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Rodent population cycles: life history adjustments to age-specific dispersal strategies and intrinsic time lags

Douglas W. Morris
Department of Biology, Memorial University of Newfoundland, St. John's, Newfoundland, A1B 3X9, Canada

Summary. Multi-annual population cycles can be generated by life history responses to density dependent changes in adult and pre-reproductive survival. The proximate mechanism linking population dynamics and demography of cycling rodents appears to be high pre-reproductive dispersal at peak density, or during periods of population increase. This model is similar to the Chitty hypothesis which can best be viewed as a special case of demographic control on population size. Normally, this control should be self-reinforcing and lead to damped oscillations toward a stable population density. Intrinsic time lags induced by variation in the length of the breeding season modify the dependence of demography on population size, and enable the cycles to persist.

Introduction

The high densities of lemmings and voles during population peaks are overshadowed only by the number and diversity of hypotheses to explain the cyclical or periodical nature of their population dynamics. The list of respected hypotheses includes the effect of time lags on population dynamics (eg. May 1981), social stress (Christian 1950), nutrient cycling (Pitelka 1964), plant toxicity (Freeland 1974), coevolution with primary consumers and phenology of resources (Rosenzweig and Abramsky 1980), and a variety of cyclical selection models based on life histories (Schaffer and Tamarin 1973; Stenseth 1978; Gaines et al. 1979a), kin selection (Charnov and Finerty 1980), and selective variation in the quality of individuals, usually mediated by behavior (the Chitty hypothesis, Chitty 1958, 1960, 1967). Of these alternatives, the Chitty hypothesis has received the most attention from field biologists (eg. Krebs 1978), but the alternatives need not be mutually exclusive. The Chitty hypothesis bears strong resemblance to the life history models in that both involve cyclical variation of the genetic quality of individuals in ecological time. As currently structured, both models also seem to require environmental variation to achieve cyclical dynamics. In the absence of perturbation, life history models predict damped oscillations of population size through time (Schaffer and Tamarin 1973). Similarly, a recent explicit analytical model of the Chitty hypothesis predicts the absence of cycles except with outside interference (Stenseth 1981). If outside interference is necessary to maintain cycles, and if populations of animals like lemmings and voles really do cycle, then their cyclical dynamics may be nothing more than responses to environmental variation, and not interesting examples of self-regulation of abundance. In this paper I show that regular cycles due solely to cyclical selection for alternative genotypes are consistent with what we know of rodent populations undergoing cycles. I also show graphically, that the Chitty hypothesis can be considered a special case of the influence of life histories on self-regulation. This decreases the number of hypotheses explaining cyclical population dynamics, and demontrates that the evolution of life history traits in response to demography may fulfill the function of self-regulation of abundance for a wide variety of biological species.

The chitty hypothesis

According to Chitty, genetically controlled spacing behavior is capable of regulating population density, and population gene pools contain a spectrum of genotypes adapted to different densities. These genotypes range from docile individuals with high reproductive capacity, to aggressive individuals with lower reproductive potential, but whose behavior is adaptive to high levels of conspecific interference. As population density rises, the aggressive genotypes increase in frequency by inhibiting survival and reproduction, and by increasing dispersal, of the docile forms. Escalating aggression with increasing density eventually leads to social breakdown, reduced recruitment and extensive mortality of aggressors. As the population crashes, the docile individuals increase in frequency, and their combined reproduction leads to increased density and perpetuation of the cycle (Fig. 1). Stationary self-regulated populations are postulated to have a balanced polymorphism of the alternative genotypes.

