$\pm$90% CI] moose, corresponding to $<$0.1 moose/km^2; Minnesota Department of Natural Resources files, Grand Rapids, Minn.). However, we suspect that this estimate may be biased low because of particularly poor survey conditions during winter 2003.

Both calf:female and male:female ratios remained relatively high during the mid-winter survey as the moose population declined (Fig. 3). Calf:female ratios ranged from 54 to 94 calves:100 females from 1984–1985 through 1996–1997, and only in recent years did calf:female ratios decline below 0.50. Male:female ratios were relatively high, ranging from 57–94 males:100 females (Fig. 3). After the hunting season ended in 1996, male:female ratios increased to 129–165 males:100 females. However, despite these favorable ratios, our population sex and age structure results suggested that males were overrepresented in the surveyed population.

Legal Harvest

Historically, annual moose harvests increased during the 1970s and peaked at about 737 animals in 1983–1984 (Fig. 4). Following the onset of the moose population decline, the hunting season was closed on portions of RLWMA beginning in 1985 and on ANWR in 1994, with the number of permits for the remainder of northwest Minnesota being reduced from 262 in 1994 to 39 in 1996. Thereafter, the hunting season was closed. During the predecline period, moose harvest rates (i.e., percent of the estimated moose population that was harvested) averaged 15.5 $\pm$ 0.02% ($n = 7$) compared to 15.1 $\pm$ 0.02% ($n = 8$) postdecline (MNDNR, unpublished data; Fig. 4). The above harvest rates might not have been sustainable except that hunting seasons occurred during alternate years from 1971–1993, and changes in moose population numbers were positive during most years during this period (Fig. 2). Thus, because of the observed population increase during many years with harvest, and the absence of population recovery after hunting closure after 1996, we ruled out legal hunting as an important factor in the moose population decline. This finding was later corroborated by time-series analyses on the determinants of moose population growth.

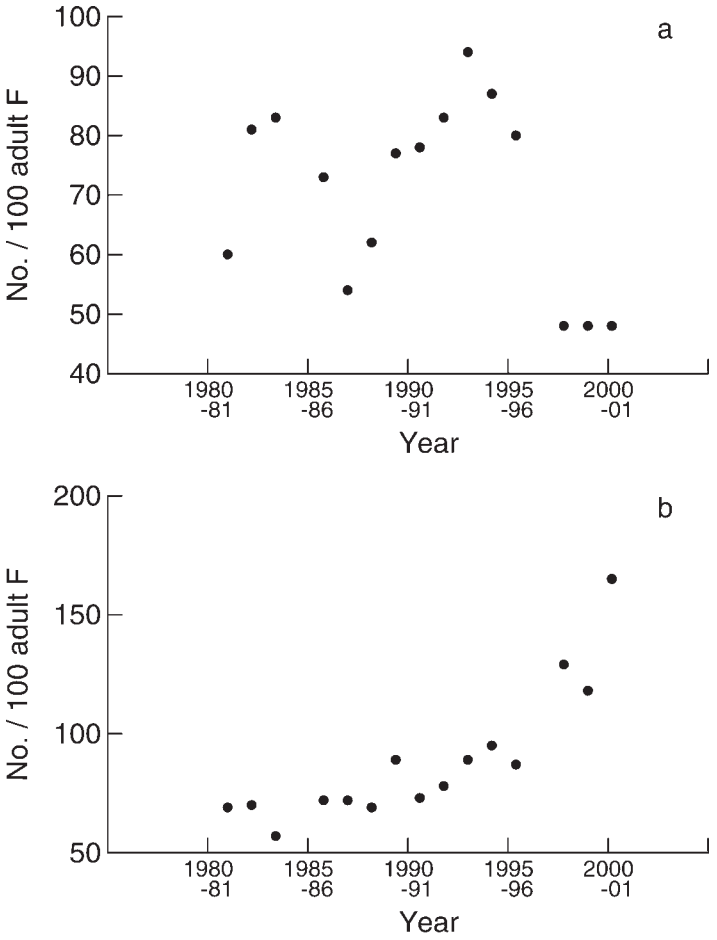

Figure 3. Calves (a) and adult males per 100 females (b) counted in northwest Minnesota, USA, during 1984–1985 through 2000–2001.

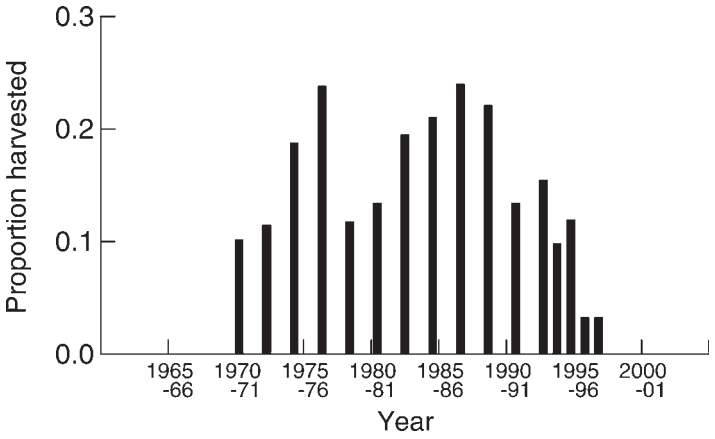

Figure 4. Proportion of estimated moose population that was harvested annually in northwest Minnesota, USA, from 1971–1972 through 2000–2001. Harvest was closed prior to 1971.

Cause of Death

During May 1995–July 1998, we captured and radiomarked 152 individual moose in the study site (ANWR: $n = 77$, AGR: $n = 33$, RLWMA: $n = 42$), and radiomarked animals were monitored intensively until the end of July 2000. We recorded 76 mortalities of radiomarked moose, and necropsies and clinical tests revealed that the majority of moose deaths were related to pathology associated with parasitism, infectious disease, and possibly starvation. Dying moose often showed severe signs of lethargy, emaciation, and morbidity (Fig. 5). Specifically, necropsies revealed that liver flukes likely were responsible for 21–32% of deaths among radiocollared animals, and for an additional 5% meningeal worms probably were implicated (Table 2). We found that 25% of moose deaths probably were attributable to pathology from infectious disease or starvation, and an additional 25% died from unknown causes (Table 2). Most animals succumbing to unknown causes probably also died from parasitism, infectious disease, and/or starvation, but due to carcass condition at time of recovery or equivocal necropsy results, we have classified such animals separately. However, it is important to note that the paucity of any predator or other sign at the site of death for these animals strongly suggests that they were unlikely to have succumbed to traumatic causes. In fact, with respect to the predation hypothesis under consideration (Table 1), only 7% of radiocollared moose were confirmed as dying from predation, implying that this factor was unlikely to be a causative agent behind the decline. Direct anthropogenic causes of mortality also were uncommon (Table 2).

We recovered an additional 84 nonradioed moose carcasses; these samples further substantiated earlier results implicating pathogens in the majority of moose mortalities (Table 2). In general, the percentages associated with different causes of death were comparable between the radiomarked and non-radiomarked samples, although the higher prevalence of meningeal worm deaths and vehicle collisions in the latter sample probably reflects a detectability bias favoring animals dying of those causes.

Parasites and Diseases

Necropsy results indicated that prevalence of flukes in moose livers was high (89.0%, $n = 100$), with flukes also being recovered in secondary tissues such as lungs (12.1%, $n = 33$), thoracic cavity (73.3%, $n = 30$), pericardium (65.3%, $n = 49$), and peritoneal cavity (16.1%, $n = 31$). For animals dying of fluke or probable-fluke parasitism, mean fluke numbers/liver was 48.1 ± 9.6 (SE; n

Figure 5. Most moose deaths in northwest Minnesota, USA, 1995–2000, were due to parasitism, infectious disease, and possibly starvation. Note the atypical moose habitat in which this individual was found. Photo by Eric W. Cox.

Table 2. Number and percent of moose mortalities by proximate cause of death in northwest Minnesota, USA, 1995–2000.

Cause of death	Calves	Yearlings	Adult F	Adult M	Total	%
Radiomarked						
Fascioloides magna	4	3	9		16	21
Probable *Fascioloides magna*	0	2	6		8	11
Probable *Parelaphostrongylus tenuis*	0	0	4		4	5
Probable disease/starvation	6	0	13		19	25
Predation	2	2	1		5	7
Vehicle collision	0	1	2		3	4
Poaching	0	0	2		2	3
Unknown	2	2	15		19	25
Subtotal	14	10	52		76	
Non-radiomarked						
Fascioloides magna	1	1	3	0	5	6
Probable *Fascioloides magna*	0	0	1	0	1	1
Probable *Parelaphostrongylus tenuis*	5	2	8	2	17	20
Probable disease/starvation	11	4	10	3	28	33
Vehicle collision	5	4	9	4	22	26
Poaching	1	0	2	0	3	4
Starvation	1	0	0	0	1	1
Natural accident	0	0	2	2	4	5
Unknown	0	1	3	0	4	5
Subtotal	24	12	38	11	85	
Total	38	22	90	11	161	

= 23), compared to means of 14.2 ± 2.2 ($n = 69$) and 10.4 ± 4.6 ($n = 8$) for animals dying of nonfluke and unknown causes, respectively ($F_{2,86} = 9.196$, $P < 0.001$). Fluke numbers for animals dying from fluke parasitism were relatively low in the thoracic cavity (0.6 ± 0.1, $n = 14$), pericardial sac (0.2 ± 0.2, $n = 13$), and peritoneal cavity (1.0 ± 0.5, $n = 12$). Also, 25% ($n = 12$) of animals dying from flukes had helminths present in the lungs (flukes were not counted in lungs). Evidence for fascioloidiasis in moose included extensive blood tunnels in the liver and other organs, scarring, calcification, and black exudate inside closed capsules. Some carcasses also had black exudate scattered profusely throughout the abdomen (G. Huschle, USFWS, personal communication). On average, 29.2 ± 6.9 ($n = 21$) cysts were counted per liver of animals dying of fluke infestation, versus 11.7 ± 1.9 ($n = 56$) and 7.7 ± 2.7 ($n = 9$) for animals dying of nonfluke and unknown causes, respectively ($F_{2,83} = 8.139$, $P = 0.001$).

We considered that meningeal worm recovery during necropsy did not preclude other sources of mortality from being important. For half the moose probably dying of meningeal worms, nematodes were recovered from the brain tissue or spinal cord ($n = 4$), compared to 4.4% ($n = 45$) and 11.1% ($n = 9$) for animals dying of nonmeningeal worm and unknown causes, respectively. Prevalence (23.4%, $n = 47$) and intensity (5.4 ± 1.5, $n = 9$) of *Echinococcus granulosus* were intermediate, and prevalence of *Sarcocystis* spp. (23.1%, $n = 39$) and *Tularemia* spp. (5.6%, $n = 18$) was relatively low.

