Causes and consequences of complex population dynamics in an annual plant, *Cardamine pensylvanica*

by

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8 November, 1995

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A dissertation submitted in partial fulfillment
of the requirements for the degree of Doctor of Philosophy
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ABSTRACT
(Botany)

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Abstract

The relative importance of density-dependent and density-independent factors in determining the population dynamics of plants has been widely debated with little resolution. In this thesis, I explore the effects of density-dependent population regulation on population dynamics in *Cardamine pensylvanica*, an annual plant.

In the first chapter, I show that experimental populations of *C. pensylvanica* cycled from high to low density in controlled constant-environment conditions. These cycles could not be explained by external environmental changes or simple models of direct density dependence ($N_{t+1} = f[N_t]$), but they could be explained by delayed density dependence ($N_{t+1} = f[N_t, N_{t+1}]$).

In the second chapter, I show that the difference in the stability properties of population growth models with and without delayed density dependence is due to the presence of Hopf as well as flip bifurcations from stable to chaotic population dynamics. I also measure delayed density dependence due to effects of parental density on offspring quality in *C. pensylvanica* and show that this is large enough to be the cause of the population dynamics we observed in *C. pensylvanica*.

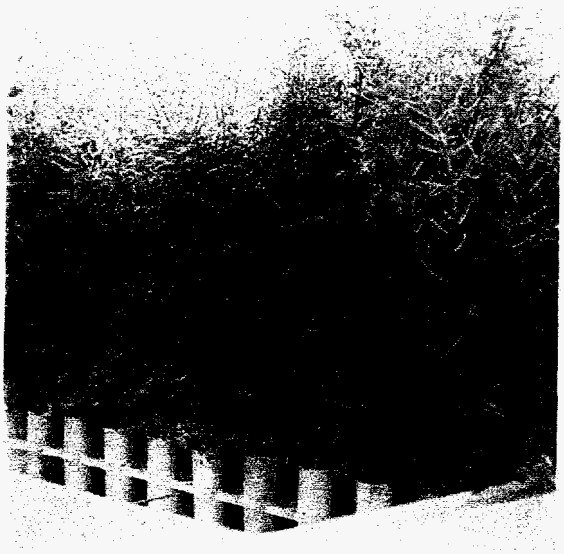
In the third chapter, I extend my analyses of density-dependent population growth models to include interactions between competing species. Interspecific and spatial environmental differences in time delay, unlike differences in growth rates and competition coefficients, allow some mixtures of species which would be stable in isolation to stably coexist.

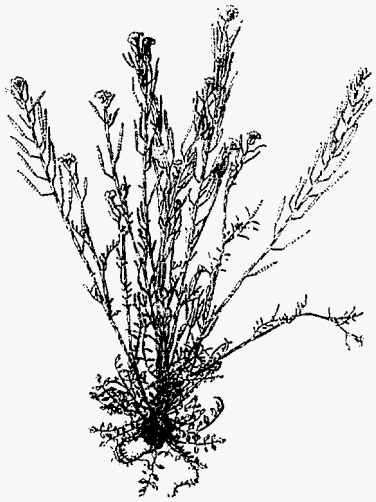
In the final chapter, I compare the effects of fixed spatial environmental variation and variation in population size (due to density-dependent cycles) on the evolutionary response of *C. pensylvanica* populations. This response was dominated by differentiation of populations within environmental treatments, suggesting that cycles in

population size accelerated the importance of random genetic drift, which then overwhelmed environmental differences.

Thus, in *C. pensylvanica,* density-dependent factors were an important component of population dynamics and evolution. Because mechanisms of population regulation that would cause delayed density dependence (parental effects, age structure, litter accumulation) may be important in many other species, such cycles may be more widespread than earlier studies had suggested.

Cardamine pensylvanica (Muhlenberg. ex Willd.) (Brassicaceae) - upper left: Sarah P. Duke Gardens; upper right: Duke University greenhouse; lower left: experimental phytotron populations; lower right: figure from Rollins (1993).





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Overview

One of the most exciting contributions of mathematical modeling to our understanding of basic ecological processes has been the discovery that very simple forms of density-dependent population regulation can lead to complex spatial and temporal patterns, even in completely homogeneous environments. However, despite their potential importance, the role of density-dependent processes in determining the distribution of real organisms has been difficult to assess. For example, in the presence of environmental heterogeneity and stochastic noise, it is difficult to separate environmentally-induced patterning from patterning due to population regulation. Furthermore, temporal patterns often take a substantial number of generations of monitoring to detect, and are therefore beyond the scale of most ecological data sets.

