

# Population cycles in microtines: the senescence hypothesis

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## Summary

The cause of population cycles in microtines (voles and lemmings) remains an enigma. I propose a new solution to this problem based on a crucial feature of microtine biology, shifts in age structure, that has been ignored until now. Empirical evidence indicates that age structure must shift markedly towards older animals during declines because of three characteristics of the previous peak year: a shortened breeding season, total replacement of the breeding population from peak to decline and density-dependent social inhibition of maturation of young. Declines become inevitable as populations composed of older animals survive and reproduce poorly because of the effects of senescence, possibly interacting with the experiences of peak density and I present both theoretical and empirical evidence for this hypothesis. **Although a variety of physiological systems deteriorate with aging, I focus on a crucial one – the inability of older animals to effectively maintain homeostasis in the face of environmental challenges because of a progressive deterioration in the endocrine feedback mechanisms involved in the hippocampal–hypothalamic–pituitary–adrenal axis.** Microtine populations will not exhibit cycles where age structure shifts are prevented owing to extrinsic factors such as intense predation. Six testable predictions are made that can falsify this hypothesis.

*Keywords:* age structure; population demography; microtines; population cycles; senescence

## Introduction

Many microtine populations (lemmings and voles) go through regular multi-annual fluctuations in numbers (commonly known as cycles) (Krebs and Myers, 1974; Taitt and Krebs, 1985). Although many hypotheses have been advanced to explain this phenomenon, all extant hypotheses are still controversial and in question (Taitt and Krebs, 1985; Boonstra and Boag, 1987; Nelson, 1987; Lidicker, 1988; Gaines *et al.*, 1991; Lambin and Krebs, 1991a). It is remarkable that even though the study of cycles is the bailiwick of population ecologists, the importance of age structure, a concept central to demography, has been virtually ignored (Krebs and Myers, 1974; Taitt and Krebs, 1985). Here, I elaborate a new hypothesis for cycles that (1) directly considers the role of age structure shifts whereas others have not, (2) considers the consequences of these age structure shifts, specifically that reproduction and survival vary as a function of age for evolutionary reasons (i.e. the theory of senescence; Rose, 1991) and, in doing so, (3) offers a more broadly based explanation that can account for population cycles in microtines.

## The problem and a new hypothesis

The central enigma in understanding microtine cycles is knowing the cause(s) of population declines and the period of low numbers that often accompanies these declines. Experiments have

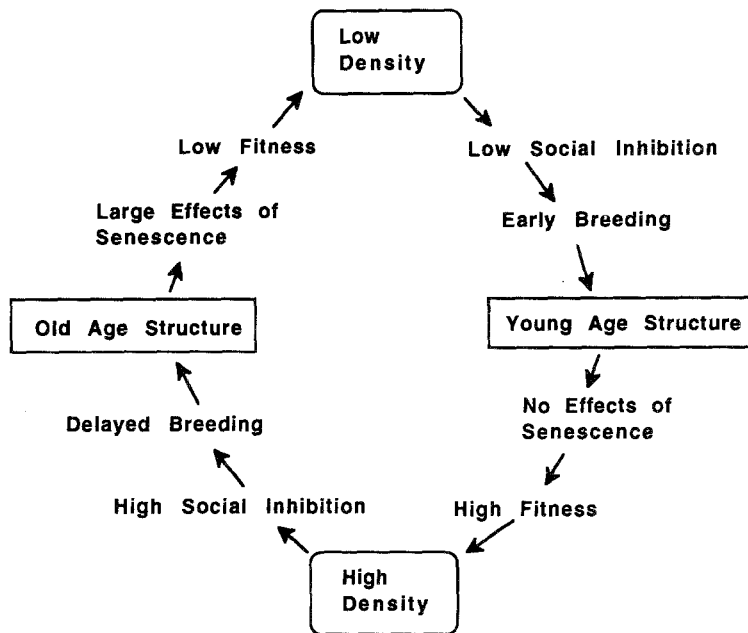


Figure 1. Model of the proposed shifts in age structure caused by density-dependent inhibition of maturation in young voles.

manipulated the environments of voles, but have been unable to arrest population declines. Therefore, whatever changes occur must be *intrinsic*. Declines cannot be stopped by transferring animals from declining populations to areas from which animals from an increasing population have just been removed (Krebs, 1966), by adding food (Krebs and DeLong, 1965; Cole and Batzli, 1978; Desy and Thompson, 1983) or by removing predators (Pearson, 1966, 1971). Interactions among these potential causes may prove relevant (e.g. predation and food; Desy and Batzli, 1989), though these have not yet been shown to be necessary and sufficient to explain declines. Nor can these interactions account for the fact that most decline animals, when removed to the optimal conditions of the laboratory, continue to exhibit poor performance with respect to reproduction and growth when compared with animals removed from other phases of the cycle (Newson and Chitty, 1962; Krebs, 1966; Mihok and Boonstra, 1992). One obvious and previously ignored change through the population cycle is the shift in age structure towards older animals during declines.

The essence of the hypothesis is that density-dependent social inhibition during the spring and early summer of the peak year of the cycle forces young animals to delay maturation until the next breeding season when they are old (Fig. 1). Old age, acting either separately or in conjunction with the experience of peak density during the previous summer and winter, impairs the viability of these animals and their young and precipitates the decline. Thus, declines will necessarily follow peaks because of the lags in age of first reproduction. This hypothesis is therefore based on two critical assumptions: (1) that age structure shifts significantly throughout a cycle and (2) that, in older populations, the effects of senescence become more pronounced, leading to reduced survival and reproductive success. Below, I review the theory and evidence for these assumptions and I describe a plausible physiological mechanism by which aging (senescence) may affect fitness in microtines.

### Changing age structure in cyclic microtines

Although a variety of factors have been implicated in being associated with microtines cycles (e.g. high body weights of adults in the spring of the peak year (Chitty, 1967), overwinter breeding between the increase and peak year (Nelson, 1987), lack of a spring decline in overwintering females in the peak year (Lambin and Krebs, 1991a)), virtually all researchers assume that knowledge of the age of the animals is irrelevant (Leslie and Ranson (1940) are notable exceptions). For example, in the two major reviews of microtine demography, no mention was made of the importance of fluctuations in age structure (Krebs and Myers, 1974; Taitt and Krebs, 1985).

