Impact of Food and Predation on the Snowshoe Hare Cycle

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fetal life. In this study, mice without C/EBPα were retarded in their development; their lungs appeared immature on histologic analysis (25), they had an inadequate supply of body fat, they had insufficient liver glycogen, and they suffered from hypoglycemia. These are all symptoms consistent with those of the preterm infant (26). The incomplete developmental activation of *c/ebpα* and its target genes may affect the metabolic state of the premature neonate. We have shown in vivo that C/EBPα is required for the normal energy-related functions of the liver and of brown and white adipose tissue. C/EBPα-deficient mice will be useful for studying the complex metabolic requirements of preterm infants.

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10. At 32 hours postpartum with glucose, the weights of the +/+ and +/− mice increased from their birth weights by 32.4 ± 9.9% (mean ± SD; n = 53) whereas the weights of the −/− mice increased by 9.6 ± 9.8% (n = 10) (Student t test, $P < 0.0001$).
11. Alanine transaminase and alkaline phosphatase activities in serum collected at 32 hours postpartum indicated no overt hepatic injury in the C/EBPα-deficient mice. Histology confirmed that there was no hepatocellular necrosis in the −/− mice. However, measurements of serum amino acids indicated large amounts of tyrosine in mutants (435.0 ± 226.9 μmol/liter, n = 4) relative to controls (115.6 ± 35.2 μmol/liter, n = 7) (Student t test, $P < 0.05$), which suggested potential alterations in tyrosine metabolism.
12. Additional Northern blots (not shown) indicated some variation in the time of expression of PEPCK and G6Pase in the control mice, although the C/EBPα-deficient neonates were consistently delayed. Within 2 hours after birth, 3 of 10 control mice and 5 of 5 −/− mice had reduced amounts of mRNA for both enzymes.
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20. Histology of the small intestine in mice that had ingested milk showed that mice of all three genotypes had oil red O–positive lipid globules in the enterocytes and lacteals of the gut villi (not shown), which suggested that absorption of milk fats was qualitatively normal. Concentrations (mean ± SD) of β-hydroxybutyrate indicated that the C/EBPα-deficient mice were capable of catabolizing an amount of fatty acids (0.3 ± 0.1 mmol/liter, n = 3) similar to the control mice (0.6 ± 0.2 mmol/liter, n = 4).
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Impact of Food and Predation on the Snowshoe Hare Cycle

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Snowshoe hare populations in the boreal forests of North America go through 10-year cycles. Supplemental food and mammalian predator abundance were manipulated in a factorial design on 1-square-kilometer areas for 8 years in the Yukon. Two blocks of forest were fertilized to test for nutrient effects. Predator exclosure doubled and food addition tripled hare density during the cyclic peak and decline. Predator exclosure combined with food addition increased density 11-fold. Added nutrients increased plant growth but not hare density. Food and predation together had a more than additive effect, which suggests that a three-trophic-level interaction generates hare cycles.

The 10-year cycle of snowshoe hare populations and those of their predators is one of the dominant perturbations of the boreal forests of North America. Predation and food shortage have been postulated as the major factors causing these fluctuations (*1*). Because in all cyclic populations many factors will change in a manner correlated with population density, necessary conditions can be recognized only by experimental manipulations (*2*). From 1976 to 1984, we manipulated food supplies of snowshoe hares (*Lepus americanus*) in the southern Yukon and showed that the cyclic decline could not be prevented by either artificial or natural food addition (*3*). Single-factor manipulations have been criticized in field ecology because they may miss important interactions between factors (*4*). For the past 8 years, we have carried out large-scale experiments on nutrients, supplemental food, and predation in the Yukon to untangle the causes of the hare cycle and the consequences the hare cycle has for the vertebrate community. By crossing a predator reduction manipulation with food addition we estimated interaction effects caused by the failure of factors to combine additively.

We chose 1-km^2 blocks of undisturbed boreal forest near Kluane Lake, Yukon, as our experimental units (*5*). The boreal forest in this region is dominated by white spruce (*Picea glauca*) and was not disturbed by logging, fire, or extensive fur trapping during our studies. We used a factorial design to untangle the effects of food and predation on hares. Three areas were used as controls (*6*). Two experimental areas were provided with ad lib supplemental food year-round. We excluded mammalian predators by building one electric fence in the summer of 1987. In the summer of 1988, we built a second electric fence to use for the combined predator reduction–food addition treatment (*7*). Since January 1989, the electric fences have worked effectively to prevent mammalian predators from entering the two areas. The fences are permeable to snowshoe hares. Beginning in 1987, we added nitrogen-potassium-phosphorus (NPK) fertilizer to two blocks of forest to increase plant growth (*8*). We chose to manipulate a few large areas rather than many small areas because of the failure of most field experiments to address large-scale issues (*9*). We captured, marked, and released snowshoe hares every March and October and estimated densities with the robust design (*10*).

