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Population cycles in small mammals: the role of age at sexual maturity

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Several hypotheses have been proposed to explain cyclic fluctuations in abundance of some small mammal populations. These hypotheses have been controversial, however, and there is no consensus among biologists as to why population cycles occur. In a demographically based model, we tested the potential influence of phase-specific changes in life history traits (age at maturity, fertility, juvenile survival and adult survival) on population cycles. Our demographic model considers, and is logically consistent with, the empirical pattern of population characteristics during a cycle. The essence of the model is that phase-specific changes in age at maturity, abetted secondarily by changes in juvenile survival, result in cyclic fluctuations in population size. Changes in adult survival and fertility may play a minor role, but they are neither necessary nor sufficient by themselves to generate population cycles. Phase-specific changes in age at maturity might be related to primary changes in the quality of the ecological and social environment that permit particularly high densities.

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The fact that some populations of small mammals undergo somewhat regular multi-annual fluctuations in abundance was known to ecologists early in this century (Elton 1924, 1942). Several hypotheses have been proposed to explain multi-annual fluctuations in abundance, commonly referred to as population cycles, but these have been controversial, and there has been no consensus among biologists as to why population cycles occur (e.g., Chitty 1960, Christian and Davis 1964, Christian 1980, Batzli 1992, Boonstra 1994, Krebs 1996, Selås 1997, Tkadlec and Zejda 1998a). Despite numerous field and laboratory research programs, which resulted in over 1000 publications (Batzli 1992), causes of population cycles remain an enigma.

Population cycles are characterized by phase-related changes in various aspects of the biology of populations. In high-density, declining populations, age at maturity is delayed, juvenile survival and fertility are reduced, adverse social interactions (aggressive and spacing behaviors) increase, and the mean age of reproductive females increases (e.g., Krebs and Myers 1974, Boonstra 1994, Krebs 1996). In the low-density, increasing phase, age at maturity decreases, juvenile survival and fertility increase, adverse social interactions decrease, and the mean age of reproductive females decreases. A hypothesis attempting to explain population cycles must consider these phase-specific, density-dependent changes in population characteristics that define population cycles (Krebs 1996).

Changes in population size are a consequence of changes in demographic characteristics. However, some demographic parameters influence changes in population size more profoundly than the others. It is, therefore, necessary to identify demographic parameter(s) that can substantially influence population dynamics. One can then proceed to search for mechanisms that can cause phase-specific changes in important demographic variables that in turn cause multi-annual fluctuations in abundance. In this paper, we examine the potential of various demographic variables to cause

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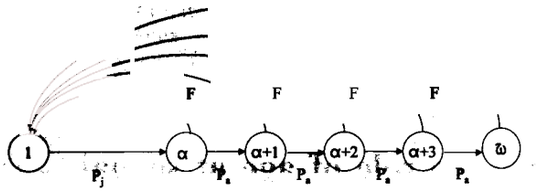

Fig. 1. A partial life cycle graph. Juveniles survive to age at maturity (α) with a survival probability of P_j per time unit. Once maturity is attained, adults survive with a survival probability of P_a and contribute to the population with an average fertility of F per time unit until age at last reproduction, ω. See text for details.

population cycles, and propose a simple, demographically based mechanistic model to explain population cycles based on density-dependent, phase-specific changes in life history traits. The model incorporates, and is logically consistent with, empirical observations.

Methods

The model and parameters

We investigated the sensitivity of population cycles to demographic parameters. Specifically, we tested whether cyclic changes in age at maturity, juvenile survival, adult survival, and fertility, alone or in combination, were necessary and sufficient to cause cyclic changes in population size of magnitude similar to those observed in nature. Juveniles survive to the age at maturity, α, with a survival probability of P_j per time unit. Once they attain sexual maturity, they reproduce with an average fertility rate of F, and survive with a probability P_a per time unit until the age at last reproduction, ω (see below for seasonality in reproduction). This simplified life cycle, called a partial life cycle, can be represented as a partial life cycle graph (Fig. 1). A transition matrix, **A**, can be constructed from the partial life cycle graph, which can then be used for population projection (Caswell 1989):

