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Can predation cause the 10-year hare cycle?

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Summary. We relate causes of mortality of snowshoe hares to density of hares over an 8-year period that included a peak in numbers. We then use simulation modeling to examine whether these density-dependent relationships could produce changes in hare density similar to those observed in our study area in Yukon, Canada.

Predation during winter was the largest source of mortality for snowshoe hares at Kluane, Yukon during 1978–84. There was a one-year lag in the response of winter predation mortality rate to hare density. There was a two-year lag in the response of winter mortality not caused by predators to hare density.

A simple simulation model with density-dependent predation produced 8–11 year cycles only within a narrow range of parameters that are inconsistent with data from the Kluane region. However, a simulation model that predicted winter mortality rates using a delayed density-dependent numerical response and a Type II functional response by predators, produced 8–11 year cycles within the range of parameter values measured in our study. Yet another simulation model that predicted both summer and winter mortality rates using a delayed density-dependent numerical response and a Type II functional response by predators, did not produce 8–11 year cycles within the range of parameter values measured in our study. Lack of data on juvenile mortality may be one reason for this result.

Key words: Snowshoe hare – Predation – Population cycle

Population size of the snowshoe hare (*Lepus americanus*) fluctuates with an 8–11 year period in northern boreal forests (Elton 1933; Keith 1963; Krebs et al. 1986). We now know something of the patterns of birth and death that accompany these fluctuations in numbers of hares, commonly known as the 10-year cycle (Cary and Keith 1979; Keith et al. 1984; Boutin et al. 1986).

Keith (1974) proposed a series of mechanisms that might cause the demographic changes of this "10-year cycle". He hypothesized that shortage of winter food for hares is responsible for the initial decline from peak densities, and that predation on hares then drives the population to yet lower densities, at which the food supply can recover.

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Keith et al. (1977) found that the predator numerical response to peak hare densities was delayed. They reported a time lag in predation rates which they estimated from studies of functional and numerical responses of four predators of snowshoe hares in their study area. Thus there is evidence for delayed density-dependent mechanisms in the hare-predator interaction. These authors attributed the delay to superabundant prey at peak hare densities with predators experiencing food shortage (which caused declines in predator numbers) only after hare densities had declined.

Other authors propose that delayed density-dependent mechanisms in the hare-vegetation interaction alone may be responsible for the 10-year cycle. Snowshoe hare densities may respond to cyclical changes in secondary chemicals in their food plants (Bryant 1981). Fox and Bryant (1984) suggest that a delay is introduced into the hare-vegetation interaction when plants that have suffered heavy browsing remain unpalatable to hares for two years.

In this paper we present data on the causes of mortality during the cyclic low in snowshoe hare abundance at Kluane Lake, Yukon. We combine mortality data collected in this study with data reported by Boutin et al. (1986) for a four-year period that spanned a peak in numbers and we construct a model of mortality patterns over a snowshoe hare cycle. Because predation during winter was the largest source of mortality for hares in our study site from 1978–84, we propose the hypothesis that predation alone can account for the cyclic population dynamics observed for snowshoe hares. Predator cycles, documented for lynx by Elton and Nicholson (1942), have left the general impression that this could be a simple predator-prey cycle. Tanner (1975), investigating the conditions for stability of predator-prey systems with no time lags, proposed that the hare cycle could be a stable limit cycle that results from a predator-prey interaction. Schaffer (1984) and Schaffer and Kot (1986) offer an alternative explanation in which chaotic behavior of a 3-level (vegetation, hares, predators) system is responsible for the 10-year cycle.

Simulation models can be used as tools to explore whether a particular hypothesis, expressed as a mathematical relationship, can produce the population dynamics that are observed in nature. In this paper we consider the hypothesis that predation mortality alone, measured at our Yukon study site and acting according to predator response mechanisms reported by Keith et al. (1977) can produce population cycles of the period and amplitude observed in northern boreal forests.

Methods

The study site was located at the south end of Kluane Lake, Yukon (61° North, 138° West). The study area and the trapping methods used are described in Krebs et al. (1986) and Boutin (1980; 1984). Mortality data during 1978–82 are from Boutin et al. (1986), who trapped and radio collared hares on the Silver Creek grid (a 10×10 grid, 30.5 m between stations and traps set at alternate stations).

In 1983–85, when hare densities were lowest, we expanded trapping effort to obtain a large enough sample of radio-collared hares. We used a 5 km^2 study area that included the Silver Creek grid, and was bounded on one side by the Alaska Highway, on two sides by local access roads, and on the fourth by Kluane Lake. Traps were set on well-used runways within this area. All hares weighing 700–1000 g were given radio collars weighing 35 g, and some weighing over 1000 g were given collars weighing up to 55 g. Some of the lighter radio collars were equipped to permit detection of activity (Wildlife Materials Inc.). All heavy radio collars (55 g) were equipped to detect mortality with motion-sensitive switches that caused the radio pulse rate to double if the collar did not move for 4 hours (Advanced Telemetry Systems). Sample sizes were limited by the number of radio collars available, or by the numbers of hares trapped.