Among live-captured females, 46.0% ($n = 37$) tested positive for *Leptospira interrogans* serovar *grippotyphosa* (titers > 1:100), 13.5% ($n = 37$) tested positive for *L. i.* serovar *bratislava*, and no animals had elevated titers for *Brucella abortus*.

Our necropsy work revealed a relatively high frequency of morbidity among moose carcasses, including symptoms such as pleuritis (94.2%, $n = 52$), adrenelitis (43.2%, $n = 37$), nephritis (46.8%, $n = 47$), spleenitis (35.0%, $n = 40$), lymphitis (44.1%, $n = 34$), hepatitis (92.2%, $n = 51$), meningitis (25.0%, $n = 40$), pneumonia (35.1%, $n = 37$), pulmonary edema (60.5%, $n = 38$), bloating (23.8%, $n = 42$) , pulmonary thrombosis (13.9%, $n = 36$), peritoneal thrombosis (25.0%, $n = 40$), and septicemia (23.3%, $n = 30$).

Nutritional Status and Body Condition

The BMF of moose dying of natural causes (i.e., parasitism, predation, disease, or starvation) differed from that of animals considered as representing the live population (i.e., dying from unnatural causes or accidents), as well as those succumbing to unknown causes of mortality ($F_{2,121} = 22.956$, $P < 0.001$). On average, percent BMF was 23.2 ± 2.8% ($n = 68$) for the cohort dying of natural causes versus 58.0 ± 4.4% ($n = 30$) and 27.7 ± 4.4% ($n = 26$) for animals dying of unnatural and unknown causes, respectively. This disparity indicated that overall, animals dying of natural causes tended to be subject to acute malnutrition. Animals dying of disease or starvation had lower BMF than the remaining main causes of death ($F_{2,121} = 4.861$, $P = 0.009$). Bone-marrow fat levels did not differ between sexes, age classes, or among seasons (all $P > 0.12$). Overall, 51.4% ($n = 124$) of moose carcasses had BMF values below the malnutrition threshold (i.e., <30%), and fluke numbers in moose livers were negatively correlated to BMF ($t_{86} = 2.057$, $P = 0.043$; Fig. 6).

Survival

On average, we monitored survival of individual radiomarked animals during 639 ± 37 days ($n = 152$) between June 1995 and July 2000. Overall, the majority of marked animals were adult females captured during winter–spring ($n = 100$), with the remainder being neonatal calves captured within days of their birth, several of which were later monitored as yearlings and even into early adulthood. Overall, annual survival rates averaged 0.66 (0.53–0.81; 95% CI), 0.64 (0.48–0.86), and 0.79 (0.74–0.84) for calves, yearlings, and adult females, respectively.

The dummy variable representing calendar year (1996) failed to

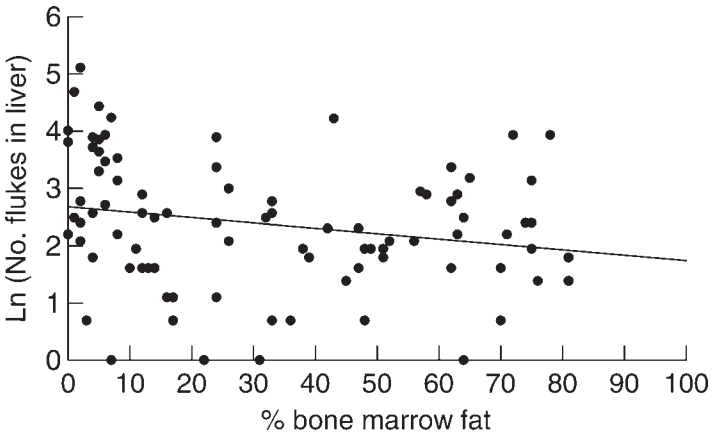

Figure 6. Relationship between liver fluke intensity in livers and bone-marrow fat content for moose carcasses in northwest Minnesota, USA (1995–2000).

conform to the assumption of proportional hazards and therefore was used as a stratum in later models. In 1996, moose survival was lower than in other years (log-rank test: $\chi^2_2 = 5.59$, $P = 0.018$). Annual survival rates for adult females ranged from 0.73 (0.59–0.91) to 0.80 (0.71–0.92) in 1996 and 1999, respectively. For yearlings, survival rates ranged from 0.26 (0.08–0.85) to 0.89 (0.71–1.0) in 1996 and 1999, respectively. For calves, survival rates ranged from 0.53 (0.32–0.88) to 0.81 (0.63–1.0) in 1996 and 1998, respectively (no calves were radiocollared after 1998).

Several candidate CPH models warranted formal evaluation via model selection. The best models included dummy variables representing the late winter pregnancy period (1 Mar–15 May) and gender (Table 3). For adult females, survival rate (reported as 90-day survival rate) was highest during 1 December–28 February (0.96 [0.93–0.99]) and lowest during 1 March–15 May (0.91 [0.87–0.95]). For yearlings, survival was highest during 1 August –30 November (0.93 [0.85–1.0]) and lowest during 1 March–15 May (0.79 [0.60–1.0]), whereas for calves, survival was highest during 1 December–28 February (1.00; no mortalities) and lowest during 1 March–15 May (0.78 [0.66–0.94]). Our CPH models revealed that hazard may have been higher among males (Table 3; Murray 2006), although because males were not monitored beyond 2 years of age, lower juvenile survival irrespective of gender could not be discounted as a survival determinant. Overall, the collective weight of evidence (w_i) from the modeling exercise indicated that both winter-pregnancy period (0.851) and gender (0.834) were strong determinants of moose hazard (Table 3). Hazard rates were comparable among all 3 study areas (all $\Delta_i >$ 6.224).

In most cases, CPH models revealed that hazard failed to differ across discrete age classes (Table 3). However, the retention of gender in the above models may still be related to age-specific differences in mortality risk. To explore this possibility, we restricted our CPH models to animals whose specific age (in years) was known (i.e., first captured as calves, or aged at time of death, n = 101). When compared to the restricted model including calendar year 1996 as stratum and late winter pregnancy status, age improved model fit ($\Delta_i = 4.583$). The hazard ratio for age (1.001 ± 0.001) indicated that overall mortality risk increased slightly with each age increment (Fig. 7). Addition of the dummy variable for gender to this model failed to improve fit ($\Delta_i = 0.806$), implying that gender differences in mortality risk were not significant when specific age classes were considered. However, qualitatively, annual survival rates were lower for males (male yearlings: 0.55 [0.23–1.0]; female yearlings: 0.66 [0.48–0.90]; male calves: 0.50 [0.31–0.81]; female calves: 0.75 [0.61–0.93]).

We examined the correlation between pregnancy status and mortality risk by further restricting our CPH models to periods when females were potentially pregnant. The model including pregnancy status provided improved explanation of moose mortality risk ($\Delta_i = 2.752$, $z_1 = 2.13$, $P = 0.033$), with the hazard ratio for pregnancy status (0.446 ± 0.129) indicating that pregnant females were less than half as likely to succumb to mortality during pregnancy than animals that were not pregnant. Pregnancy during the previous year and presence of a calf during the current or previous year failed to correlate with mortality risk (all $\Delta_i > 2.162$).

Annual cause-specific mortality rates were 0.09 (0.06–0.12) for deaths due to parasitism (liver fluke, probable liver fluke, probable meningeal worm), 0.06 (0.03–0.09) for probable disease or starvation, 0.02 (0.00–0.03) for predation, 0.01 (0.00–0.03) for anthropogenic causes (vehicle collision and poaching), and 0.06 (0.03–0.09) for unknown causes. Assuming that moose mortalities due to parasitism, disease or starvation, and unknown causes ultimately involved pathogens, the pooled annual mortality rate was 0.21 (0.17–0.26) for pathogen-related, and 0.03 (0.01–0.05) for non–pathogen-related moose deaths.

Table 3. Candidate models of moose survival relative to temporal and demographic variables in northwest Minnesota, USA (1995–2000). We present model χ^2, P, Akaike's Information Criterion (AIC_c), AIC_c difference (Δ_i), and AIC_c weight (w_i), along with the coefficient estimates and z and P values for individual parameters. All models were stratified by year 1996, and dummy coding was used to represent specific seasons, gender (F = 0), and age class (nonadults = 0).

Model	χ^2	P	AIC_c	Δ_i	w_i	Parameter	Coeff. estimate	SE	z	P[a]
1	9.841	0.007	577.764	0		Late winter pregnancy	1.001	0.001	2.27	0.023
						Gender	0.44	0.162	2.22	0.026
2	10.202	0.017	579.418	1.654		Late winter pregnancy	1.002	0.001	2.28	0.023
						Gender	0.403	0.169	2.26	0.024
						Age class	1.001	0.001	0.58	0.99
3	4.64	0.031	580.953	3.190		Late winter pregnancy	2.031	0.671	2.14	0.032
4	4.28	0.039	581.323	3.559		Gender	0.440	0.161	2.24	0.025
5	3.97	0.046	582.055	4.292		Early winter pregnancy	0.026	0.191	2.01	0.055

[a] P value for the parameter.

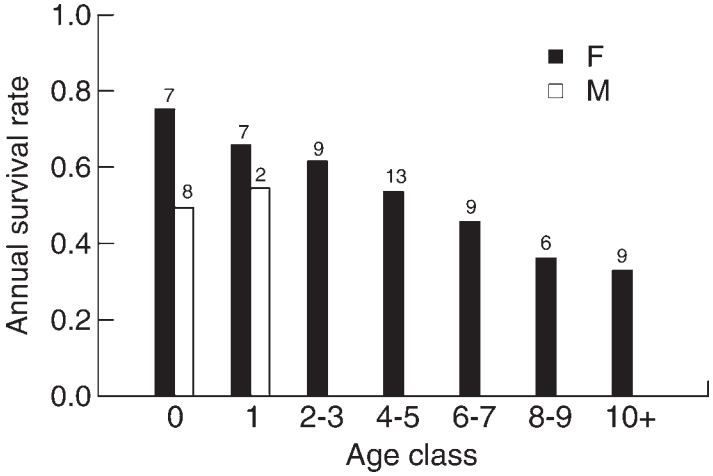

Figure 7. Age-specific annual survival rates for moose in northwest Minnesota, USA, 1995–2000. Number of deaths/age class appears above bars.

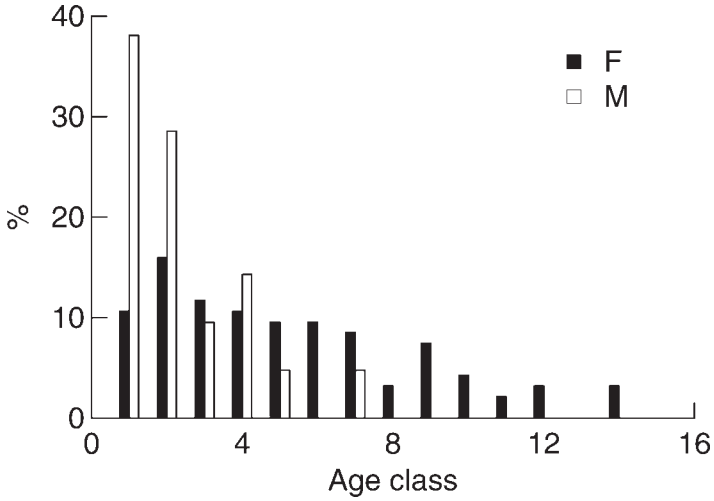

Figure 8. Age structure of dead yearling and adult moose in northwest Minnesota, USA, based on cementum annuli counts, 1995–2000.