$$\mathbf{n}(t+1) = \mathbf{A}\mathbf{n}(t) \quad (1)$$

where $\mathbf{n}(t)$ is the population vector at time (t), and $\mathbf{n}(t+1)$ is the population vector at time $(t+1)$; entries of $\mathbf{n}(t)$ and $\mathbf{n}(t+1)$ are the number of individuals of age class 1, 2,..., ω at time (t) and $(t+1)$, respectively. The population size at time (t) is given by the sum of the entries of the population vector, $\mathbf{n}(t)$. **A** is the population projection matrix whose entries are F, P_j, and P_a. The population projection matrix corresponding to the life cycle with $\alpha = 2$ and $\omega = 6$ is:

$$\mathbf{A} = \begin{bmatrix} 0 & F & F & F & F & F \\ P_j & 0 & 0 & 0 & 0 & 0 \\ 0 & P_a & 0 & 0 & 0 & 0 \\ 0 & 0 & P_a & 0 & 0 & 0 \\ 0 & 0 & 0 & P_a & 0 & 0 \\ 0 & 0 & 0 & 0 & P_a & 0 \end{bmatrix}$$

We used meadow vole (*Microtus pennsylvanicus*) as a model organism (Table 1). We assumed that the survival rates of juvenile and adult females ranged between maximum and minimum values in Table 1 of Boonstra and Rodd (1983), and that survival rates were phase-specific. We defined four-week projection intervals, and bi-weekly survival rates were converted to four-week periods.

Data on fertility (F) were not provided by Boonstra and Rodd (1983), but this parameter is required for the partial life cycle graph model. The minimum and maximum litter sizes for *M. pennsylvanicus* summarized in Table 1 of Innes (1977) were assumed to be representative of peak and valley phase, respectively. We assumed primary sex ratio to be 0.5 (Dobson and Myers 1989), and from Figure 7 of Boonstra and Rodd (1983), we estimated the proportion of breeding females to be 0.5. Fertility was then estimated as: $F = 0.5LBP_a$, where L is the average litter size, B is the proportion of adult females breeding, and P_a is the mean adult survival rate.

Age at maturity is difficult to measure in the field, and data on this life history variable are scarce (Keller 1985). Voles are known to attain sexual maturity as

Table 1. Parameter values used in the simulation model. Bi-weekly juvenile (P_j) and adult (P_a) survival rates for peak and valley phases were taken as minimum and maximum survival rates in Table 1 of Boonstra and Rodd (1983), respectively, except that juvenile survival rate for the valley phase was taken as 0.9 because maximum juvenile survival of 1 reported by Boonstra and Rodd (1983) is rare. These were then converted for four-week periods (i.e., one time unit). Age at maturity (α) was assumed to range from one month in the valley density to six months in the peak density. Age at last reproduction ($\omega = 13$ months) was assumed to be constant. Fertility (F) was calculated as $F = 0.5LBP_a$, where L is litter size, B is proportion of females breeding, and P_a is adult survival. μ and β are the mean parameter value and amplitude, respectively.

	Peak	Valley	μ	β
Age at maturity, months (α)	6		3.50	
Age at last reproduction, months (ω)		–	13	
Juvenile survival (P_j)	0.19	0.81	0.50	
Adult survival (P_a)	0.74	0.88	0.81	
Fertility (F)	0.73	1.40	1.06	

early as three to four weeks post-partum (Leslie and Ranson 1940, Batzli et al. 1977). Females of *Clethrionomys* spp. born in spring and summer produce litters by the end of the summer during low-density, increasing phase; spring- or summer-born females do not produce litters until the following breeding season at high densities (Saitoh 1981, Gilbert et al. 1986, Löfgren 1989, Gilbert and Krebs 1991). Thus, we assumed that in high-density, declining populations, young females do not reproduce until the following spring, whereas those born in low-density, increasing populations reproduce shortly after they are weaned.

Data summarized by several authors indicate that age at maturity shows approximately cyclic changes during a population cycle (e.g., Myers and Krebs 1974, Cary and Keith 1978, Bondrup-Nielsen and Ims 1986, Löfgren 1989, Gilbert and Krebs 1991, Boonstra 1994). Thus, we assumed that age at maturity changes cyclically from one month in increase phase to six months in decline phase. For comparative purposes, we also allowed P_j, P_a, and F to vary cyclically. Values of α, P_j, P_a, and F for different phases of cycles were estimated as:

$$\gamma(t) = \mu + \beta \sin(2\pi t/\tau) \quad (2)$$

where $\gamma(t)$ is the value of a parameter (α, F, P_j, or P_a) at time t, μ is the mean parameter value, β is the amplitudes of the cycle, t is time, and τ is the duration of the cycle. Values of μ and β were different for each parameter, but the value of τ was assumed to be 36 time units (i.e., 3-year cycles) for all parameters. Age at last reproduction, ω, was assumed to be constant (13 months; Table 1).