Field techniques for monitoring activity of hares and identifying the cause of death of radio-collared hares are described by Boutin et al. (1986). Radio-collared hares that were suspected to have died were tracked down using a hand-held antenna and receiver. Deaths of radio-collared hares were classified as predator kills if there were obvious signs of a chase in the snow, blood on the collar, or a predator actually feeding on the kill. Scavenging was probably rare because carcasses left in the study area and checked periodically remained undisturbed. When entire carcasses were recovered they were skinned and examined for sub-cutaneous bruises that were taken as evidence of surplus killing (Kruuk 1972). If the carcass was free of bruises or other indications of having been predated, the death was classified as due to causes other than predation.

Keith et al. (1984) argued that radio collars decrease survival of hares for a week subsequent to collaring. However, Boutin et al. (1986) found little evidence of this in the Kluane population. The proportion of hares lost within one week of collaring was never significantly higher than the proportion of hares lost within any one of the following three weeks. Therefore, we assumed that radio collaring had no effect on hare survival.

We lumped all demographic data into two 6-month periods: winter-spring (December 1 to May 31), and summer-autumn (June 1 to November 31). Hare population densities and recruitment rates of juveniles were taken from Krebs et al. (1986), and from the unpublished data of a long-term monitoring program.

The details of summer demography are incomplete because it is difficult to census jevenile hares. We began to catch juveniles in live-traps at three weeks of age, and we were unable to attach radio-collars to them until they were approximately four weeks old.

Key factor analysis (Varley and Gradwell 1960) was used to identify which mortality factors contributed most to changes in total mortality. The k-values were calculated as $\log_{10}$ (population size at the beginning of a season) – $\log_{10}$ (population after mortality factor has acted). Potential recruitment losses, $k(rec)$, were calculated as $\log_{10}$ of maximum potential natality per female minus the $\log_{10}$ of juvenile recruitment per adult female, from Krebs et al. (1986). Maximum potential natality was the maximum value obtained from in utero counts by Cary and Keith (1979). Therefore, $k(rec)$ was the maximum loss due to decreased natality and early juvenile mortality. Total K was calculated only for those years in which both summer and winter mortality data were available. We used the method of Podoler and Rogers (1975) to rank key factors in order of importance. By this method, those factors with the steepest slopes when plotted against total K contributed the most to changes in overall mortality.

Results

Key factors

The causes of death of radio-collared hares are presented in Table 1 along with "hare exposure-days", the cumulative number of days that hares with collars were in the field. The large number of deaths attributed to unknown causes in winter-spring 1983 was due to receiver failure. We were unable to recover collars until long after the hares had died so it was impossible to determine cause of death. Predation

Table 1. Causes of death of radio-collared hares

Season	Year	Hare days	Number of Deaths				
			Non-predation	Unknown predator	Avian predator	Terrestrial predator	Unknown
Winter-Spring	78–79	2093	2	8	2	1	1
Winter-Spring	79–80	4417	6	12	6	2	0
Winter-Spring	80–81	4474	5	29	5	14	5
Winter-Spring	81–82	1547	10	12	3	5	1
Winter-Spring	83–84	2289	3	5	0	7	15
Winter-Spring	84–85	3011	1	3	2	5	5
Summer-Fall	78	2870	0	4	2	1	2
Summer-Fall	79	3857	1	3	2	0	3
Summer-Fall	80	7056	4	19	4	8	3
Summer-Fall	81	4284	1	16	3	5	6
Summer-Fall	84	3213	2	2	3	1	4
Summer-Fall	85	1413	0	3	3	0	5

Table 2. Probabilities of mortality for radio collared hares

Interval		Source of Mortality		
Season	Year	Non-predation	Predation	Unknown
Winter-Spring	78–79	0.16	0.62	0.08
Winter-Spring	79–80	0.22	0.56	0.00
Winter-Spring	80–81	0.18	0.84	0.18
Winter-Spring	81–82	0.69	0.91	0.11
Winter-Spring	83–84	0.21	0.66	0.70
Winter-Spring	84–85	0.06	0.46	0.26
Summer-Fall	78	0.00	0.36	0.12
Summer-Fall	79	0.05	0.21	0.13
Summer-Fall	80	0.10	0.55	0.07
Summer-Fall	81	0.04	0.64	0.20
Summer-Fall	84	0.11	0.29	0.20
Summer-Fall	85	0.00	0.54	0.48