Similarity to life history models

The alternatives in life history performance of Chitty's competing genotypes bear a striking resemblance to the predictions of life history theory based on ratios of adult (P: probability of survival till the next reproductive episode) to pre-reproductive (J: probability of survival to age of first reproduction) survival. In iteroparous species, when P/J is small, selection favors early copious reproduction (m_x genotypes) because the probability of future reproductions is low relative to the probability of breeding once (Schaffer 1974; Stearns 1976; Horn 1978). An increase in P/J leads

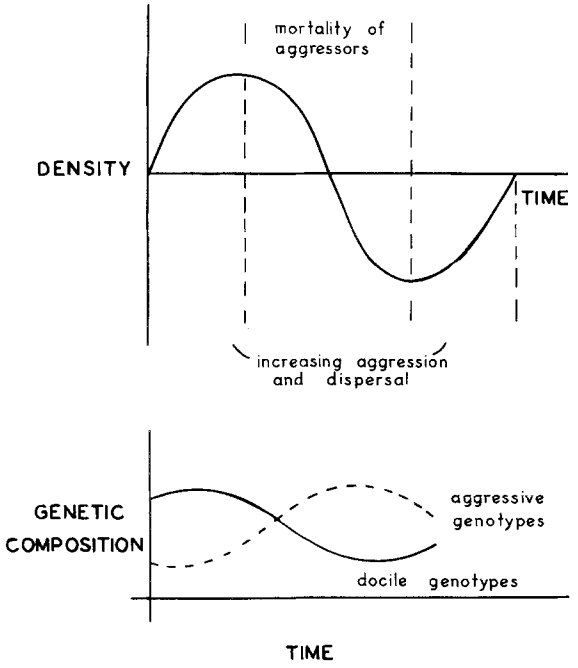

Fig. 1. My interpretation of the Chitty hypothesis. *Top:* The selective environment with changes in population density. *Bottom:* The outcome of selection in terms of the proportion of aggressive and docile genotypes. The time scale is the same in both parts of the figure, but rates of change may differ from those I have presented

to the converse of delayed iterated reproductions with reduced reproductive effort (l_x genotypes). The m_x genotypes correspond to Chitty's docile forms, and the l_x genotypes represent Chitty's aggressors. The advantage of the life history approach is that it leads to a host of readily testable predictions concerning adaptive life history traits over a range of P/J. In addition to age-specific patterns of reproduction, correlates of reproductive effort in small mammals would include gradients of many to few young per litter, small to large ratios of female body mass to the combined mass of neonates and large to small investment in lactation (Table 1).

If it can be shown that the ratio of adult to pre-reproductive survival changes predictably during the course of population cycles, we could have a rigorous and testable life historical mechanism to explain the dynamics of cycles. Krebs and Myers (1974) have chronicled mortality rates in response to changes in population density by small mammals. Both adult and juvenile mortality are typically greater in the decline phase of cycles than during increase. More recent studies on unenclosed populations have tended to confirm increased mortality of all age classes during population declines (eg., Gaines and Rose 1976; Tamarin 1977).

Demographers do not report P/J ratios, but several microtine studies do list 14-day survival rates for different age-classes, as well as an index of juvenile recruitment. A weak test of the theory would be to contrast ratios of adult 14-day survival rates to juvenile recruitment for different phases of a well-described cycle of abundance. One of the best documented demographic studies of a well-known cycling species is that by Gaines and Rose (1976) on the prairie vole, *Microtus ochrogaster*. I contrasted ratios of adult female survival (their Table 5) to early juvenile survival (their Table 6) with my interpretation of cycle phase

Table 1. Possible alternative mammalian life history traits predicted from ratios of adult to pre-reproductive survival

P/J small	Trait	P/J large
Decreases	Age to maturity	Increases
Decreases	Adult body size	Increases
Decreases	Number of reproductions	Increases
May increase	Litter size	May decrease
Likely decreases	Size of young	Likely increases
Decreases	Female mass/neonate mass	Increases
Increases	Energy to lactation	Decreases
Increases	Growth rate of young	Decreases

Table 2. Ratios of adult female survival (f) to early juvenile survival (j) for the prairie vole *Microtus ochrogaster* (data from Gaines and Rose 1976)