Given a population vector $\mathbf{n}(t)$, population size at time $(t+1)$ is a function of α, ω, P_j, P_a, and F. Naturally, changes in any of these parameters will cause changes in population size. However, one must consider how widely these parameters vary in natural populations, and whether cyclic changes in parameters within the range observed in nature can cause substantial fluctuations in abundance. All entries of the initial population vector were 100; thus the initial population size was 1300. In all subsequent times, the population vector was projected using eq. (1). For any time (t), we calculated population size as the sum of the entries of the population vector, $\mathbf{n}(t)$.

To investigate whether cyclic changes in α, P_j, P_a, or F can cause cyclic changes in population size of the magnitude observed in nature, we allowed one variable at a time to change cyclically and held other variables constant at the mean values. When the population size reached 100000 ("ceiling population size"), we set the test variable to its "worst" value. Thus, to test for the influence of age at maturity, we assigned the maximum value (Table 1) to age at maturity when population size reached the ceiling. To test for the influence of P_j, we set the value of P_j to its minimum when population size reached the ceiling. When population size reached 100 ("floor population size"), we set age at maturity to its minimum, and P_j, P_a, and F to their maximum values for evaluating the effect of cyclic fluctuations in α, P_j, P_a, and F, respectively.

To investigate the influence of fluctuations in a pair of variables on population dynamics, we allowed simultaneous, cyclic fluctuations in two variables while holding other variables constant. A similar approach was used to investigate changes in population size in response to simultaneous cyclic fluctuations in three variables while holding the fourth variable constant.

Seasonality in reproduction

Typically, reproduction in *Microtus* begins in the spring and continues until the onset of winter. However, length of breeding season varies among species. Some species (e.g., *M. ochrogaster*, *M. townsendii*) may breed year-round while others (e.g., *M. pinetorum*, *M. pennsylvanicus*) do not breed for a part of winter (Innes and Millar 1994). Even within a population, length of breeding season may vary among years (Boonstra 1985, Innes and Millar 1994). To incorporate seasonality in reproduction in our model, we calculated the mean and standard deviation of the mean length of non-breeding season for seven cyclic species of *Microtus* (*M. agrestis*, *M. californicus*, *M. montanus*, *M. ochrogaster*, *M. pennsylvanicus*, *M. pinetorum*, and *M. townsendii*) from Table 3 of Innes and Millar (1994). We assumed that the mean length of non-breeding season as calculated above (3 months; rounded-off to the nearest month) was representative of our model organism, and that within-population variation in the length of non-breeding season lies within one standard deviation around the mean (3 ± 2 months; rounded-off to the nearest month). Simulations were then run under three scenarios: (1) year-round reproduction, (2) no winter reproduction for three winter months (December–February), and (3) random variation in non-reproductive period for one (January), two (January and February), three (December–February), four (December–March), or five (November–March) winter months. For the third scenario (i.e., random variation in non-reproductive period), 1500 simulations were run for each variable or combination of variables being investigated, and the mean population vector was calculated. We calculated mean amplitudes as the mean of the ratio of maximum and minimum population size for each cycle.

Results

When only one variable was allowed to fluctuate cyclically and reproduction was allowed to occur through-

Table 2. Do cyclic fluctuations in one or more life history parameters produce population cycles? The mean amplitude of cycles was calculated as the average of the ratio of maximum and minimum population sizes for each cycle. To incorporate variation in winter reproduction, simulations were run under three scenarios: (1) year-round reproduction, (2) no winter reproduction for three winter months (December–February), and (3) random variation in non-reproductive period for one (January), two (January and February), three (December–February), four (December–March), or five (November–March) winter months. For the third scenario (i.e., random variation in non-reproductive period), 1500 simulations were run for each variable or combination of variables, and amplitudes were calculated from the mean population vector. Data for time units 50 and onwards were used to exclude erratic initial changes in population size. Values of parameters used in simulations are given in Table 1. See text for details.

