Concepts

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POPULATION CYCLES IN SMALL MAMMALS: THE PROBLEM OF EXPLAINING THE LOW PHASE

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Abstract. Cycles characterize the demography of many populations of microtine rodents and snowshoe hares. A phase of low numbers often follows the decline and introduces a lag that lengthens the cycle. This low can last 1–3 yr in microtines and 2–4 yr in hares. Understanding the low phase is critical in explaining population cycles. Two major classes of hypotheses try to account for the low phase. The first proposes that something may be ''wrong'' with the extrinsic environment. The most promising of these extrinsic explanations is that predation, acting either directly or indirectly, has delayed density-dependent effects on prey populations during the low phase. The second class of hypotheses proposes that something may be ''wrong'' with the animals themselves. The most likely intrinsic factors are maternal effects or age effects on fitness during the low phase. Experimental tests for each of these sets of hypotheses are needed, and we suggest replicated experiments on focal species in two continents to resolve these unknowns.

Key words: extrinsic mechanisms; intrinsic mechanisms; Lemmus *spp.;* Lepus americanus; *low phase; microtines;* Microtus *spp.; population cycles; population regulation; snowshoe hares.*

INTRODUCTION

Populations of some small mammals fluctuate more or less regularly. These ''cyclic'' populations have attracted considerable attention and even more controversy (Krebs 1996, Stenseth et al. 1996). There are two major types of cycles. Vole and lemming cycles typically have a 3–5 yr period, and this period is reflected in similar cycles of their associated predators (Korpimäki and Norrdahl 1991, Stenseth and Ims 1993). The snowshoe hare (*Lepus americanus*) cycle has a 9–11 yr period in the boreal forests of North America and Siberia, and is also widely reflected in cycles of the community of predators that take advantage of this abundant prey population (Keith 1990). The amplitude of these cycles varies from 50:1 to 200:1.

We concentrate here on the low phase of these cycles because it may be the Achilles heel for most of the proposed models of cycles. Fig. 1a illustrates four periods of low numbers for two vole species (*Microtus* spp.) in northern Finland (Henttonen et al. 1987); Fig. 1b illustrates six periods of low numbers for the Norwegian lemming (*Lemmus lemmus*) in southern Norway. The low phase in these populations lasted 1–3 yr (Framstad et al. 1993, 1997). Figure 2 illustrates periods of low numbers in the snowshoe hare in two areas from western North America. In central Alberta (Fig. 2a), hare populations were low for 4 yr (1964–1967) during the first cycle and ≥2 yr (1974–1975) during the second (Keith and Windberg 1978). In southwestern Yukon (Fig. 2b), hare populations were low for 4–5 yr (1983–1987); detailed live-trapping studies, supplemented by snow tracking in winter, indicated that this low occurred at a landscape level (Krebs et al. 1986, 1995*b*). These small mammals have a tremendous potential for rapid growth during the increase phase. In microtines, populations have been documented to increase eightfold to 22-fold in a 6-mo period (Krebs and Myers 1974). In snowshoe hares, populations typically increase twofold per year (Keith 1990). We wish to focus on the key question: what prevents population growth during the low phase of the cycle?

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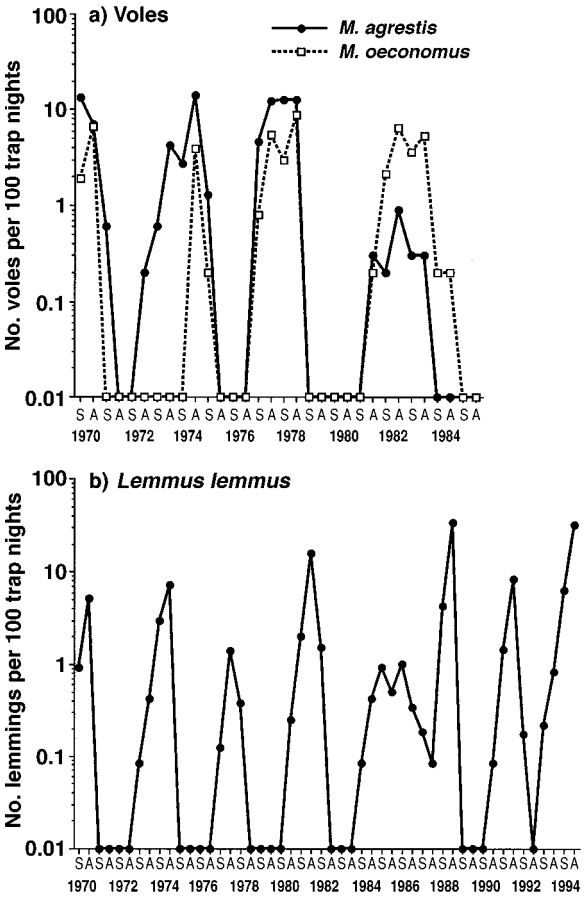