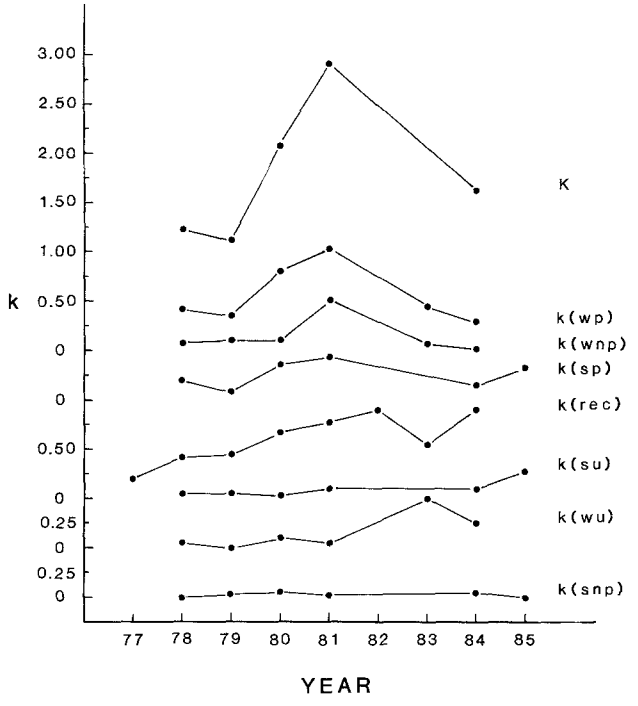

Fig. 1. K-values obtained from telemetry data plotted against time. Winter predation mortality, *k(wp)* contributes most to total mortality (*K*). *K(wnp)* is population loss due to winter non-predation mortality. *K(wu)* is population loss due to unknown winter mortality. *K(rec)* is maximum population loss due to decreased natality and juvenile survival. *K(sp)* is population loss due to summer predation mortality. *K(snp)* is population loss due to summer non-predation mortality. *K(su)* is population loss due to unknown summer mortality

was the largest source of mortality of radio collared hares during both seasons in all years of the study (Table 2). Seasonal probabilities of mortality were calculated according to Trent and Rongstad (1974) as: $1 - ((\text{hare exposure-days} - \text{deaths})/\text{hare exposure-days})^t$ where t is the number of days in the season.

Predation during the winter, *k(wp)*, was the most important factor influencing total mortality (Fig. 1). This observation is supported by the *k*-factor slopes presented in

Table 3. Slopes of *k*-factors on overall *K*

Factor		Slope	
Winter predation	*k(wp)*	0.401*	$P<0.025$
Winter non-predation	*k(wnp)*	0.212*	$P<0.05$
Summer predation	*k(sp)*	0.185*	$P<0.01$
Recruitment	*k(rec)*	0.162	$P>0.05$
Winter unknown	*k(wu)*	0.021	$P>0.05$
Summer unknown	*k(su)*	0.013	$P>0.05$
Summer non-predation	*k(snp)*	0.008	$P>0.05$

Table 3. According to the slope comparison method, predation in winter was followed, in order, by winter mortality not caused by predators, predation in summer and loss in recruitment. Unknown mortality in both summer and winter and mortality due to causes other than predation had slopes near zero and, therefore, did not explain changes in overall mortality.

Density-dependent relationships

Relationships between hare density at the beginning of each period and probabilities of mortality observed during that period are illustrated in Figs. 2 and 3. Mortality due to winter predation changed in a counter-clockwise cyclic fashion when plotted sequentially against hare density in the same time period (*t*). This counter-clockwise pattern shows a delayed mortality response, or time lag, in which mortality increased only after hare densities had begun to increase and decreased after have densities had already declined (Fig. 2a). When winter mortality was plotted against population density the previous year (Fig. 2b), this time lag disappeared and the relationship between predation mortality and hare density became linear. Summer predation mortality (Fig. 2d, e, f) exhibited a pattern similar to that of winter predation mortality.

Mortality not due to predation was lower than that due to predation, and the former followed a different pattern with respect to hare density (Fig. 3). Non-predation mortality relative to hare density also exhibited the counter-clockwise cycle in winter (Fig. 3a), again suggesting a time lag. The counter-clockwise cycle, however, is dependent on a single data point. In contrast to mortality from predation, mortality from other causes remained at low levels in all years except the year following peak hare density (1981). Winter mortality due to other causes was more closely related to hare density two years earlier (Fig. 3c) than to densities one year earlier (the time lag seen in winter predation mortality). Summer mortality from causes other than predation was not related to hare density in the current year or in previous years (Fig. 3d, e, f).

Recruitment was calculated as the mean value over all five control grids (Krebs et al. 1986) of the ratio:

$$\frac{\text{Number juveniles on grid from June (year } t\text{) to April (year } t+1\text{)}}{\text{Number breeding females on grid in May or June (year } t\text{)}}$$

When recruitment rates were plotted against population density on June 1 (Fig. 4) they showed a clockwise cycle. Change in recruitment rate was linearly related to population density three years in the future; however this relationship depended on a single point. If we discarded the first