Season	Grid A		Grid B	
	Cycle phase	f/j	Cycle phase	f/j
Winter 1970–71	Low	0.77	Decline	0.60
Summer 1971	Low	0.84	Low	0.79
Winter 1971–72	Increase	0.53	Increase	0.71
Summer 1972	Peak	0.74	Peak	0.92
Winter 1972–73	Peak	0.83	Peak	0.90
Summer 1973	Decline	1.52	Decline	0.70
	Grid C		Grid D	
	Cycle phase	f/j	Cycle phase	f/j
Winter 1970–71			Decline	0.79
Summer 1971	Low	1.04[a]	Increase	0.59
Winter 1971–72	Increase	0.62	Peak	0.39
Summer 1972	Decline	0.97	Peak	0.55[b]
Winter 1972–73	Low	0.54	Low	0.41
Summer 1973	Low	0.28	Decline	1.02

[a] Omitted from the 2nd analysis
[b] Converted to decline for the 3rd analysis

Table 3. Kruskal-Wallis one-way analysis of variance of the ratio of adult female to early juvenile survival by cycle phase

Cycle phase	Sample size	Mean rank
Low	7	10.79
Increase	4	7.75
Peak	6	12.17
Decline	6	16.08

$\chi^2 = 3.98$; $p = 0.26$

(their Figs. 1–4) across all four grids in Gaines and Rose's (1976) study (Table 2).

Ratios of adult female to early juvenile survival were extremely variable. These data must suggest, even to a pessimist, the potential for demographic feedback on population dynamics. For these dynamics to produce cycles, however, the feedback mechanism should be consistent with cycle phase. I searched for this effect by non-parametric analysis of variance (Table 3). There were no consistent differences between the ratio of adult female to early juvenile survival with cycle phase ($\chi^2 = 3.98$; $p = 0.26$) even

though there was a trend for differences between increase and decline (Mann-Whitney $U=21$; $p=0.05$). I repeated the analysis by eliminating or modifying questionable interpretations of cycle phase (Table 3), and generated comparable results. Similarly, there was no relation between the ratio of adult female to early juvenile survival and population density (July and January; interpreted from Gaines and Rose's Figs. 1–4; $\tau=-0.03$; $p=0.43$).

There are at least four problems in using this analysis to test the theory. First, I am assuming early juvenile survival is a good estimáte of the probability of breeding at least once. Second, I assume that all adults are equivalent because I am unable to include age or parity effects in the calculation of adult survival. Third, the data are based on seasonal averages, over which time more than one population response may be occurring. Fourth, I am assuming that I can classify vole population fluctuations into unbiased categories of population dynamics (though this is not a problem with the correlation analysis).

All of these limitations probably apply in different degrees to all published studies of microtine demography. Whereas the definitive test should include juvenile recruitment rates as breeding adults and data on the relative age-distribution of adults for much shorter intervals of time, it may not solve the riddle of cyclical population dynamics. The analyses here point to great variation in ratios of adult to early juvenile survival which must certainly influence population growth rates, but the changes in the survival ratio do not appear to be predictable during different phases of the cycle. This degree of variation in survival ratios should lead to large fluctuations in abundance, but not cycles. If life history compensations cause population cycles, some additional factor other than age-specific mortality must be involved. One often proposed candidate is selective dispersal (eg., Krebs et al. 1969, 1973; Myers and Krebs 1971; Lidicker 1975; Tamarin 1978; Gaines et al. 1979b; Beacham 1981).