FIG. 1. (a) Population cycles in field voles (*Microtus agrestis*) and root voles (*Microtus oeconomus*) from Pallasjärvi, northern Finland. Data are from preferred habitats: pooled data from peatlands and clearcuts for *M. agrestis*, and peatlands for *M. oeconomus.* Spring (S) and autumn (A) snap trapping were done on permanent small quadrats. Values of 0.01 are equivalent to zero. Note the low phases in 1971–1973 and 1984–1985 and the extended low phase in 1979–1981 (Henttonen et al. 1987). (b) Population cycles in Norwegian lemmings (*Lemmus lemmus*) from Finse, Norway. Data were snap-trap samples obtained from a 1-ha grid from the most productive site (grid M; Framstad et al. 1993, 1997, used with permission). Note the log scales used in Figs. 1–3.

Two hypothetical scenarios can explain observations of extended periods of low density in cyclic populations of mammals. In the simpler model (a), no real low phase exists; we perceive a low only because normal trapping methods cannot measure density below a threshold (Fig. 3a). In the more complex model (b), the detection threshold is lower and we can be sure that a low phase of more or less stable numbers persists over some time (Fig. 3b). The simpler model (a) requires us to explain only two states (a decline phase and an increase phase), whereas the more complex model (b) requires three phases (a decline, a low, and an increase). Although not all cycles are accompanied by an extended low phase, many well-documented cases can only be described by model (b). We will discuss the various explanations that may account for the low phase in model (b).

There are two broad hypotheses about the cause of the low phase. The classical model postulates that there is something ''wrong'' with the environment during the low phase. It may be that food supplies, damaged by overgrazing at the population peak, have not recovered, or alternatively, that predators have remained sufficiently abundant to prevent population growth of the prey. Similar scenarios could be devised involving parasites or diseases. The contrasting self-regulation model postulates that the environment is favorable during the low phase, but that something ''wrong'' with the animals prevents population increase. This model considers the physiological and behavioral characteristics of animals from the low phase as a potential source of demographic limitation.

Is something wrong with the environment?

Possible explanations of the low phase of the cycle could involve the food supply, predation, disease and parasites, or some interaction among these three factors

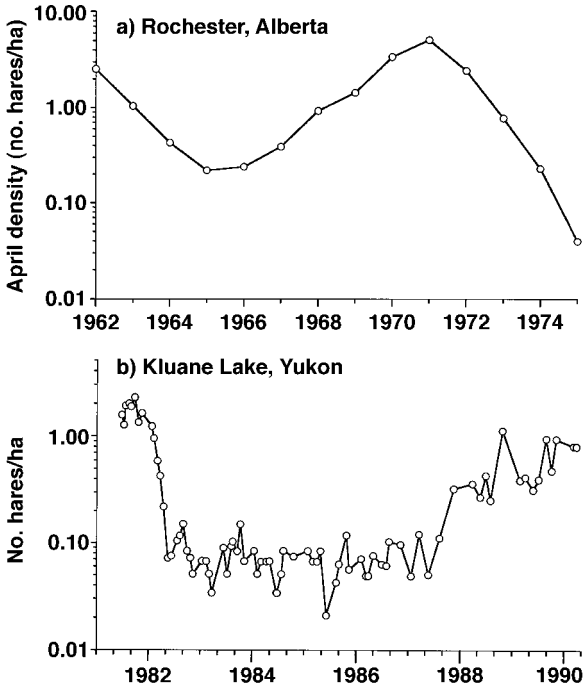

FIG. 2. (a) Population cycles in snowshoe hares (*Lepus americanus*) from central Alberta, Canada. Population density was obtained by live-trapping (see Keith and Windberg [1978] for methods). Note the low phases from 1964 to 1967 and from 1974 to 1975. (b) Cycles in snowshoe hares from the southwestern Yukon. Population density was obtained by live-trapping from an unmanipulated area, and estimated by the Jolly-Seber model (Krebs et al. 1986, 1995*b*). Note the extended low phase from 1983 to 1987.

