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POPULATION CYCLES REVISITED

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Periodic fluctuations or cycles in populations of small mammals have been widely studied, but much controversy still exists about their causes. Cycles of voles and lemmings are produced by the integrated effects of intrinsic and extrinsic factors, and the problem is to define accurately how these interact. Spacing behavior is a key component of population regulation in voles and lemmings, and this is illustrated most dramatically by the fence effect. We do not know which mechanisms produce changes in social behavior. Phenotypic changes produced by maternal effects or stress are now believed most likely, but there has been too little work done on genetic effects on spacing and we know almost nothing about kin-related social behavior in voles and lemmings. Both predation and food supply may be the extrinsic factors involved in cyclic population dynamics. Single-factor experiments suggest that food shortage by itself does not seem to be a necessary factor for cycles nor does predation, but the interaction between food and predation could be a key variable in understanding how extrinsic factors affect cycles of voles. Cycles of snowshoe hares are caused by an interaction between predation and food supplies, possibly integrated through risk-sensitive foraging. Spacing behavior is not a component of cycles of hares because snowshoe hares differ from voles and lemmings in having no known form of spacing behavior that can produce social mortality. The short-term cycle of voles and lemmings thus seems to have a different explanation from the long-term cycle of snowshoe hares. In some places, lemmings may be locked in a predator-pit at low density. Experimental exclusion of predators improved survival of adults in a population of collared lemmings, but was not sufficient to allow them to escape the predator-pit because of losses of juveniles. Whether cyclic populations of lemmings also fall into a predator-pit in the low phase remains to be determined. The low phase does not occur in every cycle and it is particularly difficult to explain. Progress in analyzing cyclic fluctuations has been made most rapidly when we define clear alternative hypotheses and carry out experimental manipulations on field populations. Much remains to be done on these small mammals.

Key words: cycles, snowshoe hares, *Lepus americanus,* voles, lemmings, *Microtus*

Periodic fluctuations in the numbers of mammals have fascinated ecologists since they were first described in the early part of this century. Early explorers and naturalists commented on the cycle of snowshoe hares (*Lepus americanus*) in Canada's boreal forests, and when Hewitt (1921) began to analyze the fur-trading records of the Hudson's Bay Company, biologists began to take a more active interest in understanding these fluctuations. At the turn of the century, Norwegian biologists had begun to study lemming populations, but it was not until Charles Elton's (1924) classic paper that we began to recognize that periodic fluctuations were common in northern ecosystems (Finerty, 1980; Stenseth and Ims, 1993).

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The importance of these early discoveries cannot be overemphasized. The prevailing view of natural ecosystems in the early 1900s was the classical idea of the Balance of Nature. Natural populations like lemmings and hares were believed to exist in a state of balance, and outbreaks or fluctuations in numbers were believed to result from human interference with nature (Egerton, 1973). However, this paradigm could not be stretched to cover periodic fluctuations in populations of northern mammals in ecosystems as yet little affected by humans. The Balance of Nature had to be redefined, and the new ecological concept bears little relationship to what was accepted by biologists of the past century as the central paradigm of ecology (Pimm, 1991).

Since Elton's (1924) classic paper, three generations of ecologists have been trying to solve the riddle of periodic fluctuations, and the purpose of this paper is to review this progress. Few reviews of population fluctuations in mammals have been published since the review by Krebs and Myers (1974). Keith (1990) has reviewed population cycles in snowshoe hares, and Batzli (1992) reviewed populations of small mammals in general. Taitt and Krebs (1985) reviewed population cycles of *Microtus,* and Lidicker (1988) reviewed progress in understanding cycles of rodents. Other reviews on more restricted aspects of cyclic populations continue to appear. The literature on this subject is enormous and periodically we need to step back and assess the directions of research and the status of the hypotheses suggested to explain cycles. The outcome of this assessment should be a list of suggestions for future research, which is my goal here.

An enormous amount of controversy exists in ecology over the causes of population cycles. Many ecologists view this with alarm. I do not share this discomfort. Controversy and criticism should be encouraged in science as long as it is constructive and not personal. We cannot make progress with our hypotheses or our methods without constant evaluation and suggestions for improvements. Our focus should be on the question of testability and experimental design. Population ecology is not a baseball game in which one team wins and another loses, one team is right and others are wrong. All our ideas will be revised and improved in the long run, and we must not permit controversies in ecology to be cast as a movie of good-versus-evil.

DEFINITION OF THE PROBLEM

Periodic fluctuations in populations of mammals often are loosely called "cycles." There is little harm in this usage as long as you realize that these population changes do not qualify as true mathematical cycles. Nevertheless, names are important because we tend to search for hypotheses or explanations for phenomena like "cycles" that we consider as a coherent group. Periodic fluctuations are population changes that recur with some regularity. Snowshoe hares, for example, often fluctuate with a period ranging from 8 to 13 years, with an average of 9–10 years (Keith, 1990). Can we define periodic fluctuations more rigorously?

Chitty (1952, 1960) defined periodic fluctuations in voles by their associated demographic and physiological characteristics. Changes in body mass occurred in cycles, he found, such that body size was directly correlated with population phase (the Chitty Effect—Boonstra and Krebs, 1979). Population cycles can be defined by certain parameters, such as phase-related changes in age at sexual maturity, reproductive rates, and survival (Krebs and Myers, 1974), thus creating a syndrome of characteristics to look for in any population before you would class it as cyclic. I call this the *biological definition of cyclic fluctuations.* Note that this definition says nothing at all about the period or the amplitude of the cycle.

A second definition of cyclic fluctuations began to appear in the literature in the 1980s, chiefly from Fennoscandian sources. Cycles were defined mathematically and precisely, and the variability or amplitude of the population changes became the defining characteristic. Hansson and Henttonen (1985) used the standard deviation of the logarithms of population size (s-index) to define cycles, and they concluded, for example, that populations of voles in southern Sweden were not cyclic while those in northern Sweden were cyclic. I call this the *mathematical definition of cyclic fluctuations.* The seeds of endless confusion were sown with this definition of cycles because it was now possible to have a population that was *cyclic* by Chitty's definition and *non-cyclic* by the s-index definition (Hansson and Henttonen, 1985).