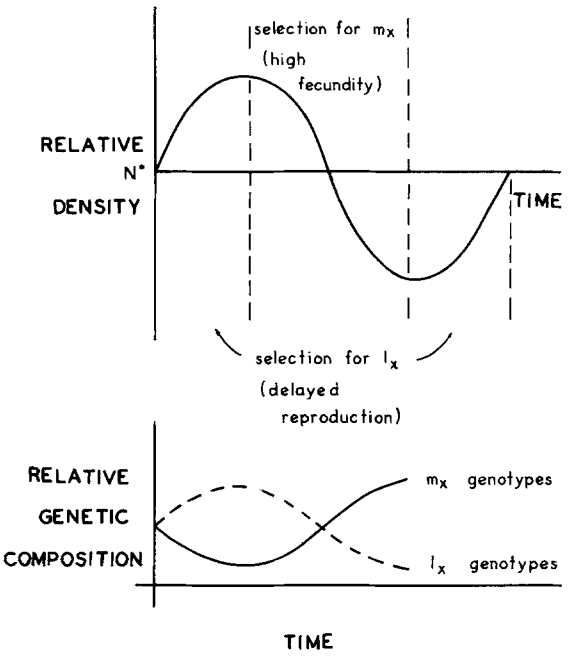

Fig. 2. A life history analogue to the Chitty hypothesis. *Top:* The selective environment with changes in population density. *Bottom:* The outcome of selection in terms of the proportion of l_x and m_x individuals. Compare this figure with Fig. 1 to see similarities and differences between the Chitty hypothesis and the life history model. Relative time scales are the same in both figures. The Chitty hypothesis can be viewed as a special case of the more general life history model because density dependent behavior acts as the proximate mechanism controlling changes in reproduction and survival. The actual time course of selection will depend upon the relation of adult/(pre-reproductive survival to population density and population growth rate

Does age-specific dispersal generate cycles?

The effect of emigration on population gene pools is equivalent to mortality because emigrating genotypes are lost from the population's "genetic memory". Thus, consistent trends in the pattern of age-specific emigration could modify P/J and induce cyclical dynamics. The most complete synopsis of dispersal patterns by small mammals has been compiled by Gaines and McClenaghan (1980). In their review, all significant correlations between dispersal rate and population density were positive, that is, "more dispersal occurs in increasing than in declining populations". Second, the number of dispersers is "positively associated with the rate of population increase". Third, in nine of 14 comparisons of ten cricetid species (seven microtines), the average age of dispersers was less than residents, and never greater (Gaines and McClenaghan 1980; their Table 3). If we assume on average that half of the instances where dispersers were of a different age than residents, they should have been older, this is a highly significant difference (one-tailed sign test, $p<0.005$; Sokal and Rohlf 1981; p 449–450). Furthermore, dispersers in these studies correspond to animals arriving on removal plots from control populations, and they classify as emigrants. To summarize, most emigrants are young animals leaving expanding or peak populations; pre-reproductive survival in the gene pool is reduced. During population increase and in populations at high density, the effective P/J ratio may be large relative to its value in populations declining from their maximum size. The predictable variation in P/J caused by density dependent and age-specific dispersal leads to cyclical selection for l_x and m_x genotypes, which in turn drive the cyclical dynamics of oscillating populations (Fig. 2). This selection is reinforced because dispersers appear to be nonrandom age and reproductive samples of the resident population (Gaines et al. 1979b, c;, Beacham 1979, 1980, 1981). Beacham (1981) in particular has documented that a greater proportion of sub-adult dispersers are in reproductive condition than in the population as a whole. Thus, age-specific and density dependent dispersal not only changes the selective environment on life histories by modifying P/J, it also acts to increase the proportion of l_x phenotypes by the selective emigration of those with high fecundity.

There are two caveats against the importance of dispersal and age-specific survival in population regulation. First, if dispersal genotypes are lost at every episode of population increase, the population of dispersing individuals should drop through time, and bring about a damping of population fluctuations. This would not occur if patterns of age-specific dispersal were simply phenotypic responses to changes in population density. Even though I have phrased the model in terms of genetic changes, identical population performance could arise by adaptive phenotypic plasticity in response to density. There are no *prima facie* reasons

for imposing a genetic model on life history traits in variable environments. Life history models predict which strategies of age-specific reproductive expenditure are optimal in given specified or environmental contexts. Whether genetic or phenotypic responses occur depends on how individual species partition the degree of genetic versus phenotypic control over life histories.

A more damaging criticism of life history control of self-regulating population cycles comes from the stability analyses of Schaffer and Tamarin (1973). In the absence of time lags and periodic environmental variation, their life history model predicted damped oscillations through time. Schaffer and Tamarin (1973) ignored time lags associated with consumer-resource interactions because of a lack of evidence supporting these interactions in microtine population regulation. Intrinsic reproductive time lags associated with, for example, finite gestation, may help generate population fluctuations (Schaffer and Tamarin 1973), but there should be strong selective pressures favoring individuals which respond to cues tending to eliminate these fixed effects (ie. animals should respond to densities suitably discounted for the built-in lag in reproduction). They are unlikely to be able to compensate for time lags induced by seasonal breeding.