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Snowshoe hares in the control areas increased from a low in the mid-1980s to a peak in 1989 and 1990 (Fig. 1). The increase phase from 1986 to 1988 showed considerable variation among the three control populations, but from the peak phase onward all the controls were similar in their year-to-year dynamics. The cyclic decline began in autumn and winter 1990 and continued until the spring of 1993 when hares had reached low numbers of approximately one hare per 15 ha. Population increase in snowshoe hares is stopped both by increased mortality and by reduced reproductive output (*11*). This previously described syndrome of demographic changes was consistent over the cycle we observed. Juvenile mortality increased while the population was still in the increase phase of the cycle (Fig. 2), whereas adult losses did not become severe until the decline phase. The decline phase in 1991 and 1992 was characterized by poor survival of both juveniles and adults and by reduced reproductive output by females through restriction or elimination of their second and third summer litters (*11*).

The impact of our experimental treatments can be measured in several ways. We concentrate here on changes in the population density of hares in the treated areas and on the survival rates of radio-collared hares. Density effects can be most simply expressed as ratios of the density in the treated area to the density in the control areas. We estimated these each spring and autumn for all treatments (Fig. 3). Effects were small during the increase phase in 1987 and 1988 because the treatments were just being established. All treatments were effective by spring 1989. The food addition effect was always positive and produced densities ranging from 1.5- to 6-fold over control levels during the peak and decline phases (Fig. 3B). The predator exclosure effect was negligible in the peak phase from 1989 to 1990 but became pronounced in the late decline and low phases, producing densities ranging from 1.4- to 6-fold over control levels (Fig. 3A). The largest effect was shown by predator exclosure and food treatment combined, particularly in the late decline phase when densities exceeded control levels by 36-fold (Fig. 3C). Averaged over both the peak and the decline phase, predator exclosure approximately doubled the density of hares, food addition approximately tripled density, and the combined treatment increased density 11-fold.

In contrast to the strong effects shown by manipulation of predation and food supply, the addition of nutrients had virtually no effect on snowshoe hare numbers. In spite of increased growth of herbs, grasses, shrubs, and trees (8), the fertilized plots contained virtually the same number of hares as did the control plots (Fig. 1). Fertilized vegetation in the boreal forest cannot duplicate either the quantity or quality of the artificial food that we added in our experiments, and for this reason fertilization is a relatively ineffective method of food addition for hares.

Survival rates can be estimated from mark-recapture methods or from radiotelemetry (*12*). Treatments had little impact on survival rates during the peak phase of the hare cycle. Monthly adult survival rates were greater than 90% in the peak phase, leaving little room for improvement.

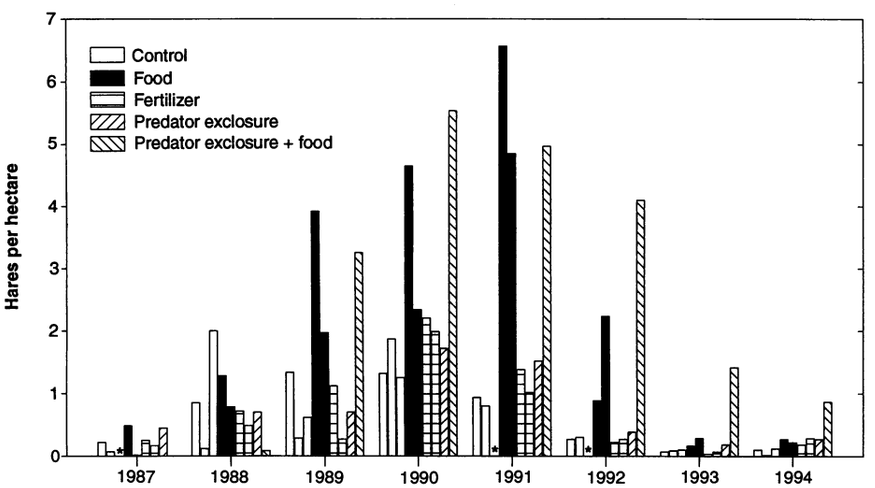

Fig. 1. Spring densities of snowshoe hares in three control and six treatment areas, Kluane Lake, Yukon, 1987 to 1994. Densities were estimated from mark-recapture live trapping for 4 to 5 days in late March and early April each year with the use of the jackknife estimator in the program Capture (*10*). Asterisk indicates one of the three control areas that was not trapped in 1987, 1991, and 1992.