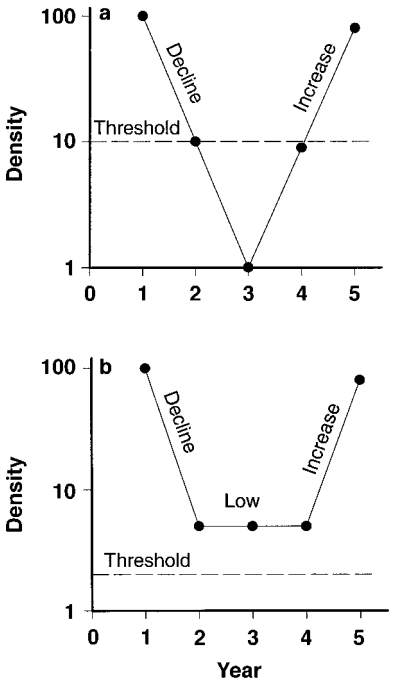

FIG. 3. Two hypothetical scenarios of the low phase in cyclic mammals. In the simpler model (a), no low phase exists; we perceive a low only because there is a threshold below which normal trapping methods cannot measure density. In the more complex model (b), the detection threshold is lower, and we can be sure that a low phase of more or less stable numbers persists. Field data support scenario (b) in some well-studied cases.

(Hörnfeldt 1994). No one has suggested that weather is responsible for the low phase of cyclic rodents and hares. The quantity of food supplies could be insufficient in the low phase of the cycle, and poor nutrition could thus be used to explain the failure of these populations to grow. This seems a priori unlikely to occur in cyclic small mammals. Virtually all studies of the food supply of snowshoe hares have found that food plants quickly recover once the population peak has passed, so that no food shortage exists for the few hares that are still alive by the low phase of the cycle (Smith et al. 1988, Keith 1990). Although we supplied artificial food in the form of rabbit chow to two snowshoe hare populations during the low phase shown in Fig. 2b, they showed no response to the supplemental food (Krebs et al. 1986). We can find almost no food supplementation experiments on cyclic vole and lemming populations during the low phase. A Townsend's vole (*Microtus townsendii*) population was provided with supplemental food through a period of low numbers (Fig. 4; Taitt and Krebs 1981), but no impact of supplemental feeding on the rate of population growth was detected. At present, the food quantity hypothesis seems unlikely to explain the low phase. However, additional feeding experiments for voles and hares during the low phase are needed to test this view. Two factors that should be considered in designing such experiments are: (1) the scale of the experiment must be sufficient, relative to the home range size of the animals, to prevent serious edge effects from occurring (Stenseth and Ims 1993); and (2) factorial designs examining the interactions between food and predation should be incorporated (Desy and Batzli 1989). We addressed both of these concerns in snowshoe hares on 1-km^2 grids in the boreal forest of southern Yukon during the increase, peak, and decline (Krebs et al. 1995*b*), and are examining responses of hares during the low phase (C. J. Krebs, D. Hik, and K. Hodges, *unpublished data*).

The low phase might be related to a deterioration in the quality of food because of the production of secondary plant defense compounds in response to intense grazing pressure at the peak population (Haukioja et al. 1983, Laine and Henttonen 1983, Bryant et al. 1985, Seldal et al. 1994). Seldal et al. (1994) found evidence that grazing-induced proteinase inhibitors are high in forage plants, and that these may affect the survival and reproduction of Norwegian lemmings during the peak and decline phases. In a test of this idea, however, Klemola et al. (1997*a*) found no relationship between cycle phase (increase vs. decline) and pancreas and liver size in *Microtus agrestis* populations (predicted to be heavier in animals consuming high levels of proteinase inhibitors in the decline phase). Neither Andersson and Jonasson (1986) nor Lindroth and Batzli (1986) were able to find any relationship between microtine cycles and the level of secondary plant compounds in vole diets. These results were echoed by those of Bergeron and Jodoin (1993), who found that intense grazing by meadow voles caused changes in the quantity of forage, but not in quality. Populations