Age Structure

Age structure of the yearling and adult female moose population was similar between the radiomarked and non-radiomarked cohorts (radiomarked animals back-calculated to time of first capture; paired $t_{112} = 1.638$, $P = 0.10$), and between animals dying of natural versus nonnatural causes (paired $t_{112} = 1.398$, $P = 0.16$). Accordingly, we used the pooled sample of necropsied animals contributing cementum annuli to describe the age structure of the yearling and adult sample. The age structure of the moose population differed between genders ($F_{1,\ 110} = 9.723$, $P = 0.002$), with most males being aged 1–2 years and no males >7 years being recovered (Fig. 8). Although it is notable that our sample of males for age determination was small ($n = 11$), the median age of harvested males in the area (1987–1995 mean age of males approx. 3.5 yr; MNDNR, unpublished data) was comparably low. However, it should be noted that male:female ratios from moose population surveys (Fig. 3) were considerably more skewed toward males than that indicated by our age-distribution data set (Fig. 8). Females were well represented through age 14 years, with about 50% of the yearling-adult population being >5 years old (Fig. 8). When blocking for gender, age structure did not differ between animals dying of natural versus nonnatural causes ($F_{1,113} = 0.534$, $P = 0.47$). Age structure was similar among the major natural causes of death (i.e., parasitism, disease/starvation, unknown causes; $F_{2,78} = 1.512$, $P = 0.22$), and bone-marrow fat levels at time of death were not related to moose age class ($F_{1,109} = 2.166$, $P = 0.28$).

Pregnancy Assessment

Serum progesterone concentrations fell into 2 relatively distinct groups that appeared to differ among study years. We determined qualitatively that a threshold in progesterone concentrations occurred in the range of 2.0–3.0 ng/mL (Fig. 9a). With 2.5 ng/mL used as a cutoff, we found that overall, 49.0% ($n = 153$) of serum samples collected during winters 1995–1996 through 1998–1999 were from pregnant females. With the use of this threshold, mean progesterone concentration between females deemed to be pregnant (6.1 ± 0.3 ng/mL, $n = 75$) was different from those considered nonpregnant (0.7 ± 0.1 ng/mL, $n = 78$).

Fecal progestagen concentrations fell similarly into 2 groups, although the pregnancy threshold for this assay was less well defined, ranging from 3.0–6.0 μg/g (Fig. 9b). Using a threshold of 4.0 μg/g, we determined that 43.6% ($n = 163$) of females were pregnant. Mean fecal progestagen concentrations for animals suspected of being pregnant versus nonpregnant was 14.2 ± 0.8 μg/g ($n = 71$) and 2.1 ± 0.1 μg/g ($n = 92$), respectively. There was significant correlation between log-transformed serum progesterone and fecal progestagen levels taken from the same individual during the same reproductive season ($t_{90} = 6.24$, $P < 0.001$), but the correlation between fecal progestagen and pregnancy-specific protein B was not significant ($t_{23} = 0.58$, $P = 0.56$).

Visual observations of calves largely corroborated our pregnancy status assessment, with 44.7% ($n = 195$) of females surveyed in May–June being observed with a calf. Our calf survey revealed that for females lacking calves, serum (81.6%, $n = 60$) and fecal (69.2%, $n = 91$) assays taken from the female provided corresponding pregnancy assessment. When females were found to be accompanied by a calf, pregnancy detection was 100% ($n = 36$) and 79.2% ($n = 72$) for serum and fecal assays, respectively. Overall, these results imply that pregnancy status assessment via hormone assay was effective for both serum and fecal samples, with slightly higher reliability for the former assay.

Pregnancy Rates

Overall, moose pregnancy rates determined from progesterone or progestagen assay, calf observations during spring, or evidence detected during carcass necropsy (i.e., corpora lutea, embryos, lactation) were low for our study area and averaged 47.9% ($n = 280$). Restricting the analysis to results from hormone assays, logistic regression revealed that neither year, study area, nor age class (adult vs. yearling) were associated with pregnancy status (all $P > 0.17$). Pregnancy rates ranged from 38.5% ($n = 26$) in 1995–1996 to 58.6% ($n = 70$) in 1998–1999. An analysis further restricted to animals whose age had been estimated via cementum annuli ($n = 111$) indicated that pregnancy rates were similar across female ages ($t_2 = 0.322$, $P = 0.75$). However, qualitatively it appeared that pregnancy rates were low among yearlings, increased

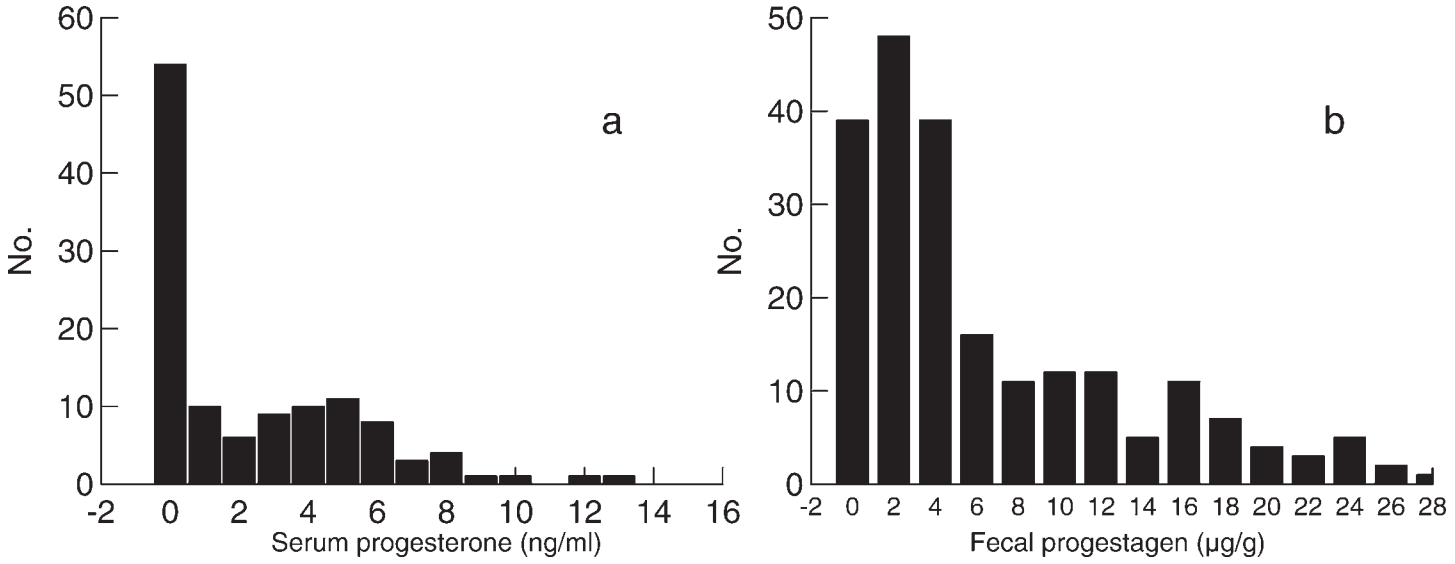

Figure 9. Distribution of (a) serum progesterone and (b) fecal progestagen concentrations in moose samples from northwest Minnesota, USA (1996–2000).

up to ages 6–7 years, and then declined thereafter (Fig. 10). The initiation of reproductive senescence appeared to occur as early as age 8 years in our study population and most age classes had pregnancy rates <50% (Fig. 10).

Adult females exhibited low fecundity; of 31 female carcasses examined for embryos, 7 were found to have a single fetus and only 2 (22%) had twins. Similarly, our May–June observations of calf–female pairs suggested only a 19% ($n = 87$) twinning rate among pregnant females.

Pregnancy status during a given year failed to predict pregnancy during subsequent years. For females whose pregnancy status was evaluated in 2 consecutive years, the percent pregnancy in both years (60.9%, $n = 69$) was marginally comparable to pregnancy in the first year only ($\chi^2_1 = 3.260$, $P = 0.070$); for those not pregnant in the first year, the percent pregnancy in the second year only (48.6%, $n = 70$) was comparable to the rate of no pregnancy in year 2 ($\chi^2_1 = 0.057$, $P = 0.81$). The apparent independence of interannual pregnancy status was further substantiated by the proportion of animals that showed no interannual variability in pregnancy status; 47.1% ($n = 34$) for animals monitored during 2 consecutive years, 40.0% ($n = 35$) for 3 years, and 18.2% ($n = 11$) for 4–5 years ($\chi^2_2 = 2.888$, $P = 0.24$). Thus, past pregnancy status apparently did not influence future reproductive potential and therefore annual pregnancy status could be considered as an independent event.

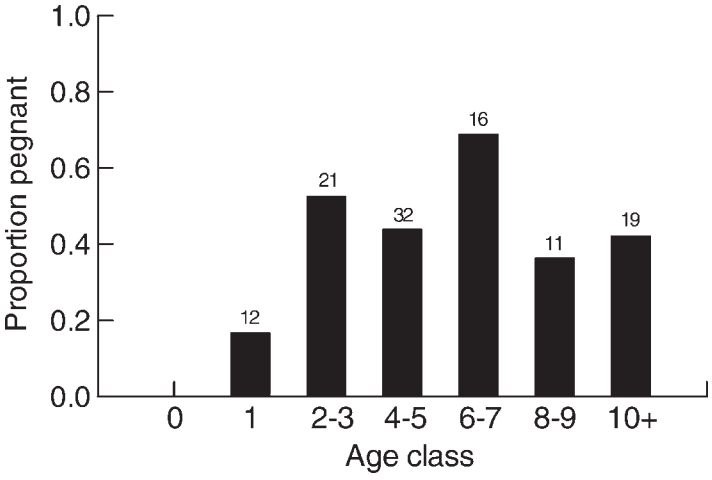

Figure 10. Age-specific pregnancy rates for moose in northwest Minnesota, USA, during winter 1996–1998. The number of samples appears above bars.

Next we examined the relationship between pregnancy status and female condition at time of death. When blocking for age and season, we found that pregnancy status was related to BMF levels ($t_1 = 3.676$, $P = 0.002$), with pregnant versus nonpregnant females having BMF levels of 46.2 ± 4.3% ($n = 39$) versus 25.1 ± 2.9% ($n = 71$), respectively. The odds ratio for the logistic regression model (1.031 ± 0.122) indicated that for each percentage increase in BMF at time of death, the probability of pregnancy increased by about 3%. The relationship between pregnancy status and BMF was further illustrated when we separated BMF according to the starvation threshold (30% BMF). In this analysis, 51.8% ($n = 54$) versus 19.6% ($n = 56$) of females fell above the threshold for the pregnant and nonpregnant groups, respectively ($\chi^2_1 = 12.463$, $P < 0.001$).