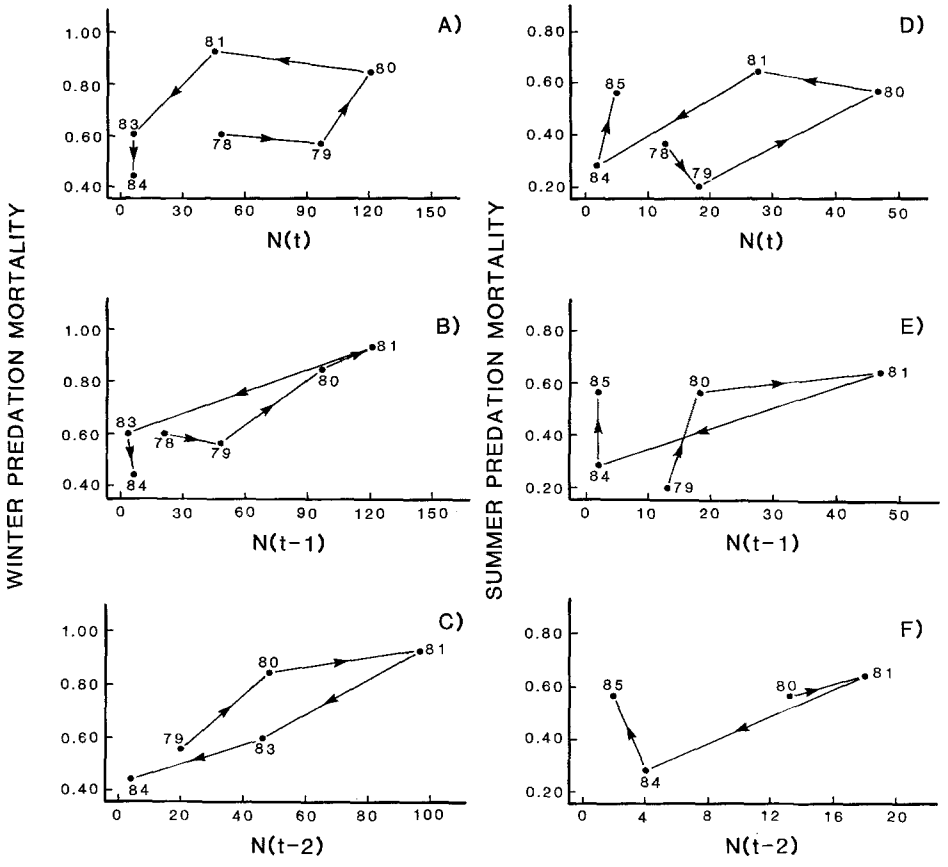

Fig. 2A–F. Predation mortality probability vs population size for a 25.6 ha area, plotted sequentially for years 1978 to 1985.
A–C winter predation mortality against population size in the same year (t), the year before ($t-1$) and two years before ($t-2$).
D–F summer predation mortality against population size in the same year (t), the year before ($t-1$) and two years before ($t-2$)

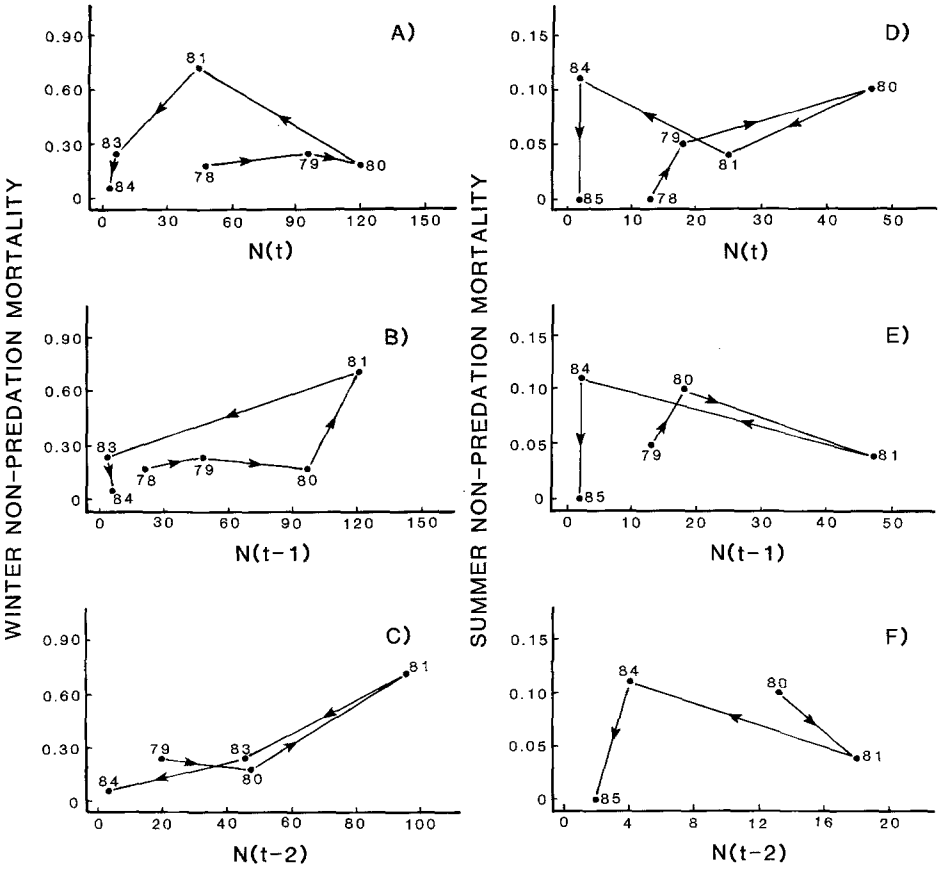

Fig. 3A–F. Non-predation mortality probability vs population size for a 25.6 ha area, plotted sequentially for years 1978 to 1985.
A–C winter non-predation mortality against population size at time t, $t-1$, $t-2$ respectively.
D–F summer non-predation mortality against population size at time t, $t-1$, $t-2$ respectively