The reason that the effects of age have been ignored is because small mammals are practically impossible to age in the field because of variable growth rates of animals within and between years (Krebs *et al.*, 1969; Brown, 1973; Beacham, 1980a) and of overlapping generations within a year. Though attempts have been made to age animals collected from field populations (with techniques such as development of roots in molar teeth – useful only in the genus *Clethrionomys* (Zedja, 1964; Viitala, 1971; Viro, 1974; Fuller, 1977) or eye lens weights (Kozakiewicz, 1976), these methods are practical only for young animals because of the effects of seasonality and maturation (Zedja, 1971; Mihok, 1980). As a consequence, population data for small mammals are usually structured in terms of mass-based (with classes such as adult, subadult, and juvenile based on weight (e.g. Krebs *et al.*, 1969; Boonstra and Rodd, 1983; Tamarin *et al.*, 1984)) rather than the more conventional age-based demography of larger mammals or mammals with non-overlapping generations (Caughley, 1966; Sinclair, 1977; Berger, 1986). **Indeed, Sauer and Slade (1986, 1987) have argued that mass-based demography is preferred for rodents because most individuals do not live long enough for changes in fecundity and survival owing to senescence to be expressed.** However, the effectiveness of this method depends on the degree to which survival and fecundity are primarily determined by body mass.

For cycling microtines, there are three aspects of their demography that indicate that age structure of the breeding animals must show major changes over a cycle and has the potential to be a significant variable in their demography: first, the length of the breeding season varies over a cycle; second, virtually the entire breeding population is replaced from one year to the next; and third, age at sexual maturity shows major changes over the cycle. The evidence for suppression of various aspects of reproduction from cyclic populations using the general scheme of Gipps (1985) is summarized in Table 1. A summary of this type has inherent limitations which hampers satisfactory comparisons among studies because of differences in trapping methods and the lengths of time within years in which trapping was carried out (i.e. trapping did not cover the entire breeding season), relevant data not being collected (i.e. suppression is only given for one of the sexes) and variations in definitions of maturity. For example, in the live-trapping studies by Krebs and his students (Krebs *et al.*, 1969; Tamarin *et al.*, 1984), the median weight (an index of age) of sexual maturity was usually calculated, but information on the proportion of the young of the year actually maturing in their year of birth was not. Nevertheless, the data in Table 1 is robust enough to show that reproductive suppression occurs in virtually all peak populations.

*Length of the breeding season.* Cyclic populations are characterized by changes in the length of the breeding season (Krebs and Myers, 1974) and of necessity this will affect the age when animals can first breed. The general pattern can be summarized as follows: the increase phase of a cycle is characterized by an extended breeding season, sometimes accompanied by winter breeding prior to the peak (e.g. Iverson and Turner, 1974); the peak phase is characterized by a shortened breeding season and lack of winter breeding (Table 1); and the decline phase is often

characterized by a delayed onset of breeding in the spring (Table 1). Since there is virtually complete turnover in the breeding population from one year to the next (see below), the population at the onset of breeding in the spring of the peak must be relatively young (especially if there has been winter breeding), whereas that in the spring of the decline must be much older.

*Duration of life and population turnover.* In the benign environment of the laboratory, voles can usually live approximately 1 year (Leslie and Ranson, 1940; Leslie *et al.*, 1955; Buchalczyk, 1970; Morrison *et al.*, 1977). Under field conditions, most microtines live considerably less than 1 year and virtually no animal that breeds in one year is alive the next (Zedja, 1964; Bergstedt, 1965; Krebs, 1966; Myllymäki, 1977; Rose and Dueser, 1980; Wolff and Lidicker, 1980; Cockburn and Lidicker, 1983; Mihok, 1984). In expanding populations of *Microtus californicus*, adult males and females had mean life expectancies of 8–13 weeks compared with 2–7 weeks in declining populations (Krebs, 1966). In a long-term study on both *Microtus ochrogaster* and *Microtus pennsylvanicus*, voles lived a mean of 5–9 weeks after capture (Getz *et al.*, 1979). In *M. pennsylvanicus* captured shortly after weaning, male and female litter mates during increase periods lived a mean of 10.8 and 17.7 weeks, respectively, in contrast to 10.8 and 13.6 weeks, respectively, during peak periods (Boonstra and Craine, 1988). Nevertheless, it is important to appreciate that these values are means and that in peak periods a portion of the non-maturing young may live much longer (e.g. 10–15% of the non-maturing *M. pennsylvanicus* born from May – mid-June into a peak population were still alive at the onset of breeding the next year; Boonstra, 1989). Thus, most animals live less than 1 year in the field and annual turnover of the breeding population must be virtually 100%.

*Inhibition of sexual maturity.* The most crucial variable affecting the intrinsic growth rate of a population is the age at sexual maturity (Cole, 1954; Schaffer and Tamarin, 1973; Stearns, 1976). Microtines show tremendous plasticity in this trait (for reviews see Hasler (1975), Keller (1985), Nadeau (1985) and Stenseth *et al.* (1985)). In general, microtines have the potential to mature 2–3 weeks after weaning. For example, corpora lutea have been found in wild 14–17 day old *M. californicus* (Greenwald, 1957) and *M. pennsylvanicus* have been observed to breed at 25 days of age (Beer *et al.*, 1957). Similar estimates have been obtained for other microtine species (Negus *et al.*, 1977; Hagen and Forslund, 1979; Gustafsson *et al.*, 1980; Solomon, 1991).

However, this potential to mature rapidly varies greatly among different cohorts born in the same year and among cohorts born at the same time in different years. Within a year, there are general seasonal patterns, with young born earlier in the breeding season usually maturing rapidly and breeding in their year of birth while those born later in the breeding season delaying maturation until the next year (Schwarz *et al.*, 1964; Anderson, 1970; Hasler, 1975; Keller, 1985). Between years in cyclic microtine populations, there are major shifts in the age at maturation. In increasing populations, young born throughout the breeding season may mature well into the autumn, whereas young born into peak populations delay maturation until the next year (Table 1). Thus, during the peak year reproductive inhibition of young acting together with a compressed breeding season and complete turnover of the breeding population must result in an age structure shift towards older animals during declines.