Among necropsied carcasses, we failed to detect a relationship between recent pregnancy status and fluke parasitism ($t_1 = 0.629$, $P = 0.53$), with comparable numbers of flukes being retrieved from livers of pregnant (27.8 ± 5.7, $n = 45$) and nonpregnant (23.8 ± 3.5, $n = 65$) female carcasses. Using results from both necropsy and serological tests for disease antibodies from live animals, we surmised that pregnancy status was not related to prevalence of meningeal worm ($\chi^2_1 = 0.49$, $P = 0.48$), sarcosystis ($\chi^2_1 = 1.468$, $P = 0.23$), or echinococcus ($\chi^2_1 = 0.355$, $P = 0.55$). Forty-six percent ($n = 28$) of sera from nonpregnant adult females tested positively for *L. i. grippotyphosa* compared to 38% ($n = 8$) of sera from pregnant adults ($\chi^2_1 = 0.201$, $P = 0.65$). Eighteen percent of the nonpregnant females tested positively for *L. i. bratislava*, but none of the pregnant animals did so, and all but one of the animals that tested positively for this serovar also tested positively for *L. i. grippotyphosa*.

Table 4. Mean values and results from principal components analysis of 7 serum assays from moose captured in northwest Minnesota, USA (1995–1998). Only the first 7 assays were used in principal components analysis, and factors were rotated using the varimax method (gamma = 1). Two factors had latent roots with eigenvalues > 1.0.

Blood parameter	$\bar{x}$ ± SE (*n*)	Factor 1	Factor 2
WBC[a]	8.8 ± 0.5 (80)	0.067	0.449
RBC	7.3 ± 0.2 (80)	0.811	0.573
HGB	15.7 ± 0.3 (80)	0.983	0.092
HCT	48.8 ± 1.0 (80)	0.927	0.205
MCV	68.3 ± 1.1 (80)	−0.289	−0.852
MCH	22.2 ± 0.4 (80)	−0.107	−0.966
MCHC	32.4 ± 0.2 (80)	0.485	−0.476
RDW	24.0 ± 0.3 (65)	–	–
PLT	157.7 ± 7.4 (65)	–	–
SEGS	17.5 ± 1.2 (22)	–	–
LYMPHS	45.5 ± 3.7 (22)	–	–
MONOS	5.7 ± 1.2 (20)	–	–
EOS	30.4 ± 2.8 (22)	–	–

[a] WBC = white blood cells, RBC = red blood cells, HGB = percent hemoglobin, HCT = percent hematocrit, MCV = average red blood cell size, MCH = hemoglobin amount relative to size of red blood cell, MCHC = hemoglobin amount relative to size of cell per red blood cell, RDW = red blood cell distribution width, PLT = platelet counts, SEGS = mature neutrophils, LYMPHS = lymphocytes, MONOS = monocytes, EOS = eosinophils.

We failed to detect a relationship between moose pregnancy status and the various moose conditions or pathologies observed at time of death (all $P > 0.11$). Pregnancy status did not differ between animals dying of natural versus those dying of nonnatural causes and thus representing the live population ($\chi^2_1 = 0.130$, $P = 0.72$), and the proportion of animals known to be currently or recently pregnant did not differ among natural causes of death (fluke parasitism, meningeal worm, probable disease or starvation, or unknown cause of death; $\chi^2_3 = 0.743$, $P = 0.87$).

Body Condition Indices From Whole Blood

We collected blood samples from 12 calves and 68 females and yearlings during the course of the study. On average, the 7 blood assays conducted on all samples showed low levels of pairwise correlation ($r = 0.22 \pm 0.12$), with 6 of the 21 correlation coefficients being significant (Bonferroni correction, $P < 0.05$). Most notably, concentrations of RBC, HGB, and HCT were correlated positively, as were MCV and HCT. Levels of RBC were negatively correlated to MCV and MCH levels. For the remaining 6 blood parameters (only assayed on a subsample of moose), no correlation coefficients were significant (all $P > 0.23$). Principal-components analysis restricted to the 7 major blood assays revealed that 2 factors had latent root eigenvalues >1.0, explaining 75.6% of the total variation. Levels of RBC, HGB, and HCT were positively associated with Factor 1, whereas MCV and MCH were negatively associated with Factor 2 (Table 4).

We examined the relationship between the 2 principal-component factors and moose survival for 18 months following blood sampling. Logistic regression revealed that Factor 1 ($t_1 = 3.346$, $P = 0.001$) was associated with mortality risk, whereas Factor 2 ($t_1 = 0.214$, $P = 0.83$) was not. The odds ratio for Factor 1 (0.27 ± 0.12) indicated that low levels of Factor 1 were associated with increased risk of death. On average, values for Factor 1 were 0.45 ± 0.09 ($n = 43$) and -0.32 ± 0.19 ($n = 28$) for surviving and dying moose, respectively. The observed correlation between blood profiles and mortality risk was corroborated by logistic regression of survival likelihood versus individual blood assays; values for RBC, HGB, and HCT were each higher among surviving than dying moose (all $P < 0.05$).

We also examined the 2 principal-component factors for association with moose pregnancy status. Logistic regression indicated that Factor 1 was associated with pregnancy ($t_1 = 2.678$, $P = 0.007$), whereas Factor 2 was only marginally associated ($t_1 = 1.660$, $P = 0.097$). The odds ratios (Factor 1: 6.72 ± 1.15; Factor 2: 0.34 ± 0.15) indicated that pregnancy was clearly associated with Factor 1 but less so with Factor 2. Factor loadings were notably different between pregnant (Factor 1: 0.65 ± 0.08, Factor 2: 0.02 ± 0.11, $n = 32$) and nonpregnant (Factor 1: -0.03 ± 0.13, Factor 2: 0.53 ± 0.09, $n = 32$) females. This finding was confirmed by logistic regression of pregnancy status versus individual blood assays; values for HGB, HCT, MCV, MCH, and MCHC were all higher among pregnant females (all $P < 0.05$).

Trace Elements

We analyzed 106 moose livers for concentrations of 19 trace elements, 8 of which had concentrations consistently above detectability thresholds (Table 5). Pairwise correlation coefficients, restricted to the 8 detectable trace elements, were significant for 17.9% (Bonferroni correction, $P < 0.05$, $n = 28$) of pairs. Principal-components analysis of the 8 elements identified 4 factors having latent roots with eigenvalues >1.0 (Table 5); together these factors explained 81.5% of the total variation in trace element measures. Factor 1 reflected positive influence from Cu, Se, and Mo; Factor 2 included positive influence from Mn; Factor 3 had positive influence from Fe; and Factor 4 was influenced positively by Mn and negatively by Mg (Table 5).

Most moose parasites and pathologies were not associated with trace-element PCA factors ($P > 0.11$). However, prevalence of peritonitis ($t_{105} = 2.453$, $P = 0.014$; odds ratio: 0.48 ± 0.19), hepatitis ($t_{105} = 1.969$, $P = 0.049$; odds ratio: 0.54 ± 0.18), and pulmonary edema ($t_{105} = 2.139$, $P = 0.032$; odds ratio: 0.083 ± 0.22) were negatively correlated with Factor 1, whereas nephritis ($t_{105} = 2.110$, $P = 0.035$; odds ratio: 2.08 ± 0.47) was positively associated with Factor 4. Recent or current moose pregnancy status at time of death was not related to any of the 4 PCA factors (all $P > 0.15$), and whether animals died of natural versus unnatural causes also was not associated with any of the trace element PCA factors (all $P > 0.21$). Overall, levels of select trace elements such as Cu were low (Table 5), especially compared to those found in other moose populations (Custer et al. 2004).

Vegetation and Fecal Pellet Analyses

All 4 primary winter browse species had P content exceeding 6% (Table 6). Principal-components analysis for percent ADF, percent NDF, and percent P from the collected vegetation samples ($n = 283$) indicated that 2 factors had latent roots with eigenvalues >1.0, explaining 87.1% of the variation. Overall, the 4 browse species differed in composition of the 3 variables of interest (Wilks's lambda; $F_{9,608} = 20.608$, $P < 0.001$). When the

Table 5. Trace element composition of 106 moose livers from northwest Minnesota, USA, 1995–2000. For elements found at low concentrations, detectability thresholds and the proportion of livers above the threshold are provided. Principal components analysis was conducted using the 8 trace elements with all samples above the detectability threshold; mercury was excluded because of the reduced number of samples ($n = 48$). Factors were rotated using the varimax method (gamma = 1), and 4 factors had latent roots with eigenvalues > 1.0.

Element	$\bar{x} \pm$ SE (ppm)	Detectability threshold	Proportion below threshold	Factor 1	Factor 2	Factor 3	Factor 4
Cadmium	2.66 ± 0.21	–	–	0.361	0.117	0.003	0.627
Copper	92.9 ± 9.37	–	–	0.919	−0.024	0.154	0.022
Iron	1335.7 ± 120.1	–	–	0.114	−0.066	0.816	−0.214
Magnesium	580.2 ± 14.2	–	–	−0.108	−0.048	0.022	0.844
Manganese	8.4 ± 0.4	–	–	−0.158	0.924	0.044	−0.082
Molybdenum	3.31 ± 0.13	–	–	0.513	0.669	0.051	0.316
Selenium	3.2 ± 0.3	–	–	0.948	−0.013	−0.042	0.056
Zinc	331.3 ± 3.9	–	–	−0.019	0.150	0.749	0.277
Aluminum	–	5.0	0.81	–	–	–	–
Arsenic	–	0.5	1.0	–	–	–	–
Boron	–	2.0	0.93	–	–	–	–
Barium	–	0.5	0.83	–	–	–	–
Beryllium	–	0.1	0.94	–	–	–	–
Chromium	–	0.5	0.22	–	–	–	–
Lead	–	0.02	0.41	–	–	–	–
Mercury	–	0.02	0.57	–	–	–	–
Nickel	–	0.02	0.25	–	–	–	–
Strontium	–	0.40	0.80	–	–	–	–
Vanadium	–	0.50	1.0	–	–	–	–

analysis was restricted to dogwood and willow (species where our sample contained moose-browsed and random samples), we found that whether the sample was taken randomly versus at a site where moose had browsed was related to browse composition (Wilks's lambda; $F_{9,608} = 11.555$, $P < 0.001$), with browsed dogwood having lower percent ADF, higher percent NDF, and higher percent P than unbrowsed samples (all univariate $P < 0.05$).

Fecal pellets contained on average 8.0 ± 3.6% ($n = 64$) protein. We found that protein levels differed between animals that died in the following 18 months versus those that did not ($F_{1,61} = 18.18$, $P < 0.001$). Animals dying shortly after fecal collection had higher fecal protein content than did surviving animals (dying: 10.2 ± 4.4 [$n = 33$]; surviving: 6.3 ± 0.7 [$n = 35$]).

