

## POPULATION CYCLES IN SMALL MAMMALS: THE $\alpha$ -HYPOTHESIS

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Causes of cyclic fluctuations in abundance (population cycles) of some small-mammal populations remain poorly understood despite 6 decades of research and >20 hypotheses. Population cycles are demographic processes and cannot be fully explained without considering demographic mechanisms that underlie cyclic fluctuations in abundance. From simulation studies, we have recently shown that phase-related, density-dependent changes in age at maturity, abetted secondarily by changes in juvenile survival, are likely the main demographic causes of cyclic fluctuations in population size. The suggested mechanism of population cycles is based primarily on changes in age at maturity ( $\alpha$ ); we refer to this idea as the  $\alpha$ -hypothesis. Here, we fully develop the  $\alpha$ -hypothesis and present a testable, demographically based, mechanistic explanation of population cycles. The  $\alpha$ -hypothesis identifies the demographic basis of population cycles and provides a mechanistic explanation of how changes in key demographic variables (age at maturity and juvenile survival) might cause cyclic fluctuations in abundance and biologic attributes of the cycles. The  $\alpha$ -hypothesis is supported by, and logically consistent with, empirical patterns of life history and dynamics of cyclic populations of small mammals. Future research should focus on empirically determining causes of phase-related changes in age at maturity and juvenile survival.

Key words:  $\alpha$ -hypothesis, age at maturity, arvicoline rodents, demographic mechanisms, population cycles, population dynamics

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Populations of some small mammals fluctuate cyclically, with amplitudes of >2 orders of magnitude. Such fluctuations in numbers, commonly called population cycles, have been the subject of considerable research, dating back to Charles Elton's work early in the past century (Elton 1924, 1942). Theoretical and empirical research programs over the past 6 decades have produced >20 hypotheses to explain population cycles and >1,000 publications (Batzli 1992). However, there exist more disagreements than agreements among ecologists regarding the causes of population cycles,

and existing hypotheses are controversial (Chitty 1960; Krebs 1996; Lidicker 1988; Seldal et al. 1994).

Clearly, changes in population size are a consequence of changes in  $\geq 1$  demographic variable (Cole 1954; Lewontin 1965; Oli and Dobson 1999). Fluctuations in abundance, therefore, cannot be explained without considering underlying demographic mechanisms that produce such changes (Oli and Dobson 1999). Recognizing the importance of demographic mechanisms in the regulation of populations of small mammals, Lidicker (1978:137) stated that "placing demographic mechanisms in relation to life history phenomena in general should be

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a productive direction for the future” and predicted that such an approach should improve our understanding of mammalian population biology. However, Lidicker’s (1978) prediction has not yet materialized.

We suggest that a complete explanation of population cycles should involve 2 steps. First, there should be a clear understanding of underlying demographic mechanisms of population cycles. Specifically, we should identify demographic variables that show phase-related changes during a cycle and potentially can cause large-scale fluctuations in abundance similar to those observed in natural populations. If changes in  $\geq 1$  demographic variable, within the range observed in natural populations, can cause large-scale fluctuations in abundance, and consequences of these changes are consistent with empirical data, such variables may be considered necessary and sufficient (cf. Chitty 1960) demographic conditions (hereafter referred to as key demographic variables) for cyclic fluctuations in abundance. Second, after key demographic variables are identified, causes of phase-related, density-dependent changes in key demographic variables should be ascertained. Such causes may be expected to involve factors in social and ecologic environments and have been the focus of most hypotheses to explain cyclic fluctuations in abundance.

It is well known that demographic variables differ in their potential to influence population dynamics, with some variables affecting changes in population size more profoundly than others (Cole 1954; Lewontin 1965; Oli 1999a). Using life-history data for *Microtus pennsylvanicus* and a demographically based model, we recently showed that phase-specific changes in age at maturity, abetted secondarily by changes in juvenile survival, are likely the main demographic causes of cyclic fluctuations in population size (Oli and Dobson 1999). Because the suggested demographic mechanism is based primarily on changes in age at maturity ( $\alpha$ ), we call this idea the  $\alpha$ -hypothesis. In this paper, we fully develop the

$\alpha$ -hypothesis and present a demographically based, mechanistic explanation of population cycles. Our goal is to advance a testable demographic mechanism of population cycles that should lead to the ascertainment of environmental factors or ecologic, physiologic, or social processes that underlie changes in key demographic variables.