Consider a seasonal environment in which a population ceases breeding during the increase or peak phase of the cycle. Pre-reproductive mortality will be increased, not because of reduction in survival rate, but because the pre-reproductive interval has been artificially increased by the cessation of breeding. Fewer individuals may die per unit time, but the proportion of animals dying before they are capable of successfully breeding increases. Similarly, first breeders which die before spring will tend to decrease the ratio P/J because they will have bred once and not again. Reduced survival of multiparous individuals will affect P/J similarly.

We can visualize these effects more clearly by considering a typical vole. Assume that age at maturity is six weeks, and litter spacing of adult females with post-partum estrus is four weeks. Further, assume that the winter non-breeding season is longer than the age at maturity. First, consider juvenile summer survival per two weeks to be 0.6, and similarly, adult summer survival to be 0.8 per two-weeks. Then, taking two equal sized cohorts (N), the probability of a juvenile surviving to reproductive age is $((N \times 0.6) \times 0.6) \times 0.6/N = 0.22$, whereas the probability of an adult animal living to breed again is $(N \times 0.8) \times 0.8/N = 0.64$; $P/J = 2.96$. Next, assume $p = j < 1$, where p and j are two-week survival rates for adults and pre-adults respectively. Then the effective ratio of adult to pre-reproductive survival would be $(N \times p) \times p/((N \times j) \times j) \times j = 1/j > 1$. Even if survival rates per unit time are identical, the effective breeding season survival of adults is always greater than that of juveniles whenever the inter-litter interval is less than the age to maturity. Now let's look at over-winter survival. Neither adults nor juveniles can breed again until spring, so that the inter-litter interval is identical to the age of maturity for voles which have not yet reproduced. Unless juvenile winter survival is substantially less than that for adults, cessation of breeding in organisms where the inter-litter interval is typically less than the age at maturity decreases the effective ratio of adult to pre-reproductive survival. In this example, P/J (summer) > 1; P/J (winter) $= 1$. More realistic models would incorporate detailed age-specific survival data, but their results would remain qualitatively the same. Seasonal breeding in multi-voltine species will effect seasonal differences in P/J.

Seasonal breeding in voles will tend to reduce P/J, and spring cohorts should consist of individuals placing more emphasis on current reproduction than on future survival. This reasoning may also provide a phenomenological explanation for the "spring decline" in vole populations. Seasonal breeding may represent the environmental "kick" required to explain the long-term persistence of population cycles by earlier life history models. The effect of seasonal breeding on life history feedback on population dynamics depends upon the initial ratio of P/J. In increasing and peak populations where age-specific dispersal tends to reduce the effective adult/pre-reproductive survival ratio, seasonal breeding will reduce it even further, readjust the relative importance of l_x and m_x individuals, and cause a higher peak density than would have been expected otherwise (because of overcompensation towards m_x individuals). Seasonal cessation of breeding will have related effects throughout the cycle. Seasonally-induced time lags are capable of destroying the normal population damping of self-reinforcing life history traits.

Seasonal time lags also occur simply as a function of reduced population size. In voles, the absence of winter reproduction leads to reduced spring populations. For populations in the increase phase of the multi-annual cycle, this should tend to increase the relative advantage of m_x individuals because the negative reinforcement of increasing density on recruitment has been reduced. This effect would be magnified by spring flushes of nutrients, and by alterations in P/J. Alternatively, in those years when the voles breed year-round, density dependent feedback on the relative values of l_x versus m_x individuals will occur more quickly than on average. Variation in the duration of the breeding season modifies the selective environment in terms of response time to changes in adult and pre-reproductive survival, and contributes to the persistence of cycles. It is interesting that winter breeding is most often reported only in increasing populations (Krebs and Myers 1974). Winter breeding may be manifested in part, by strong selective advantage to individuals maximizing current fecundity in increasing populations.