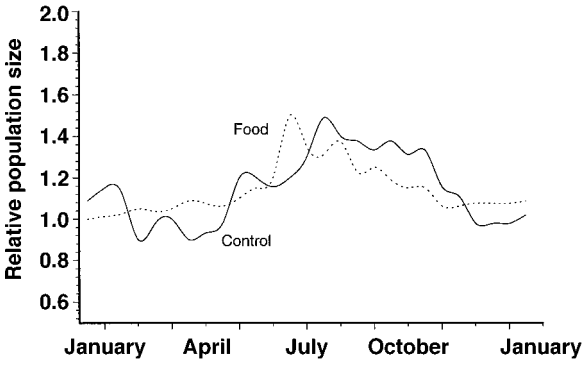

FIG. 4. Population estimates for Townsend's vole (*Microtus townsendii*) in a low-density period during which a feeding experiment was performed in 1973 (Taitt and Krebs 1981). Relative population size is calculated by standardizing densities to 1.0, using the density of each area for the period January–March 1973 before the food was added at the end of March 1973. Food-supplemented populations did not increase more than control populations, suggesting that food was not a resource in short supply.

of voles introduced to areas that had previously experienced high vole density and significant exploitation of the food supply grew well and showed no negative demographic effects in two studies (Oksanen et al. 1987, Ostfeld et al. 1993). In a third study (Agrell et al. 1995), however, negative effects on reproduction were found. The cause for these differences is not certain and may, in part, be related to habitat differences among the study sites and to the season when the experiments were carried out. In studies of hares, secondary plant compounds either did not change, or changed in a direction opposite to that predicted, relative to hare numbers (Sinclair et al. 1988). Thus, changes in plant quality do not appear to cause the extended low phase in these mammals.

The close link between the population dynamics of these cyclic herbivores and their predators has made it difficult to tease apart cause and effect. One possibility is that predators simply follow the changes in prey abundance and, thus, have little or no consequences on prey demography. The alternative is that predators drive these prey cycles and, thus, are necessary causes for cycles. In the latter case, the lows would be caused by predators that keep prey numbers depressed for an extended period of time after the decline. In snowshoe hares, the population decline appears to be directly related to intense predation. Keith et al. (1977) found that, during the low and increase phases in Alberta, only 23–31% of the hare population disappeared over winter; approximately half of this loss was caused by predators. In contrast, during the decline phase, up to 50–77% of the population disappeared over winter, with 60–70% of the loss being due to predators. Krebs et al. (1995*b*) found that, during the decline phase in the Yukon, <1% of the hares survived an entire year, and predators were responsible for 83% of this loss (comparable figures are not yet available for the low phase). Thus, predation plays a major role during declines of hares, but appears to be unimportant during the low phase.

In populations of microtines, declines may be caused by predators (Korpimäki and Krebs 1996), although declines can also occur in their absence (Pearson 1966). The hypothesis that resident predators deepen declines and extend the length of the lows has been proposed a number of times (Pearson 1966, Fitzgerald 1977, Grodzinski 1977, Korpimäki et al. 1991). The critical resident specialist predators capable of carrying out this task are thought to be the small mustelids, particularly least weasels *Mustela nivalis* (Hansson 1987, Korpimäki et al. 1991, Hanski et al. 1993). The most compelling evidence that predation plays a role in vole cycling comes from Fennoscandia and has recently been reviewed (Norrdahl 1995, Korpimäki and Krebs 1996). We summarize the Fennoscandian evidence as follows: southern regions show a great deal of habitat fragmentation and appear to have noncyclic vole populations regulated by generalist predators; northern regions have much less habitat fragmentation and cyclic vole populations are thought to be regulated by specialist predators, particularly weasels. Shrews fluctuate in synchrony with microtine rodents, apparently due to predation (Heikkilä et al. 1994). In much of Finnish Lapland since the mid-1980s, one of the major cycling voles, *Microtus agrestis*, has become much less common, for unknown reasons, and appears to have stopped cycling (Henttonen et al. 1987, Hanski and Henttonen 1996). One of its principal predators, the least weasel, has also become uncommon. The absence of weasels may explain why two associated species (*Clethrionomys glareolus* and *C. rutilus*) that formerly cycled, possibly as a by-product of cycling in *M. agrestis* (Henttonen 1987), now do not (Hanski and Henttonen 1996).

Although current models of predation may account for cycles with lows lasting only 1 yr (Hanski and Korpimäki 1995), none of the cycle models produces extended lows of the type shown in Figs. 1 and 2. To support the explanation that predation is responsible for an extended low phase, one would have to show that the low phase is a misnomer and that population trajectories are really like those shown in Fig. 3a. If the animals are indeed driven to virtual extinction over most of the landscape, dispersal from the few patches where they remain to other suitable habitats may significantly delay the increase. Much research, both theoretical and experimental, has focused on the role of habitat heterogeneity in space as a factor affecting the presence of cycling (Ostfeld 1992*a*, Stenseth and Lidicker 1992), but virtually none has focused on heterogeneity in time in good habitats as a factor retarding the increase and causing the low phase.