#### BIOLOGY OF A POPULATION CYCLE

Because phase-specific changes in age at maturity and juvenile survival have been established as necessary and sufficient for population cycles (Oli and Dobson 1999), we proceed to explain how changes in these demographic variables might occur and how these changes could cause cyclic fluctuations in abundance (Fig. 1). We recognize 4 broad phases of a population cycle. In the peak population phase, density is high and competition among individuals for resources, which may be of low quality or in short supply (Seldal et al. 1994), increases. Individuals respond to crowding, resource shortages, and competition for resources by altering spacing, aggression, and social dominance (Boonstra and Boag 1992; Christian 1978, 1980; Krebs 1985). These can be referred to as behavioral responses to a stressful environment. Predators, if present, may exhibit numeric or functional responses to the high density of prey (O’Donoghue et al. 1997). These ecologic and social environmental factors may act directly to influence demographic variables, or indirectly act as nonspecific stressors, and trigger a stress response (Boonstra et al. 1998; Christian 1980). The primary stress response is increased hypothalamus-pituitary-adrenal activity, which is related inversely to hypothalamus-pituitary-gonadal activity (Christian and Davis 1964, 1966; Handa et al. 1994; Saplosky 1992).

Reproduction is suppressed because of a high population density (Boonstra and Rodd 1983; Christian 1971, Gilbert and Krebs 1991; Wiger 1979), stress response (Christian 1978, 1980; Davis 1978), risk of predation (Boonstra et al. 1998; Mappes

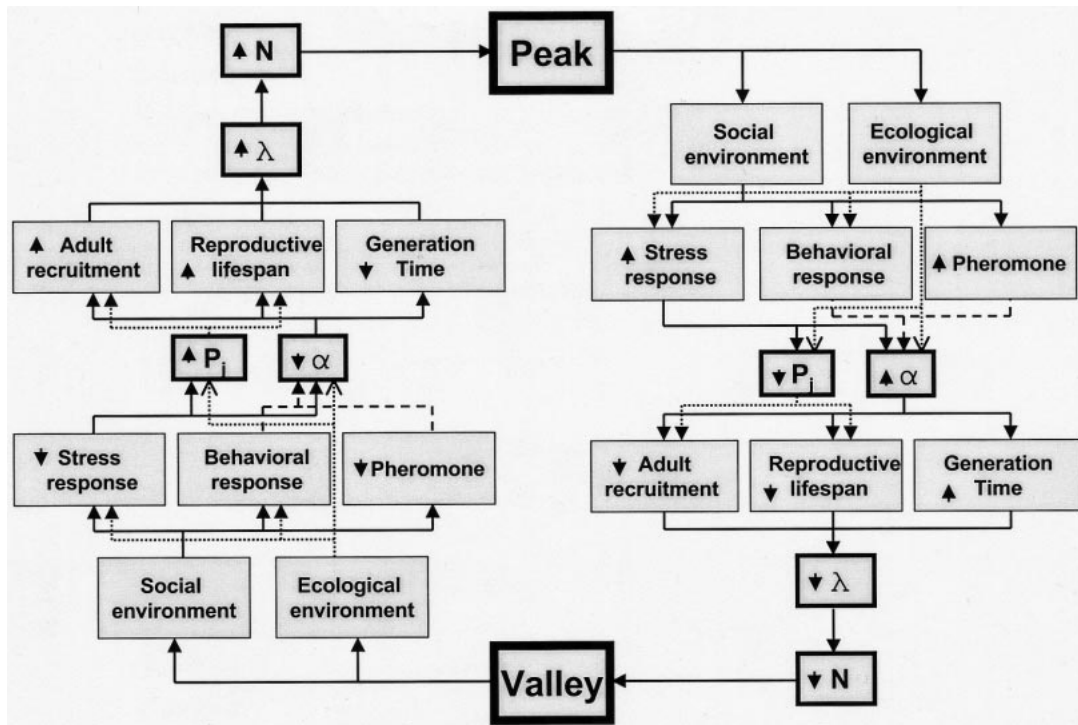


FIG. 1.—Diagrammatic representation of the proposed model of population cycles.  $\alpha$ ,  $P_j$ , and  $\lambda$  refer to age at maturity, juvenile survival, and population growth rate, respectively.