This might be dismissed as an argument over words, and we could all carry on with some distinction like *cycles-type m* and *cycles-type b.* But the critical conclusion that follows from this type of analysis is that different population processes must be affecting cyclic and non-cyclic voles, such as those in northern and southern Sweden. Sandell et al. (1991) argued that the distinction between *cyclic* and *non-cyclic* populations based on the s-index is artificial, and that we should look for an integrating theory that spans the range of population behaviors shown by small mammals from stability to chaotic fluctuations and cycles.

In this paper, I will adopt the biological definition of cycles and seek an explanation for this class of events. In making this search I do not wish to imply that these cyclic populations differ in any fundamental way from other populations of rodents that may be stable or fluctuate irregularly. The problem of regulation is general to all these populations, but for the moment I want to consider only cyclic populations before we try to broaden the generalizations.

HYPOTHESES TO EXPLAIN CYCLES

Many different questions can be asked about cycles and, without guidance beforehand, no one can determine which of these questions are most valuable scientifically. Valuable questions lead to general insights and suggest many avenues of research, but you can know this only after the question you propose has been thoroughly investigated. Common questions about cycles might include: 1) What produces population declines? 2) What stops population growth? 3) What determines cycle length? 4) What affects the amplitude of cycles? 5) What causes the phase of low numbers? 6) What affects the rate of population increase? 7) What determines the seasonal timing of the decline phase? Many additional questions are possible and the critical point is that you need to specify exactly what question you are addressing to avoid misunderstanding. Hypotheses to explain cycles in the sense of question 2 will be different from those designed to answer question 4.

In this paper I wish to address question 2, which is the most general question of population regulation (Krebs, 1994, p. 322). I assume that if I can answer question 2, I also will be able to answer question 1. I make two critical assumptions at this point. 1: *Cycles represent a repeatable process in time.* I assume that if I can explain the cycles of snowshoe hares in Alaska in the 1970s, the same explanation should apply in Quebec in the 1990s. 2: *Cycles of different species will have the same explanation.* I can thus test explanations for cycles of brown lemmings (*Lemmus sibiricus*) with data from cycles of California voles (*Microtus californicus*).

Both these assumptions might be wrong. I adopt both of them for reasons of simplicity to maximize the testability of hypotheses about cycles. I believe that we ecologists should seek the underlying generalities about cycles and not get lost in describing differences among cycles. Every naturalist can show differences between every cycle in every population. In my view, these differences will be interesting and possibly explicable once we understand the underlying generalities.

Hypotheses to explain cycles fall into

five classes: 1) food supply; 2) predation; 3) food supply and predation; 4) qualitative changes in individuals; 5) multiple factors. Batzli (1992) lists 22 hypotheses to explain cycles, a more detailed breakdown than I list here. Much of the controversy over cycles of small mammals has centered on whether or not one needs to include individual differences (4) in any explanation for cycles. Batzli (1992) and Chitty (1960) both agree that individual differences (4) are a necessary component of an explanation for cycles of voles and lemmings. Others, like Hanski et al. (1993), argue that predation is both necessary and sufficient to explain cycles of microtines.

Before any of these hypotheses can be tested it is necessary to state explicitly the hypothesis, the alternative hypotheses, and their predictions. Few authors do this, and journal reviewers and editors should start to demand such rigor. Given a prediction, mensurative or manipulative experiments can be done to test for the effect. The unanswered question at present is how much valid inference can come from experiments on fenced populations of small mammals. Ford and Pitelka (1984) used 0.07–0.13-ha enclosures to analyze the effect of food and water supplements on population changes in *M. californicus*. Batzli and his students (Desy and Batzli, 1989) have used *Microtus ochrogaster* in 0.13-ha enclosures to test multifactorial hypotheses with explicit predictions. Ostfeld et al. (1993) used 0.16-ha enclosures to test for relationships between reproduction and population density in *Microtus pennsylvanicus*. I do not know if enclosures of this size are large enough to mimic the processes that occur in natural populations. Nor do I know if the time periods ecologists can use in enclosure studies are long enough to see natural dynamics. If they are, we can make progress more rapidly because enclosures permit experiments that would be logistically difficult in open-field populations.

I will discuss here only the predation-hypothesis (2) for cycles, the food-supply-and-predation hypothesis (3), and the qualitative-change hypothesis (4), and I will leave the multiple-factor hypothesis (5) for later discussion. I have picked these in part because they are relevant to the research I will discuss and in part because they are the most popular of the current theories to explain cycles in small mammals. Changes in the food supply (hypothesis 1) have never been shown to be necessary or by itself sufficient to cause cycles, and most ecologists who have supported hypothesis 1 in the past now argue for hypothesis 3 or 5.

PREDATION HYPOTHESIS

The predation hypothesis asserts that direct mortality caused by predation and indirect effects produced by the presence of predators are necessary and sufficient causes of the observed cyclic fluctuations in populations. The predation hypothesis for rodent cycles has been asserted most strongly by Hanski et al. (1993) for populations of Fennoscandian voles on the basis of being able to fit a mathematical model to time-series data. This paper would seem to be the perfect ecological illustration of the logical fallacy of post hoc-ergo propter hoc, the problem of confusing correlation with causation. Because many ecological changes (e.g., parasite loads, territory size) are associated with cyclic changes in populations, it is possible to model any or all of these. Models such as that in Hanski et al. (1993) are monuments to mathematical creativity, but should not be confused with reality. Such models are primarily of heuristic value (Oreskes et al., 1994).

Two conditions are necessary for the predation hypothesis to be applied as a sufficient explanation for cycles (the *strong model*). First, the removal or reduction of predation should prevent cycles from occurring. Second, predation must be shown to have indirect effects on reproduction and growth to explain the symptoms of cycles (Korpimäki et al., 1994; Ylönen, 1994). An alternative model is to propose that predation is necessary for cycles, but not sufficient by itself. I call this the *weak model.* In this alternative model, predation could interact with food supplies or with spacing behavior or any other ecological factors to produce cycles, or the reproductive changes that occur during cycles may have a completely separate explanation not involving predation. Note that the weak model makes the same prediction for a predator-removal experiment as the strong model.