#### *Are animals in the decline really older? Two examples*

The summary of the previous literature indicates that voles during the decline must be significantly older than those during the peak, but much of the evidence is inferential. To circumvent the difficulty of aging all small mammals in the field, I have chosen to focus only on a segment of the population that can be accurately aged, those first caught as young, and use them

Table 1. Suppression of reproduction in cycling populations of microtines

Species	Suppression of maturation at peak	Percentage of young maturing of those born in spring and early summer				Comments	Early end to breeding in peak	Late start of breeding in decline	Reference
		Males		Females					
		Increase	Peak	Increase	Peak				
<i>C. glareolus</i> *	Y	Almost all	0	Almost all	0–19		E by 2 months	–	Zedja (1964, 1967)
	Y	91	0	75	4		E by 1.5–2 months	–	Wiger (1979)
	Y	93–95	0–60	78–100	25–45		E by 1 month	L by 1–2 weeks	Gustafsson (1983)
	Y	Many	0	63–75	0		E by 1–2 months	–	Nyhom and Meurling (1979)
	Y	–	–	25–70	11–36		–	–	Hansson and Henttonen (1985)
	Y	–	0	–	0	Peak only studied	–	–	Bondrup-Nielsen and Ims (1986)
<i>C. rufocanus</i>	Y	–	–	Most	Few		E by 2–4 weeks	–	Löfgren (1989)
	Y	90	0–23	82	0–50	0% on high density site	E by 1 month	L–	Kalela (1957)
<i>C. rutilus</i>	Y	–	0	–	0–8	Peak only studied	–	–	Ims (1987)
	Y	2–56	0	15–63	0–8.5		E by 1–1.5 months	–	Koshkina and Korotkov (1975)
	Y	–	–	29–54	3–5		–	–	Hansson and Henttonen (1985)

<i>D. groenlandicus</i>	Y	?	0	-	-	Too few females caught	E ≥ 1 month	L-	Krebs (1964)
	Y	69	0	-	-	Too few females caught	-	-	Fuller <i>et al.</i> (1975)
<i>L. trimucronatus</i>	Y	?	0	72	8		E ≥ 1.5 months	L-	Krebs (1964)
	Y	?	0	55	0		E by 1 month	L-	Mullen (1965)
<i>M. californicus</i>							E by 2-3 weeks	L by 5-10 weeks	Krebs (1966)
<i>M. pennsylvanicus</i>						Higher median weight at sexual maturity for both sexes	E by 2 months	L by 1-2 months	Keller and Krebs (1970)
	Y					Higher median weight at sexual maturity for both sexes	E by 1-2 months	L by 2-4 weeks	Tamarin (1977)
	Y	12	33	76	24	Peak in 1970	E-	L by 2-4 weeks	Mihok (1984)
	Y	8-15	0-5	50-60	4-20		E by 3-4 months	L-	Boonstra (1985, 1989)
<i>M. ochrogaster</i>	Y	-	-	-	-	Higher median weight at sexual maturity for both sexes	E by 2 months	L by 1-2 months	Keller and Krebs (1970)
<i>M. townsendii</i>	Y	32-33	4-7	48-56	5-8		E by 3 weeks	L by 2 months	Beacham (1980c)

Abbreviations are as follows: Y, suppression of maturation in young of the year; L, late start of breeding season in decline; E, early end to breeding season in peak year; -, suppression not studied or data incomplete or insufficient (Y, E or L alone: suppression studied and observed; Y-, L-, E-: suppression studied but not observed).

\* Genera abbreviations are *C.*, *Clethrionomys*; *D.*, *Dicrostonyx*; *L.*, *Lemmus*; *M.*, *Microtus*.

Table 2. Regressions used to predict age in voles<sup>a</sup>

Species	Males	Females
<i>M. pennsylvanicus</i>		
April – mid-July	Age = 1.54 (weight) –1.92** $r^2 = 0.92, n = 100$	Age = 1.62 (weight) –2.16** $r^2 = 0.91, n = 109$
Mid-July – October	Age = 1.92 (weight) –4.50** $r^2 = 0.92, n = 127$	Age = 2.04 (weight) –4.69** $r^2 = 0.86, n = 112$
<i>M. townsendii</i>		
Mid-March – April	Age = 0.78 (weight) +9.70** $r^2 = 0.74, n = 171$	Age = 0.84 (weight) +9.40** $r^2 = 0.67, n = 249$
May – mid-July	Age = 0.88 (weight) +7.91** $r^2 = 0.62, n = 454$	Age = 0.84 (weight) +9.34** $r^2 = 0.54, n = 529$
Mid-July – October	Age = 0.89 (weight) +8.89** $r^2 = 0.50, n = 39$	Age = 0.59 (weight) +16.78* $r^2 = 0.16, n = 23$

\*  $p < 0.05$ , \*\*  $p < 0.0001$ .

<sup>a</sup> See text for details.  $n$  equals the sample size on which each regression is based.

as a representative sample to reflect age changes in the entire population. Regressions relating weight to known age of young were obtained for two vole species and these were then applied to young from cycling populations from the same area.

*Methods* For *M. pennsylvanicus* in Ontario, we located nests in the field with a spool-and-line technique (Boonstra and Craine, 1986) at the Station for Atmospheric Experiments (site described in Boonstra and Boag, (1987)) and marked the young or removed pregnant females to the laboratory, allowed them to litter and then reintroduced mother and marked young (<5 days old) to the field to the same trap site at which the mother was first caught. We subsequently caught some of these young in live-traps. Young were weighed both in the nest and at every capture in traps. Since age was accurately known for these animals, I calculated regressions relating weight to age for young weighing <31 g when first caught in live-traps. Over this weight range (3–30 g), simple regressions explained most of the variance (Table 2). For each sex, separate regressions were calculated for young caught from April to mid-July (period of most rapid growth) and for those caught from mid-July to October (period of slower growth). For the former, the data came from three low density populations (see Craine (1987) for details) and, for the latter, from two enclosed populations which increased from moderate to high density (Boonstra and Hogg, 1988). Although differences in growth rates of animals have been found at different densities (e.g. Boonstra and Boag, 1987), I did not have the growth rate data of young under all density conditions. In particular, I didn't have regressions for spring – early summer young at high densities and, thus, ages I derived when applying these regressions to the grid F data (below) are likely to be underestimates of actual age and, thus, to be conservative with respect to the true differences in age between peak and decline voles.

These regressions were then used to calculate the age of young <31 g captured on grid F, a site near the above site (within 15 km) (see Boonstra (1985) for details of trapping methods and a discussion of demography). Grid F was chosen for two reasons: first, it went through a multi-annual cycle in numbers and, second, it was trapped with both Longworth live-traps and pitfall traps (the latter from spring to autumn, from May 1979 to May 1982). Pitfalls capture meadow voles at earlier ages than Longworths (Boonstra and Rodd, 1984). Age was calculated at time of first capture only for young <31 g trapped during the breeding season until December. On all subsequent captures, an animal's age was simply incremented by the difference between the time

of subsequent capture and the time of first capture. To compare between peak and decline years, I examined only the age profiles of breeding animals. Animals were classed as breeding if males had scrotal testes and if females were perforate, lactating or pregnant. Jolly (1965) estimates of population numbers were used.

For *Microtus townsendii* in British Columbia, Lambin (personal communication) kindly calculated similar regressions for young  $\leq 31$  g caught using data obtained by finding nests with radiotelemetry (Lambin and Krebs, 1991b; Lambin, 1993), marking the babies and subsequently recapturing some of them in live-traps. Though he aged young in the nest, he did not weigh them and, hence, the regressions are only based on relationships between age and weight obtained from capture in traps. Regressions were calculated for three times of the year: from mid-March to April (spring period of growth), from May to mid-July (late spring – early summer growth) and from mid-July to the end of October (late summer – autumn growth which spans the period when botflies (*Cuterebra grisea*) infest a high percentage of the voles and depress both reproduction and growth; Boonstra *et al.*, 1980) (Table 2). Though density differed among years in Lambin's study, he found no effect of density on the regressions (by ANCOVA; X. Lambin, personal communication) and, thus, pooled the growth data from all years.

