Management and Conservation Article

Temperature Mediated Moose Survival in Northeastern Minnesota

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ABSTRACT The earth is in the midst of a pronounced warming trend and temperatures in Minnesota, USA, as elsewhere, are projected to increase. Northern Minnesota represents the southern edge to the circumpolar distribution of moose (*Alces alces*), a species intolerant of heat. Moose increase their metabolic rate to regulate their core body temperature as temperatures rise. We hypothesized that moose survival rates would be a function of the frequency and magnitude that ambient temperatures exceeded the upper critical temperature of moose. We compared annual and seasonal moose survival in northeastern Minnesota between 2002 and 2008 with a temperature metric. We found that models based on January temperatures above the critical threshold were inversely correlated with subsequent survival and explained >78% of variability in spring, fall, and annual survival. Models based on late-spring temperatures also explained a high proportion of survival during the subsequent fall. A model based on warm-season temperatures was important in explaining survival during the subsequent winter. Our analyses suggest that temperatures may have a cumulative influence on survival. We expect that continuation or acceleration of current climate trends will result in decreased survival, a decrease in moose density, and ultimately, a retreat of moose northward from their current distribution. (JOURNAL OF WILDLIFE MANAGEMENT 73(4):503–510; 2009)

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The earth is in the midst of a pronounced warming trend, and more substantial changes in temperature and precipitation patterns are expected during the next century (Houghton et al. 2001). Direct effects of this warming may include altered mortality and reproductive rates that ultimately result in a shift in wildlife species distribution (Humphries et al. 2004). In the Great Lakes region of North America, predictions call for an increase of 1.5° C to 2° C in spring and summer temperatures as early as 2025–2035 and as much as 5° C to 8° C in autumn and winter temperatures over the next century (Union of Concerned Scientists 2003). Wildlife species on the southern edge of their distribution will likely experience a shift northward (Parmesan and Yohe 2003, Humphries et al. 2004). Knowledge of the response of wildlife to changes in environmental conditions, therefore, is paramount to species management.

Prior to European settlement, moose (*Alces alces*) were found throughout the forested portion of northern Minnesota, USA (Idstrom 1965). Alteration of habitat by logging, mining, and homesteading, combined with subsistence and market hunting, and perhaps disease, dramatically reduced their range to only the most isolated portions of northern Minnesota (Surber 1932). Moose numbers slowly recovered and by the early 1970s were distributed in 2 disjunct populations in the northeastern and northwestern portions of the state. By the mid-1980s the northwestern population contained as many as 4,000 moose (Murray et al. 2006). This population underwent a substantial decline (Murray et al. 2006), and <100 moose were estimated from a 2007 aerial survey (Lenarz 2007). Murray et al. (2006) concluded that climate change acting in tandem with pathogens and malnutrition was responsible for this decline. An estimated 7,600 moose occur in northeastern Minnesota and aerial surveys suggested that until recently, moose numbers were stable (Lenarz 2008).

Several factors have been identified as influencing the biogeographical distribution of moose including food supply, climate, and habitat composition (Kelsall and Telfer 1974). Based on metabolic research, Renecker and Hudson (1986) indicated that moose are intolerant of heat but superbly adapted to cold and that summer temperatures may well define their southerly distribution. When winter temperatures were >−5° C or summer temperatures were >14° C, moose experienced an increase in metabolism and heart and respiration rates (Renecker and Hudson 1986, 1990), reduced feed intake (Belovsky and Jordan 1978, Renecker and Hudson 1986), and reduced body weight (Renecker and Hudson 1986). When ambient air temperatures exceeded 20° C, moose resorted to open-mouthed panting to regulate core body temperature (Renecker and Hudson 1986). Schwartz and Renecker (1997) suggested that heat stress was particularly difficult in the spring when moose were still in their winter coats. Thus, these temperature thresholds represent physiological thresholds that, when exceeded, represent heat stress that increases the energy expenditure needed to stay cool.