The strongest support for the predation model comes from work in Fennoscandia and is based on the simple observation that populations of voles in northern Fennoscandia cycle, while those in southern Fennoscandia do not cycle (Hansson and Henttonen, 1985). I do not accept this north-south dichotomy for two reasons. First, it does not appear in North America and this lack of generality ought to be worrisome. The north-south dichotomy in Fennoscandia may not be related to direct latitudinal effects such as snow cover. Second, this dichotomy for Fennoscandia is compromised because it is confused by the mathematical definition of cycles instead of the biological definition of cycles, and it is confounded with the habitat fragmentation associated with agriculture in southern Fennoscandia. Erlinge et al. (1984), Erlinge (1987), and Agrell et al. (1992) report on a supposed non-cyclic population of *Microtus agrestis* in southern Sweden. This population shows all the biological attributes of a cyclic population (Fig. 1). I would re-interpret the Fennoscandian cline to be a difference in cyclic amplitude between northern and southern populations (Xia and Boonstra, 1992). This difference in amplitude could in fact be partly due to different predator communities in these two regions, but this remains to be tested experimentally. Habitat fragmentation also seems to have the potential to eliminate cyclic fluctuations in mammals, although the data on this effect are poor for microtines. Keith (1990) discusses how the historic cycles of snowshoe hares disappeared in southern Wisconsin as the forest habitat was fragmented, and he observed the same effect in central Alberta. The mechanism behind the fragmentation effects in hares appears to be heavy predation (Keith et al., 1993). More work is needed on fragmentation in relation to cycles.

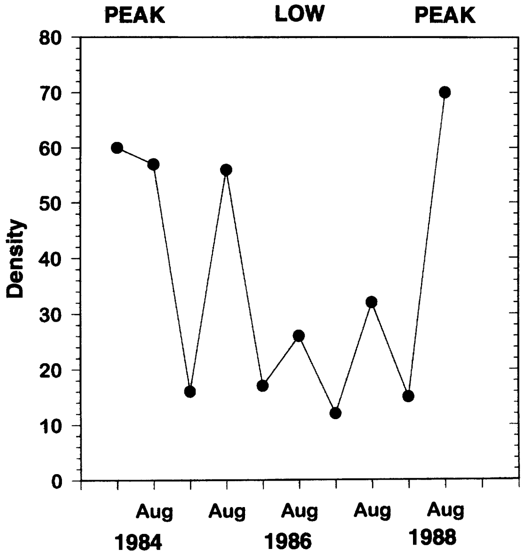

FIG. 1.—Population fluctuations in *Microtus agrestis* near Lund, Sweden. Erlinge (1987) and Agrell et al. (1992) call this population non-cyclic and yet point out that voles of high body weight were present in 1985 and 1988. I believe the population trends are consistent with the biological definition of a cycle. The classification of such populations may have effects on our theories about cycles and their causes (data from Erlinge, 1987, and Agrell et al., 1992).

The key question for the predation hypothesis is the impact of predator removals. For voles and lemmings the existing experiments are not positive. Marcström et al. (1988) removed predators from an island in the Baltic Sea for 8 years with no significant effect on abundance of voles during two cycles. Norrdahl and Korpimäki (1995) have removed owls and weasels from a large study area in Finland for 3 years with undetectable effects on the vole cycle. Lidicker (1973) studied an island population of *M. californicus* that showed no evidence of a 3-year cycle in the absence of mammalian predators. These comparisons of island and mainland populations provide hypotheses for experimental tests, particularly because islands may compromise dispersal that thus show stable densities (Tamarin et al., 1987). More experiments are clearly needed, but for the present there is not a single field experiment to show that predator removal has any impact on cycles of voles or lemmings. Note that evidence that predators eat a large fraction of the population (Pearson, 1971) is necessary, but not sufficient, to test the predation hypothesis. The key question is whether the predation is additive or compensatory, whether the animals would die from other causes if predation were absent. Predation does not seem to be either necessary or sufficient to generate a cycle in these small mammals.

INDIVIDUAL-DIFFERENCES HYPOTHESIS

The individual-difference hypothesis states in general that changes in the attributes of individuals are a necessary component of cycles. The changes may be phenotypic or genotypic, and involve physiological and behavioral parameters. The most general statement of this hypothesis was made by Chitty (1960, 1967) and I have called it the *self-regulation hypothesis* (Krebs, 1978). The self-regulation hypothesis is a concept and to test it a mechanism must be specified. Three mechanisms have been put forward for self-regulation. 1) *Genetic changes* produced by density-dependent and frequency-dependent natural selection could be involved in regulation. 2) *Behavioral and physiological effects* caused by social interactions associated with crowding and by reproductive state could affect individuals in cyclic populations. These are sometimes called "stress" effects and are phenotypic. 3) *Maternal effects* in early life could have lasting effects on the physiology of individuals. Note that because there are several mechanisms for self-regulation, rejecting one mechanism does not mean that one has rejected the entire hypothesis.

A necessary precondition for the self-regulation hypothesis to apply is that the species concerned has some form of spacing behavior. There is abundant evidence in voles and lemmings for spacing behaviors, but for snowshoe hares discussed below there is no evidence of any form of spacing behavior so that we might expect a priori that this hypothesis will not apply to this species.

Individual differences certainly occur in every population, and changes in age structure, genetic composition, sex ratios, birth rates, and expectation of life must occur in any fluctuating population regardless of what is causing the fluctuation. Consequently we must not simply rely on descriptions of individual differences over a cycle to test this hypothesis. Experimental manipulations must be the focus of all tests of cyclic hypotheses, and endless descriptions of correlations, although useful, will not answer questions about causation.

The original hypothesis for self-regulation was called the Chitty hypothesis or the *polymorphic-behavior hypothesis,* which made the risky prediction that spacing behavior was highly heritable and that spacing behavior limited breeding density (Krebs, 1985; Tamarin, 1985). This hypothesis has been rejected by Boonstra and Boag (1987) with heritability data not directly related to the proposed mechanism of spacing behavior. On the basis of this one study, on one species of vole, the literature now states that this hypothesis has been rejected. I suggest that it is foolish to base a conclusion on only one test of any hypothesis and that further investigation would be desirable.