and Ylönen 1997; Ylönen and Ronkainen 1994), malnutrition (Andreassen and Ims 1990), or the action of puberty-delaying pheromones (Kruczek et al. 1989; Lepri and Vandenberg 1986; Vandenberg 1987, 1988, 1994). Juvenile survival may decrease because of a stress response (Boonstra et al. 1998; Christian and LeMunyan 1958), shortage of food or malnutrition, increased predation, diseases manifested as a result of immunosuppression caused by stress response (Boonstra et al. 1998; Moshkin et al. 1998), and stress-related illnesses (Christian 1978, 1980; Saplosky 1992). We expect adults to be better suited than juveniles to cope with stressful environmental conditions (Moshkin et al. 1998). Thus, decline in survival or fertility of adults should not be as dramatic as those in juveniles (Keith 1990; Keller and Krebs 1970; Krebs et al. 1986). Because of delayed sexual maturity and low juvenile survival, fewer females survive to reproduce,

generation time is increased, reproductive life span is shortened (Getz et al. 1997; Stenseth et al. 1985), and the mean age of reproductive individuals increases (Boonstra 1994; Tkadlec and Zejda 1998a). Consequently, decline in populations becomes inevitable.

The changes described for the peak phase continue during the early decline phase, and the downtrend in population size should accelerate. Toward the end of this phase, quality of the social and ecologic environment begins to improve. Behavioral and physiologic responses to the stressful environment should thus decline. Consequently, population parameters, primarily age at maturity and juvenile survival, begin to improve. The population decline may continue, however, until animals born, raised, or conceived during more stressful environments gradually are replaced by those born under improved conditions (see the following discussion).

When the population enters the low (or trough) phase, density is lowest in the cycle, resources are recovered, and most of the animals raised, born, or conceived during stressful environments have been replaced by those conceived or born in an improved environment. Because of these improvements, age at maturity declines, juvenile survival increases, and mean age of reproductive individuals decreases. Generation time should decrease, and reproductive life span and recruitment of adults should increase, leading to increase in population size.

Trends in population and environmental characteristics described for the low phase continue until the middle of the increase phase. By this time, population density would have increased, resources begin to deplete, and competition for resources, as well as adverse social interactions, increase. Consequently, physiologic and behavioral responses to stressful environmental conditions increase. Population-level response to deteriorating environmental conditions will begin to manifest in a manner similar to those described for the peak phase. Nevertheless, only the more vulnerable segment of the population will be affected adversely at the beginning of these changes, and the population continues to grow until the population enters the peak phase, and the cycle is repeated.

Changes in population parameters lag behind changes in quality of ecologic and social environments, and changes in population size lag behind changes in population parameters (Cary and Keith 1978); improvements in quality of environment should not be followed immediately by rapid increase in population size. Thus, animals at the end of the decline phase respond not only to quality of environment at that time but also to the environment of the peak or early decline phase.

We suggest 4 possible mechanisms for a delayed response of demographic parameters to changes in quality of social and ecologic environments. First, pre- and postnatal

stress has been shown to have long-term, irreversible consequences on behavior and reproductive performance of the offspring (Drickamer and Mikesic 1990; Hansson 1989; Henry et al. 1994; Mihok and Boonstra 1992; Pollard 1986; Ward and Read 1985). Individuals that were born, raised, or conceived during peak and early decline phases will continue to exhibit physiologic and behavioral responses to the stressful environment within which they were born and developed. It also takes time for individuals that had experienced a stressful environment pre- or postpartum to be replaced by those conceived and born in an improved (less stressful) environment. Second, environmentally mediated maternal effects, which are expressed within the context of the quality of the current environment, can introduce time lags in the demographic responses to changes in environmental quality (Boonstra and Hochachka 1997; Hansson 1989; Rossiter 1996). Third, predator responses to decline in density of prey usually lags behind decline in density of prey (Hörnfeldt et al. 1990). Thus, even if prey density were low, predator density would still be high enough to act as an environmental stressor. Finally, it takes time for resources to be replenished if they had been depleted at high-density phases, and this can introduce a time lag.