Regardless of the validity of predator-based explanations for the decline phase of the cycle, it seems unlikely that these explanations can account for the extended low phase. A recent analysis by Framstad et al. (1997), using a threshold model of the lemming times series (Fig. 1b), has highlighted the distinctiveness of the low phase, which is unlikely to be related to the effect of specialist predators. Sonerud (1988) found that, although shrews declined in synchrony with microtines in Norway (consistent with the argument that predators caused these declines), shrews recovered sooner than microtines (inconsistent with the argument that predation causes the lows, because both groups have similar potential population growth rates). Sonerud concluded that predators either do not cause the lows, or kill microtines (but not shrews) selectively during the lows. Given the dearth of prey present during the low phases, we suggest that the second possibility is unlikely. Mihok et al. (1988) radio-collared voles in Manitoba, Canada, recovered virtually all radios (95%) from voles that disappeared, and found no evidence of weasel predation that could account for the persistence

of the low-density phase. Norrdahl and Korpimäki (1995) radio-collared voles in Finland and found that weasels were a major cause of mortality during the decline, but not during the low year (1993) or the increase year. Sittler (1995) found no evidence that mustelid predation caused the 3-yr low of collared lemmings (*Dicrostonyx groenlandicus*) in northeast Greenland. Experimental removal of predators in the low phase could answer this question directly. Marcström et al. (1988) removed mammalian predators (foxes and martens, but not weasels, which were at low densities) from two islands in Sweden and found no effect on vole cycles. In one of the two cycles observed, a vole low phase extended over two years. Whether these results are limited to island situations or to the lack of removal of weasels is not clear, but this type of removal experiment does serve as a model for the kind of study needed to examine the impact of predation during the low phase. On Wrangel Island, a large (~5200 km^2) island in Chukchi Sea, both species of lemmings (*Dicrostonyx torquatus* and *Lemmus sibiricus*) go through 4–5 yr cycles, with *L. sibiricus* showing lows of 3 yr (Chernyavskii 1979). However, this island has no resident specialist predators (including no weasels), and it is estimated that other species of predators, which are active only in summer, kill no more than 10% of the lemming populations in peak years and 18% in decline years (Chernyavskii and Doroggoi 1981) Thus, neither mustelids nor other predators appear to be necessary for the low phase.

An alternate view of the role of predation involves indirect sublethal effects and the hypothesis that responses by prey to the risk of predation may affect reproductive fitness and ultimately cause the low phase. Hik (1995) proposed a predation-sensitive foraging hypothesis to explain how snowshoe hares respond behaviorally during declines. He argued that hares are able to assess predation risk in different habitats and, during the population decline, they attempt to minimize their probability of getting killed by restricting their activity to areas with dense cover. The correlate of good protective cover is poor-quality food. Thus, hares trade off reproduction (which requires high-quality overwintering forage before the breeding season) for survival during population declines. In the southern Yukon, Hik (1995) found that under conditions of high predation risk during the decline, hares shifted their winter activity from more open areas, with abundant, good-quality forage and the highest probability of predation, to dense spruce stands where only poor-quality forage remained, but the probability of predation was lowest. This resulted in poorer nutrition, which was reflected in lower mass of females and reduced fecundity at the end of the decline. A complementary hypothesis to that of Hik (1995) is the chronic stress hypothesis put forward by Boonstra et al. (1998). They proposed that chronic stress induced by high predation risk during the decline explains the reproductive and physiological changes that occur, and that this stress has long-term consequences. They found that all physiological measures associated with the stress response (hypothalamic–pituitary–adrenal response, energy mobilization, immune response, mass loss, and reproductive hormone response) were severely affected during the hare decline, and that all improved markedly two years after the predators had declined. The subsequent low phase of the cycle is hypothesized to be a lag caused by the indirect effects of high predation risk experienced during the decline (Boonstra and Singleton 1993, Hik 1995, Boonstra et al. 1998). These indirect effects are proposed to leave an indelible imprint on the hares, acting through changes in the physiological and neurological makeup of the animals, chiefly at the level of the brain (particularly the hippocampus and the hypothalamic–pituitary–adrenal axis; Sapolsky 1992, 1996).