A second hypothesis for self regulation was presented by Charnov and Finerty (1980) and is called the *sociobiological hypothesis.* It is a phenotypic hypothesis that predicts that spacing behavior limits breeding density and that relatedness of neighbors changes over the cycle. Kin are hypothesized to tolerate kin in spacing behavior and to react more aggressively toward non-kin (Tamarin and Sheridan, 1987). The risky prediction is that at low density kin groups will form and these will be lost at high density due to intruder pressure. Ims (1989) rejected this hypothesis for *Clethrionomys rufocanus.* Pugh and Tamarin (1990) could find no evidence for this prediction in *M. pennsylvanicus,* and Kawata (1990) concluded that there was no evidence for the sociobiological hypothesis. Ferkin (1990) discussed the difficulties faced by any hypothesis based on kin recognition in rodents. Repeated contacts are needed for rodents to recognize close relatives, and familiarity based on association is the usual cue that rodents use to define close relatives.

A related hypothesis by Lambin and Krebs (1991) turns the predictions of the Charnov-Finerty hypothesis upside down and suggests that matriarchal kin groups may affect the rate of population growth in microtine rodents. Limited data (Lambin and Krebs, 1993) support this version of the sociobiological hypothesis, and have illuminated the fact that there are virtually no data on the kin structure of natural populations of small mammals. Kin selection has been implicated in population cycles of the red grouse (*Lagopus lagopus scoticus*) in Scotland in which the central mechanism is differential aggression by males between kin and non-kin (Watson et al., 1994).

The *stress hypothesis* was first proposed to explain cycles by Christian (1950). It was an attractive hypothesis because it was physiological and phenotypic, and the delayed effects of stress on the adrenal-pituitary system also could be invoked to provide a time lag to generate cycles in density. Unfortunately, field data failed to verify most of the predictions of the stress hypothesis and it was rejected (Krebs and Myers, 1974). More recently, Boonstra (1994) has revived the stress hypothesis under a new guise as the *senescence hypothesis.* This hypothesis focuses on the change in age structure that occurs during a population cycle such that older individuals accumulate in declining populations. Older individuals suffer from an inability to maintain endocrine homeostasis, and this results in a reduction of reproductive effort and increased mortality due to physiological deterioration associated with age. Boonstra (1994) makes six predictions based on the senescence hypothesis, and he currently is attempting to test this potential mechanism for self-regulation.

To summarize, the self-regulation hypothesis has focused on the role of spacing behavior in affecting birth, death, and dispersal rates in populations of small mammals. The predictions of at least four of the possible mechanisms for self-regulation are quite clear and at present there are limited data against the genotypic mechanisms of self-regulation and more data against the Charnov-Finerty hypothesis. It would be foolish to reject any of these possible mechanisms until we have more tests, and more studies of phenotypic mechanisms of self regulation are urgently needed. The self-regulation hypothesis is far from dead, and none of the issues that it has raised will be solved by ignoring it.

Cycles of Voles

One key question ecologists can ask about cycles is *what experimental manipulation will stop cycles?* For field populations none of the manipulations carried out in the past 40 years have been shown to stop cyclic fluctuations with the single exception of the fence effect (Krebs et al., 1969). Vole populations enclosed in a large fence increase in density to a level 2-5 times normal, overgraze the grassland and decline from starvation. The fence effect is illustrated in Fig. 2 (a, b, c) for *Microtus townsendii.* Within 6 months, this particular fenced population had increased to 2,500 voles/ha, ca. 50% above the control population outside (Boonstra and Krebs, 1977). The resulting habitat destruction is shown in Fig. 2b. Not all populations of small rodents enclosed in a fence show the fence effect (Ostfeld, 1994) and we do not know why.