Climatic Influence

Weather data in northwest Minnesota followed a general increasing trend in summer and winter temperatures, and a lengthening of the annual growing season, during the 1960–1961 to 2000–2001 monitoring period (Table 7, Fig. 11). In contrast, winter snow depth and summer precipitation levels did not change noticeably during the same period (Table 7). The rate of climate change did not differ between periods preceding (1960–1961 to 1983–1984) versus following (1984–1985 to 2000–2001) peak moose population counts (homogeneity of slopes; all $P > 0.34$), although most temperature indices exhibited a qualitative tendency favoring warmer conditions during 1984–1985 to 2000–2001 (Table 7). For the 2 moose thermoregulation thresholds (i.e., years with mean March temperature >−5 C; years with mean September temperature >14 C), pre- versus postpeak conditions also differed, with more postpeak monthly temperature means exceeding both of the thresholds (Table 7).

The complete moose survey data set (1960–1961 to 2000–2001) failed to exhibit first-order autocorrelation (ACF = −0.37; $P = 0.14$). We detected evidence of inverse density dependence in the complete time-series models (Table 8, Fig. 12), although it is notable that comparable analyses in the context of the count-based PVA failed to identify a similar pattern. The parameter representing summer temperature at $t - 1$ was present in 2 of the 3 final models, with w_i indicating that it was likely relevant to moose population change (Table 8). Moose population change was negatively related to time-delayed temperature during summer (Fig. 12).

For the restricted time series (post-1983–1984, when the moose population was declining, and also the period in which deer harvest data were available), inverse density-dependence was no longer related to population growth rate but summer temperature ($t - 1$) remained prevalent in candidate models (Table 8, Fig. 12). No parameter representing male harvest rate was retained in any of the final models (Table 8), and moose harvest was not related to population rate of change in the current or previous years (all $P > 0.86$). After penalizing for autocorrelation (Bartlett 1946), moose population rate of change was not correlated to male harvest rate during the current or previous year ($P > 0.19$), implying that deer abundance probably did not influence moose numbers directly.

Table 6. Mean values for percent acid detergent fiber (ADF), percent neutral detergent fiber (NDF), and percent crude protein (P), among the 4 main moose browse species in winter in northwest Minnesota, USA (1997–1998, 1998–1999, and 1999–2000). Dogwood and willow samples included those collected randomly in the study area versus those collected specifically from plants browsed by moose, whereas aspen and alder samples were obtained exclusively from opportunistic sampling.

Plant species	% ADF $\bar{x} \pm$ SE (*n*)	% NDF $\bar{x} \pm$ SE (*n*)	% P $\bar{x} \pm$ SE (*n*)
Alder	55.7 ± 0.9 (44)	68.0 ± 1.0 (44)	9.3 ± 0.3 (39)
Aspen	51.9 ± 0.9 (32)	60.7 ± 1.0 (32)	8.3 ± 0.2 (27)
Dogwood	51.5 ± 1.1 (47)	62.2 ± 0.9 (47)	6.7 ± 0.1 (42)
Willow	55.1 ± 0.4 (160)	67.9 ± 0.4 (160)	8.3 ± 1.0 (152)

Table 7. Change in selected weather variables in northwest Minnesota, USA (1960–2001). Variables were selected a priori to represent general changes in winter and summer temperature and precipitation as well as temperature thresholds that were specific to moose thermoregulatory abilities in autumn and spring. Autocorrelation in the regression of a given variable versus time at $t - 1$ (ACF) is given, and P and r values are for a linear regression of the given climatic index versus time. For temperature and precipitation, prepeak and postpeak columns refer to mean climatic variables during 1960–1961 to 1983–1984 and 1984–1985 to 2000–2001, respectively, whereas for moose thermoregulation threshold the values represent percent of years falling above the tolerance threshold. P values from statistical comparison of pre- versus postpeak conditions are provided.

Weather variable	ACF in 41-yr time series	P	r	Prepeak $\bar{x} \pm$ SE (n)	Postpeak $\bar{x} \pm$ SE (n)	P^a
Temperature						
Jan–Feb temperature	−0.037	<0.001	0.53	−15.5 ± 0.7 (23)	−12.4 ± 0.8 (18)	0.01
Jul–Aug temperature	0.232	0.01	0.42	19.2 ± 0.2 (23)	20.0 ± 0.3 (17)	0.10
Last spring freeze (Julian days)	0.113	<0.001	0.51	168.8 ± 2.0 (23)	161.7 ± 3.5 (17)	0.07
First autumn freeze (Julian days)	−0.041	0.01	0.39	291.7 ± 1.8 (24)	296.8 ± 3.4 (17)	0.16
Annual growing season (days)	−0.082	<0.001	0.54	122.6 ± 3.1 (23)	135.1 ± 5.9 (17)	0.05
Precipitation						
Jan–Feb snow	–0.134	0.58	0.09	18.4 ± 2.1 (19)	18.1 ± 1.7 (18)	0.91
Jul–Aug precipitation	–0.144	0.41	0.14	8.4 ± 0.7 (23)	8.5 ± 0.8 (16)	0.91
Moose thermoregulation threshold						
Mar temperature >−5 C (%)	–	–	–	30.4% (23)	77.8% (18)	0.01
Sep temperature >14 C (%)	–	–	–	8.3% (24)	47.1% (17)	0.01

[a] P value for the test comparing prepeak versus postpeak conditions.

Calf:female ratios were not correlated to moose population change or summer temperature at $t - 1$ (all $P > 0.14$).

Population Viability Analyses

The analysis of moose survey data (1960–2001) provided estimates of μ and σ^2 equal to −0.010 (−0.099 to 0.079) and 0.071 (0.047–0.120), respectively. With the use of the normal distribution curve described by the above μ and σ^2, we determined that the probability that resampling from the same distribution would yield a declining average growth rate ($\mu < 0$) was 0.56. The probability that resampling would indicate that the population was

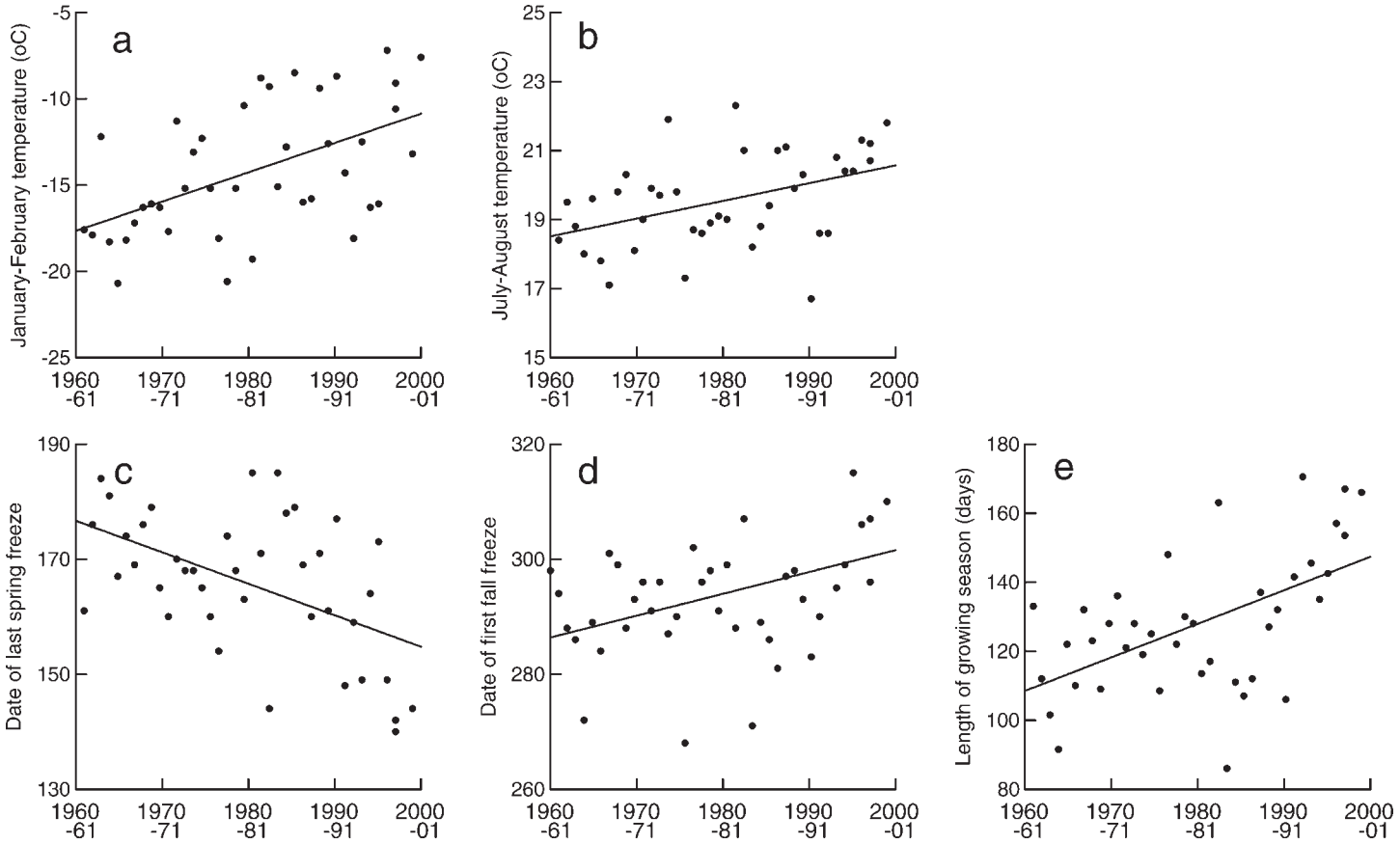

Figure 11. Temporal change in winter temperature (a), summer temperature (b), last spring freeze (c), first fall freeze (d), and total length of the growing season (e) in northwest Minnesota, USA (1960–1961 to 2000–2001). Each univariate relationship was statistically significant.

Table 8. Candidate models of moose population change with respect to climate, and moose and deer population density, in northwest Minnesota, USA (1960–2001). We present model F, R^2_{adj}, Akaike's Information Criterion adjusted for small sample size (AIC_c), AIC_c difference (Δ_i), and AIC_c weight (w_i), along with the coefficient estimates and t and P values for individual parameters. Deer harvest data were available for post-1983–1984 models only.