These regressions were then used to calculate the age of young trapped in four populations by Beacham (1980b) on a site near (within 20 km) that used by Lambin. Two control grids and two enclosure grids (the latter having dispersal areas) were trapped from 1976 to 1978 with both Longworth live-traps and pitfall traps. Pitfalls were extremely effective in capturing young voles of this species (Boonstra and Krebs, 1978). The same methods used in the meadow vole were then applied to the Townsend's vole. All Beacham's populations showed similar demography, going through one cycle.

*Results* The *M. pennsylvanicus* population on grid F went through a pronounced cycle in numbers from 1979 to 1982 (Fig. 2). The cycle on grid F was characterized by an increase year in 1979 with an extended breeding season, a peak year in 1980 with a shortened breeding season and a decline lasting from late 1980 to early 1982. (The age of breeding animals was not calculated for the period from 1978 to early 1979 for three reasons: first, the grid was not trapped until July 1978 and, hence, I could not calculate the age of any animals that may have lived from the spring of 1978 to the spring of 1979; second, only live-traps were used in 1978 and, thus, I did not capture a large sample of voles when they were still young; and third, the decline in early 1979 was induced by severe weather (freezing rain followed by intense cold; see Boonstra and Rodd (1983) for details) whereas the decline starting in 1980 was not.) The age of breeding animals shifted markedly over the cycle. In the 1979 increase, they appeared relatively young, but this was an artefact since the age of the overwintering breeding animals was not known. Nevertheless, it does reflect maturation of the young born that year. The ages of a representative sample of overwintering animals were known for both 1980 and 1981 and there was a significant shift in age from the peak to the decline, so that voles present at the onset of breeding in spring of the decline were 2.5–3.5 months older than those at a comparable time in the peak (Fig. 2 and Table 3). The progressive increase in age of breeding females during most of the peak and all of the decline reflects lack of maturation of most female young born in their year of birth. In addition, none of the animals present at the onset of breeding in the peak were still present in the decline and, thus, there was complete population turnover from one year to the next.

The cycle in the *M. townsendii* populations was characterized by an increase in 1976 with an extended breeding season, a peak in 1977 with a shortened breeding season and a decline in 1978 with a delay in the onset of breeding (Fig. 3 shows population changes on a representative grid, grid B). The age of breeding animals from the peak to the decline shifted in the same manner as

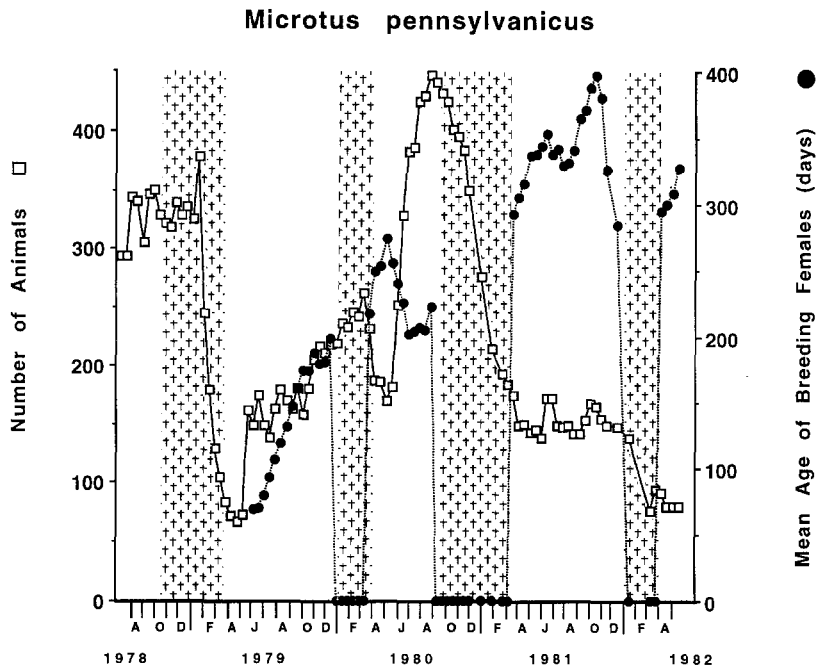


Figure 2. Population changes and shifts in age structure of breeding females in *M. pennsylvanicus* on grid F from Ontario (Boonstra, 1985) over one cycle. Nonbreeding periods are shaded.

Table 3. Mean age (days  $\pm$  SE) of breeding animals at the end of March (approximate onset of breeding season) for voles first caught as young in two species of *Microtus*<sup>a</sup>

Species grid	Males		Females	
	Peak	Decline	Peak	Decline
<i>M. pennsylvanicus</i>				
F	198.8 $\pm$ 13.0 (28)	303.2 $\pm$ 3.1 (25)**	209.2 $\pm$ 19.4 (15)	292.0 $\pm$ 5.4 (25)**
<i>M. townsendii</i>				
A	206.3 $\pm$ 6.0 (11)	297.9 $\pm$ 5.7 (7)**	221.0 $\pm$ 5.4 (10)	296.8 $\pm$ 6.4 (10)**
B	215.6 $\pm$ 2.5 (34)	310.5 $\pm$ 3.1 (17)**	218.9 $\pm$ 6.2 (25)	341.0 $\pm$ 27.2 (13)**
C	221.9 $\pm$ 3.2 (36)	296.8 $\pm$ 3.5 (12)**	221.9 $\pm$ 9.5 (22)	332.8 $\pm$ 25.3 (17)**
D	225.7 $\pm$ 6.9 (18)	299.5 $\pm$ 11.5 (2)**	217.5 $\pm$ 13.3 (8)	292.7 $\pm$ 7.7 (10)**

\*  $p < 0.01$ , \*\*  $p < 0.0001$ .

<sup>a</sup> The *M. pennsylvanicus* data was obtained from Ontario (see Boonstra, 1985) and the *M. townsendii* data from British Columbia (Beacham, 1980b). Sample sizes are in parentheses. See text for additional details. Statistical comparisons are within a sex between peak and decline phases.

that seen in the meadow vole: breeding voles in the decline were 2.5–4 months older than those in the peak (Fig. 3 and Table 3). The progressive increase in age of breeding females during the peak reflects lack of maturation of female young of the year. Again, no animals present at the onset of breeding in the peak were still present in the decline indicating that there was complete turnover in the population from one year to the next. Thus, the evidence from these two species indicates that voles are significantly older in the decline than at other times.