Discussion

Life history tactics have been less than convincing mechanisms of population self-regulation because life history models were incapable of explaining population cycles of northern herbivores as intrinsic outcomes of demographic structure. Two of the limitations of earlier models were incomplete explanations for variable pre-reproductive survival (Gaines et al. 1979a; my test of adult female/early juvenile survival ratios), and the need for external environmental heterogeneity to provide occasional "kicks" to the normally self-damping effects of self-reinforcing life history traits. I have shown that age-specific emigration may account for variable pre-reproductive survival, and that time-lags induced by seasonal reproduction (or natural variation in the length of the breeding season) may destroy the damping of population numbers through time. These represent proximate mechanisms causing cycles; the interaction between life histories and demography represent ultimate con-

trols on population regulation. Even so, should not all seasonally reproducing multi-voltine small mammals exhibit multi-annual cycles of population density, especially because models of age-specific dispersal strategies predict that most successful dispersers will be young animals (Morris 1982)? I suspect that the answer lies in the magnitude of dispersal by cyclic species. Individuals should disperse only when their expected lifetime reproductive success is greater by doing so than by remaining in their natal population (Morris 1982). If habitats offer variable rewards in time and space, then successful dispersers may frequently improve their reproductive fitness. The question becomes, do cyclic species live in more heterogeneous habitats than non-cyclic species? At the moment I am not prepared to answer that question except to note that the proposition fits *Microtus* migrating among patches of suitable grassland, or among transient forest openings.

Rigorous tests of life history control of microtine population regulation will require detailed age-specific survival rates as functions of population density and population growth rate. In particular, we need explicit estimates of pre-reproductive survival. We also need to know how "survival" rates are modified by density dependent and age-specific emigration. I suggest studies comparing cycling and non-cycling rodents in terms of field estimates of adult/pre-reproductive survival. The predictions are that adult/pre-reproductive survival will be consistently related to population dynamics in cyclical species, that this consistency is in large part determined by dispersal, and that seasonally-induced time lags destroy the self-damping effects of life history control on population regulation. Survival ratios need not be consistent in non-cycling species, and seasonal and other environmental effects should be expected to modify survival rates. In both cases, however, life history theory predicts population responses to variation in demography. If possible, data should also be collected on age-specific reproductive effort as a function of population density. This will provide an additional test of life history control of population regulation (Schaffer and Tamarin 1973) and enable a thorough test of the life history theory assumption that survival rates dictate age-specific reproductive effort.

My purpose in this paper is not to deify life history models as the only explanation for population cycles. I agree with Rosenzweig and Abramsky (1980) that a synthetic view of possible alternatives will likely give the most satisfactory explanation to multi-annual population cycles. At the same time, some explanations may remove more of the "residual variation" around the phenomenon than others. The frequency, regularity and synchrony of the cycles argue for general theories. A life history explanation is promising because it includes special cases (eg. Chitty's hypothesis), because the normal self-reinforcement of life history traits also serves as a useful model of self-regulation in non-cycling species and because the dynamics of populations are inextricable outcomes of demographic strategies. Perhaps the most useful characteristic of the life-history approach is that it leads to explicit testable hypotheses. Detailed demographic analyses of cycling populations could test the assumptions about changes in P/J during the cycle and as adjustments to seasonal breeding. Information on dispersal could be used to test the implicit assumption of uni-directional migration, and also evaluate more general theories of age-specific dispersal strategies (eg. Morris 1982). These same studies could then contrast the life history performance of individuals "selected" under different regimes of P/J to corroborate the predictions of the theory.

Other hypotheses such as Rosenzweig and Abramsky's (1980) coevolutionary disequilibria or phenological misadaptation may yet prove to be better models of multi-annual cycles than those based on the evolution of life histories. Tests of assumptions and predictions will be their judge. One point is inescapable. Whatever the mechanism that controls multi-annual population cycles in northern herbivores; it leads to major changes in the demographic structure of cycling populations. Whether these changes ultimately drive the cycles or not, their influence on population dynamics must be addressed.

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