Variable(s)	Generated cycles?	Mean amplitude		
				3***
Age at maturity (α)	Yes	8.42	9.86	6.47
Juvenile survival (P_j)	Yes	3.34	3.99	3.05
Adult survival (P_a)	No	–	–	–
Fertility (F)	No	–	–	–
α and P_j	Yes	95.08	29.58	30.63
α and P_a	Yes	14.24	11.06	8.97
α and F	Yes	21.38	16.76	10.39
P_j and P_a	Yes	5.11	5.46	4.01
P_j and F	Yes	5.95	7.98	4.00
P_a and F	No			–
α, P_j and P_a	Yes	1868.54	371.91	42.97
α, P_j and F	Yes	1625.79	633.93	51.89
α, P_a and F	Yes	85.73	194.86	13.86
P_j, P_a and F	Yes	9.50	9.54	5.27

* Year-round reproduction.
** No reproduction for three winter months.
*** Random variation in non-reproductive period for one, two, three, four, or five winter months.

out the year, only α and P_j caused cyclic fluctuations in population size (Table 2, Fig. 2). The mean amplitude of population cycles caused by cyclic fluctuations in P_j alone were much smaller then those observed in natural populations. However, the mean amplitudes of cycles caused by cyclic fluctuations in α alone were similar to those observed in some natural populations (e.g., Taitt and Krebs 1985, Agrell et al. 1992, Saitoh et al. 1997). Incorporating seasonality in reproduction in the model did not change the potential of life history traits to cause cyclic fluctuation in abundance or the relative amplitude of population cycles (Table 2).

When two variables were allowed to fluctuate simultaneously and reproduction was allowed year-round, all variables produced cycles with mean amplitudes of > 14 if they were allowed to fluctuate along with α (Table 2, Fig. 3). Simultaneous fluctuations in α and P_j produced cycles with amplitudes of 95 and these are similar to amplitudes of cycles observed in some populations with extremely wide fluctuations in abundance (e.g., Beacham 1980, Mihok et al. 1985, Keith 1990; mean amplitude ≤ 100). Amplitudes of cycles produced by simultaneous fluctuation in P_j and P_a, and P_j and F generally were smaller than those observed in natural populations. When seasonality in reproduction was incorporated in the model, absolute mean amplitudes changed somewhat but the relative potential of simultaneous fluctuation in a pair of variables to cause cyclic fluctuation in population size remained unchanged (Fig. 3, Table 2).

When three variables were allowed to fluctuate simultaneously and reproduction was allowed year-round, all combinations of variables produced cycles. However, amplitudes of population cycles produced by any combination of variables involving α were > 85, whereas the amplitude of cycles produced by combination of variables without α was at least an order of magnitude smaller. Incorporating seasonality in reproduction changed the absolute mean amplitudes but not the relative potential of combination of variables to cause cyclic fluctuation in abundance nor the relative amplitudes (Fig. 4, Table 2).

It can be inferred from the results in Figs 2–4 and Table 2 that: (1) when only one variable is considered, only cyclic fluctuations in α can cause cyclic fluctuations in population size of the magnitudes similar to those observed in some natural populations, (2) cyclic changes in P_j can cause population cycles, but amplitudes of cycles are substantially smaller than those observed in natural populations, (3) cyclic fluctuations in no variable other than α, or combination of variables that does not include α, can generate population cycles of amplitude observed in nature unless these are accompanied by changes in α, and (4) that simultaneous cyclic fluctuations in both α and P_j can cause population cycles with differences in peak and valley population size of up to two orders of magnitude. We therefore conclude from these results that cyclic changes in α are necessary, and in some cases sufficient, for causing population cycles, and that similar fluctuations in P_j act synergistically with α to produce population cycles with extreme amplitudes.

Discussion

Demography of population cycles

The dynamics of a population are determined by the population growth rates, which in turn, are functions of demographic variables. Fluctuation in population size, therefore, cannot be explained without considering demographic causes of such fluctuations. Our study addresses this issue. Our results indicate that phase-specific changes in age at maturity are the primary demographic cause of population cycles. We do not suggest that changes in survival or fertility do not influence population cycles. Only strong, cyclic changes in age at maturity are necessary, however, and in many cases, sufficient to cause cyclic fluctuations in population size. Changes in other population parameters, primarily juvenile survival, may play a supporting role, especially when changes in age at maturity are small. However, much greater levels of changes in fertility and adult survival rate than those used in this study would be required for these variables to cause cyclic fluctuation in numbers if such changes are not accompanied by realistic changes in age at maturity.