This dissertation grew from a study that was begun by Doug Taylor several years ago. Our goal was to monitor experimental populations of fast-growing plants in controlled conditions, and simply to observe what would happen. As an initial attempt at assessing the relative importance of demographic, genetic, and external environmental factors in determining spatial and temporal patterns, we monitored populations with different degrees of spatial environmental heterogeneity and genetic diversity.

In the first chapter of this thesis, I present the most striking result of this study. All of our experimental populations cycled from high to low density over time, with a period of about four generations. However, simple models of direct density dependence ($N_{t+1} = f[N_t]$) could not reproduce the population cycles we observed. On the other hand, models which included delayed density dependence ($N_{t+1} = f[N_t, N_{t-1}]$) were able to reproduce these cycles. This led to the study presented in the second chapter, in which I measured delayed density dependence due to effects of parent density on offspring quality. Parental density effects were not only detectable, but were large

enough to predict population cycles that were remarkably similar to our earlier observations.

In the third chapter, I highlight some of the differences between the stability of models with only direct density dependence and models with a combination of direct and delayed density dependence. Relative to non-delayed models, inclusion of delayed density dependence changes the shape of population cycles (flip *vs.* Hopf bifurcations) and decreases the range of parameters which predict stable equilibria. Differences in the importance of delayed density dependence among competing species also change the way in which demographic parameters scale to overall dynamics by allowing some pairs of species that would both exhibit cyclical or chaotic dynamics in isolation to stably coexist. Analogous conclusions hold for differences in delayed density dependence due to spatial environmental variation.

The final chapter of this thesis is a first attempt at extracting some of the effects of the genetic and environmental treatments in our long-term experiment from the overwhelming signal of the population dynamics. The results of this study highlight two interesting interactions between population dynamics and evolution. Although this study was not designed to test hypotheses about the effects of population dynamics on evolution, it appears likely that population cycles accelerated the rate of random genetic drift. Population density also appeared to be a critical determinant of the direction of evolutionary responses to the environmental treatments.

Taken together, these results show that temporal patterns due to density dependent feedback can and do matter in plant populations. Furthermore, the effects of density dependent feedback were large relative to the effects of our environmental treatments. However, the most significant result of this study is that population cycles were due to delayed density dependence. The vast majority of both theoretical and empirical studies of plant population dynamics have assumed that population

regulation is only due to direct density dependence. However, delayed density dependence is likely to be important in many plant populations, and has very different effects on population dynamics than direct density dependence. Incorporation of this into our understanding of plant population biology will undoubtedly lead to a richer and more comprehensive understanding of the dynamics of plant populations.

CHAPTER 1.

Complex dynamics in experimental populations of an annual plant, *Cardamine pensylvanica*

Abstract

To study the numerical dynamics of plant populations, twelve experimental populations of an annual greenhouse weed, *Cardamine pensylvanica*, were maintained for fifteen generations in controlled-environment growth chambers by growing plants in an array of pots and allowing seed for the next generation to disperse into an adjacent array of fresh pots. Discrete generations were enforced by harvesting mature plants after seed dispersal, but germination, recruitment, competition and dispersal occurred naturally. The numerical dynamics of the experimental populations cycled from high to low density with a period of four to five generations, as indicated by negative autocorrelations in population size at lags of two and three generations. Demographic data collected during the experiment indicate that population density affected plant growth and seed set. Independent estimates of low-density recruitment were also high enough to predict complex population dynamics from simple models of direct density dependent population regulation. However, simple population models fit to the time series data predicted stable dynamics. Similar models including time-lagged density dependence qualitatively reproduced the dynamics of the experimental populations. Delayed feedback through maternal effects or interacting herbivores or pathogens may be possible causes of the observed dynamics. This suggests that although plant population dynamics may be stabilized by direct density dependence, delayed density dependence could destabilize dynamics.

Introduction

The relationship between density-dependent population regulation and complex (cyclical or chaotic) population dynamics is one of the most widely debated issues in ecology. Ecologists have long been aware that many animal populations exhibit cyclic oscillations from high to low density over time (Elton 1942, Andrewartha and Birch 1954, Krebs 1985), and that these oscillations are similar to those generated by simple models incorporating density-dependent population regulation (Cunningham 1954, Nicholson 1954, May 1973). The potential importance of density dependence in generating complex population dynamics is also supported by several examples of experimental populations of animals which have exhibited complex dynamics under controlled conditions (Nicholson 1954, 1957, Prout and McChesney 1985, Constantino and Desharnais 1991, McCauley 1993, but see Mueller and Ayala 1981). However, the presence of density-dependent population regulation in natural populations has been notoriously difficult to demonstrate from time series data (Wolda and Dennis 1993, Hanski *et al.* 1993).