As climate change progresses, heat stress will increase. In moose, this will likely result in increased energy expenditure and reduced food intake, both of which would result in a

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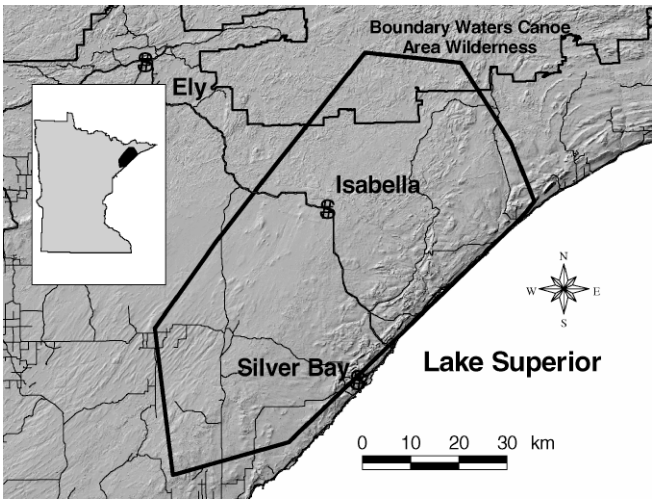

Figure 1. Minimum convex polygon of area in northeastern Minnesota, USA, used by adult moose included in our study, 2002–2008.

loss of body weight or a failure to accumulate fat reserves. Ungulates in northern latitudes are particularly dependent on the accumulation of fat reserves to survive winter (Mautz 1978, Verme and Ullrey 1984). In addition, cattle exposed to heat stress had markedly reduced white blood cell numbers (Morrow-Tesch et al. 1996), which suggests a reduction in the animal's resistance to environmental pathogens (Hahn 1999). Regardless of mechanism, exposure to heat stress would be part of a cumulative process that may ultimately result in death. We hypothesized that moose survival rates would be a function of heat stress, defined as the frequency and magnitude that ambient temperatures exceed the thresholds identified earlier (Renecker and Hudson 1986). Our primary objective was to estimate annual and seasonal survival rates in adult moose in northeastern Minnesota and determine whether heat stress explained variation in these survival rates.

STUDY AREA

We radiocollared and monitored moose within a 3,780-km^2 study area in northeastern Minnesota (47°30′N, 91°21′W; Fig. 1). The area was a low plateau of modest relief that rose abruptly from Lake Superior to a crest approximately 700 m above sea level. A continental divide runs northeast–southwest down the middle of the plateau with water flowing northwest into the Hudsonian watershed or southeast into Lake Superior. Wetlands, including bogs, swamps, small to medium-sized lakes, and small streams were interspersed with rolling uplands (Heinselman 1996).

The study area fell within the Northern Superior Upland section (Minnesota Department of Natural Resources [MNDNR] 2007). The landscape was a mosaic of conifer communities characterized by northern white cedar (*Thuja occidentalis*), black spruce (*Picea mariana*), and tamarack (*Larix laricina*) on the lowlands and balsam fir (*Abies balsamea*), and jack (*Pinus banksiana*), white (*P. strobus*), and red pines (*P. resinosa*) on the uplands. Deciduous species intermixed with conifers on uplands and included quaking aspen (*Populus tremuloides*) and white birch (*Betula papyrifera*). Open lands were characterized as upland and lowland deciduous shrub and sedge meadows.

Moose hunting occurred each year in northeastern Minnesota with a limited number of permits issued to State and tribal licensed hunters. Beginning in 2007, most hunters were restricted to harvesting males and approximately 82 moose (approx. 4% of estimated population) were harvested in the study area (M. S. Lenarz, Minnesota Department of Natural Resources, unpublished data).

People sparsely inhabited the area, and communities within the study area contained <100 permanent residents. Few paved roads existed and much of the area was accessible only from logging roads or abandoned railroad grades. Approximately 74% of the land fell within the Superior National Forest with the balance in state, county, or private ownership.

Average monthly maximum temperatures exceeded 14° C between May and September with July being the warmest month (average high of 26° C; National Oceanic and Atmospheric Administration [NOAA] 2001–2008). The average monthly high temperature stayed <−5° C from December to February with January being the coldest month (average high of −10° C, NOAA 2001–2008). Total annual precipitation averaged 71 cm with 55% occurring between June and September. Precipitation usually occurred as snow between late October and mid-April and snow sometimes accumulated >100 cm (NOAA 2001–2008).