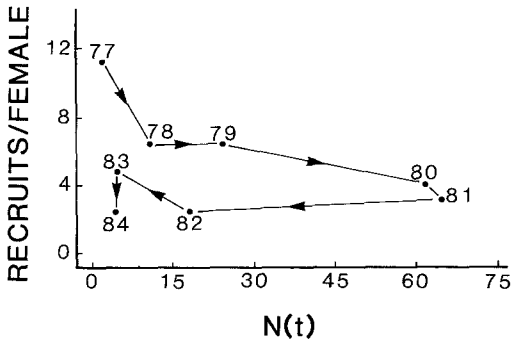

Fig. 4. Mean recruitment index vs mean population size for a 25.6 ha area, measured over 5 trapping grids. Recruitment index is the number of juveniles captured between birth and the following April 1 per adult female present in May and June each summer (Krebs et al. 1986)

year of data, which was based on a single adult female on each of two grids, there was no significant relationship between density and recruitment.

Population models based on density-dependent mortality

Linear density-dependent model

We used simulation models to see whether any of these relationships between mortality and density could produce a ten-year cycle of the amplitude and period observed in our study area. We projected population size (N) into the future holding recruitment rates constant and applying survival probabilities that are linearly related to population size in previous years. Population size in subsequent years was calculated by the equation:

$$N(t+1)=N(t)*R*S \tag{1}$$

where R, the population growth rate, was equal to 1+(recruitment rate per female/2) and S, the survival rate, was a linear function of population size in a previous year. Thus, for winter predation mortality acting with a one-year lag,

$$S=\text{intercept}+(\text{slope}*N(t-1)) \tag{2}$$

Parameter estimates for survival after winter mortality due to predation (one year lag: slope = −0.00320, intercept = 0.488; two year lag: slope = −0.00496, intercept = 0.532) did not produce 8–11 year cycles for any values of R. Parameter estimates for survival after winter mortality due to other causes (two year lag: slope = −0.00648, intercept = 1.00) produced 8–11 year cycles only with R values between 2.3 and 2.7 which represents only a small portion of the range of values of R measured on the control grids. Over the eight years of the study, R values ranged from 2.13 to 6.75 (mean = 3.55), although these values did not vary consistently with density.

Predator response model

The relationship between prey density and predation rate can be more realistically described by a model that includes functional and numerical responses of predators. Therefore we assumed a Type II functional response (Holling 1959) and a linear delayed density-dependent numerical response in a Nicholson-Bailey model (1935) to determine predation rate in our simulations. We do not have enough predation rate observations to justify fitting a more complex type-III functional response model. The Nicholson-Bailey model computes predation effects by calculating an instantaneous predation rate (short term probability of mortality) as:

$$\text{predation rate}=\text{kill}/N(t) \tag{3}$$

where $N(t)$ is the hare population size at the beginning of the time interval in which the rate is measured. The kill component of equation 3 is calculated as a product of predator numbers, assumed to be proportional to $N(t-1)$, and kill rate per predator. We assumed predator numbers proportional to $N(t-1)$ based on the observation of a one-year lag in numerical response of some snowshoe hare predators (Keith et al. 1977). Predator numbers are therefore predicted by $c*N(t-1)$, and the attack rate per predator by the type II equation $a*N(t)/(H+N(t))$, where a is the maximum annual kill per predator and H is the prey density (N) at which predators can achieve 1/2 of a. Substituting the product of these into Eq. (3) gives the instantaneous rate of predation:

$$=\frac{(a*N(t)/(H+N(t)))*(c*N(t-1))}{N(t)} \tag{4}$$

This equation simplifies to:

$$\text{predation rate}=\frac{a*c*N(t-1)}{H+N(t)} \tag{5}$$

or

$$\text{predation rate}=\frac{A*N(t-1)}{H+N(t)} \tag{6}$$

where $A=a*c$ represents the combined effects of the predator numerical response (c) and the maximum kill rate per predator (a).

We fitted the data on hare density and predation rate (Table 4) to this model using a non-linear estimation technique (Marquardt algorithm) to estimate A and H. The best estimates of A and H for winter predation mortality were 2.38 kills/hare (S.D. = 0.48) and 8.02 hares/grid (S.D. = 4.3) respectively. We also fitted data on summer hare density and predation rate to this model. Best estimates of A and H from summer mortality data were 0.859 kills/hare (S.D. = 0.554) and 1.77 hares/grid (S.D. = 4.84) respectively.