We consider changes in age at maturity as the primary demographic cause of population cycles for three reasons. First, population growth rate is more sensitive to age at maturity than other demographic variables (Fig. 2; Cole 1954). Second, age at maturity shows much wider fluctuations during a population cycle compared to other demographic variables. For example, Gilbert and Krebs (1991) reported that, during years of low density, year-born females of *Clethrionomys rutilus* bred and were responsible for up to 66% of total litters produced. During years of high density, few, if any, year-born females reproduced. Similar observations have been reported by several authors (Bujalska 1985, Bondrup-Nielsen and Ims 1986, Gilbert et al. 1986, Boonstra 1994, and references therein). These observations suggest that sexual maturity in cyclic populations can vary from a few weeks to several months during a cycle; scope of variation in other demographic variables is considerably smaller. Finally, age at maturity responds rapidly to changes in the quality of the environment, such as population density (Gustafsson et al. 1983, Bondrup-Nielsen and Ims 1986, Nakata 1989, Gilbert and Krebs 1991). Therefore, age at maturity, although difficult to measure, appears to be the sort of demographic variable that should cause rapid changes in the abundance of cyclic populations of small mammals.

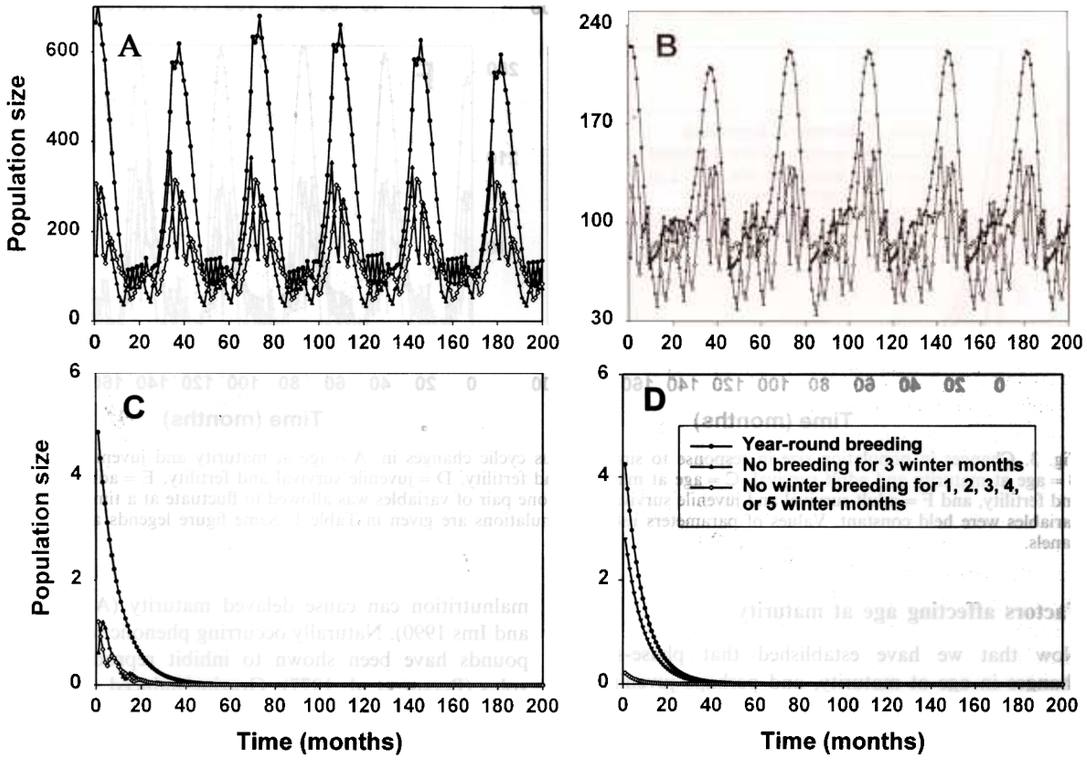

Fig. 2. Changes in population size in response to cyclic changes in: A = age at maturity, B = juvenile survival, C = adult survival, and D = fertility. Only one variable allowed to fluctuate at a time; all other variables were held constant. Values of parameters used in simulations are given in Table 1. Same figure legends apply to all panels.