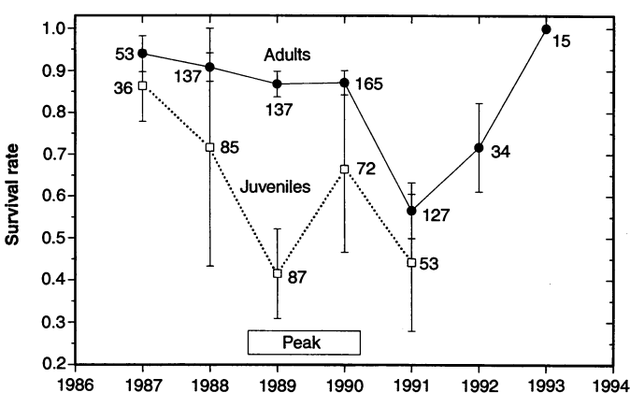

Fig. 2. Survival rates of adult (circles) and juvenile (squares) snowshoe hares in the most intensively studied control area (control area 1). Juvenile survival is already deteriorating in the late increase phase (1988) and is low in the peak and decline phases of the cycle. We could not estimate juvenile survival in 1992 because few of the 13 juveniles caught were ever recaptured. We presume juvenile survival was poor in 1992. Survival rates (per 28 days) were estimated from mark-recapture data with the use of the Jolly-Seber Model B (*10*). Juvenile survival refers to trappable juveniles only, which are those more than 6 to 8 weeks old. Sample sizes of hares are given next to data points.

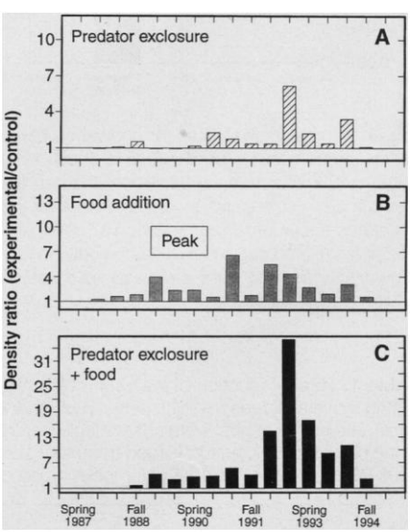

Fig. 3. Ratio of population densities for the three treatments to average control population densities at the same time. If there is no treatment effect, we expect a ratio of 1.0. During the peak and decline phases, the predator exclosure (**A**) doubled density on average, food addition (**B**) tripled density, and the combined treatment (**C**) increased density 11-fold.

The major effects of the treatments on survival were visible in the decline phase (Fig. 4). The probability of a hare living for 1 year in the control areas during the decline was 0.7%. In fertilized areas, this probability was 1.9%, which is slightly but not significantly higher than in the control areas. This probability improved to 3.7% in the food addition grids and to 9.5% in the predator exclosure areas. The best chances of survival occurred in areas treated with a combination of mammalian predator reduction and food, where the probability of survival was 20.8% for 1 year during the decline. The effects of food and predation on survival during the decline phase were nearly additive and showed no sign of an interaction. The addition of food by itself was not sufficient to prevent large losses to predators, and the rapid population collapse in the food areas from 1991 to 1992 (Fig. 1) was due to heavy predation.

The numbers of both avian and mammalian predators follow the hare cycle, but with a 1- to 2-year time lag (*1*). Virtually all snowshoe hares in our study area die from predator attack in the immediate sense. From 1989 to 1993, we found that 83% of the deaths of all radio-collared hares were due to predation and only 9% were attributed to starvation (*13*). We presume that hares suffering from food stress will be more susceptible to death from predation.

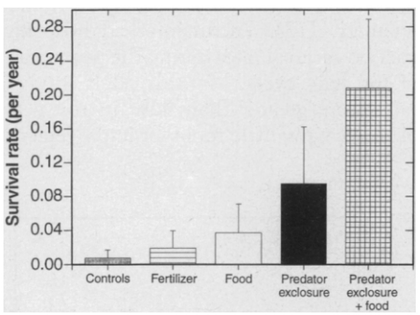

Fig. 4. Annual survival rates for snowshoe hares with radio collars during the decline phase from autumn 1990 to autumn 1992. Ninety-five percent confidence limits are shown. Sample sizes for the estimates are (in order): 278, 206, 197, 246, and 262 hares. Radio collars were placed only on fully grown animals and thus are used to measure adult mortality rates.