The classic analysis of Hassell *et al.* (1976) typifies the discrepancy between models of population regulation and census data. To estimate the frequency of intrinsically-generated complex dynamics in natural populations, Hassell *et al.* (1976) parameterized a discrete generation model of density-dependent population regulation with field census data for insect populations. Of the twenty-four times series analyzed, twenty-three were predicted to reach a stable equilibrium and one was predicted to have stable limit cycles. Based on these results, many ecologists have concluded that density dependence does not usually cause complex dynamics in nature (Hassell *et al.* 1976, Nisbet and Gurney 1982, Godfray *et al.* 1991). Others responded to this conclusion by noting that complex dynamics are far more likely when population regulation occurs

after a time delay of greater than one generation (Schaffer and Kot 1986, Turchin 1990, Berryman 1992). To demonstrate the potential importance of delayed regulation, Turchin and Taylor (1992) analyzed thirty-six insect and vertebrate time series using response surface methodology (Box and Draper 1987) with temporal lags. In contrast to Hassell *et al.* (1976), they found a wide range of complex dynamics, with only about half of the populations exhibiting monotonic growth toward stable equilibria. They concluded that intrinsically-generated complex dynamics are common in nature, but result from delayed rather than direct density dependence.

In contrast to animal populations, the long-term numerical dynamics of plant populations have received relatively little attention. Plant population biologists widely accept the importance of direct density-dependence in both natural and agricultural systems (Harper 1977), but density dependence in plants has been assumed to stabilize population dynamics, rather than generating cycles or chaos (Antonovics and Levin 1980, Rees and Crawley 1989, Crawley 1990, but see Molofsky 1994). Rees and Crawley suggested that the stability of plant populations comes from several fundamental differences between plants and animals (Rees and Crawley 1989, Crawley 1990): (1) plants show extreme physiological plasticity and can reproduce at very low size; thus, even under crowded conditions most individuals can reproduce; (2) plants are spatially fixed and have long dispersal distances relative to the scale of competition, so the effects of crowding at one location can be ameliorated by long-distance recruitment; (3) plant population dynamics may often be stabilized by recruitment from long-lived seed banks. Numerical dynamics in plants are therefore expected to be asymptotically stable. Nevertheless, there are a few examples of plant populations that appear to exhibit oscillatory dynamics independent of changes in the external environment (Wilkon-Michalska 1976 in Silvertown 1991, Symonides *et al.* 1986, Thrall

et al. 1989, Tilman and Wedin 1991, but see Rees and Crawley 1991). However, it is difficult to interpret these studies because extremely few studies have monitored plant population dynamics over several generations. Examples of complex dynamics in plant populations may be rare exceptions to generally stable plant population dynamics or may reflect general phenomena that cause complex dynamics in plants, but have not been incorporated into models of plant population regulation.

To address this issue, we monitored population dynamics in replicated experimental populations of a greenhouse weed, *Cardamine pensylvanica*. We first show the dynamical data and the evidence for density dependence in these populations. We then show the results of characterizing the dynamics of the populations by fitting simple lagged and non-lagged discrete generation models to the data. We also predict dynamics from independent estimates of population growth rates. Comparison of the actual, fitted and predicted dynamics highlights some possible causes of complex dynamics in plant populations as well as some problematic issues in extracting the effect of density-dependence on population dynamics from time series data of either plant or animal populations.

Methods

Species

Cardamine pensylvanica (Brassicaceae) (Muhl.) is an ephemeral weed of damp habitats (Al-Shebaz 1988) and is a widespread and noxious greenhouse weed. Greenhouse populations of *C. pensylvanica* have no specific germination or flowering requirements and are present throughout the year under a wide variety of environmental conditions. Seed from these populations can germinate within a week of reaching a suitably moist environment and individuals begin to set seed approximately two months

after germination. In the field, *C. pensylvanica* behaves as a winter annual, setting seed in the early spring before most other plants are established. The source population for this experiment was created by selecting more than 100 individuals from each of three long-established greenhouse populations: the greenhouses at Duke University (Durham, NC), North Carolina State University (Raleigh, NC), and a private greenhouse in Durham, NC. The history of the source populations were not known specifically except that each greenhouse was decades old, and in each greenhouse the staff recognized *C. pensylvanica* as a chronic pest. Thus, our controlled experimental conditions were similar to the recent experience of the populations under "natural" conditions. Fifty non-flowering plants from each source population were planted in a single flat in the Duke University greenhouse until they flowered and dehisced seed. The parent plants were then removed, and the germinated seedlings were harvested from the flats and used to initiate the population cages.