Model	F	R^2_{adj}	AIC_c	Δ_i	w_i	Parameter	Coeff. estimate	SE	t	P^a
1960–1961 to 2000–2001										
1	6.615	0.228	−112.763	0	0.905	Moose no.	1.2×10^{-4}	0.427	2.981	0.003
						Summer ($t-1$) temperature	−0.047	−0.258	1.805	0.04
2	4.837	0.089	−108.036	4.727	0.085	Moose no.	9.0×10^{-4}	0.336	2.199	0.019
3	6.371	0.315	−103.618	9.145	0.009	Moose no.	1.2×10^{-4}	0.412	2.902	0.004
						Summer ($t-1$) temperature (C)	−0.054	−0.290	2.023	0.025
						Snow depth ($t-1$)	-9.9×10^{-3}	−0.305	2.161	0.019
Post 1983–1984										
1	6.593	0.427	−49.924	0	0.576	Summer ($t-1$) temperature (C)	−0.097	−0.597	3.054	0.004
						Summer ($t-1$) precipitation (cm)	0.027	0.408	2.089	0.026
2	10.605	0.375	−47.932	1.992	0.213	Summer ($t-1$) temperature (C)	−0.123	−0.644	3.257	0.003
3	7.655	0.454	−47.915	2.008	0.211	Summer ($t-1$) temperature (C)	−0.113	−0.594	3.177	0.003
						Mar temperature >5 C (previous 2 winters)	0.140	0.333	1.781	0.048

[a] Probability for one-tailed t-test adjusted for autocorrelation.

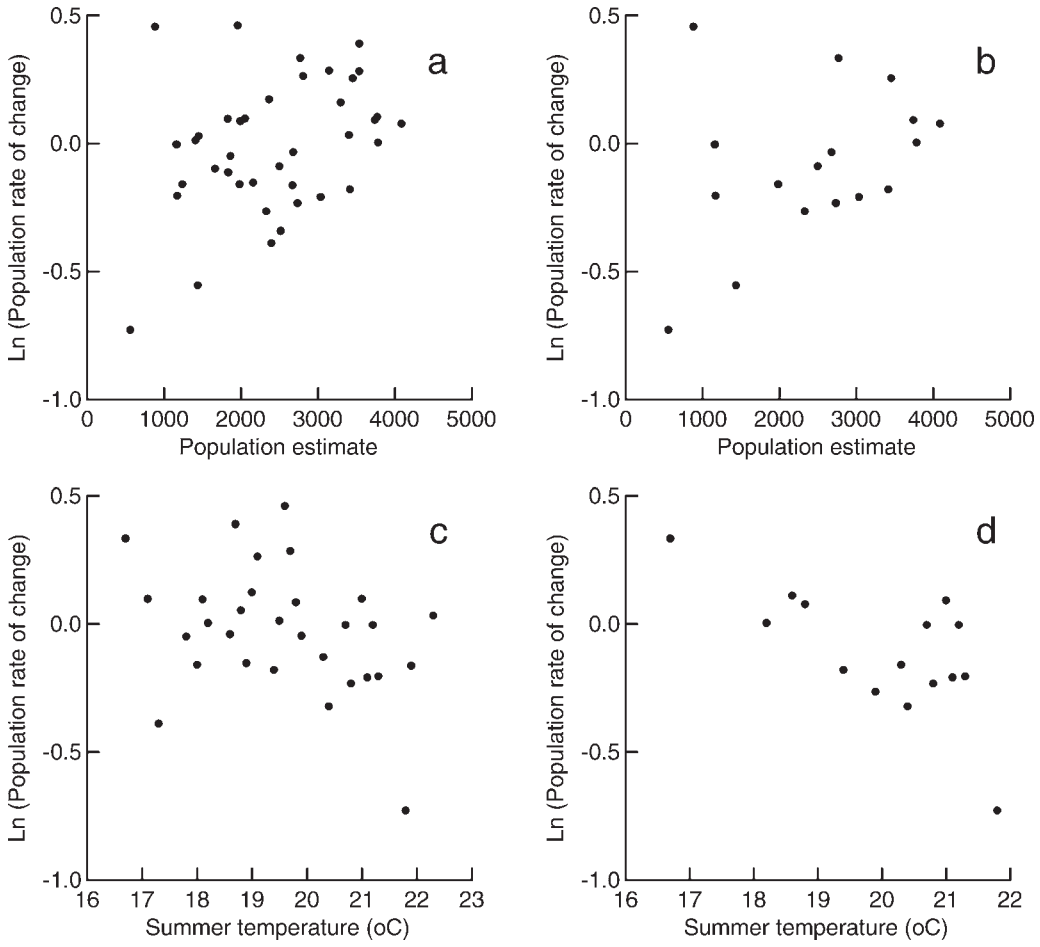

Figure 12. Relationship between moose population rate of change and population estimate (a), (b), and mean summer (Jul–Aug) temperature during the previous year (c), (d), in northwest Minnesota, USA. Panels (a) and (c) include the complete time series (1960–1961 to 2000–2001) whereas panels (b) and (d) refer to the decline period only (after 1983–1984). Panel (d) was not represented in the better models but is included for comparative purposes.

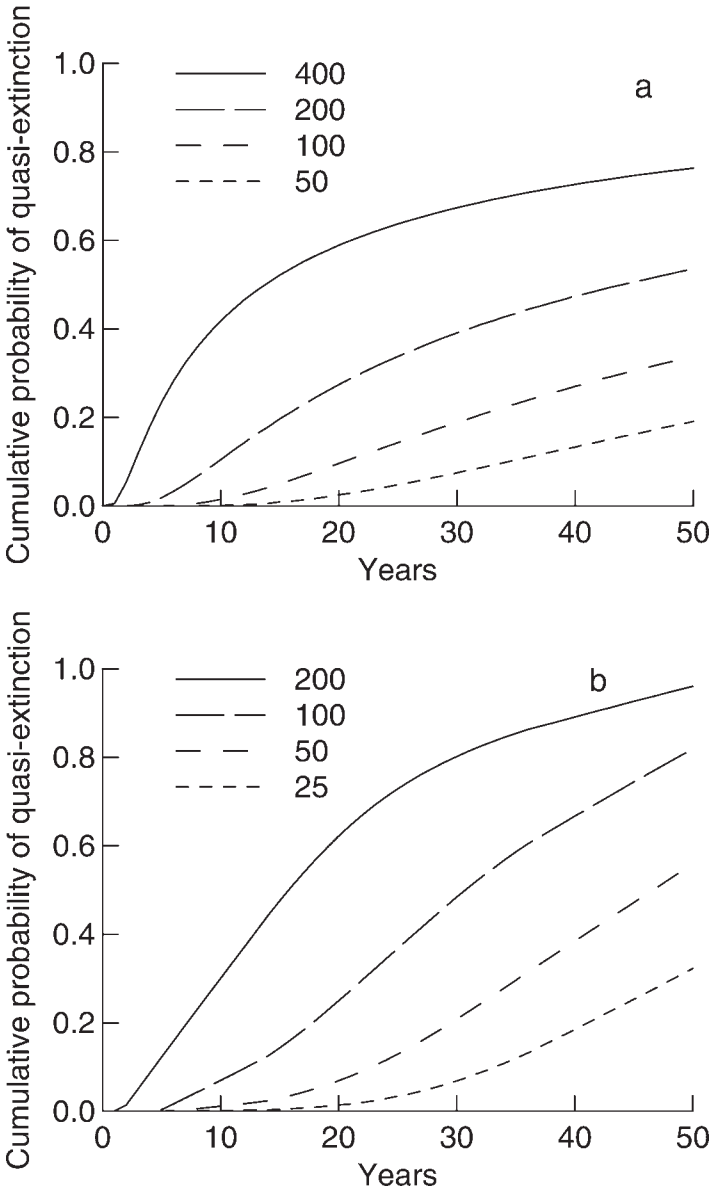

Figure 13. Cumulative probability of quasi-extinction in the northwest Minnesota, USA, moose population with the use of (a) count-based and (b) demographic projections. Projections were stochastic and lines were smoothed with the use of a LOWESS function for graphing purposes.

in rapid decline ($\mu < -0.05$) was 0.29. The wide confidence intervals for μ and σ^2 and the test statistic associated with the no-constant regression ($t_{39} = 0.226$, $P = 0.82$) indicated that despite the observed qualitative decline in aerial counts since 1984–1985 (Fig. 1), there was no significant overall trend in the time series. In 2-tailed tests, both μ ($t_{36} = 1.486$, $P = 0.073$) and σ^2 ($F_{13,22} = 2.276$, $P = 0.043$) were comparable between the pre- versus postpeak time periods. With the use of population parameters for the complete time series and its CDF, and the 2000–2001 moose population estimate (883 animals) as a starting population size, median quasi-extinction time ranged between 74 and 184 years for thresholds of 200 to 50 animals, respectively. Probabilities of quasi-extinction during the next 50 years were estimated as 0.76 (0.11–1.00), 0.54 (0.02–0.99), 0.34 (0.01–0.97), and 0.19 (0.01–0.93) for thresholds equal to 400, 200, 100, and 50 moose, respectively (Fig. 13).

Similar projections using the post-1984 count data only ($\mu = -0.099$ [−0.287 to 0.088], $\sigma^2 = 0.105$ [0.055–0.273]) revealed a higher probability that $\mu < 0$ (0.83) and $\mu < -0.05$ (0.68). A 50-year projection using this data segment indicated a high probability of quasi-extinction for all thresholds (400:0.99 [0.25–1.0], 200:0.98 [0.01–1.0], 100:0.93 [0.01–1.0], 50:0.90 [0.00–1.0]).

The demographic model derived from vital rates collected during the field study also suggested a declining moose population. The dominant eigenvalue for the Lefkovich matrix was 0.970, indicating that overall the moose population should be subject to a modest annual decline of about 3%. Matrix analysis indicated that 69% of the elasticity was attributable to contributions from survival in the adult stage class. Stochastic population simulations gave a probability of quasi-extinction equal to 0.95 (0.91–0.99), 0.79 (0.75–0.83), 0.53 (0.49–0.57), and 0.31 (0.28–0.36) in the next 50 years based on extinction thresholds of 200, 100, 50, and 25 females in the population, respectively (Fig. 13).

Simulated management efforts designed to improve moose vital rates had limited favorable effects on the estimated moose population growth rate. When mortality rate was reduced by 0–10%, or when pregnancy rate was increased by 0–20%, the transition matrix for the moose population remained nonsustainable (i.e., $\lambda < 1.0$; Table 9). However, when both vital rates were altered concurrently, the population generally achieved a sustainable transition matrix (i.e., $\lambda > 1.0$).

DISCUSSION

Initially, we considered 6 factors, acting alone or in tandem, as potentially explaining the recent moose population decline in northwest Minnesota. Results from our field study combined with time-series analyses allowed us to eliminate legal hunting, predation, and resource competition with deer or among moose as being important factors in the numerical decline (Table 1). Hypotheses related to pathogens and climate change were more strongly supported by our results, and therefore these factors were presumably implicated. Our work also revealed that moose in northwest Minnesota showed signs of chronic malnutrition that were probably related to basic nutrient deficiencies in the region; we posit that chronic malnutrition may aggravate the deleterious effects of pathogens and climate change on moose survival and productivity. We surmise that the northwest Minnesota moose population currently is not viable and that the numerical decline will persist for the foreseeable future.