METHODS

Field Procedures

We captured adult male and female moose (≥1.7 yr old) by net-gunning (2002; Wildlife Capture Services, Marysvale, UT) or darting (2003–2005; Quicksilver Air, Inc., Fairbanks, AK) from helicopters in February or early March. Beginning in 2003, we sedated moose with a mixture of 1.2 ml (4.0 mg/ml) carfentanil citrate (ZooPharm, Laramie, WY) and 1.2 ml (100 mg/ml) xylazine HCl (Midwest Veterinary Supply, Inc., Burnsville, MN) administered from a Palmer Capchur® dart (Douglasville, GA). The antagonist was 7.2 ml (50 mg/ml) naltrexone HCl (ZooPharm) and 3 ml (5 mg/ml) yohimbine HCl (Midwest Veterinary Supply). We monitored rectal temperatures continuously while moose were immobilized. Beginning in 2003, we extracted a last incisor (I_4) tooth following procedures similar to Nelson (2001). We aged adult moose by counting cementum annuli (Matson's Laboratory, Milltown, MT) of incisors (I_4) extracted at capture or I_1 postmortem. We fitted each moose with a very high frequency radiocollar (Advanced Telemetry Systems, Isanti, MN). Collars were equipped with motion-sensing–mortality detectors, which increased the pulse rate when the collar was motionless for >6 hours. Animal capture and handling protocols met the guidelines recommended by the American Society of Mammalogists (Gannon et al. 2007).

Each year we estimated moose numbers using a helicopter survey to fly transects on a stratified random sample of plots

(Lenarz 2008) and we corrected observations using a sightability model (Anderson and Lindzey 1996, Quayle et al. 2001). We monitored radiocollared moose weekly for mortality using fixed-wing aircraft from February 2002 to March 2008. When radiocollars emitted a mortality signal, we homed in on the collar on the ground and examined the site for evidence of predators and a struggle (Roffe et al. 1996) or other causes of mortality. We conducted a gross field necropsy looking for evidence of trauma, parasites, or disease if carcass condition permitted. We sent frozen or formalin-preserved tissues to a diagnostic lab (University of Minnesota Veterinary Diagnostic Laboratory, St. Paul, MN) for pathogen examination.

Data Collection and Statistical Analysis

Daily maximum and minimum temperature data from 1 March 2001 to 31 October 2002 were recorded at Ely, Minnesota (approx. 37 km NW of study area; NOAA 2001–2002). We collected temperature data from 1 November 2002 to 28 February 2008 using a temperature recorder (Barnstead/ERTCO, West Patterson, NJ) located within the study area. The recorder was located on the north side of a spruce tree with 100% canopy closure to ensure shading. Maximum and minimum temperatures recorded daily on the study area were highly correlated with data from Ely (1 Nov 2002–29 Feb 2008; NOAA 2002–2008; max. $r =$ 0.97, min. $r = 0.99$). Hence, use of Ely temperature data prior to the establishment of a temperature recorder in the study area was justified.

We reasoned that because effects of heat stress would be cumulative, mortality would occur following a period during which moose were exposed to heat stress, and we selected 4 periods to represent this stress: warm season (1 Apr–31 Oct), cold season (1 Nov–31 Mar), late spring (1 Apr–31 May), and January (Table 1). We used warm- and cold-season periods because predictions call for increased summer and winter temperatures (Union of Concerned Scientists 2003). We used late spring because Schwartz and Renecker (1997) suggested that heat stress was particularly difficult in spring when moose were still in their winter coats. Finally, we used January as a period because it was the coldest month with the highest variability in mean temperature.

Corresponding to moose metabolic thresholds (Renecker and Hudson 1986), we tabulated the number of days when daily maximum and minimum temperatures exceeded 14° C and 20° C during the warm season and during late spring. Similarly, we tabulated the days >-5° C during the cold season and during January. We then summed the temperature difference each day that a threshold was exceeded for a heat stress index (HSI). We considered this metric to reflect the increased energy expenditure needed for moose to maintain thermal balance.