In the simulation model, the hare population in year $t+1$ was calculated as:

$$N(t+1)=N(t)*R*\exp(-A*N(t-1)/(H+N(t))) \tag{7}$$

where R is the rate of growth of the hare population due to recruitment, as in the previous linear model. Here the exponential term $\exp(-x)$ represents the annual survival

Table 4. Overwinter probabilities of survival vs. December 1 population density on the Silver Creek Control grid (25.6 ha) in the previous year

Year	Probability of survival	Population density
1978	0.3839	20
1979	0.4365	47
1980	0.1419	95
1981	0.0951	120
1983	0.3833	4
1984	0.5465	5

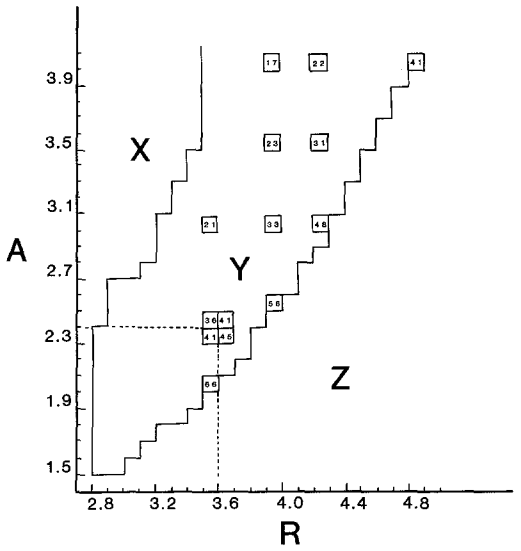

Fig. 5. Behaviour of the "predator response" model. 8–11 year cycles are produced with parameter combinations falling within the area *Y*. Cycles with period less more than 8 years or dampening cycles are produced within the area X. Cycles with period more than 11 years are produced within the area *Z*. Numbers within the boxes are maximum population sizes for a 25.6 ha area during 8–11 year cycles for selected combinations of *A* and *R*, with H = 8 (see text). "*A*" is a constant that describes the functional and numerical response of the predators and *R* is the rate of growth of the hare population. Observed values of *A* and *R* at Kluane occur at the intersection of the broken lines

rate associated with the instantaneous kill rate *x* predicted by Eq. (6). This method for approximating the annual net effect of predation is strictly valid only if $N(t)$ is low, so predation rate per predator is simply proportional to prey density at all times during the year. For high values of $N(t)$, Eq. (7) will underestimate the predation impact (a conservative assumption in relation to the predator hypothesis).

The (*R*, *A*) parameter space in which 8–11 year cycles were produced by this simple model when $H = 8$, is illustrated in Fig. 5. Maximum population densities on a 9.56 ha grid, for select parameter combinations are included in the figure. Increasing *H* produced a larger maximum *N* for the cycle but had little effect on periodicity. Decreasing *H* produced a smaller maximum *N* and a slightly shorter period of the cycle. Values of *H* greater than 20 or less than 8 produced cycles with maximum *N* that were outside of the range of spring densities measured by Krebs et al. (1986). The model produced cycles with an 8–11 year period within a fairly narrow range (*Y*) of values of *A* and *R*. For the parameter combinations in the region *X* (Fig. 5), the model produced cycles with period less than 8 years or dampening cycles. Parameter combinations in region *Z* (Fig. 5) produced cycles with constant amplitude and constant period that were longer than 11 years. As *R* increased for a fixed *A*, the period of cycles became longer and the amplitude increased.

As previously mentioned, the mean *R* for the control grids ranged from 2.13 to 6.75 with a mean for the eight years of 3.55. The value of *A* measured in this study is 2.38 (S.D. = 0.48), as reported above. Thus, assuming that predation in winter was the only source of mortality, and recruitment was constant, our recorded values of *A* and R were within the range in which cycles of 8–11 year period are produced. If we add summer predation to this model, using the estimated parameters reported above to calculate summer survival, then 8–11 year cycles are produced only with values of *R* greater than 8. This model that includes summer predation mortality did not therefore produce cycles with realistic values of *R*.

For winters in which predation rates are high, prey population density will be much lower at the end of the winter than it was at the beginning. Estimates of predator functional response however, are based on an average for the entire winter, so that kill rates at the beginning of the winter are underestimated and kill rates at the end of the winter are overestimated by the exponential form in Eq. (7). If we address this problem by using an exact solution to the "random predator equation" (Rogers 1972) in the model, the estimates of *A* and *H* are not biologically reasonable and population dynamics produced are unstable. Fitting our mortality data to this model resulted in predator attack rates that are higher at the end of the winter than would be predicted by a Type II functional response.

Discussion

To understand what causes the 10-year hare cycle it is necessary to describe the mechanisms behind the demographic changes observed in snowshoe hare populations. The *k*-factor analysis reiterates the claim of Boutin et al. (1986) that predation was the factor with the largest influence on snowshoe hare survival in the Yukon study area during 1977–84. This observation led us to explore the logic of the hypothesis that predation, measured at our Yukon study site, could be responsible for the 10-year hare cycle.