#### ASSUMPTION

The  $\alpha$ -hypothesis is based on the assumption that age at maturity shows phase-related changes during a cycle. Results of several studies (e.g., *Microtus*—Boonstra 1989; Christian 1971; Keller and Krebs 1970; Krebs 1966; Krebs et al. 1969; Lidicker 1979; Myllymäki 1977; Schaffer and Tamarin 1973; Tamarin 1977a, 1977b; *Clethrionomys*—Gilbert et al. 1986; Gilbert and Krebs 1991; Gustafsson et al. 1983; Löfgren 1989; Nakata 1989; Saitoh 1981; Tkadlec and Zejda 1998a; *Lemmus*—Krebs 1964; Pitelka 1973) suggested that age at maturity in cyclic populations shows density-dependent, phase-related changes, with

early maturity during the low-increase phase and a substantially delayed maturity during the peak-decline phase of a cycle (cf. Oli 1999b; Oli and Dobson 1999).

Factors that can influence sexual maturation in cyclic populations of small mammals are reviewed in Oli and Dobson (1999). We point out, however, that the  $\alpha$ -hypothesis does not rely on any specific factor or mechanism of reproductive suppression, and as long as age at maturity shows phase-related changes, the  $\alpha$ -hypothesis remains valid regardless of the mechanisms involved. It is possible that several factors may act synergistically to engender changes in age at maturity and juvenile survival, which then cause cyclic fluctuations in abundance (Lidicker 1978, 1988, 1991; Hansson 1998). As Krebs (1996) has pointed out, species-specific or habitat-specific differences in ecology are to be expected, and our goal is to understand the underlying generality.

#### PREDICTIONS

If the previous assumption (that age at maturity shows density-dependent, phase-related changes during a cycle) holds, 4 predictions directly follow from the  $\alpha$ -hypothesis:

*Prediction 1.*—Among demographic variables, changes in age at maturity (and juvenile survival) should contribute the most to changes in the population growth rate as the population proceeds from 1 phase of the cycle to the next. Changes in population size are a consequence of changes in the growth rate of populations and initial population sizes, which in turn are determined by changes in demographic variables. As a population proceeds from 1 phase of the cycle to the next, population growth rate also changes, and changes in population growth rate results from changes in demographic variables. If a demographic variable contributes most to changes in population growth rate as a population moves from 1 phase of the cycle to the next, that variable

can be characterized as the primary demographic cause of this cyclic shift.

Using life-history data for *M. pennsylvanicus* and a demographic model, Oli and Dobson (1999) showed that phase-specific changes in age at maturity, abetted secondarily by changes in juvenile survival, can cause cyclic fluctuations in abundance with amplitudes similar to those observed in natural populations. Recently, Oli (1999a) investigated the pattern of relative importance of life-history variables to population growth rate for 127 natural populations of mammals. His results showed that growth rate of populations characterized by early maturity and high fertility rates (the type of life history commonly found in the increase phase of a cyclic population) is most sensitive to changes in age at maturity. In populations characterized by delayed maturity and moderate to high fertility rates (the type of life history commonly found in the peak-decline phase of cyclic populations), population growth rate is most sensitive to changes in juvenile survival and age at maturity (Oli 1999a). Although these results suggest that age at maturity and juvenile survival rates are the 2 most influential life-history variables in cyclic populations, they do not constitute a formal test of the prediction.

A conclusive test of prediction 1 requires some way of partitioning contributions of various demographic variables to changes in population growth rate as the population proceeds from 1 phase of the cycle to the next. We recommend a life-table response experiment (LTRE) analysis using phase-specific demographic data (Caswell 1989a, 1989b; Levin et al. 1996; Oli et al., in press). Because quality of the environment changes as a population proceeds from 1 phase of the cycle to the next, phases of a cycle can be considered as a treatment. Then LTRE analysis can be used to decompose the total change in population growth rate (as a population proceeds from 1 phase of the cycle to next) into contributions from different demographic variables (Caswell

1989a, 1989b; Levin et al. 1996). If LTRE analysis revealed that age at maturity (and juvenile survival) contributes the most to phase-specific changes in the population growth rate, prediction 1 would be supported.

*Prediction 2.*—Sensitivity of age at maturity (and juvenile survival) to changes in quality of the environment should be greater in cyclic populations than that in noncyclic populations of the same species. Why do some populations of a species cycle, whereas others do not? The  $\alpha$ -hypothesis predicts that cyclic and noncyclic populations of a species differ in sensitivity of age at maturity (and, to a lesser extent, juvenile survival) to changes in quality of environment. Thus, age at maturity (or, equivalently, maturation rate) in cyclic populations is predicted to be more sensitive to changes in the environment than that in conspecific noncyclic populations.