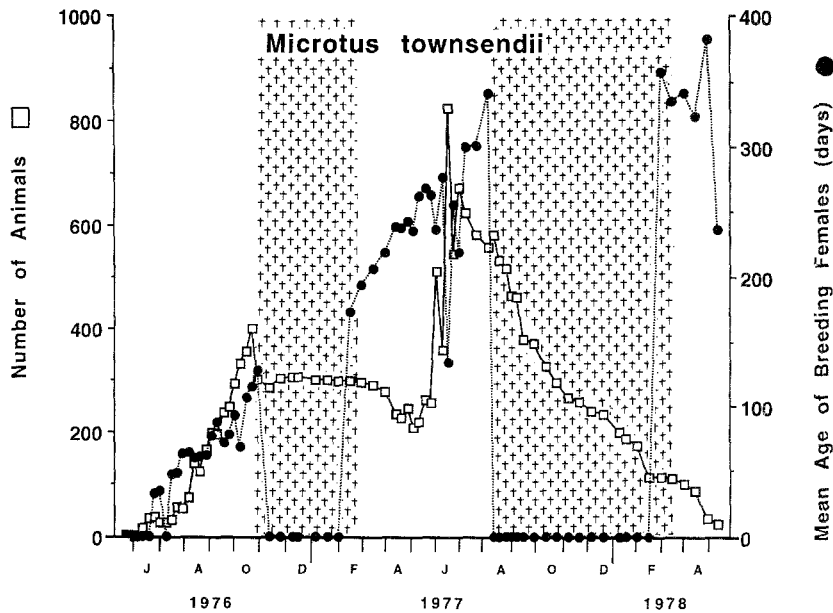


Figure 3. Population changes and shifts in age structure of breeding females in *M. townsendii* on enclosure grid B from British Columbia (Beacham, 1980b) over one cycle. Nonbreeding periods are shaded.

### The effects of senescence in cycling microtines

Clearly age structure of microtines changes markedly over a cycle but this becomes relevant only if populations composed of older individuals are significantly less fit than those composed of younger ones. I propose that animals during declines exhibit the effects of senescence and that this causes declines. There are both theoretical and empirical reasons for suggesting this. Senescence is the innate deterioration in age-specific fitness components of survival and fecundity with increasing age (Finch, 1990; Rose, 1991). Senescence occurs because the force of natural selection decreases with age (Medawar, 1952; Williams, 1957).

There are two predictions from senescence theory that are pertinent to microtine cycles (Williams, 1957; Emlen, 1970): first, that mortality related to senescence should not increase until after the age of first reproduction and, second, that the age at which senescence is first expressed depends on how much mortality occurs – if it is high, then few animals are long-lived, even in the absence of the effects of senescence and the effects of senescence should be expressed early. However, these predictions cannot be extrapolated directly from theory to nature. The predictions are derived from models developed from theoretical populations with stable age distributions and fixed ages of maturation (Charlesworth, 1980), whereas age at sexual maturity in real populations can vary markedly within a species because of the effects of seasonality and/or social inhibition (see above for microtines). I suggest that species-specific senescence rates are determined by overall mean ages at first reproduction and mortality independent of the effects of senescence and that in microtines, selection fixes senescence rates at an early age owing to their potentially early maturity and high mortality rates (Krebs and Myers, 1974; Taitt and Krebs, 1985). Hence, during population declines microtines are becoming senescent even when they haven't yet reproduced.

There are several lines of empirical evidence in microtines that are consistent with this

explanation and difficult to reconcile with conventional mortality factors such as severe predation or weather (Mihok *et al.*, 1985).

(1) Adults usually, though not always, survive more poorly during declines and the changes in survival are often sex specific (Krebs *et al.*, 1969). Typically for populations that decline at the onset of the breeding season, survival deteriorates up to 3 weeks earlier in males than in females (Krebs and Boonstra, 1978). This argues against the role of extrinsic agents such as predation causing this differential. Rather it suggests that the challenges of reproduction (competition among males and pregnancy and lactation among females) must be borne earlier by males than females and the costs in terms of reduced viability must also be paid earlier.

(2) Pregnancy rates and litter sizes are often lower during declines (Krebs and Myers, 1974). In *M. pennsylvanicus*, females brought into the laboratory from declining populations had significantly fewer litters than those from increasing populations brought in the next year under the same conditions (Mihok and Boonstra, 1992).

(3) Animals are typically less aggressive during declines than at other times (Krebs, 1970; Mihok, 1981; Hofmann *et al.*, 1982; Krebs, 1985; Mihok *et al.*, 1985) suggesting that conspecific interactions are not responsible for poorer survival during declines.

(4) Declines occur in the absence of detectable dispersal (Getz *et al.*, 1979; Beacham, 1980b).

(5) Animals typically grow more slowly and are lighter in declines than at other times (Newson and Chitty, 1962; Krebs and Myers, 1974; Boonstra and Boag, 1987; Lidicker and Ostfeld, 1991).

(6) Finally and most critically, poor juvenile survival characterizes years of population decline in microtines (Krebs and Myers, 1974). Early juvenile survival was the prime determinant of population growth in *M. ochrogaster* (Krebs, 1971; Gaines and Rose, 1976) and in *Synaptomys cooperi* (Gaines *et al.*, 1977) and the second most important determinant in *M. californicus* (after female survival) and *M. pennsylvanicus* (after the percentage of females lactating and female survival) (Krebs and Myers, 1974). There is a great deal of evidence indicating that interactions with adults, primarily adult females, can reduce survival of juveniles (Smyth, 1968; Krebs *et al.*, 1969; Boonstra, 1977a, 1978; Redfield *et al.*, 1978; Getz *et al.*, 1979; Gilbert *et al.*, 1986; Rodd and Boonstra, 1988). However, during the decline, evidence indicates that lactation and maternal condition is associated with the poorer survival and that the crucial time is at or near weaning. In a laboratory study on *Microtus canicaudus*, young not surviving past weaning usually died at approximately 9–12 days of age (Hagen and Forslund, 1979). It is at this stage that the energy demands of lactation are maximal (Kaczmarek, 1962; Migula, 1969; Millar, 1978). In the field, the same pattern of loss of young has been found. Godfrey (1955) found that the young *Microtus agrestis* from a declining population disappeared right at weaning. Hoffmann (1958), from work on a declining *Microtus montanus* population, concluded that although females were nursing, the young were not appearing in the population and, hence, must be dying at or just after weaning. In a *M. pennsylvanicus* population (Fig. 2), survival of adults was similar in both peak and decline years, but the number of juveniles (per lactating female) entering the population during the decline was approximately half that in the peak (Boonstra, 1985). Similarly, in the wood lemming, *Myopus schisticolor*, reproduction and survival of adults were comparable during a peak and decline, but the lack of juvenile recruitment during the decline caused the population to crash (Bondrup-Nielsen and Ims, 1988). Thus, poor juvenile survival characterizes declining populations and suggests that some aspect of maternal condition may be responsible.