A contrasting view is that of Korpimäki et al. (1994) and Ylönen (1994), who have proposed that predation risk is directly involved in population lows in vole cycles. They argue that high predation risk during the low phase, particularly from small mustelids, causes voles to curb their reproduction; only when predators become rare can vole populations increase again. In this view, the reduced reproduction in the low phase is a facultative response based on the presence of predators. However, controversy exists as to whether this mechanism is theoretically possible (Lambin et al. 1995, Kokko and Ranta 1996, but see Kaitala et al. 1997).

Diseases and parasites are, without question, the least studied of the factors that could limit cyclic populations during the low phase (Hörnfeldt 1978, Mihok et al. 1985, Descôteaux and Mihok 1986, Haukisalmi et al. 1988, Laakkonen 1996). In snowshoe hares, L. B. Keith et al. (1985) and I. M. Keith et al. (1986) found evidence that the rate of parasitism was broadly associated with hare abundance, that only one of six parasite species examined affected survival, and that no disease affected ovulation rate, litter size, or pregnancy rate. They concluded that disease did not play a causative role during the decline (Keith et al. 1986). A possible explanation for the reduced vigor shown during the low phase is transmission of microparasites from mother to offspring. Potential candidates include parasites such as the eimerians, intestinal protozoans that can be transmitted directly through ingestion of contaminated feces and that are known to have effects on survival in other species (see Fuller and Blaustein 1996). Both hares and microtines engage in coprophagy, a possible transmission route. More work, such as experimental treatment of individuals, needs to be done on microparasites before we will know their role in the low phase. At present, the predation hypothesis, either

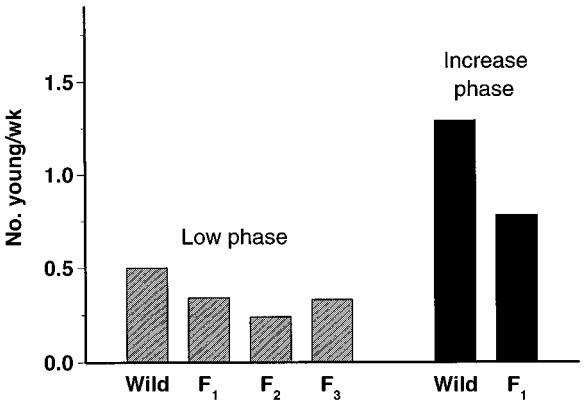

FIG. 5. Number of young produced per week by female meadow voles (*M. pennsylvanicus*) held in the laboratory under ideal conditions. Voles from the low phase of the cycle cannot reproduce as effectively as those brought in from an increasing field population, and this poor performance is transmitted to their offspring. (Data are from Mihok and Boonstra 1992.)

acting directly or indirectly, seems to be the most likely of the extrinsic hypotheses for the low phase.

Is something wrong with the animals?

The low phase could be the result of changes in survival, in reproduction, or both. Because the population is essentially stable during the low phase, births and deaths must balance. If the birth rate is high, there must be a high loss rate, at least among some age groups; if the birth rate is low, mortality must also be low. We do not have data to decide which of these two alternatives is correct for many species, but evidence indicates that both may play a role. In some field populations, evidence indicates that survival, particularly of the young, deteriorates significantly during the decline, even when adult survival and reproduction (as indicated by pregnancies and lactation) remain moderate or high (Boonstra 1985, Bondrup-Nielsen and Ims 1988). There has been considerable anecdotal speculation that voles and lemmings from the low phase are difficult to breed in the laboratory. Mihok and Boonstra (1992) have provided a unique set of data to quantify this belief (Fig. 5). Meadow voles (*Microtus pennsylvanicus*) from the low phase had poor breeding performance in the laboratory under ideal conditions of food, water, shelter, and no predation, whereas voles from the increase phase under the same conditions had excellent breeding performance. This poor breeding performance persisted through the F_3 generation, indicating that some form of inheritance had to be involved. These results argue against the classical model that something is wrong with the environment, and support the self-regulation model for the low phase. A similar conclusion was reached by Framstad et al. (1997), who used a time series modeling approach to understand the pattern of lemming (*Lemmus lemmus*) cycles in Norway (Fig. 1b). Thus, the key to understanding the low phase is to elucidate the quality of the individuals in the population.