We are aware of 2 studies that have provided support for this prediction (Gustafsson et al. 1983; Heikkilä et al. 1993). Gustafsson et al. (1983) studied sensitivity of sexual maturation of young bank voles (*Clethrionomys glareolus*) collected from cyclic and noncyclic populations to changes in population density. Their results showed that sexual maturation of young voles collected from cyclic populations was substantially delayed in animals kept in high density compared with those kept in low density. Population density had no effect on maturation rate of young voles collected from noncyclic populations. Heikkilä et al. (1993) studied the effect of predation risk (an environmental stressor—Boonstra et al. 1998) on maturation rate of cyclic and noncyclic populations of bank voles and reported similar results. Population density and predation risks are just 2 of several factors that determine quality of environment in natural populations, and the combined effect of multiple environmental factors on differences in sensitivity of age at maturity between cyclic and noncyclic populations could be substantial.

As a corollary to this prediction, we suggest that amplitude of cycles is determined primarily by sensitivity of age at maturity to changes in quality of environment. High sensitivity of maturation rates (and juvenile survival) to changes in environmental quality will cause wide fluctuations in population size if quality of the environment changes sufficiently. Although there is sufficient evidence to suggest that maturation rate of individuals from cyclic populations will be much more sensitive to changes in environmental quality compared with conspecific individuals from noncyclic populations (Gustafsson et al. 1983; Hansson 1990; Heikkilä et al. 1993), no study has specifically tested this idea.

*Prediction 3.*—In cyclic populations, between-year variation in age at maturity (and juvenile survival) during a cycle should be greater than within-year variation in these variables; this pattern should not exist in annually fluctuating populations. Even within a geographic region or a climatic regime, some populations of small mammals exhibit multiannual cycles, whereas others do not (e.g., *Microtus californicus*—Lidicker 1988; Lidicker and Ostfeld 1991). The  $\alpha$ -hypothesis predicts that, in cyclic populations, between-year variation in age at maturity (and juvenile survival) during a cycle should be greater than within-year variation and that such a pattern should not exist in populations that do not undergo multiannual fluctuations in abundance. We are not aware of any study that has tested this prediction.

*Prediction 4.*—Age at sexual maturity is delayed at peak or decline phase compared with low or increase phase. Because of the shorter nonreproductive period in low-increase phase, more animals survive to reproduce, which would result in a greater proportion of juveniles compared with peak-decline phase. Thus, the proportion of juveniles should be greater at low-increase phase compared to peak-decline phase. This prediction also is made by the senescence hypothesis (Boonstra 1994) and senes-

cence-seasonality hypothesis (Tkadlec and Zejda 1998a, 1998b). Through an extensive review of the literature, these studies have shown that this prediction holds for most cyclic populations (Boonstra 1994; Tkadlec and Zejda 1998a, 1998b).

#### CONCLUSION

Population cycles are demographic processes, and an explanation of population cycles necessitates an understanding of demographic mechanisms that underlie cyclic fluctuations in abundance. The  $\alpha$ -hypothesis identifies the demographic basis of population cycles and provides a mechanistic explanation of how changes in the key demographic variables might cause cyclic fluctuations in abundance and biologic attributes of the cycles. It provides testable predictions and is consistent with the empirical pattern of demographic, life-history, and behavioral changes associated with population cycles. The  $\alpha$ -hypothesis also is consistent with a recently proposed explanation of the Chitty effect (Oli 1999b) and the prediction of life-history theory that age at maturity should have a greater influence on the population growth rate than any other life-history variable in species with high reproductive rates (Cole 1954; Lewontin 1965; Tkadlec and Zejda 1998a, 1998b), a prediction that has been empirically supported in some taxa (Levin et al. 1996; Oli 1999a). We note, however, that the  $\alpha$ -hypothesis in itself is not a complete explanation of population cycles. Instead, it is the 1st of the 2 steps (see the previous discussion) necessary for a complete understanding of cyclic fluctuations in abundance. Although we have discussed possible mechanisms of changes in age at maturity (Oli and Dobson 1999), causes of phase-related, density-dependent changes in key demographic variables remain to be ascertained.

Predictions of the hypothesis must be subjected to rigorous tests before validity of the hypothesis as a demographic explanation of population cycles can be determined. If the  $\alpha$ -hypothesis is further corroborated as the plausible demographic

mechanism of population cycles, future research should focus on empirically determining causes of phase-related, density-dependent changes in age at maturity and juvenile survival. This should lead to a more complete understanding of population cycles, a problem that has puzzled ecologists for decades.

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