Thus far I have demonstrated that (1) age structure shifts must accompany microtine cycles with older animals being present during declines and (2) there are theoretical and empirical reasons for expecting that these older, decline animals are less fit than those present at other times. Given this, it will necessarily follow that populations will decline after a peak. There are

two remaining issues: (1) what are the actual physiological mechanisms that cause this reduction in fitness in the older animals? and (2) is senescence alone sufficient to produce cycles or does it interact with other aspects of the environment?

### Physiological mechanisms of senescence in microtines

There are many physiological functions that deteriorate with aging (Comfort, 1979; Adams, 1984; Finch and Schneider, 1985; vom Saal and Finch, 1988; Finch, 1990), consistent with the hypothesis that senescence evolved as the result of age-specific effects affecting a large number of loci (Williams, 1957). Physiological decisions to reproduce, grow or put energy into storage are mediated by hormones and the endocrine system may be a major pathway through which antagonistic pleiotropy (Williams, 1957) shapes the evolution of senescence (i.e. hormones mediate trade-offs between early reproduction and subsequent costs to survival because of physiological deterioration; Stearns, 1989; Finch, 1990). Exactly how senescence proceeds in microtines is unknown and remains to be tested, but we know what happens in other species and it is likely that similar mechanisms operate in voles and lemmings. I will focus on a hormonal system – the hippocampal–hypothalamic–pituitary–adrenocortical axis – that is central to a mammal's ability to deal with environmental challenges and that shows senescent degeneration caused by a cumulative lifetime exposure to normal concentrations of adrenal hormones (Sapolsky *et al.*, 1986).

A crucial deteriorating function in aging mammals is the inability to maintain homeostatic balance in response to acute challenges (Sapolsky, 1987). These challenges activate the hypothalamic–pituitary–adrenocortical axis, one of the principal hormonal avenues used to cope with stressors, in an attempt to regain homeostasis. Adrenal glucocorticoids play two predominant physiological roles: first, they are critical in normal day to day activities associated with the diurnal cycle of waking such as increased locomotion, exploratory behaviour, appetite and food seeking behaviour (McEwen *et al.*, 1988) and, second, they play a central role in allowing the organisms to adapt to acute stressors and return to homeostasis by stimulation of hepatic gluconeogenesis, inhibition of glucose uptake by peripheral tissues, suppression of inflammatory responses, suppression of numerous immune reactions and inhibition of secretion of several hormones and neuropeptides (Munck *et al.*, 1984). This stress response role is thus geared to short-term adaptation to acute challenges because it is essentially catabolic in nature, shunting energy to muscles and away from other tissues. Chronic exposure to glucocorticoids has deleterious consequences such as steroid diabetes, fatigue, myopathy, atherosclerosis and hypertension, infertility, inhibition of growth and impaired disease resistance (Munck *et al.*, 1984).

Older animals cannot adapt as effectively to acute stressors as can younger ones. For example, when aged male rats are exposed to a variety of different stressors (restraint, ether, cold exposure, cage transfer, laparotomy), they can respond as rapidly as young rats to these challenges in terms of increased adrenal glucocorticoid levels and, thus, older rats have sufficient adrenal reserve capacity for secretion of corticosterone (Sapolsky *et al.*, 1986; Langfield, 1987). However, they have an impaired ability to terminate the response rapidly once the stressor is removed. The consequence of this is that aged animals are exposed to the potentially biologically damaging effects of high glucocorticoid levels for much longer than young animals. This inability to terminate the stress response in aged rats is due to progressive degeneration of the hippocampal area of the brain that normally inhibits glucocorticoid release (Langfield *et al.*, 1978; Brizzee and Ord, 1979) which can be prevented if the rats are adrenalectomized at middle age (Langfield *et al.*, 1981; O'Steen and Donnelly, 1982). Thus, this degeneration in aged animals is

the result of a cumulative effect of lifetime exposure to normal levels of glucocorticoids. Sapolsky *et al.* (1986) hypothesized that glucocorticoids precipitate a feedback cycle in which prolonged exposure to high levels of glucocorticoids caused by normal aging processes further damages the inhibitory neurons and/or receptor sites which then leads to even longer exposure to high glucocorticoid levels. In addition, repeated exposure to chronic stressors may accelerate the rate of aging (Sapolsky *et al.*, 1987). The net results are serious pathological consequences for old animals unable to maintain homeostasis. I suggest that this mechanism operates in microtines as well and that in nature they start to experience this senescent degeneration well before they are a year old because of their short lifespans and potentially early ages of maturation. Hence, the older animals characteristic of declining populations are senescent and are experiencing physiological deterioration in their ability to maintain homeostasis.

### Interactions between aging and the environment

Though microtines during the decline phase must be considerably older than those present at other times in a cycle, it may not be possible at present to attribute declines solely to the effects of senescence. If fluctuating age structure plays a crucial role in population cycles, age structure may act either independently of the environment or synergistically with it. Not only are animals older in the decline, but they were born and grew up in a peak density environment, in contrast to the low density environment experienced by peak phase animals. Thus, the rate of senescence may be accelerated by the challenges of living at high density experienced during the peak (Sapolsky *et al.*, 1987).

Christian (1980 and previous papers cited therein) also recognized the potential of peak density environments to have long-term, negative consequences on demography, making this the sole basis of his 'stress' hypothesis. However, in contrast to the senescence hypothesis which requires both changes in age structure over a cycle and a generalized deterioration in body function with age, Christian ignored the effect of age structure shifts and instead concentrated solely on the hypothalamic–pituitary–adrenal axis as being central to population declines. He maintained that social strife at peak densities resulted in high glucocorticoid levels in both adults and young. These high levels precipitated declines because they increased mortality by suppressing the immune and anti-inflammatory responses and decreased reproduction by impairing reproductive capacity of both adults and offspring. Though his hypothesis was supported by numerous laboratory experiments (Christian, 1978, 1980), field evidence was weak and inconclusive (Krebs and Myers, 1974; Lee and McDonald, 1985). Nevertheless, aspects of Christian's hypothesis may still have merit.

Recent evidence collected from field populations of *M. pennsylvanicus* at the onset of spring breeding is consistent with two aspects of Christian's ideas. First, Boonstra and Boag (1992) found that social strife, as indexed by wounding rates in males, was positively correlated with population density. Second, stress responses were also positively correlated to population density; in populations with the highest densities, both males and females had the highest free corticosterone levels. However, the high density population showed the lowest rate of spring decline and, thus, stress responses *per se* could not explain demography at that time. Boonstra and Boag's (1992) study did not, however, determine whether there were long-term consequences for progeny exposed to higher free corticosteroid levels at peak densities. Considerable laboratory evidence makes it reasonable to expect that progeny were affected.