We calculated annual and seasonal survival using the Kaplan–Meier procedure (Kaplan and Meier 1958) modified for a staggered-entry design (Pollock et al. 1989). We censored all moose that died from anthropogenic causes (i.e., capture mortality, hunting, poaching, or collisions with vehicles), moose that emigrated, lost their collar, or lost radio contact, and moose still alive as of 1 March 2008. We reasoned that anthropogenic mortality would be unrelated to heat stress. We partitioned survival data into 6 annual periods (1 Mar–29 Feb) from 2002 to 2008. In a similar manner, we partitioned data into 24 seasonal periods (4 seasons $\times$ 6 yr) based on intervals identified by Murray et al. (2006) with spring (1 Mar–15 May), summer (16 May–31 Jul), autumn (1 Aug–30 Nov), and winter (1 Dec–28 Feb).

We conducted regression analysis of survival data based on the Cox proportional hazards model (Cox 1972; PROC PHREG, SAS Institute, Cary, NC) to explain the effect of sex on hazard rate. The hazard rate (function) represents the instantaneous risk of death and this analysis tests whether the regression coefficient for sex is zero (i.e., there was no difference between sexes). We then tested the assumption that the hazard ratio (ratio of hazard functions between the 2 sexes) was constant over time by recalculation of the regression using a time-dependent explanatory variable (SAS Institute 2004). We used linear regression (Draper and Smith 1998; PROC REG) to identify relationships between estimates of the dependent variable, annual or seasonal survival, and the independent variable, HSI. In all analyses, survival occurred subsequent to heat stress and there was no overlap between periods. Because estimates of survival were limited ($n = 6$ yr), we restricted our analyses to models containing only one independent variable (Burnham and Anderson 1998, Harrell 2001). We hypothesized that survival would be a linear function of HSI, a measure of the thermal environment to which moose were exposed. We selected models a priori relative to temperature thresholds identified as being important to moose (Renecker and Hudson 1986, 1990), and models reflect the magnitude by

Table 1. Temperature models tested for relationship to annual and season survival of moose in northeastern Minnesota, USA, 2002–2007. Summer thresholds were included in 8 models and the winter threshold in 4 models.

Thresholds:

- 14° C = upper critical temp during summer at which moose experienced increased metabolism and heart and respiration rates (Renecker and Hudson 1986).
- 20° C = temp during summer at which moose began to pant (Renecker and Hudson 1986).
- −5° C = upper critical temp during winter months at which moose experienced increased metabolism and heart and respiration rates (Renecker and Hudson 1986).

Time intervals:

- Warm season (WS) = 1 Apr–31 Oct. Regressions of autumn survival used warm-season data truncated at 31 Jul to eliminate overlap.
- Late spring (LS) = 1 Apr–31 May, to reflect when moose may experience greater stress because of retention of winter coat (Schwartz and Renecker 1997).
- Cold season (CS) = 1 Nov–31 Mar.
- Jan (J) = period during the cold season when heat stress index (HSI) was most variable.

Measures:

- Max. (X) = sum of ° C above threshold during time interval when daily max. ambient air temp exceeded threshold.
- Min. (M) = sum of ° C above threshold during time interval when daily min. ambient air temp exceeded threshold. This metric represents an entire day above the threshold temp.

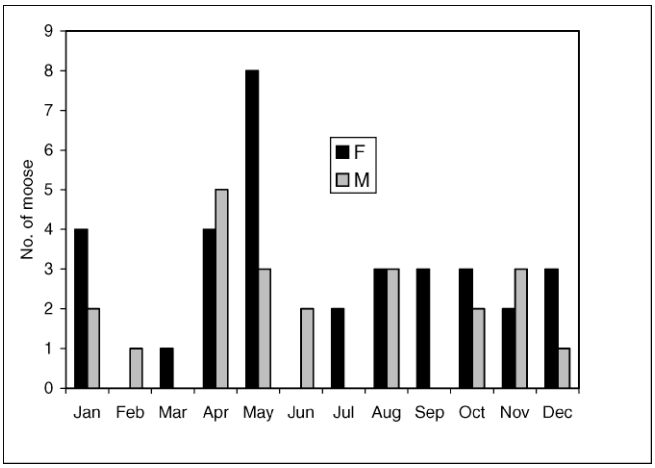

Figure 2. Timing of nonanthropogenic mortality in adult male and female moose in northeastern Minnesota, USA, 2002–2008.

which thresholds were exceeded (Table 1). Models reflect temperature events that preceded calculated survival; for autumn survival, we truncated the warm-season HSI at the end of July to eliminate overlap with calculated survival.