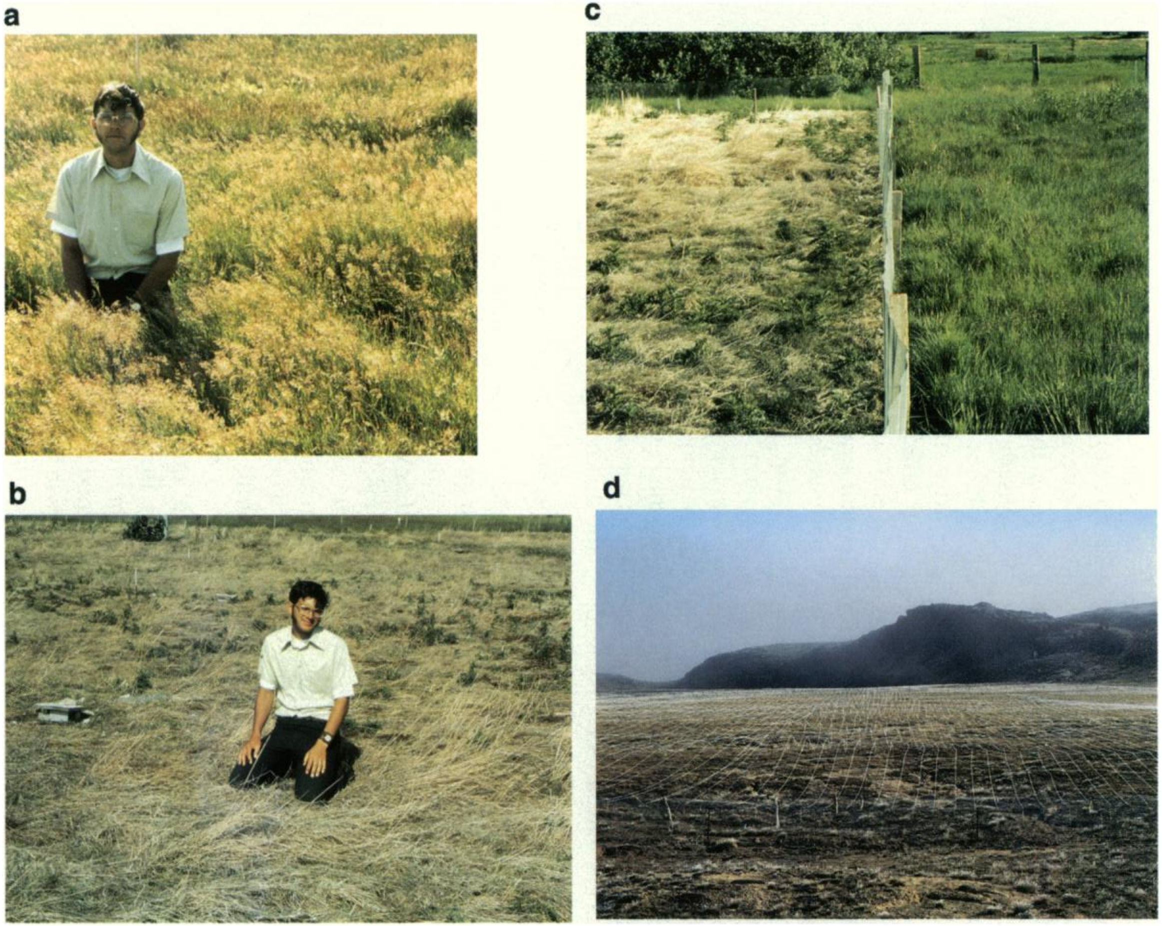

FIG. 2.—a) The fence effect. Rudy Boonstra in a unfenced trapping area at Westham Island, British Columbia, 22 July 1975. This is the midsummer aspect of these perennial grasslands even in years of high abundance of voles. At the time of this photo densities of *Microtus townsendii* were >400/ha (photo courtesy of R. Boonstra). b) Inside the 0.3-ha enclosure for voles, 22 July 1975. This is the peak of the growing season in summer. The density of voles was ca. 1,280/ha at this time in the enclosure, at least 50% higher than the control densities (photo courtesy of R. Boonstra). c) The fence of the 0.30-ha enclosure with signs of severe overgrazing inside the fence, July 1975. The only green plants visible in the enclosure are Canadian thistles (*Cirsium arvense*), which voles will not eat (photo courtesy of R. Boonstra). d) Predator exclosure for collared lemmings at Pearce Point, Northwest Territories, August 1992. An 11-ha area of habitat for lemmings was enclosed in a 1.3-m fence of poultry wire and covered with monofilament nylon lines spaced at 45 cm to keep raptors out (photo courtesy of D. Reid).

In a remarkable prescient paper Lidicker (1962) suggested that emigration might regulate population density. The fence effect, discovered 4 years later, confirmed Lidicker's ideas and helped to stimulate interest in analyzing how dispersal could be involved in population regulation. Dispersal is one possible consequence of spacing behavior, and spacing behavior allows priority of access to resources. Evidence is accumulating that for males the limiting resource is females, and for females the limiting resource is space (Boonstra, 1977). Space has two components on which there is less agreement; space could mean *food* (Ostfeld, 1985) or space could mean *space-free-from-infanticidal-females* (Wolff, 1993). I suspect the social concept of space is more important than the food concept for voles and lemmings, but more experiments to test this view are needed.

Spacing behavior is an attractive process because much experimental evidence in rodents shows that it can affect dispersal, mortality, and reproduction; the three key demographic processes driving population fluctuations (Krebs, 1985). The control of recruitment in voles is largely under the influence of the breeding females in the population (Boonstra and Rodd, 1983; Rodd and Boonstra, 1988).

If spacing behavior is an important process in population cycles, several critical questions need answering: *Why does spacing behavior not produce a fence effect at all times?* Boonstra and Krebs (1977) predicted that a cyclic population fenced at the peak of the cycle would not demonstrate a fence effect because dispersal is not necessary for the decline phase. No one has tested this experimentally. If the fence effect does not operate at all times, it strongly suggests that there must be variation in spacing and dispersal behavior, and these individual differences need to be analyzed. *What controls the proximate expression of spacing behaviors?* We do not know the heritability of spacing behaviors in any species of microtine rodent. Many ecologists believe that we can construct a complete theory of population dynamics without studying heritability because spacing behavior is clearly affected by ecological and environmental factors. I doubt that this is true. *Why does spacing behavior not lead to population stability?* Heske and Bondrup-Nielsen (1990) provide one answer to this question. The standard theoretical prediction that spacing must lead to stability (Stenseth, 1986) must be wrong in lieu of the fence effect. The simplest explanation of why stability is not achieved is that kinship effects are present such that different types of individuals are using different dispersal strategies, and the composition of the population changes with density. If daughters can breed in their mother's territory (Lambin, 1994; J. O. Wolff, in litt.), spacing behavior cannot stabilize density. Seasonality also is one component of the answer to this question, as Heske and Bondrup-Nielsen (1990) suggest, but the exact reason for this is far from clear.

CYCLES OF SNOWSHOE HARES

The 10-year cycle of snowshoe hares and their predators has been one of the classic examples of cycles illustrated in biology textbooks. In 1976, we began to investigate the cycle of snowshoe hares at Kluane Lake in the southern Yukon. The prevailing wisdom about the hare cycle has come from the work of Lloyd Keith and his students in central Alberta. Keith (1981, 1990) suggested that cycles of hares were caused by food shortage in winter and heavy predation. Because of the time lag of the increase of predators, Keith suggested that shortage of food usually acted first to stop population growth of hares, and predation became more important in the decline and low phases of the cycle (Keith et al., 1984). My colleagues and I have spent the past 20 years testing this model with field experiments.