Laboratory studies have found that a variety of pre- and post-natal stressors in rodents can cause irrevocable changes in development that impair subsequent fertility and behaviour (Herrenkohl, 1979; Ward and Reed, 1985; Crump and Chevins, 1989; Takahashi *et al.*, 1990). In

addition, some of these changes can have long-lasting effects, impairing performance over two generations (Christian and LeMunyan, 1958; Pollard, 1986), which may help to explain why some microtine populations remain low for up to a year after a decline. The problem with many of these laboratory studies is that the environmental perturbations to which the animals were subjected were often extreme (exposure to bright lights, heat, cold, electric shocks, crowding and pharmacological doses of glucocorticoids; for a review of stress and development, see De Kloet *et al.* (1988)) and may result in effects that are essentially artefacts relative to the range of conditions animals in nature are likely to experience. However, other studies that have used much more subtle perturbations, such as simply handling the neonates shortly after birth have still produced profound effects on adult behaviour and ability to handle future challenges (De Kloet *et al.*, 1988; Meaney *et al.*, 1988). Though similar studies have not been conducted in microtines, there is evidence from other species that variation in the pre-natal hormonal environment of young affects their performance as adults. Horton (1984) found that simply rearing pregnant female *M. montanus* under different photoperiods was sufficient to affect rates of growth and sexual maturation in progeny. Ims (1989) found evidence for intrauterine masculinization of female embryos, leading to a higher probability of subsequently dispersing in populations of *Clethrionomys rufocanus*. Thus, there are reasons for expecting that the hormonal environment experienced by young microtines at peak densities could interact with old age to affect their fitness during the decline.

### General discussion

The above discussion argues that senescence, possibly interacting with environmental factors, is the explanation for why microtines cycle and is worthy of consideration for testing. Even though microtines exhibit the gamut of social systems ranging from monogamy to polygyny to promiscuity (see papers in Tamarin *et al.* (1990)), population cycles are not restricted to any specific social system. This suggests that the cause of cycles is related to a feature common to them all and I submit that this feature is the reproductive inhibition of young that occurs at peak densities. The cause of the density-dependent reproductive inhibition in cyclic microtines is related to density of breeding adults. Removal of adults from populations in which inhibition is occurring results in rapid maturation of the young, especially females (Bujalska, 1973; Boonstra, 1978; Redfield *et al.*, 1978; Saitoh, 1981; Bondrup-Nielsen, 1986; Bondrup-Nielsen and Ims, 1986; Gilbert *et al.*, 1986; Rodd and Boonstra, 1988). Adults may act either directly or indirectly to suppress reproduction in young by restricting access to crucial resources such as space or food (Bujalska, 1973; Saitoh, 1981), by direct pheromonal inhibition of maturation (Carter *et al.*, 1980; Baddaloo and Clulow, 1981) and by behavioural interactions which stimulate the glucocorticoid levels resulting in the inhibition of reproductive hormone secretion (Christian, 1980).

If this hypothesis is correct and decline animals have significantly lower fitness than those at other times, what permits the population to recover from the decline? Perhaps the lower fitness in the decline environment and particularly the negative maternal environment experienced by young born during the decline is gradually eliminated in successive pregnancies by succeeding cohorts in what is essentially an optimal, uncrowded environment (Mihok and Boonstra, 1992).

This hypothesis does not claim to explain all population declines in microtines. Extrinsic factors such as intense predation by weasels or bouts of severe weather such as freezing rain followed by cold temperatures may be sufficient to explain some declines. However, these extrinsic factors are not necessary to explain population declines (*sensu* Chitty, 1967) where the prerequisite antecedent conditions producing age structure shifts are found and declines will

occur in the absence of these factors. In addition, in populations where age structure shifts do occur, there may be variation in how declines proceed (Krebs and Myers, 1974), with some populations declining rapidly during the breeding season, some declining more slowly over two breeding seasons and some declining mainly during the winter. Differences in decline pattern may occur because of variation in age structure of the young produced from peak populations. This variation may result from factors such as variable degrees of suppression of maturation of the young cohorts because of different starting densities in the peak years or because of variation in the length of the breeding season owing to environmental factors. The detrimental effects of senescence on organisms are progressive as a function of aging and may not produce all or nothing consequences in terms of survival or reproduction (Finch, 1990).

There are **two issues which may argue against the hypothesis**: (1) is delayed reproduction in young adaptive if it leads to reduced fitness and a population decline? and (2) how do we account for non-cyclic populations? One of the puzzling features of microtine cycles is why these populations don't stabilize (Caughley and Krebs, 1983; Stenseth, 1986; Heske and Bondrup-Nielsen, 1990). According to the senescence hypothesis, **cyclic behaviour is fundamentally tied to the reproductive inhibition of the young in the peak year**. However, if the long-term consequence of this inhibition is a lower probability of survival for the individual and a population decline, why don't young adopt a different strategy, i.e. reproduce in spite of high density rather than face almost certain reproductive failure. The problem is that young may have no alternative because reproduction at peak density may result in no fitness gain because the young are socially subordinate and there are few additional resources for breeding (e.g. space, food, etc; see Bronson (1989) for a review). Schaffer and Tamarin (1973) presented an adaptive model to relate alternating reproductive effort to fluctuating microtine populations. They proposed that because survival of the pre-reproductives (juveniles) declines at high density, it is adaptive for young to delay maturation until such time when conditions improve. **Though it may be adaptive to delay reproduction for these young animals (i.e. they are making the best of a bad situation; (Gross, 1982)), the length of the delay is crucial**. Good conditions may not arrive soon enough relative to their lifespan because they must delay until the next breeding season, when they are old and, consequently, a population decline inevitably follows.