RESULTS

From 2002 to 2005 we captured and radiocollared 116 adult moose (61 F and 55 M). Sex ratio (M:F) of collared animals at the beginning of each year (1 Mar) varied from 0.41 to 0.80 ($\bar{x} = 0.65$, SE = 0.06, $n = 6$). We determined ages for 87% of radiocollared moose and median age at capture was 5.7 years (1.7–19.7, $n = 55$, SE = 0.5) for females and 4.7 years (1.7–14.7, $n = 46$, SE = 0.4) for males. Of the 116 radiocollared moose, 85 (73%) died by 1 March 2008. In addition, one moose apparently slipped its collar, one moose emigrated out of the study area, and we lost contact with one moose. We designated moose that died within 2 weeks of capture (5) as capture mortality. Hunters killed 15 moose, 2 were poached, and 8 were killed in collisions with vehicles (i.e., cars, trucks, or trains). We considered remaining mortality (55) to be nonanthropogenic and causes included wolf (*Canis lupus*) predation (5), bacterial meningitis (1), or unknown (49) and occurred in all months of the year (Fig. 2). We monitored survival of individual radiocollared animals during a mean 876 days (SE = 57, $n = 116$) between February 2002 and March 2008.

Much of the unknown mortality appeared to be non-traumatic. In 49% ($n = 24$) of cases, we found the intact carcass with only minor scavenging by small mammals or birds. Wolves and bears (*Ursus americanus*) were the primary scavengers in 37% ($n = 18$) of the remaining cases. We conservatively did not attribute predation as the cause of death in these cases because there was no clear evidence the animal had been killed rather than simply scavenged. In 14% ($n = 7$) of cases, we were unable to examine the carcasses or only found a collar with tooth-marks.

Incorporating only nonanthropogenic mortality (i.e., moose that died from disease, predation, or unknown causes), annual survival rates for male and female moose averaged 0.84 (SE = 0.05, $n = 6$) and 0.78 (SE = 0.04, $n = 6$), respectively. The Cox proportional hazard model indicated that sex did not contribute to prediction of survivorship ($\chi^2_1 = 0.01$, $P = 0.94$). The dummy value for sex conformed to the proportional hazard assumption ($\chi^2_1 = 0.29$, $P = 0.59$) that the ratios of hazard functions were constant over time. We used survival estimates from pooled male and female data in subsequent regression analyses.

Annual survival rates for the combined data averaged 0.81 (SE = 0.04, $n = 6$) and ranged from 0.68 in 2006 to 0.96 in 2004 (Table 2). Average survival was highest during summer, and there was no mortality in 3 of 6 summers (Table 2). In contrast, average survival was lowest during spring and fall.

The HSI and its variability were dependent on the threshold and season. During the warm season, mean HSI per day was 6.2° C above the 14° C threshold and 2.6° C above the 20° C threshold (Table 3). During the cold season, the mean HSI/day was 5.2° C above the −5° C threshold. Excluding 3 models with insufficient data for analysis (LS14M, LS20M, and WS20M), models based on January temperatures had the highest coefficients of variation ranging from 82 to 86, followed by late-spring temperatures (58–160). In contrast, models based on temperatures in the cold season had substantially lower coefficients of variation ranging from 17 to 21.

Regression analyses indicated that HSI, as calculated for each model, explained a high proportion of variation in seasonal and annual survival. Minimum and maximum temperatures in January (J5M and J5X, respectively) consistently explained >78% ($P < 0.019$) of variability in

Table 2. Annual and seasonal survival for adult moose in northeastern Minnesota, USA, 2002–2008, as calculated following Pollock (1989). Survival based only on nonanthropomorphic mortality including wolf predation, disease, or unknown causes.