We have shown that predation in winter is the largest mortality factor for snowshoe hares at Kluane, Yukon. Such winter predation rates can produce cyclical changes in hare densities similar to those reported by Krebs et al. (1986). When $H = 12$ (mean value from fitting procedure + 1 S.D.) in the model that included winter predation, peak spring densities of 30–100 hares per 10 ha (similar to data of Krebs et al. (1986)) were predicted. In this model predation also accounted for the decline from those densities to near zero. This result is contrary to Keith's (1974) hypothesis that predation mortality alone is insufficient to cause a decline from peak hare densities. In a more recent paper (Keith et al. 1984), greater emphasis has been given to the role of predation in hare population declines, although they still propose that outright starvation is the primary cause of death during the first winter of the decline.

It is possible that food shortage acted indirectly to limit hare population size, by making hares more vulnerable to predators. Keith et al. (1984) suggest that mortality due to predation may be partially compensatory. They noted that hares taken by predators are in poorer condition than hares in a randomly shot sample (condition is measured as percent fat in bone marrow), suggesting that predators were catching hares that were starving. Sinclair et al. (unpublished work) are presently assessing whether nutrition dropped below body maintenance level during the Kluane hare cycle. If starvation did not occur, then we would expect to see nutrition remaining above this maintenance level over the cycle.

In all the simulation models we have assumed recruitment was constant in order to test whether mortality pro-

cesses alone could cause the hare cycle. According to the work of Cary and Keith (1979), reproduction varies in a cyclical manner, preceding population density change by 2 1/2 years on their Rochester, Alberta study site. Changes in reproduction in their study were large and could potentially impose a cyclic pattern of density changes on the fall population. We were unable to directly relate reproduction to recruitment. Recruitment measured by Krebs et al. (1986) was an index of reproduction that included early juvenile mortality, immigration, and emigration of hares. The recruitment index may precede changes in density by three years, but the significance of this relationship in our data rests on a single point. Thus, it is not clear whether summer mortality compensates for changes in reproduction, resulting in virtually constant recruitment of juveniles to the population.

The lack of information about juvenile hare demography may explain why the model which included summer mortality did not predict 8–11 year cycles in numbers of hares. If predators respond to juveniles as well as to adults, then our estimates of the predation functions A and H from summer studies are inadequate. Both the functional and numerical responses of predators were probably affected by the combined juvenile and adult density. The plausibility of this explanation of the failure of the more complex predation model can be evaluated when better data on juvenile hares are available. Studies that assess juvenile numbers with greater precision at an early age and that determine causes of juvenile mortality are necessary.

Density-dependent mortality in winter from causes other than predation (with a 2-year time lag) can produce 8–11 year cycles, but only within a narrow range of R values (2.3–2.7). The amplitude of these cycles is 1 1/2 to 2 times as large as the changes reported by Krebs et al. (1986). Our data do not therefore support the hypothesis that a 2-year lag in nonpredation mortality, resulting from a plant-herbivore interaction and acting according to a simple linear density-dependent relationship, can cause the observed changes in hare numbers. However, our simulations do not exclude the possibility that plant-herbivore interactions influence hare population dynamics by a different mechanism.

Predation mortality is best described as a non-linear function of hare population density. Increasing densities of prey typically cause predators both to increase in number and to vary their rates of consumption as prey abundance changes (Solomon 1949; Holling 1959). We measured predation rate directly by monitoring hare mortality. Consequently, the predation rates apply to the entire suite of hare predators. These rates also reflect the total (functional and numerical) response of predators. Therefore, we cannot distinguish between the impact of various predator species on the hare population, nor can we separate the functional and numerical responses which, in concert, produce the time lag observed in the predation rates.

The estimates of predation rates presented here exceeded those of Keith et al. (1977), probably because we estimated predation from hare mortality directly, rather than extrapolating kill rates for individual predators. Predator studies can underestimate kill rates if scats or pellets are missed, if predator numbers are underestimated, if non-resident predators are responsible for significant hare mortality, or if surplus killing occurs. Keith et al. (1984), found higher predation rates during the second winter of declining hare populations than those they had predicted from their previous studies of predators. They attributed 92% of deaths of radiocollared hares to predation during this second winter of the decline.

The patterns of numerical and functional response that result in the predation rates we measured can be deduced from studies of snowshoe hare predators. The decrease in numbers of coyotes (*Canis latrans*), lynx (*Lynx canadensis*), and great horned owls (*Bubo virginianus*) lagged a year behind the deline in hare density in studies by Keith et al. (1977). Numbers of goshawks (*Accipiter gentilis*) changed synchronously with hare density and numbers of red-tailed hawks (*Buteo jamaicensis*) remained constant throughout an 8-year period at their study site near Rochester, Alberta.

Functional responses by snowshoe hare predators have been described in two studies. Ward and Krebs (1985) showed that daily travel distance of lynx in Yukon, Canada, remained relatively constant at hare densities ranging from 14.7 per ha to 1 per ha and then increased when hares reached densities below 1 per ha. Ward (1985) suggested that daily capture rates of snowshoe hares by lynx declined as lynx increased their travel distance. Keith et al. (1977), working at Rochester, Alberta, found Type II functional response curves for great horned owl, red tailed hawk and lynx, and evidence for a Type III functional response for coyotes feeding on snowshoe hares. We propose that migrating raptors may also exhibit non-linear functional responses to changing hare densities by pausing in their migration through snowshoe hare habitat to hunt hares only when densities are high.