**Many populations of microtines do not cycle (Taitt and Krebs, 1985) and any hypothesis to explain why cycles occur in some populations and at some times should also be able to explain why others don't**. I suggest that any factor such as predation, parasitism, low cover, severe weather or high habitat heterogeneity that operates to keep population density low will prevent density-dependent inhibition of maturation of young and prevent the development of age structure characteristic of decline populations. In southern Fennoscandia, evidence suggests that intense, continuous predation by generalist predators is the most likely cause for the lack of cycles there (Erlinge, 1987; Erlinge *et al.*, 1983). In the non-cyclic populations, a high proportion of female young born each year mature in their year of birth (Nyholm and Meurling, 1979), whereas those born in the north into peak populations do not mature until the next year (Nyholm and Meurling, 1979; Bondrup-Nielsen and Ims, 1986). Hence, under conditions of intense, continuous predation, young cohorts are being produced throughout each breeding season and age structure shifts from one year to the next do not occur. **Annual, intense bouts of parasitism and disease, acting either alone or in concert with other factors, may also act to prevent cycles. *Microtus townsendii* populations, which are largely non-cyclic (Taitt and Krebs, 1985), are parasitized every year from July to September by botflies (*C. grisea*) and by grey flesh flies (*Wohlfahrtia vigil*) (Boonstra, 1977b; Boonstra *et al.*, 1980), increasing mortality and decreasing reproduction**. Inadequate cover may prevent the development of peak densities because of more intense predation, decreased food supply and altered levels of social interaction in these areas

(Hoffmann, 1974; Birney *et al.*, 1976). Koshkina and Korotkov (1975) report that for *Clethrionomys rutilus* in suboptimal habitats in Siberia where overwinter survival is low and, thus, where density at the onset of the breeding season is always low as well, the proportion of young females maturing in their year of birth is always high (23–72%) and these populations do not cycle. This contrasts with low maturation rates (0–8.5%, Table 1) at peak densities in optimum habitats where the populations cycle. Finally, highly heterogeneous habitats may permit young to escape from density-dependent suppression of reproduction in optimal habitats by dispersal into suboptimal habitats (Abramsky and Tracy, 1979; Rosenzweig and Abramsky, 1980; Ostfeld, 1992), resulting in the continuous production of cohorts of young animals.

The latter point emphasizes the role that dispersal has in generating cycles according to this hypothesis. Dispersal processes have been regarded as necessary for population regulation in microtines (Lidicker, 1975; Gaines and McClenaghan, 1980; Tamarin, 1980; Stenseth, 1983; Krebs, 1992). I view dispersal, motivated largely by spacing behaviour acting particularly on young during the increase phase, as a mechanism of filling optimal environments with voles. Once these environments are full, density-dependent inhibition of maturation occurs and sets the stage for the decline. This view of the role of spacing behaviour and dispersal is similar to the first portion of the ‘social fence hypothesis’ put forward by Hestbeck (1982), but differs from the second portion of his hypothesis that resource exhaustion causes the decline. Resource exhaustion may be sufficient to cause declines (Hestbeck, 1986), but is not necessary (Krebs and DeLong, 1965; Cole and Batzli, 1978; Desy and Thompson, 1983).

The senescence hypothesis must be potentially falsifiable. It makes the following testable predictions.

(1) That breeding animals present during declines are significantly older than those at comparable times in non-decline years.

(2) That the basis of the difference between decline animals and those at other times is phenotypic, not genotypic. Thus, all microtine populations have the intrinsic capacity to exhibit cycles so long as the necessary age structure shifts occur.

(3) That the young of females removed from decline populations should have lower fitness (lower survival and growth of progeny) than those removed from increase and peak populations when removed to either the laboratory or to vacant field habitats.

(4) That the effects of senescence in nature should start occurring well before a year of age, with the onset varying not only among species depending on their life histories (Promislow, 1991) but also among different populations within the same species because of habitat or area-dependent factors. For example, in meadow voles living in southern Yukon where the mean breeding season may be 4 months or less (Krebs and Wingate, 1985), the onset of senescence in overwintering animals should be delayed for at least 8 months. In contrast, in meadow vole populations living in Indiana where the mean breeding season is 8 months or more (Krebs *et al.*, 1969), the onset of senescence should be delayed for only 3–4 months.

(5) If senescence is the sole cause of declines, then old animals should have lower fitness (reproduction and survival) even if they haven’t been exposed to the prior effects of peak density. Alternatively, if the exposure to peak densities is the sole cause of declines, then animals that are kept under optimum conditions and prevented from breeding until the next season should have a fitness similar to that of young animals. If both senescence and prior peak density interact, then both may be required to produce decline phase animals. Thus, while there may be a negative effect of age on reproductive fitness, this effect may be exacerbated by prior exposure to peak density. Experiments to test this interaction should be carried out in field situations with normal

seasonality and social structure. Thus, it should be possible to experimentally produce cycles in any microtine population.

(6) That old animals cannot maintain physiological homeostasis as well as young ones in response to challenges (Sapolsky *et al.*, 1986). Thus, it should be possible to use response to challenges such as a restraint stressor as a probe for senescence, with voles from decline populations being less able to maintain homeostasis than those from other years. Thus, recovery from exposure to a standardized stressor such as restraint will vary among years of a cycle and be slowest in decline populations.

Finally, does the senescence hypothesis have any relevance as an explanation to cycles in other mammals? I suggest it may be part of the explanation of the 10 year snowshoe hare (*Lepus americanus*) cycle. Evidence suggests that the decline in hares is related either to predation alone (Krebs *et al.*, 1986; Sinclair *et al.*, 1988) or to an interaction between food and predation (Keith, 1987). One of the enigmas of this cycle is the very poor survival of juveniles during the decline and the period of low numbers even after the vegetation has recovered and recovery of the population does not occur until after juvenile survival improves (Krebs *et al.*, 1986; Keith, 1987). During the decline and period of low numbers (lasting for 4–5 years), there is a shift so that a significantly larger portion of the population is old (i.e. in 1970, the peak at Rochester, Alberta, 7% of the population were 3+ years old versus in 1975, the last year of the decline, when 56% were 3+ years old) (Keith and Windberg, 1978). I suggest that the poor survival of juveniles may be related to the age of their mothers and it is only when age structure shifts towards younger animals, that the hare population starts increasing again.

### Conclusion

Microtine cycles remain one of the major unsolved problems of population ecology. All current explanations are still controversial and in question and all ignore a central aspect of the demography of microtines – their age structure. I propose a new solution to this problem which ties together shifting age structure of breeding animals with senescence. Age structure must undergo major shifts from the peak to the decline for three reasons: (1) the breeding season during the peak is shortened and no winter breeding occurs, unlike the situation found during the increase, (2) turnover of the population from one breeding season to the next is virtually 100% and, thus, only young that have not bred previously contribute to reproduction each year and (3) density-dependent inhibition of sexual maturation of young born early during the peak year forces them to delay reproduction until the next year, whereas young born during the increase year mature and breed in the year of their birth. Old age of animals present during the decline, either acting separately or in conjunction with the experiences of peak density, results in lower fitness for both themselves and their young born in the decline year, causing the population to cycle. May (1976), using a simple mathematical model, found that time-lags of 9 months would generate cycles of 3–4 years. The senescence hypothesis provides the biological mechanism based on density-dependent inhibition of maturation in peak years to produce time-lags of the correct magnitude. The hypothesis is falsifiable and I make six predictions that can be used to test it.

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