	Spring[a]		Summer		Fall		Winter		Annual	
Yr	Survival	95% CI	Survival	95% CI	Survival	95% CI	Survival	95% CI	Survival	95% CI
2002	0.96	0.08	0.91	0.12	0.95	0.10	0.95	0.07	0.79	0.12
2003	0.92	0.07	0.96	0.05	0.92	0.08	0.93	0.06	0.76	0.10
2004	0.98	0.03	1.00	0.00	0.98	0.04	1.00	0.00	0.96	0.09
2005	0.99	0.02	0.96	0.04	0.94	0.06	0.95	0.05	0.85	0.08
2006	0.86	0.08	1.00	0.00	0.86	0.10	0.92	0.08	0.68	0.13
2007	0.91	0.09	1.00	0.00	0.94	0.08	0.97	0.04	0.83	0.08

[a] Seasons follow Murray et al. (2006) with spring (1 Mar–15 May), summer (16 May–31 Jul), autumn (1 Aug–30 Nov), winter (1 Dec–29 Feb), and annual (1 Mar–29 Feb).

Table 3. Statistics for heat stress index (HSI) for each of the models used in regression analyses, which evaluate moose survival in northeastern Minnesota, USA, 2002–2008.

Physiologic threshold[a]	Interval	Daily ambient temp	Model[b]	*n*	Mean annual HSI	Mean HSI/day	SE	CV	Min.	Max.
14° C	Late spring (Apr–May)	Max.	LS14X	6	189	3.1	45	58	45	360
		Min.	LS14M[c]	6	1		1	160	0	3
	Warm season (Apr–Oct)	Max.	WS14X	6	1,329	6.2	99	18	873	1,572
		Min.	WS14M	6	64	0.3	15	58	19	123
20° C	Late spring (Apr–May)	Max.	LS20X	6	57	0.9	20	85	4	141
		Min.	LS20M[c]	6	0		0		0	0
	Warm season (Apr–Oct)	Max.	WS20X	6	552	2.6	72	32	253	793
		Min.	WS20M[c]	6	2		1	150	0	9
−5° C	Jan	Max.	J5X	6	58	1.9	20	86	11	152
		Min.	J5M	6	6	0.2	2	82	0	13
	Cold season (Nov–Mar)	Max.	CS5X	6	779	5.2	53	17	618	1,010
		Min.	CS5M	6	118	0.8	10	22	86	160

[a] Physiologic thresholds based on Renecker and Hudson (1986). When threshold temp were exceeded moose experienced an increase in metabolism and heart and respiration rates, reduced feed intake, and reduced body wt.
[b] Model descriptors: LS = late spring, X = max. temp, M = min. temp, WS = warm season, J = Jan, CS = cold season.
[c] Insufficient HSI for analyses.

spring, fall, and annual survival (Fig. 3a; Table 4). These models also explained >55% ($P < 0.090$) of variability in winter survival, despite that these temperatures occurred 11 months prior to the survival period. Models based on late-spring temperatures (LS14X and LS20X) were also important in explaining variability in autumn survival ($r^2 > 0.835$, $P < 0.011$; Fig. 3b) and to a lesser extent, winter survival (LS14X only, $r^2 = 0.537$, $P = 0.098$). Models based on cold-season temperatures (CS5M) explained ≥68% of variability in spring and autumn survival ($P < 0.044$). The model based on total warm-season maximum temperatures (WS14X) was important in explaining variability in survival the following winter ($r^2 = 0.715$, $P = 0.034$). None of the models were effective at explaining summer survival. In general, models with a higher coefficient of variation (Table 3) tended to explain a higher proportion of the variability in survival.

DISCUSSION

Our results suggest that ambient temperatures in excess of reported physiological thresholds are important in explaining moose survival in northeastern Minnesota. January temperatures, in particular, explained a high proportion of variation in survival both short- and long-term with an inverse relationship between temperature and survival. In addition, above-average temperatures in late spring appear to be important in explaining moose short-term survival during the fall. In contrast, when we measured HSI over longer periods (e.g., warm season or cold season), models provided lower potential for explaining subsequent survival. The HSI measured over several months tended to be less variable (Table 3), perhaps because of an inherent muting effect, and this may result in the lower potential to explain variation in survival.