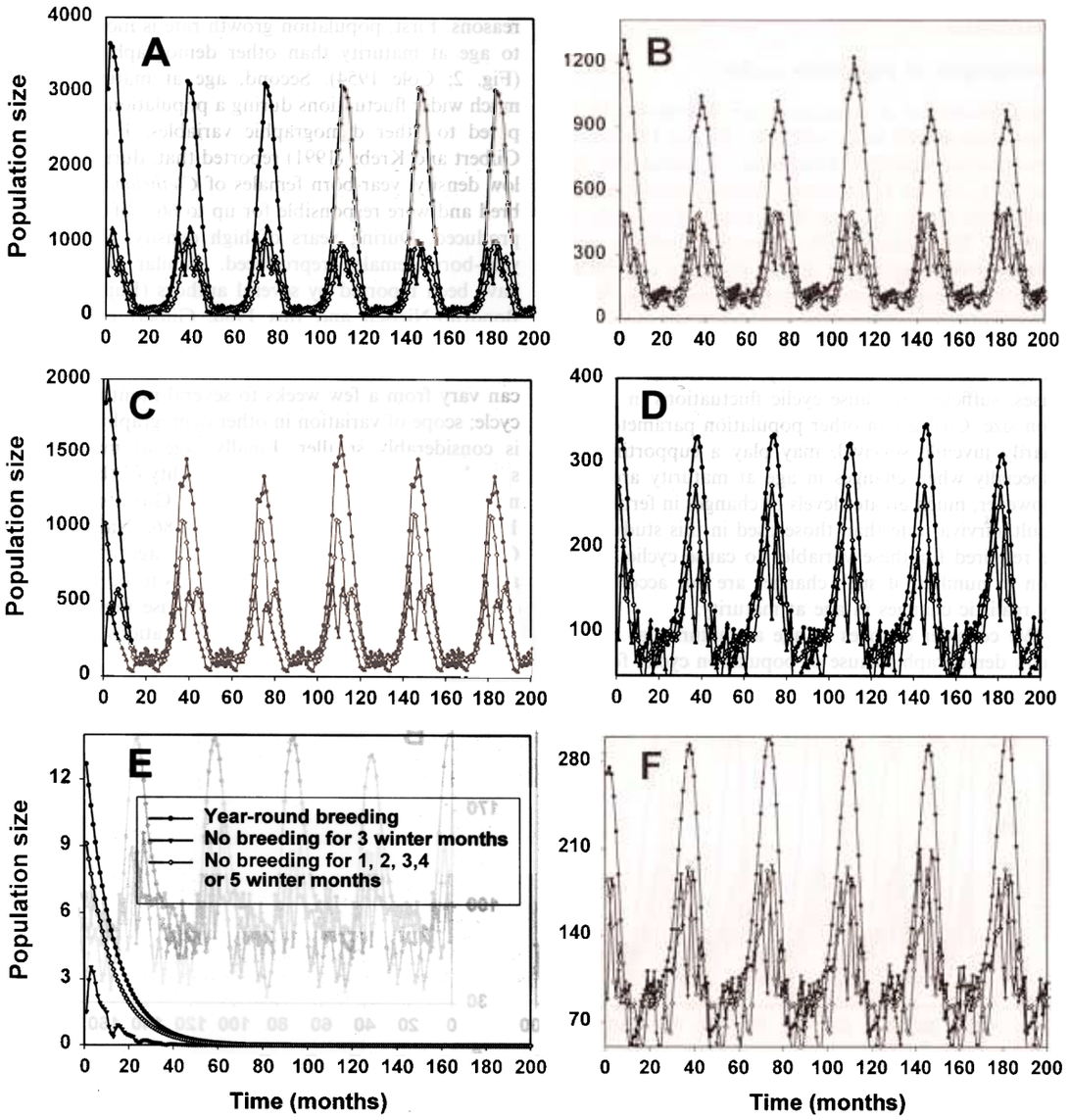

Fig. 3. Changes in population size in response to simultaneous cyclic changes in: A = age at maturity and juvenile survival, B = age at maturity and adult survival, C = age at maturity and fertility, D = juvenile survival and fertility, E = adult survival and fertility, and F = adult survival and juvenile survival. Only one pair of variables was allowed to fluctuate at a time; all other variables were held constant. Values of parameters used in simulations are given in Table 1. Same figure legends apply to all panels.