How might self-regulation occur during the low phase? The central feature with these hypotheses is that a mismatch occurs between the quality of the animal present during the low phase and the quality of the environment to sustain population growth, i.e., the environment (food, space, etc.) is adequate, but population growth is constrained by something in the animals themselves. We should note that all mechanisms of self-regulation potentially can operate directly or indirectly. Direct mechanisms affecting mortality rates, for example, would include infanticide, wounding or killing of juveniles or subadults. Indirect mechanisms affecting mortality rates would include territorial exclusion, which causes additional movements that expose individuals to hazards such as predation or bad weather. We consider four hypotheses about how self-regulation might operate in the low phase.

The sociobiological hypothesis proposes that related animals are treated differently from unrelated ones within the social system, and this affects survival or reproduction. Charnov and Finerty (1980) first suggested this idea for microtine cycles, but it has been questioned by Kawata (1990), Pugh and Tamarin (1990), and Stenseth and Lomnicki (1990). For Townsend's vole *Microtus townsendii,* Lambin and Krebs (1991) suggested an alternative mechanism involving matrilines, but the generality of this idea has been challenged by Ostfeld (1992*b*). Mountford et al. (1990) modeled a similar idea, called the kin collaboration hypothesis, to examine its applicability to population cycles in Red Grouse (*Lagopus lagopus*, a species with strong territorial behavior), and recent experimental evidence has not refuted it (Moss et al. 1996). At the densities typically found during the low phase in microtines and hares, it seems improbable that negative intraspecific interactions between unrelated animals at that time are likely to have any demographic consequences. Hence, we reject the sociobiological explanation.

The polymorphic behavior hypothesis (Chitty 1967) proposes that natural selection acts during cycles to change the composition of the population. According to this view, spacing behavior (operating through territoriality, dispersal, infanticide, and social mortality) is the key to understanding cycles. This hypothesis has been the impetus for an enormous amount of research and controversy, but support for it has been equivocal (Gaines 1985). In a quantitative genetic study of a fluctuating meadow vole population, this hypothesis was rejected because no evidence was found of high heritability for body size traits (Boonstra and Boag 1987). However, there was no measure of the heritability of any aggressive or spacing behavior trait in this study. A study of collared lemmings (*Dicrostonyx groenlan-*

dicus) from the Canadian Arctic, in a population that appeared to be kept at low densities by intense predation (Krebs et al. 1995*a*, Reid et al. 1995), found no significant heritability for three behaviors (aggression, dispersal, and activity) or for body size traits (Boonstra and Hochachka 1997). No heritability studies have been carried out on snowshoe hares (*Lepus americanus*), but field studies have found no significant spacing behavior that is likely to be affected by density, as the hares are not territorial and their home ranges overlap broadly (Boutin 1984). Thus, the polymorphic behavior hypothesis seems unlikely to be correct.

The outbreeding hypothesis proposes that cyclic populations undergo regular cycles of inbreeding and outbreeding. The wood lemming (*Myopus schisticolor*) at times shows a pronounced female bias in sex ratio, and Maynard Smith and Stenseth (1978) postulated that this is a result of periodic inbreeding during the population low. Fredga et al. (1993) examined overwintered peak animals and concluded that inbreeding did not occur in this species, because there was no deviation from Hardy-Weinberg equilibrium. However, one generation of panmictic breeding is sufficient to restore the Hardy-Weinberg equilibrium (Weir 1996). A major problem in trying to assess the significance of inbreeding is the lack of knowledge of social and breeding structure at low densities.

Smith et al. (1975) suggested that aggressiveness is correlated with heterozygosity, such that inbreeding at low density would lead to reduced aggression and increased population growth, whereas high levels of outbreeding that are likely to occur at peak densities would lead to increased heterozygosity, increased levels of aggression, and reduced levels of population growth. Recent evidence on meadow voles (Boonstra et al. 1994) found that aggression was significantly related to heterozygosity (although the amount of the variance explained was low), but heterozygosity did not vary with population size or with reproduction. Thus, this hypothesis was not supported. The outbreeding explanation seems unlikely to explain the low phase.