In the 1st 10 years of study, we tested the role of food available in winter on the hare cycle by providing supplemental food

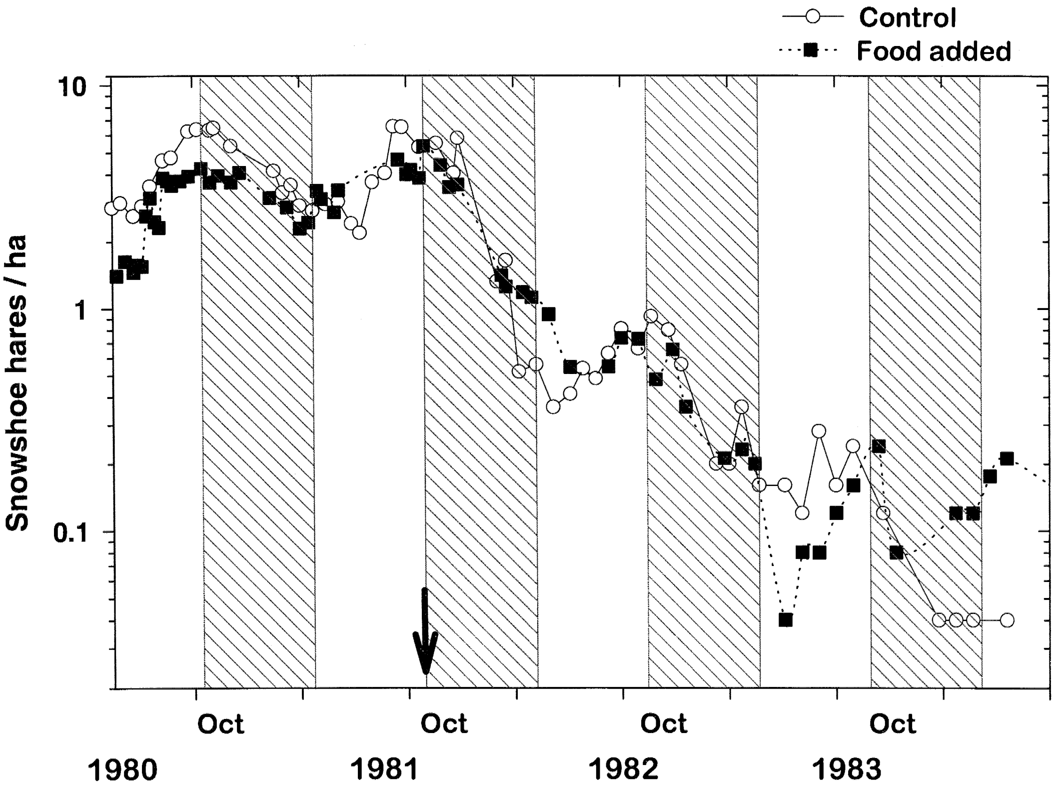

FIG. 3.—Population declines of snowshoe hares on a food-supplemented area (solid squares) and a control area (open circles), 1980–1984, Kluane Lake, Yukon. Food was supplemented naturally through the winter beginning in October 1981 (arrow) by cutting down large white spruce trees to feed to hares. Supplemental food by itself is not sufficient to prevent a cyclic decline (after Krebs et al., 1985).

ad lib. to three study areas (Krebs et al., 1986; Sinclair et al., 1988; Smith et al., 1988). The results were unequivocal: you could increase the carrying capacity of an area for hares about 3-fold by feeding in winter, but you could not stop the population decline or alter its rate. We tested this conclusion with both artificial and natural supplemental foods (Fig. 3; Krebs et al., 1986). These results could be criticized because of the use of artificial food (rabbit chow) or alternatively only one natural food (white spruce, *Picea glauca*) and a lack of replication. But it seemed clear to us that predation was the major process causing numbers of hares to change, and in 1986 we began a 10-year study on a larger scale to test the Keith hypothesis that both heavy predation and food shortage were necessary and sufficient to cause the cycle in hares (Krebs et al., 1992). This is called the Kluane Boreal Forest Ecosystem Project; when it ends in 1996, we will be able to present all the results (Boutin et al., 1995).

We have rejected the individual-differences hypothesis for cycles of snowshoe hares on the basis of some preliminary experiments by Boutin (1980, 1984). Removal experiments in hares do not produce the high colonization rates seen in voles, so there appears to be no surplus breeders available. We have not detected any form of spacing behavior in snowshoe hares. They do not defend territories, and their home ranges overlap broadly. We can find no mechanism for social mortality in hares, in contrast to voles and lemmings (Krebs et al., 1992). Consequently, we have concluded that snowshoe hares do not fit under the aegis of the self-regulation hypothesis, which requires some form of spacing behavior as a prerequisite.

We have manipulated populations of snowshoe hares at Kluane since 1986 in a 2 by 2 factorial design with the two factors being predation and food supplements. We have built two electric fences around 1 km^2 of boreal forest to reduce mammalian predation in two populations, and we have provided three areas with supplemental artificial food. Because of the costs of the fencing, we were unable to replicate the predator-reduction treatment or the predator-reduction + supplemental-food treatment. The details of these experiments are given in Krebs et al. (1995). The hare cycle can be stopped by a combination predator reduction + supplemental-food treatment, so it is the interaction between predation and food that is crucial to the cycles of snowshoe hares.

The emerging model of the snowshoe hare cycle from these manipulations has been presented by Hik (1994, 1995). In effect, Hik has reversed the Keith hypothesis and extended it by placing the prime responsibility for the cycle of hares on the predators. Mortality from predation causes the peak and decline phase of the cycle. Supplies of food in winter by contrast are never absolutely short during the cycle, but the behavior of the hares changes as predators become more abundant. In habitats that are heterogeneous, predator-sensitive foraging restricts hares to relatively safe, closed habitats in the peak and decline phases, and the food in these habitats is relatively poor. Adequate food exists in open habitats nearby, but these areas carry a high risk of predation. The changes in reproduction that occur over the cycle, described by Cary and Keith (1979) and Keith (1990), possibly are the result of feeding behavior of hares in the presence of risk of predation. Hares born in the peak and decline phases of the cycle live in an environment of high risk of predation, and they adopt feeding strategies to counter this risk (Gilbert and Boutin, 1991). These safe feeding strategies lead to decreases in body weight and fecundity, although food is abundant in open habitats. Coping with risk of predation also may lead to stress (Boonstra and Singleton, 1993), which may compound problems of population recovery in the low phase (R. Boonstra, C. J. Krebs, and D. Chitty, in litt.).

The predation-risk model for cycles of hares makes some clear predictions regarding foraging behavior that can be tested in the field (Hik, 1994). Because the reproductive collapse of hares already has begun during the increase phase of the cycle (Keith and Windberg, 1978), the effects of risk of predation must be seen at this time of the cycle for this explanation to be correct. One alternative hypothesis is that it is the food plants of summer that affect hare reproduction more than the food plants of winter that have been studied in detail. There are almost no data on the dynamics of food plants in summer in the boreal forest, because these herbs were considered to be in vast excess of needs during the short growing season.