Factors affecting age at maturity

Now that we have established that phase-specific changes in age at maturity, and perhaps juvenile survival, are necessary and sufficient for population cycles, possible mechanisms for changes in age at maturity should be discussed.

Several factors can influence age at maturity. Nutrition affects growth as well as maturation processes, and malnutrition can cause delayed maturity (Andreassen and Ims 1990). Naturally occurring phenolic plant compounds have been shown to inhibit reproduction in voles (Berger et al. 1977). Grazing-induced proteinase inhibitors, which are produced by plants as a defense against herbivory, can adversely influence growth as well as maturation processes in arvicoline rodents (Seldal et al. 1994). Recently, Selås (1997, 1998) suggested that masting plants produce seeds at the expense of

chemical defense against herbivory. This should result in a temporal increase in rodent density after mast years. Thus, changes in the quality and quantity of food resources as a result of plant-herbivore interactions can influence growth, as well as maturation processes, and consequently, abundance (Berger et al. 1977, Seldal et al. 1994, Selås 1997, Selås and Steel 1998; but see Hansson 1998 for a different view), despite the failure of experiments involving food supplementation to stop population cycles (e.g., Taitt et al. 1981, Desy and Batzli 1989, Ylönen et al. 1991, Krebs et al. 1995).

Gustafsson et al. (1983) experimentally demonstrated that high population density inhibits sexual maturition in *C. glareolus*. Gilbert and Krebs (1991) found a strongly negative correlation between sexual maturity and population density in *C. rutilus*. Similar results have been reported by several other studies (e.g., Christian 1971a, b, Perrin 1979, Wiger 1979, Boonstra and Rodd 1983, Moshkin et al. 1998, Tkadlec and Zejda 1998b). Thus, population density appears to be an important influence on maturation rate, although underlying mechanisms are not fully understood.

Social suppression of reproduction can occur either by behavioral means (e.g., Batzli et al. 1977, Krebs et al. 1978), or by the action of puberty-delaying pheromones secreted by adult females at high density (e.g., Lepri and Vandenbergh 1986, Vandenbergh 1987, 1994, Kruczek et al. 1989, Drickamer and Mikesic 1990). Adult females might have the ability to assess the quality of the environment using such cues as resource shortage, the rate of encounter with other conspecific individuals, and the prevalence of adverse social encounters. If the environment is perceived to be unfavorable or risky, adult females might release puberty delaying pheromones as a signal of poor environmental quality. Inhibiting the maturity of young females will result in lower density, and thus less competition for resources, in subsequent generations. Young females might respond physiologically to the chemosignals which inhibit their sexual maturity or directly delay maturity due to unfavorable social conditions. Refraining from reproduction at a time when environmental conditions are less than adequate might allow suppressed females to put on additional body mass and gain experience which could enhance future reproductive success (Lepri and Vandenbergh 1986, Drickamer and Mikesic 1990).