Pathogens and Cause of Death

It is understood that the demographic impacts of infectious pathogens have not been well-elucidated for free-ranging moose populations (Lankester and Samuel 1998). Indeed, although both parasites (Gulland 1992, Stein et al. 1999) and diseases (Dobson and Meagher 1996, Joly and Messier 2005) have been implicated in declines of other ungulate species, our study is the first to quantify rigorously the role of pathogens in a moose population die-off. Mortality from pathogens is notoriously difficult to confirm simply from carcass necropsy (Minchella and Scott 1991, Holmes 1995), and although assessment of pathogen effects on moose was largely inductive rather than deductive, our high rates

Table 9. Finite population growth rate (λ) and predicted population size after 50 years (*n*) for hypothetical changes in the percent of mortalities and percent pregnancy in the northwest Minnesota moose population. These results are based on sensitivity analyses and attendant deterministic projections of the population transition matrix derived from the field study. Changes in mortality were applied to all female stage classes, whereas changes in pregnancy rate were applied to yearlings and adults only. We assumed a starting female population size of 442 moose, which assumed sex ratio parity in the 2000–2001 population estimate for northwest Minnesota.

	Change in % pregnancy							
	0		+5		+10		+20	
Change in % mortalities	λ	*n*	λ	*n*	λ	*n*	λ	*n*
0	0.970	96	0.977	138	0.984	197	0.998	400
−5	0.981	169	0.989	254	0.996	362	1.010	767
−10	0.993	311	1.000	442	1.001	465	1.022	1312
−20	1.016	977	1.024	1447	1.031	2034	1.046	4188

of carcass recovery and necropsy results allowed us to draw strong inference upon cause of death. Pathogens were identified as the principal proximate cause of 37–62% of deaths among radiocollared animals, with an additional 25% of mortalities potentially also being pathogen-induced but lacking conclusive necropsy evidence (Table 2). Such high prevalence of mortality from pathogens is exceptional and may indicate that the northwest Minnesota moose population was limited by parasites and diseases. Because of the particular importance of white-tailed deer parasites in observed moose deaths, this finding is consistent with the general hypothesis of parasite-mediated apparent competition (Holt and Lawton 1993, Hudson and Greenman 1998). However, the necessary assumption underlying this conclusion is that pathogen-induced mortality was additive rather than compensatory to other causes of death.

Liver flukes infect moose across most of the region of overlap with white-tailed deer (Lankester and Samuel 1998, Pybus 2001), but where the 2 ungulate species co-occur, flukes can be an uncommon parasite of moose. For example, in Alberta liver fluke prevalence (4%) and intensity (~3 worms/animal) among hunted moose was notably low (Pybus 1990), compared to the high prevalence (87%, Karns 1972; 89%, this study) and intensity (typically >15 worms/moose, this study) observed in northwest Minnesota. Our necropsies revealed extensive evidence of fascioloidiasis (liver rot) and fluke-induced pathology in other moose organs, even among some individuals not necessarily considered to have died from liver fluke parasitism. Overall, our necropsy findings were consistent with those for experimentally infected mule deer (*O. hemionus*) that died of liver fluke infection (Foreyt 1992, see also Pybus 2001), therefore supporting our contention that liver flukes were likely the major pathogen proximally responsible for moose deaths. Previously, Karns (1972, 1973) inferred that liver flukes elicited high calf mortality and poor blood parameters for infected moose in our study area, but evidence for similar effects of this parasite in other moose populations is scant.

Early literature implicated abundance of white-tailed deer and meningeal worm occurrence as determinants of moose abundance and population persistence (Telfer 1967, Karns 1967, Gilbert 1974, Prescott 1974), but the lack of strong corroborative evidence has led to criticism of this hypothesis (Nudds 1990, Schmitz and Nudds 1994, Whitlaw and Lankester 1994*a*,*b*). Currently, the importance of meningeal worm in limiting moose populations in regions of overlap with deer remains in question (Lankester and Samuel 1998, Lankester 2001). We failed to detect high incidence of meningeal worm infection in necropsied carcasses, and the occurrence of parelaphostrongylosis in the radiocollared population was equally rare. We acknowledge that the relatively limited importance of meningeal worm in moose deaths in the present study may be anomalous compared to other moose populations in the southern range. However, we caution that the higher detectability of animals succumbing to this cause of death (Table 2) can lead to an exaggeration of its alleged importance in populations where sampling protocol is exclusively opportunistic collection of dying animals. Clearly, additional work is needed to further elucidate the roles of *F. magna* and *P. tenuis* as proximate and/or ultimate factors implicated in moose population declines in regions of overlap with white-tailed deer (Pybus 2001, Lankester 2001).

We attributed a number of moose mortalities to disease and starvation when evidence of infectious (or possibly noninfectious) disease, pathology, and acute malnutrition (low BMF) were present but the primary agent causing mortality and morbidity could not be distinguished. We were unable to rule out specific lethal infectious diseases as being sources of mortality among northwest Minnesota moose, and our general necropsy findings for many animals were consistent with the symptoms described as "wasting disease" among moose in Sweden and Nova Scotia (Frank 2004). The specific cause(s) of these moose die-offs remains to be fully elucidated because symptoms usually include complex clinical signs and nonspecific pathological findings. However, some researchers believe that viral infection may be implicated given that a retrovirus has been isolated from diseased animals in Sweden (Merza et al. 1994), and symptoms of bovine viral diarrhea and mucosal disease also tend to be prevalent (Rehbinder et al. 2004). Currently, it is unclear if virus is the causative agent as opposed to a secondary infection. Analysis of samples from the present study failed to identify evidence of retrovirus (G. Huschle, personal communication). Regardless, the specific role of infectious diseases in moose population declines in northwest Minnesota and elsewhere requires additional research (Frank 2004).

Acute and Chronic Malnutrition

We failed to detect evidence of density-dependent constraints acting on moose population estimates from aerial counts, nor were deer and moose numbers negatively correlated. Similarly, protein content of moose browse tended to be at or above the 5–7%

protein threshold for maintenance of adult moose during winter (Schwartz and Renecker 1998). Thus, absolute food shortage was unlikely to be important in the moose population decline. However, we did observe evidence of acute and chronic malnutrition, leading to the suspicion that habitat in northwest Minnesota was not adequate for sustaining moose. In relation to acute malnutrition, the low BMF that was prevalent among moose dying of natural causes is characteristic of animals faced with starvation (Mech and DelGiudice 1985). However, we argue that in many such cases evidence of fascioloidosis or other significant pathology was overwhelming and perhaps suggestive of a secondary role of acute malnutrition in moose death. Indeed, Lankester and Samuel (1998) and Pybus (2001) speculated that heavy fluke infections may be associated with malnutrition, and it is reasonable to presume that the extensive damage caused by liver flukes and infectious diseases would naturally lead to moose debilitation, malnutrition, and low BMF at the time of death. Thus, the end point could be either death from the pathogen or from starvation, even though the former agent may have been the driving force behind the mortality.

In theory, pathogens and nutritional status may interact to affect hosts (Crompton 1991, Holmes 1995), with malnutrition leading to immunosuppression and increased parasitism and disease, while pathogens cause host energy depletion and tissue damage. Thus, when co-occurring, the effects of pathogens and malnutrition should be interactive such that host condition and survival are further compromised (Gulland 1992, Murray et al. 1997, Monello et al. 2001, Stien et al. 2002, Gunn and Irvine 2003). Such a scenario is consistent with observations in our study, where liver fluke intensity and BMF exhibited a negative relationship. However, confirmation of the synergistic effects of pathogens and nutrition in moose, as opposed to simple co-occurrence of the 2 agents in compromised individuals, will require a more rigorous experimental approach (Hudson et al. 1992, Murray et al. 1997).

Evidence for chronic malnutrition in moose from northwest Minnesota included the negative relationship between serum-based indices of nutritional status versus survival probability and pregnancy status. Furthermore, up to 69% of dead moose had Cu levels below either estimated minimum requirements for domestic cattle (Gustafson et al. 2000), or mean Cu levels in other North American and Scandinavian moose populations (Custer et al. 2004, Frank et al. 2004*a*). Copper deficiency can influence moose productivity (Flynn et al. 1977), presumably through its effects on immune system function and conditions such as anemia, blood disorders, and cardiac abnormalities (Underwood 1977, Flueck 1994). Postmortem evidence of Cu deficiency typically includes emaciation, weakness, lesions of the mucous membranes of the digestive tract, and atrophied lymphoid organs; these symptoms are consistent with many observations of dead or dying moose in our study, but may also be similar to a range of other pathological conditions. By way of comparison, the aforementioned moose population die-offs in Sweden and Nova Scotia are primarily related to noninfectious disease caused by chronic malnutrition (Frank 2004*a*,*b*). Specifically for the Swedish population, trace-element and biochemical analyses support the hypothesis that molybdenosis and Mo-induced disturbances of Cu metabolism are principally involved in the decline (Broman et al. 2002, Frank et al. 2004*a*), whereas in Nova Scotia, evidence of molybdenosis is lacking but Co and vitamin B_{12} deficiencies have been identified (Frank et al. 2004*b*). Similarities between such case studies and the general pattern of debilitation among moose in our study indicate that moose habitat in northwest Minnesota likely failed to allow animals to reach the prime body condition that is characteristic of populations in the core of the species' range (Karns 1998). More generally, these results suggest that moose may be particularly sensitive to nutritional deficiencies. Indeed, the low levels of several trace elements and basic pathological findings observed in the present study are suggestive of a similar condition between moose in Sweden and Minnesota. In northwest Minnesota, chronic malnutrition may render moose susceptible to pathogen-induced mortality, such that the proximate cause of death was primarily through parasitism. The absence of similar parasites in moose in Sweden logically would lead to mortality from alternate causes. Although the above studies highlight the potential range and complexity of nutritional diseases affecting moose populations, unequivocal assessment of the importance of chronic mineral or trace element deficiency in free-ranging moose is lacking.

Climate Change

Time-series analyses of regional weather data for the last 40 years provided support for the hypothesis that climate change was significant in the region and potentially implicated in the moose population decline. Ungulate population dynamics in general, and moose numbers in particular, are strongly influenced by climate (Crête and Courteois 1997, Saether 1997, Post and Stenseth 1998, Gaillard et al. 2000, Vucetich and Peterson 2004). It follows that when moose numbers are below those where density dependence can constrain numerical growth, density-independent environmental stochasticity may be an important factor affecting population dynamics. The mean winter and summer temperatures in northwest Minnesota were higher, the growing season averaged 12 days longer, and the March and September thermoregulation thresholds for moose were exceeded more frequently during the postpeak period. The disparity between pre- versus postpeak mean values for most of these indices was substantive (Table 7), and these patterns were generally consistent with observations of recent climate change severity across central North America (Intergovernmental Panel on Climate Change 2002). If the southern range limit of moose is determined by thermoregulatory restrictions and heat stress (Kelsall and Telfer 1974; Renecker and Hudson 1986, 1990), then it should not be surprising that moose numbers in northwest Minnesota have declined. In fact, the observed population decline may simply reflect a recent northward shift in the thermoneutral zone for this species. Other southern moose populations presumably subject to similar climate change also have exhibited comparable trends in recent years, although proximate causes of those declines have been diverse (Karns 1998). It seems possible that climate change played a supporting role in several of these die-offs.