In summary, our research on the cycle of snowshoe hares have confirmed experimentally the conclusions of Keith (1981) that both food supplies and losses by predation are the key factors causing populations to fluctuate. In contrast to Keith, we have placed more weight on predation as the main process, and we propose that the effects of food occur because of predator-sensitive foraging by hares in relation to habitat heterogeneity.

POPULATIONS OF LEMMINGS

In 1987, we began studies on population dynamics of lemmings in collaboration with Rudy Boonstra at Pearce Point, Northwest Territories (69°48'N, 122°40'W). In this part of the western Canadian Arctic the two dominant rodents were the collared lem-

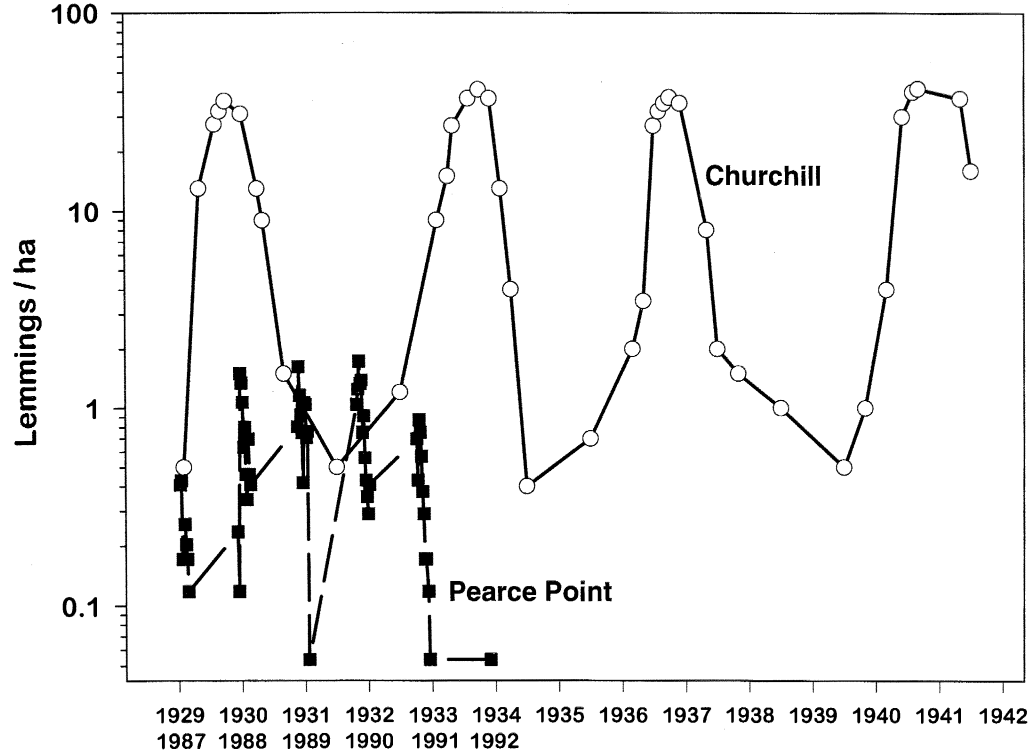

FIG. 4.—Population densities of collared lemmings estimated by Shelford (1943) for a cyclic population near Churchill, Manitoba, 1929–1942, and comparable estimates of density for collared lemmings at Pearce Point, Northwest Territories. Lemmings from Pearce Point seem to be locked into a continuous phase of low numbers.

ming (*Dicrostonyx groenlandicus*) and the tundra vole (*Microtus oeconomus*). We studied these populations for 6 years and they showed no population cycles over that time period (Krebs et al., 1995). Moreover, collared lemmings in this area of the Arctic persisted at extremely low densities, and consequently we were able to study for the first time a low density, non-cyclic population of lemmings. Fig. 4 illustrates the numerical dynamics of our populations in comparison with those studied by Shelford (1943) at Churchill, Manitoba.

The low phase of cycles of voles and lemmings always has been the phase least studied (R. Boonstra, C. J. Krebs, and D. Chitty, in litt.), and we viewed this population at Pearce Point as an analogue of a cyclic population perennially kept in the low phase of the cycle. One of the classic hypotheses for the low phase is that it is due to heavy predation (Pearson, 1966). Our studies at Pearce Point turned into an attempt to test this simple hypothesis.

During the 1st 3 years of this study, we found that heavy predation on lemmings in summer prevented population growth, so that the lemming population was trapped in a predator-pit. Generalist predators like red foxes (*Vulpes vulpes*) and rough-legged hawks (*Buteo lagopus*), in addition to losses to Arctic ground squirrels (*Spermophilus parryii*—Boonstra et al., 1990) and brown bears (*Ursus arctos*—Reid et al., 1995), preyed on lemmings at a rate much higher than the reproductive rate in summer, and as a consequence most populations of lemmings declined to near-extinction in sum-