Those models containing January minimum and maximum temperatures were consistently important in explaining variability in annual, spring, and autumn survival (Table 4), which suggests that increasing winter temperatures may

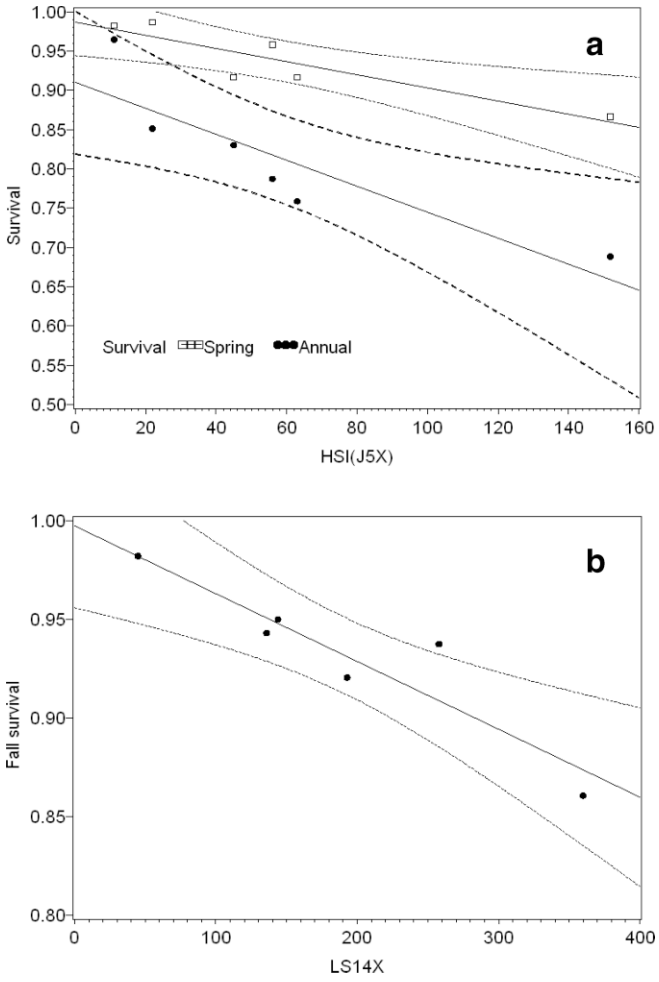

Figure 3. Relationship between (a) heat stress index (HSI) based on maximum temperatures in January (J5X), (b) HSI based on maximum temperatures in late spring (LS14X), and annual and seasonal survival of moose in northeastern Minnesota, USA, 2002–2008. Dashed lines represent 95% confidence interval.

Table 4. Regression statistics of relationships between thermal models and moose survival in northeastern Minnesota, USA, 2002–2008. Models not listed had $r^2 < 0.50$.

Survival period	Model[a]	*n*	Coeff.	SE	*t*	*P*	r^2
Spring (1 Mar–15 May)	J5M	6	−0.00966	0.00205	−4.72	0.009	0.847
	J5X	6	−0.00084	0.00021	−4.01	0.016	0.800
	CS5M	6	−0.00153	0.00052	−2.91	0.044	0.679
	WS14M[b]	6	−0.00105	0.00040	−2.62	0.059	0.631
Fall (1 Aug–30 Nov)	J5X	6	−0.00078	0.00013	−5.93	0.004	0.898
	LS14X	6	−0.00035	0.00007	−5.06	0.007	0.865
	LS20X	6	−0.00078	0.00017	−4.50	0.011	0.835
	J5M	6	−0.00832	0.00193	−4.30	0.013	0.823
	CS5M	6	−0.00140	0.00040	−3.47	0.026	0.750
Winter (1 Dec–28 Feb)	WS14X	6	−0.00010	0.00003	−3.17	0.034	0.715
	J5X[b]	6	−0.00043	0.00017	−2.47	0.069	0.605
	J5M[b]	6	−0.00459	0.00206	−2.23	0.090	0.554
	WS20X	6	−0.00011	0.00005	−2.11	0.102	0.528
	LS14X	6	−0.00019	0.00009	−2.15	0.098	0.537
Annual (1 Mar–28 Feb)	J5M	6	−0.01871	0.00483	−3.88	0.018	0.790
	J5X	6	−0.00168	0.00044	−3.80	0.019	0.783
	CS5M	6	−0.00296	0.00112	−2.64	0.058	0.636