The time-lag in mortality due to predation (total response) indicated in Figs. 2 and 3 was similar to that found by Keith et al. (1977) for predation on snowshoe hares by lynx, coyote, great horned owl and goshawk.

The parameters in our model are based on mortality rates and density estimates from a single cycle in hare numbers. It is, therefore, possible that the relationship between predation rate and density that produces cycles in this model can not be generalized to represent other areas or other years. No other comparable series of snowshoe hare mortality rates exists for comparison with our measurements.

Our model shows that a simple interaction between snowshoe hares and their predators in winter in the Yukon can produce cyclical population changes. Winter predation can initiate the decline from peak hare densities as well as reduce the population to extremely low densities. Field experiments are necessary to test the prediction of this model that winter predation is sufficient to cause the hare population cycle. If a hare population is prevented from experiencing food shortage and no cycles are produced, the hypothesis that predation is sufficient to cause the 10-year cycle would be falsified. If, in addition, a hare population that is protected from predation still exhibits a 10-year cycle, the hypothesis that predation is necessary to cause a 10-year cycle would be falsified.

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References

Boutin S (1980) Effect of spring removal experiments on the spacing behavior of female snowshoe hares. Can J Zool 58:2167–2174

Boutin S (1984) Effect of late winter food addition on numbers and movements of snowshoe hares. Oecologia (Berlin) 62:393–400

Boutin SA, Krebs CJ, Sinclair ARE, Smith JNM (1986) Proximate causes of losses in a snowshoe hare population. Can J Zool 64:606–610

Bryant JD (1981) The regulation of snowshoe hare feeding behavior during winter by plant antiherbivore chemistry. In: Myers K, MacInnes CD (eds) Proc Int Lagomorph Confer (Guelph, Ontario, Canada, 1979)

Cary JR, Keith LB (1979) Reproductive change in the 10-year cycle of snowshoe hares. Can J Zool 57:375–390

Elton C (1933) The Canadian snowshoe rabbit enquiry, 1931–1932. Can Field Nat 47:63–69

Elton C, Nicholson M (1942) The 10-year cycle in numbers of the lynx in Canada. J Anim Ecol 11:215–244

Fox JF, Bryant JP (1984) Instability of the snowshoe hare and woody plant interaction. Oecologia (Berlin) 63:128–135

Holling CS (1959) The components of predation as revealed by a study of small-mammal predation of the European pine sawfly. Can Entomol 91(5):293–320

Keith LB (1963) Wildlife's Ten-Year Cycle. Madison: Univ Wisconsin Press

Keith LB (1974) Some features of population dynamics in mammals. Proc Int Congr Game Biol, Stockholm 11:17–58

Keith LB, Todd AW, Brand CJ, Adamcik RS, Rusch DH (1977) An analysis of predation during a cyclic fluctuation of snowshoe hares. Proc Int Congr Game Biol 13:151–175

Keith LB, Cary JR, Rongstad OJ, Brittingham MJ (1984) Demography and ecology of a declining snowshoe hare population. Wildl Monog 90:1–43

Krebs CJ, Gilbert BS, Boutin S, Sinclair ARE, Smith JNM (1986) Population biology of snowshoe hares. I. Demography of populations in the southern Yukon, 1976–1984. J Anim Ecol 55:963–982

Kruuk H (1972) Surplus killing by carnivores. J Zool (London) 166:233–244

Nicholson AJ, Bailey VA (1935) The balance of animal populations. Part I. Proc Zool Soc Lond pp 551–598

Podoler H, Rogers D (1975) A new method for the identification of key factors from life-table data. J Anim Ecol 44:85–114

Rogers D (1972) Random search and insect population models. J Anim Ecol 43:239–253

Schaffer WM (1984) Stretching and folding in lynx fur returns: evidence for a strange attractor in nature? Am Nat 124:798–820

Schaffer WM, Kot M (1986) Chaos in ecological systems: the coals that Newcastle forgot. Tree 1:58–63

Solomon ME (1949) The natural control of animal populations. J Anim Ecol 18:1–35

Tanner JT (1975) The stability and the intrinsic growth rates of prey and predator populations. Ecology 56:855–867

Trent TT, Rongstad OJ (1974) Home range and survival of cottontail rabbits in southwestern Wisconsin. J Wildl Manage 38:459–472

Varley GC, Gradwell GR (1960) Key factors in population studies. J Anim Ecol 29:399–401

Ward RMP (1985) Behavioral responses of lynx to declining snowshoe hare abundance. M.Sc. thesis. University of British Columbia, Vancouver

Ward RMP, Krebs CJ (1985) Behavioural responses of lynx to declining snowshoe hare abundance. Can J Zool 63:2817–2824

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