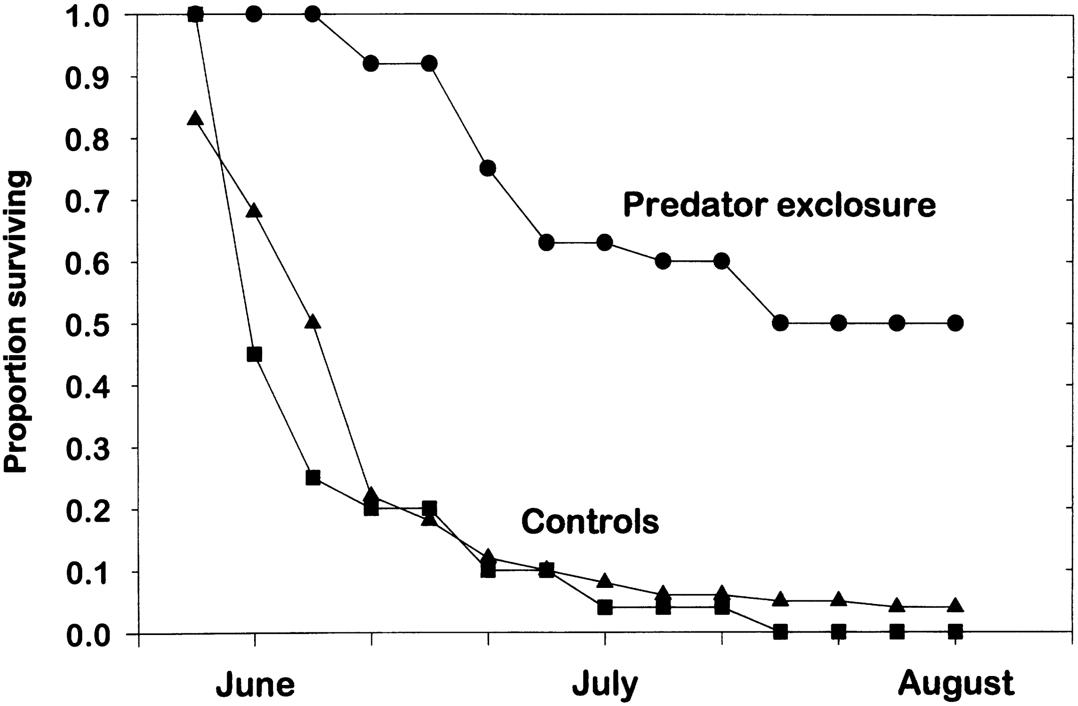

FIG. 5.—Survivorship curves for overwintered collared lemmings from inside the predator exclosure and outside the exclosure, Pearce Point, Northwest Territories, summer 1991. Two control populations were monitored. Predation losses are severe in open habitats (after Reid et al., 1995).

mer. Breeding in winter countered these losses in summer, so that these populations did not go extinct. On the basis of these natural-history observations, we decided to try to exclude predators from an 11-ha area to allow the population of lemmings to escape the predator-pit. In 1990, we constructed an exclosure of poultry wire around the perimeter to keep out mammalian predators and monofilament lines over the top to keep out raptors (Fig. 2d).

Survival rates of lemmings inside the exclosure were much improved over control lemmings (Fig. 5) and reproductive success was significantly enhanced as well when predators were excluded (Reid et al., 1995). However, the experimental population did not increase as predicted, apparently because juvenile lemmings dispersed outside the exclosure and were killed immediately by predators. The results reported in Reid et al. (1995) are thus consistent with the hypothesis that lemmings in the low phase of the cycle are locked into a predator-pit, as suggested by Korpimäki et al. (1991). In most cyclic populations, this model would predict that the collapse of numbers of predators from starvation would terminate the low phase and allow the increase phase to begin. We are now moving to a new site in the central Canadian Arctic to test this hypothesis on a cyclic population of lemmings. An alternative explanation is that Reid et al. (1995) have studied a population of lemmings in poor habitat and that these lemmings are kept in an analog of the increase phase by heavy predation.

The emerging model for cycles of voles and lemmings is a multifactorial one involving spacing behavior and predation with changes in both factors being both necessary and sufficient to cause cyclic fluctuations of populations. Given this model as a hypothesis, the next step is to try to restrict or eliminate predators from a large area to see how the resulting population dynamics would change. If predation is necessary for the peak phase, the experimental population would continue to increase to a higher density limited by spacing behavior and food. If predation is necessary for the decline phase, the experimental population would not cycle, but would remain at peak density. If predation is necessary for the low phase, as at Pearce Point, the length of cycles should change dramatically in the experimental area.

CONCLUSIONS

Ecologists have made considerable progress in unravelling the population cycles that so intrigued Charles Elton (1924). The model emerging now is a composite of many threads contributed by a wealth of talented and hard-working mammalogists. I believe we ecologists might agree on the following conclusions. 1) Population fluctuations of voles and lemmings are a joint product of intrinsic processes involving spacing behavior and extrinsic processes, probably involving predation and food. 2) Population cycles of snowshoe hares do not result from spacing behavior, but are caused by extrinsic processes involving predation and shortage of food, possibly produced by the response of individuals to risk of predation. More specific conclusions I would draw from the available data are as follows. 3) Spacing behavior in voles and lemmings produces dispersal, which is necessary for population regulation. Breeding females appear to be the key to the population dynamics of voles and lemmings. 4) Predation in voles and lemmings will affect the rate of the decline and the duration of the low phase. At least in some populations, predation is not necessary for the decline phase, but this could vary with the species. 5) In snowshoe hares, predation plays a major role, and in the absence of predation there would be no decline or low phase and hares would become food-limited. 6) Disease and parasitism play, at best, a minor role in population cycles.

What experiments hold the key to furthering our understanding of population cycles in mammals? I would direct attention in several directions. Frustrated studies of dispersal using islands or fences have not been fully exploited to further our understanding of the demography of cycles. The detailed mechanisms behind the fence effect are not understood. The effects of relatedness on population dynamics needs to be analyzed both by observations studies with DNA methods and by manipulative experiments. Large-scale, predator-manipulation studies would shed light on the details of how predation affects cyclic populations. Factorial experiments of the type pioneered by Batzli and his colleagues should be extended in time and in space to study explicit predictions about multi-year dynamics. Fragmentation of habitats eliminates cycles and we should find out why. The dynamics of populations of voles and mice along agricultural fence-rows need study as a model system for corridors and for testing metapopulation theory in conservation biology. Detailed studies of reproduction are needed to explain why breeding occurs in some winters in voles and lemmings and does not occur in others. Individuals from peak- and low-phase populations should be analyzed in the laboratory to find out why they differ in their reproductive fitness. The polymorphic-behavior hypothesis needs further tests for voles and lemmings, and the present belief that we can understand all cycles without any genetic parameters is poorly founded. Individual body growth needs detailed analysis by physiological ecologists to explain the Chitty Effect and its association with population cycles.

There are many details of cyclic fluctuations that will be particular to a given species and will not be general. Lemmings must cope with snowfall and California voles do not, so that we must expect to see some differences in their ecology. However, underneath all these local variations there is emerging a vision of how both intrinsic and extrinsic factors are involved in producing population cycles. There remains much to be done to sharpen our understanding, and if we continue to ask precise questions, make predictions, and test them experimentally, we will continue to see the ecology of small mammals recognized as an outstanding paradigm of population regulation.

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