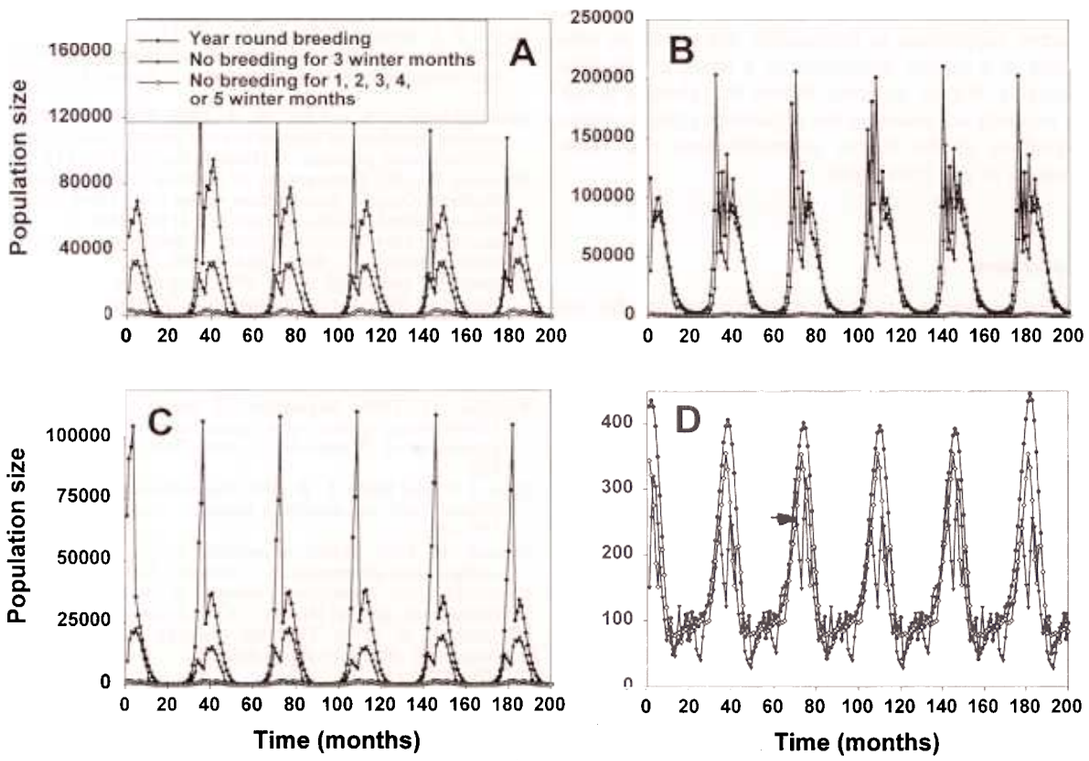

Fig. 4. Changes in population size in response to simultaneous cyclic changes in: A = age at maturity, juvenile survival, and fertility, B = age at maturity, adult survival and fertility, C = age at maturity, juvenile survival, and adult survival, D = juvenile survival, adult survival, and fertility. The fourth variable was held constant. Values of parameters used in simulations are given in Table 1. Same figure legends apply to all panels.

Another possible mechanism for phase-related changes in age at maturity is a response to pre- or post-natal stress (Christian 1980, Pollard 1986, Mihok and Boonstra 1992, Boonstra et al. 1998). Individuals in cyclic populations may have the ability to assess the quality of the environment. When the environment is unfavorable, factors in the social and ecological environment (e.g., high density, adverse social interactions, high predation risk, resource scarcity) act as non-specific stressors, and trigger a stress response. The primary stress response is an increased hypothalamus-pituitary-adrenal activity, which is inversely related to hypothalamus-pituitary-gonadal function (Christian 1980, Lee and McDonald 1985, Handa et al. 1994). Suppression of hypothalamus-pituitary-gonadal activity then produces delayed maturity. The experimental evidence that predation risk, a non-specific stressor, significantly inhibits reproduction in *C. glareolus* (Heikkilä et al. 1993, Ylönen and Ronkainen 1994) and snowshoe hare (Boonstra et al. 1998) suggests that stress response can cause changes in age at maturity in cyclic populations.

There may be other factors as yet not fully understood that can directly or indirectly influence age at maturity. In the absence of conclusive experiments, we would not discount any possible mechanism of reproductive suppression as implausible, nor would we subscribe to a specific mechanism or a factor as the most probable. We do, however, believe that changes in age at maturity are necessary for population cycles to occur regardless of the factors or mechanisms that cause changes in age at maturity.

Conclusion

Despite numerous studies over the past six decades, we know very little about the causes of population cycles in small mammals. There are more points of disagreement among ecologists than of agreement. One possible reason for the obvious lack of progress in unraveling the enigma of population cycles could be that most existing hypotheses have attempted to explain changes in abundance without considering underlying demographic mechanisms. Consequently, field studies either have ignored demographic mechanisms, or have focused on variables that have no potential for causing large, often spatially synchronous, fluctuations in population size. Our study indicates that phase-specific changes in age at maturity are the primary demographic cause of cyclic fluctuations in abundance, but few, if any, studies of cyclic populations have considered age at maturity to be an important demographic variable. The demographic model presented here should be tested in cyclic populations by examining changes in age at maturity during all phases of population cycles. If our conclusions hold, field studies should focus on empirically determining causes of phase-specific changes in age at maturity. It should then be possible to develop a unifying, demographically-based, mechanistic hypothesis of population cycles.

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