Because we used live trapping as our primary technique of study, we have less data on the reproductive output of snowshoe hares in relation to these three treatments. During the peak phase, the food treatment areas have the same reproductive output as do the control areas (*14*). Table 1 gives the total production of live young hares at birth for a female over the summer breeding period for the control areas, the food areas, and the predator exclosure plus food grid. The collapse of reproduction in the control areas was prevented in the combined treatment area. Because we do not know the reproductive schedule for the food or the predator exclosure treatment areas during the decline phase, we cannot assess the separate contributions of food and predation to the reproductive output of females.

There are three possible explanations of these differences in reproductive output. Reproductive changes may be driven by food limitations in the decline period. Alternatively, hares may respond to predation risk in the decline phase by altering their habitat use so that they cannot achieve adequate nutrition (*15*). In both of these cases, this reproductive curtailment is due to food shortage, but in the first case it is absolute food shortage and in the second case it is relative food shortage caused as an indirect effect of predation. The third possibility is that these reproductive effects are a direct result of stress and the physiological derangement associated with stress (*16*). We cannot yet determine which of these explanations is correct. Absolute winter food shortage does not necessarily occur during the peak or decline phase (*3*), and the weight of evidence is against the first explanation. Behavioral evidence suggests that there may be a relative shortage of food (*15*).

If both food and predation are together sufficient to explain population cycles in snowshoe hares, why were we not able to prevent the decline entirely in the combination treatment area? Hare densities fell from about seven per hectare in 1989 to about one per hectare in 1994 in the combination treatment area, even though hare density in this area remained at or above normal peak densities for 7 years from 1988 to 1995. The combination treatment delayed the decline but did not prevent it. There are three possible reasons for this, and they illustrate one difficulty of large-scale experiments. First, hares could move freely into and out of the predator exclosures. Individuals were often killed by predators when they moved out, but others could emigrate into nearly unoccupied landscape outside the fence in the low years of 1992 to 1994 (*17*). Second, we could not prevent raptor predation inside the predator exclosure. Our monofilament treatment covered only a small fraction of the predator exclosure and was ineffective in preventing raptors such as goshawks from invading the area. Because raptors and owls cause about 40% of the predation mortality in our hare populations, the effect of the exclosures was to reduce total predation losses, not eliminate them. Great horned owls and goshawks continued to kill hares inside the predator exclosures during the decline and low phases. Third, another factor in addition to food and predation may be sufficient to cause the decline.

These results support the view that population cycles in snowshoe hares in the boreal forest are a result of the interaction between food supplies and predation. They do not support either the plant-herbivore model or the predator-prey model for cycles but suggest that hare cycles result from a three-trophic-level interaction (*18*). Our experimental results are consistent with the general ideas of Keith (*19*) and Wolff (*20*) that both food and predation play a role in generating hare cycles, but they do not support Keith's sequential two-factor model that states that food shortage effects are followed by predation effects in causing cyclic declines. Further work will be required to determine if the nutritional effects on hares are an indirect effect of predation that is explicable in terms of the hares' behavioral responses to predation risk. Our studies have provided little data on the causes of the low phase of the hare cycle, which can persist for 3 to 4 years. Food supplies recover quickly after the peak has passed, and predator numbers collapse during the hare decline. Whether the direct or indirect effects of predation can also explain the low phase remains an open question.

Table 1. Total production of live young by female snowshoe hares over the summer breeding season during the late increase (1988), peak (1989 and 1990), and decline (1991 and 1992) phases of the hare cycle. Litter sizes at birth were not available for all treatments in all years. We assume that total production in the predator exclosure plus food treatment area would have been the same as in the food areas in the peak years of 1989 and 1990. Numbers in parentheses indicate number of females sampled.

Year	Control areas [no. of young ± SE (*n*)]	Food areas [no. of young ± SE (*n*)]	Predator exclosure + food area [no. of young ± SE (*n*)]
1988	16.4 ± 0.44 (10)	—	—
1989	13.7 ± 0.39 (21)	14.1 ± 0.43 (36)	—
1990	13.7 ± 0.40 (33)	15.1 ± 0.43 (36)	—
1991	7.8 ± 0.37 (18)	—	16.3 ± 0.73 (15)
1992	3.3 ± 0.25 (4)	—	17.1 ± 0.31 (50)