Finally, the senescence–maternal effects hypothesis proposes that a change in maternal quality occurs in animals present during the peak phase, carries over into the decline and low, and may serve to maintain the low phase by affecting survival and reproduction (Boonsta 1994; see also Tkadlec and Zejda 1998). This change is produced through density-dependent social inhibition of maturation in peak years, whereby young born in spring and early summer (that would normally mature and breed in their birth year when density is low) are forced to delay maturation until the next breeding season. Combined with a shortened breeding season in the peak year (breeding stops 1–2 mo earlier than in other years) and with complete turnover of the breeding population (virtually no microtine survives to breed in two years in any phase), the result is a shift in age structure: animals present in the spring of the decline are much older than animals present in the spring of the peak or increase. This older age, possibly interacting with stress effects experienced at peak densities, may have long-term consequences acting through changes in maternal condition. As previously outlined, maternal effects may also play a role in snowshoe hare cycles (Boonstra and Singleton 1993). Here, the causal agent is not age shifts (although they may also play a role; see Boonstra 1994), but changes in foraging behavior (Hik 1995) and in physiology associated with chronic stress (Boonstra et al. 1998) as a consequence of high predation risk during the decline. Maternal effects can cause a resemblance not only between parents and their offspring, but also between grandparents and grandoffspring (Kirkpatrick and Lande 1989, McRoberts et al. 1995). In forest insects, maternal inheritance may explain population fluctuations in some lepidopteran pests (Rossiter 1991, 1994). At present, the maternal effects hypothesis seems to be the most likely of the intrinsic hypotheses for the low phase.

Directions for future research

A plethora of hypotheses exists about what might be wrong with individuals in the low phase of the cycle. Unfortunately, there are few data on these individuals because they are so difficult to study at low densities. It would seem prudent to require replication of these tests. We suggest that, although none of these ideas can be ignored in our present state of knowledge, some ideas seem more likely than others. Stenseth et al. (1996), in a recent analysis of field evidence using time series analysis, concluded that a hypothesis involving both extrinsic and intrinsic self-regulation was needed to explain microtine density cycles.

What experiments should be conducted to examine extrinsic factors during the low phase? The most likely explanation emphasizing extrinsic factors is that predators maintain prey populations at low density, and the critical experiment must be to eliminate or reduce predators from areas large enough to affect demography. We commend this experiment to future workers on small mammals. Such an experiment has already been carried out on snowshoe hares during the decline (Krebs et al. 1995*b*). For voles, removal of avian predators has had no significant effect on population dynamics (Norrdahl and Korpimäki 1995). The key predators that potentially act during the low phase of the vole cycle appear to be weasels; hence, manipulations must target these animals. Klemola et al. (1997*b*) showed that reducing small mustelids increased female productivity and slowed population growth in *Microtus agrestis*. Clearly, if removal of these mustelid predators truncates the low phase, it would call into question the self-regulation hypotheses. Thus, these predator removal experiments are critical.

What data are required to test for possible mecha-

nisms of self-regulation in the low phase? We suggest that three types of information would be useful.

1. *Maternal effects.*—The most likely explanation involves maternal effects, which directly affect the performance of individuals. Thus, we need detailed studies of individual performance in terms of numbers of litters, survival of offspring, and causes of death to obtain a better description of what happens to individuals in the low phase in contrast with individuals from other phases. Although the field must be the ultimate benchmark in interpreting experimental results, we see that a combination of field and laboratory experiments will be needed to test this hypothesis. These studies may be coupled with more specific tests of the roles of senescence and stress, involving detailed studies of reproductive performance in captivity to assess the physiological mechanisms by which these factors may operate.

2. *Relatedness of individuals.*—An unlikely explanation is that interactions between unrelated animals play a role during the low phase. Because the techniques for determining relatedness are now readily available (Ishibashi et al. 1997), we can reject a lot of ideas involving nonrelatives. We need to follow the spatial relationships among relatives through the low phase in small-mammal populations. Are relatives scattered at random across the landscape, or do they cluster in colonies? This information would test directly one important assumption of the sociobiological and outbreeding hypotheses, which depend on relatives interacting during the low phase.

3. *Genetics of behavioral traits.*—The least likely, but still vaguely possible, explanation involves genetic differences between animals present at the low phase and those present at other phases. If spacing behavior is relevant to maintaining populations in the low phase, we need to determine the heritabilities of the relevant behavioral traits. There are few studies of the inheritance of traits that are important in spacing behavior in small mammals. Although we appreciate that these studies are difficult, they are crucial in evaluating the polymorphic behavior hypothesis.

Thus, we think it is imperative to focus primarily on two major explanations for the low phase: the role of predators and the role of maternal effects. One way to make these experiments most robust would be to replicate them in key small-mammal species on two continents. We suggest that, by designing similar manipulations on similar *Microtus* species in Europe and North America, we can narrow the scope of potential explanations for the low phase of their cycles.

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