The specific implication of climate change in moose population dynamics in northwest Minnesota is not well understood, but the negative association between population rate of change and summer temperature at $t - 1$ points to heat stress as a contributing factor (Renecker and Schwartz 1998, Schwartz and Renecker

1998). A direct link may exist between summer heat stress and body-condition deterioration that could translate into energy loss and general malnutrition and immunosuppression, which then would logically lead to reduced productivity, higher mortality, and ultimately population decline. However, climate may also influence moose numbers indirectly through a range of ecosystem-level changes, and therefore a rigorous assessment of climate effects on moose populations will require an integrated approach involving multiple levels of analysis (Parmesan and Yohe 2003, Schmitz et al. 2003).

Population Demography

Survival rates for adult females were low relative to other moose populations (range = 0.75–0.94/yr; Peterson 1977, Mytton and Keith 1981, Bangs et al. 1989, Ballard et al. 1991, Modafferi and Becker 1997). In contrast, observed calf survival rates were higher than average (range = 0.20 to 0.27/yr; see Hauge and Keith 1981, Testa 2004), owing to the atypically low rate of neonate predation compared to other areas across the species distribution (Franzmann et al. 1980, Gasaway et al. 1983, Ballard et al. 1991, Ballard and Van Ballenberghe 1998, Timmermann and Buss 1998). Given these survival rates and our necropsy results, negative effects of pathogens and malnutrition probably began in earnest among the yearling age class and worsened progressively with age, implying that moose did not acquire immunity to pathogens and that their deleterious effects were cumulative (Lankester and Samuel 1998, Lankester 2001, Pybus 2001).

The few old-aged females (i.e., >10 yr) and virtual absence of older (i.e., >7 yr) males in the carcass sample led to a population age structure skewed toward the younger age classes and characteristic of declining populations. Although caution is warranted in interpreting the demographic implications of population age structure in the absence of additional data (Caughley 1966, 1977), our survival results offer more definitive evidence of high mortality risk, especially among older animals. The low proportion of male carcasses in our recovered sample suggested that the population was skewed toward females, which is consistent with our observation of higher male mortality among radiocollared juveniles and the general understanding that in unhunted moose populations males typically suffer higher mortality (Ballard et al. 1991, Miquelle et al. 1992). Yet this interpretation conflicts with the high male:female ratios from annual surveys for the region after hunting-season closure (concurrent with our field study). Although this disparity is difficult to reconcile in the absence of adult male survival data, a detection bias for males during aerial survey (Gasaway et al. 1986) may be responsible. Regardless, because male:female ratios never ranged below 50% during the study, we suspect that fecund females should have had access to sufficient numbers of males for breeding. However, prime-aged males clearly were rare in the studied population, and although younger males certainly are capable of impregnating adult females (Schwartz et al. 1982), both their ritualistic courting behavior (Bubenik 1998) and spermatogenesis (Bubenik and Timmermann 1982) may be compromised. It follows that at this stage we are unable to fully discount younger age of males as contributing to low pregnancy rates in the population. Alternatively, younger males may breed later during the rut and thereby induce long-term influences on calf viability (Cook et al. 2004).

Pregnancy and calving rates in the present study were consistently <50% and the lowest in comparison to other North American moose populations (Simkin 1974, Chriton 1992, Schwartz 1998, Testa 2004). Boer (1992*b*) indicated that adult pregnancy rates were remarkably consistent across North America and averaged 84%, whereas Gasaway et al. (1992) suggested that pregnancy ranged from 60–100% and was heavily influenced by density-dependent constraints. The absence of density-dependence in moose numbers in northwest Minnesota implies that low pregnancy rates were likely related to nutritional stress that was independent of food availability. Normally, moose fertility rates peak between 4–7 years of age (Sylven 1980, Saether and Haagenrud 1983, Schwartz and Hundertmark 1993), with yearling pregnancy rates often serving as an indicator of reproductive performance in the population (Schwartz 1998). Overall, our low yearling pregnancy rates (<20%), and low twinning rates (19%), were atypical for this species (Gasaway et al. 1992, Schwartz 1998). Collectively, these results argue strongly that the moose population in northwest Minnesota was subject to chronic nutritional limitation affecting female fertility and fecundity.

MANAGEMENT IMPLICATIONS

Our results provide support for the role of pathogens and climate change in a moose population decline, with further contribution from nutritional deficiency in northwest Minnesota's moose habitat. Across the southern moose range, current conditions favor white-tailed deer population persistence and growth, which should facilitate sustained transmission of *F. magna* and *P. tenuis* to moose. Notably, because moose are aberrant hosts of these parasites (Lankester and Samuel 1998), the host–parasite relationship between moose and the 2 helminth species need not be density dependent. Rather, parasite transmission and moose infection may be linked simply to the abundance of reservoir hosts (deer), and thus, parasitism may be inversely density dependent across the range of species overlap in North America. In theory, inverse density dependence alone can drive populations to extinction (Courchamp et al. 1999, Stephens and Sutherland 1999), and when combined with the low productivity that is characteristic of peripheral moose populations (Karns 1998) and the alleged northward shift in the moose thermoneutral zone, the long-term prognosis for many southern moose populations may be particularly dire (Fig. 14). A similar scenario has been envisioned for southern caribou (*Rangifer tarandus*) populations subject to inversely density-dependent predation combined with the effects of habitat deterioration (Wittmer et al. 2005).

If the above model is correct, then a clear change in the distribution of moose in North America is predicted. Yet, the sensitivity analysis consequent to our demographic PVA indicated that relatively modest changes in moose survival and productivity could generate a transition matrix promoting moose population growth, particularly when co-occurring. Although this favorable outcome may suggest that southern moose population restoration via intervention and management may be attainable, several points fuel our skepticism. First, the sensitivity analysis was restricted to

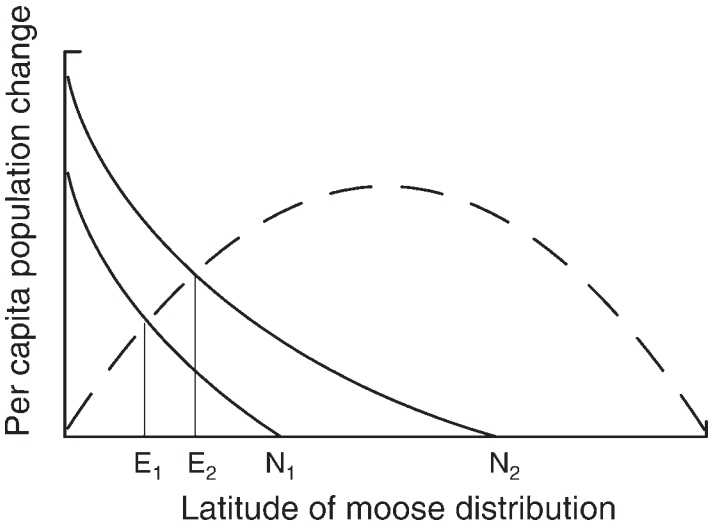

Figure 14. Conceptual model of the relationship between deer parasites and moose distribution in North America. The dashed curve represents per capita population increase of moose; declines at the distribution limits are through natural constraints (poor habitat, competition, predation). The solid curves represent reductions in moose survival and productivity due to current (lower line) and future (upper line) effects of parasitism corresponding to increased deer abundance due to further habitat and climate change. Effects of parasitism decline to zero as current (N_1) and future (N_2) northern limits of the deer distribution are reached. Where population losses from parasitism exceed per capita gains (below current and future extinction thresholds [E_1 and E_2], respectively), moose populations are not sustainable and local extirpation is predicted. Because future climate change alone may shift the dashed curve to the right, parasitism may not be required to cause the predicted extirpation.

deterministic projections, but it is understood that even populations with positive growth rates can go extinct due to the variability inherent in vital rates (Saether et al. 1996, Morris and Doak 2002); it seems unlikely that management efforts could alter moose-population demography and variability sufficiently to curtail the decline fully. Second, climate-change predictions for central North America (Intergovernmental Panel on Climate Change 2002) reveal that the environment in the southern range likely will become even less hospitable to moose in the next century; further northward shifts in the thermoneutral zone for this species may simply preclude their occurrence across much of their current southern distribution. Third, further climate and habitat change should benefit deer population numbers, although it is difficult to predict specifically how such changes will impact deer parasites and their potential transmission to moose (Holmes 1996, Harvell et al. 2002). In theory, any management action designed to rescue the moose population from its extinction vortex (e.g., increased deer harvest, moose hunting closure, parasite control) will need to project the population rate of change above an unstable equilibrium (E_1, Fig. 14). However, this equilibrium may continue to increase consequent to further environmental change (E_2, Fig. 14), thereby requiring that management actions span prohibitive spatial and temporal scales.

We acknowledge that our conceptual model is speculative and may not apply to the entire southern distribution of moose. For example, pathogen-induced moose mortality may not be ubiquitous across the southern moose range because both *F. magna* and *P. tenuis* currently occupy restricted distributions across this region (Lankester 2001, Pybus 2001). Environmental deficiencies in trace elements also may be localized. However, even in the absence of these particular stressors it seems likely that climate change alone should be sufficient to force the southern distribution of moose northward, given the low productivity (Karns 1998) and thermoregulatory constraints (Renecker and Hudson 1986, 1990) allegedly characterizing marginal habitat in the species' southern range. This can be considered simply as a sharpened decline in the per capita rate of moose population change at lower latitudes (i.e., shift to the right of the dashed curve in Fig. 14). Such changes are likely to span the southern distribution of moose, and therefore may elicit a prevalent numerical decline regardless of the occurrence of other contributing factors. Thus, it seems possible that many southern moose populations may simply be doomed to extinction irrespective of any intervention, and that redirection of management efforts and funding toward species potentially benefiting from such attention is advisable.

KEY POINTS

- We studied the cause of a moose population decline at the southern edge of the species' distribution in northwestern Minnesota (1995–2000).
- Our work revealed that pathogens were the most prevalent proximate cause of mortality in the population; deaths due to starvation also were relatively common.
- Pregnancy rates were among the lowest recorded for moose and evidence of chronic malnutrition was observed in the population.
- In the last 40 years, summer and winter temperatures increased steadily in the study area and time-series analysis revealed a negative relationship between summer temperature and moose population growth rate.
- We conclude that climate is acting in tandem with pathogens and chronic malnutrition to cause a decline in the moose population in northwestern Minnesota.
- We suggest that climate change may prompt a widespread northward shift in the southern distribution of moose in North America.

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Eric Cox conducts a field necropsy on research moose DO957 in June 1998 in northwest Minnesota, USA. Photo by Peter Harrison.