[a] Models descriptors: J = Jan, M = min. temp, X = max. temp, CS = cold season, WS = warm season, LS = late spring.
[b] Based on heat stress index from the preceding yr.

be more important to moose than are warming summers. This could, however, be an artifact of the small sample size ($n = 6$ yr). It should not be inferred that summer heat stress is unimportant to moose. We tested a model of warm-season heat stress that included 7 months and mean HSI per day was highest during this period (Table 3). Further partitioning of the HSI during the warm season (e.g., late spring) suggested that specific portions of the warm season may be more important than others in explaining variability in survival. It is clear from literature on effects of heat stress on cattle that warm summertime temperatures are important (Hahn et al. 1992, 1997; Morrow-Tesch et al. 1996, Hahn 1999). Unlike cattle, however, moose in northeastern Minnesota may be able to ameliorate warmer conditions by using thermal refuges such as conifer swamps or aquatic habitats (Renecker and Hudson 1990, Dussault et al. 2004). That the mean July maximum temperature (measured in the shade) in the study area exceeded 24° C implies that threshold temperatures were exceeded most days and would have resulted in high metabolic costs if thermal refuges were not available. Demarchi and Bunnell (1993) predicted that moose in British Columbia would need ≥26–35% crown closure to remain below their upper critical temperature.

Our study revealed a high proportion of moose that apparently died from non-traumatic causes throughout the year (Fig. 2). In some cases, moose were emaciated; in others, tests indicated that moose were infected with parasites such as brainworm (*Parelaphostrongylus tenuis*), winter ticks (*Dermacentor albipictus*), flukes (*Fascioloides magna*), and tapeworm (*Echinococcus granulosus*; M. S. Lenarz, unpublished data), and in some cases, moose were both emaciated and infected with parasites. Carcass necropsy is a poor method for confirming mortality from specific pathogens (Minchella and Scott 1991, Holmes 1995), and we were generally unsuccessful in assigning specific parasites or diseases as a causative agent. In the absence of other identifiable causative agents, we inferred that infective pathogens (disease or parasites) or malnutrition were likely proximate causes of mortality. Murray et al. (2006) were more successful in quantifying the role of specific pathogens in a moose population die-off in northwestern Minnesota and suggested that a direct link might exist between heat stress and body condition deterioration, which would result in energy loss, general malnutrition, immunosuppression, and ultimately in mortality. Correlations we found, between warmer temperatures and reduced survival, support this inference. Many animal diseases are kept in check by climatic restrictions on vectors, environmental habitats, and disease-causing agents (Stem et al. 1989, DelGiudice et al. 1997). Changes in temperature regimes may result in a spread of disease and parasites or produce an increase in incidence of disease that would reduce animal productivity or increase animal mortality (Baker and Viglizzo 1998).

MANAGEMENT IMPLICATIONS

Assuming that the cumulative effect of heat stress is body-condition deterioration and that this translates into general malnutrition, immunosuppression, and ultimately, increased mortality (Murray et al. 2006), the increased temperatures predicted by climate models in the Great Lakes region (Union of Concerned Scientists et al. 2003) will likely result in further reductions in survival. Causal attribution of moose survival to warming temperatures is complicated, however, and difficult to test in the wild. Specific hypotheses on the importance of pathogens or the significance of thermal cover in reducing heat stress could be tested but likely would require a captive population or moose outfitted with Global Positioning System radiocollars. Moreover, climate may also influence moose numbers indirectly through a range of ecosystem-level changes (Murray et al. 2006), and a rigorous assessment of climate effects on moose populations would require an integrated approach involving multiple levels of analysis (Parmesan and Yohe 2003, Schmitz et al. 2003).

Regardless of the specific mechanism, we expect that continuation or acceleration of current climate trends will lead to a retreat of moose northward from their current distribution.

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