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5. Blocks were spaced at least 1 km apart. Within each block, we surveyed checkerboard grids of 20-by-20 points with 30.5-m spacing and used these grids for snowshoe hare live trapping. Two experimental areas were provided with supplemental food (commercial rabbit chow, 16% protein) year round. In the summer of 1987, we built one electric fence around 1 km^2 to exclude mammalian predators, and over the following year we covered 10 ha with monofilament to reduce avian predation. The monofilament was never effective in preventing avian predation inside the electric fences, and consequently we did not rely on it as a part of the treatment. In the summer of 1988, we built a second electric fence around 1 km^2 to use for the combined predator reduction–food addition treatment. We modified the design of the electric fences in 1988 to make them more effective, and since then they have worked effectively to prevent mammalian predators from entering the area. The fences are permeable to snowshoe hares. We could not replicate either the predator reduction or the predator reduction–food addition treatment because of maintenance costs and the difficulty of maintaining electric fences in the Yukon winter with −45°C temperatures. The fences had to be checked every day during winter. From 1976 to 1985, we trapped hares in six areas within the main study region and found that their population trajectories were very similar (*3*). We thus have no reason to suspect strong area effects on the unreplicated predator reduction plots.
6. We used three control areas but were not able to trap hares in all of them every year. We have more detailed data on hares from control area 1. The three control areas had quite different histories during the increase phase from 1986 to 1988. Control area 3 reached its greatest hare density in 1988 and remained at a plateau until 1990. Control area 2 reached its peak density in 1990, and control area 1 reached its peak in 1989. By the late peak in 1990 and during the decline phase, the control areas were much more similar to each other in hare densities.
7. The electric fence was 10-stranded, 2.2 m in height, and carried 8600 V. Snow tracking of mammalian predators meeting the fence illustrated its effectiveness. We excluded mammalian predators virtually continuously from January 1989 onward. Our attempts to use monofilament fishing line as a deterrent to birds of prey was largely ineffective because ice formation and snow accumulation on the lines in winter caused them to break or collapse to the ground. We used monofilament on 10 ha of the predator exclosure but did not attempt to use it on the combination treatment area. The predator exclosures thus were mammalian predator exclosures and were still subject to avian predation.
8. We fertilized two 1-km^2 blocks of forest with commercial fertilizer. In May 1987, we used ammonium nitrate at 25 g/m^2. In May 1988, we switched to NPK fertilizer and used 17.5 g of N/m^2, 5 g of P/m^2, and 2.5 g of K/m^2. In 1989, we used half this amount, and in the years 1990 to 1994 we used the full amount as in 1988. The fertilizer was spread aerially and we did ground checks to make sure it was uniformly spread. We do not present the data here to show the plant growth responses, but all elements of the flora responded dramatically to the added nutrients (C. J. Krebs *et al.*, unpublished data).
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Double Dissociation of Conditioning and Declarative Knowledge Relative to the Amygdala and Hippocampus in Humans

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A patient with selective bilateral damage to the amygdala did not acquire conditioned autonomic responses to visual or auditory stimuli but did acquire the declarative facts about which visual or auditory stimuli were paired with the unconditioned stimulus. By contrast, a patient with selective bilateral damage to the hippocampus failed to acquire the facts but did acquire the conditioning. Finally, a patient with bilateral damage to both amygdala and hippocampal formation acquired neither the conditioning nor the facts. These findings demonstrate a double dissociation of conditioning and declarative knowledge relative to the human amygdala and hippocampus.

Studies in animals have established that the amygdala is critical for emotional conditioning (*1*), whereas several human and nonhuman primate studies have established that the hippocampus and surrounding regions are necessary for establishing declarative knowledge (*2*). Because of the rarity of patients with selective bilateral damage restricted to either the amygdala or hippocampus, the exact roles of these structures in emotional and declarative learning have not been established clearly for humans (*3*). Here, we report the relative contributions of the amygdala and hippocampus to emotional conditioning and to the establishment of declarative knowledge in humans. We studied three people with distinct brain lesions: SM046 had bilateral destruction of the amygdala, but bilaterally intact hippocampi; WC1606 had bilateral hippocampal damage, but bilaterally intact amygdalae; and RH1951 had bilateral damage to both hippocampus and amygdala (*4*) (Table 1 and Fig. 1). Four normal participants of comparable age and education served as controls.

Two conditioning experiments were carried out. The first, a visual-auditory conditioning experiment, used monochrome slides as the conditioned stimuli (CS) and a startlingly loud sound (a boat horn delivered at 100 dB) as the unconditioned stimulus (US). The second, an auditory-auditory conditioning experiment, used computer-generated tones as the CS (the US was the same as in the visual-auditory experiment). In both experiments, the skin conductance response (SCR) was the dependent measure of autonomic response (*5*). Each conditioning experiment was performed three times in SM046 and twice in WC1606 and

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