Population cage design and maintenance

The experimental populations were maintained for fifteen generations in a growth chamber in the Duke University Phytotron with a 16-hour photoperiod and a constant temperature of 27°C. Each population consisted of a number of 2.5 cm diameter by 10 cm deep tubular pots (RL-200 Conetainers™, Stuewe & Sons, Corvallis, OR) arranged in a long narrow array (2 pots x 36 pots). The populations were watered from the bottom by filling a system of interconnected tanks with dilute Hoagland's solution until the pots were saturated, then draining the system. Populations were isolated from each other within the growth chamber by clear plastic sheeting suspended from the ceiling. The location of the populations within the chamber was randomized twice each generation.

Discrete generations were enforced as follows: Populations began with three seedlings sown in half the pots (Figure 1, open circles). After establishment (one week), the surviving seedlings were thinned to one plant per pot. Before plants set seed, the remaining pots (Figure 1, slashed circles) were filled with bare soil to receive naturally dispersed seed from the original plants. After all plants had dehisced most of their seed (approximately two to three weeks after the first plants dehisced seed), the pots for the first generation were removed and replaced with bare soil. These pots received seed for the third generation plants, *etc.* The time of harvest was determined from pilot experiments which showed a distinct end to seed set approximately two weeks after the first plants set seed. After four generations the timing of seed dehiscence appeared to vary somewhat; to account for this, we harvested plants whenever the distinct wave of seed set had finished (usually about three weeks after first seed set). Because numerical dynamics were not visibly different during the first four generations than in later generations (see Results), we assume that initial population dynamics were not driven by the length of time available for recruitment (see Discussion). Generations (time from input of bare soil to the time of removal of plants in that soil) were approximately three months long; fifteen generations were completed in three years. This is similar to the life span of individuals grown under identical conditions without forced generation lengths (E. Crone, unpublished data).

The experimental populations were set up with different combinations of two environments: (1) *deep soil* - pots filled with fine vermiculite, and (2) *shallow soil* - pots half filled with fine vermiculite. Because arrays were watered from the bottom and lit from above and soil depth determined the position of plants within the pots, plants in deep soil experienced high light and low water availability, and plants in shallow soil experienced the reverse. Each population had one of four environment types: (1)

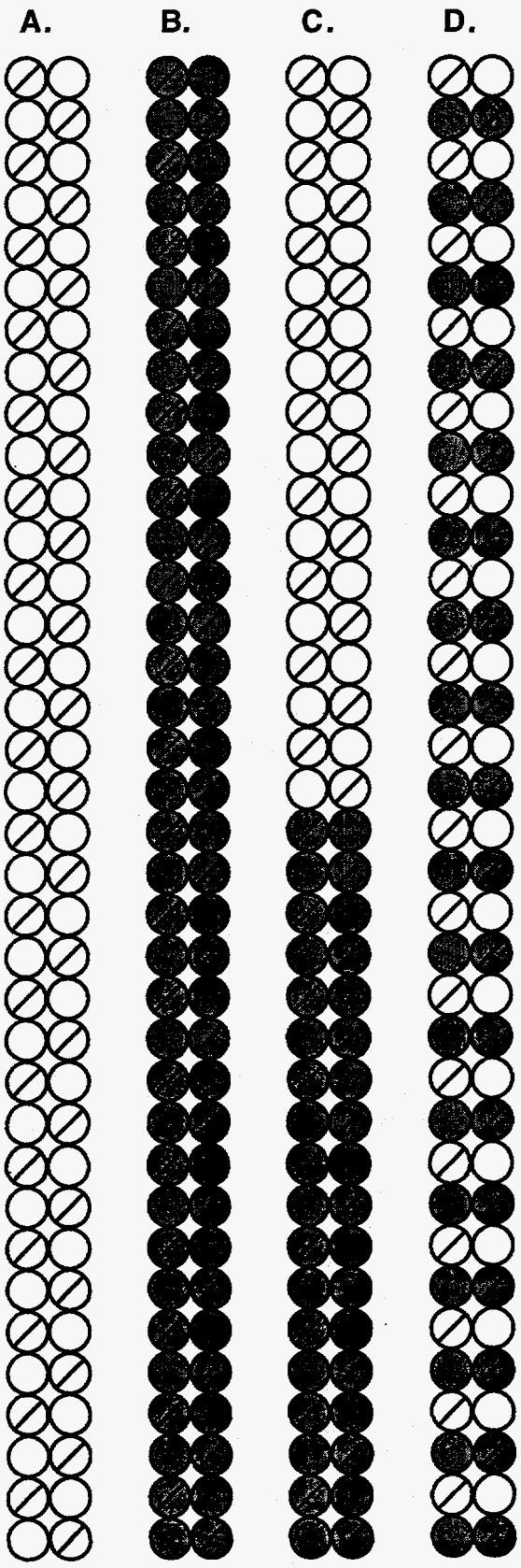

Figure 1. Diagram of the experimental setup. Pots were either filled with soil (shaded) or half-filled (unshaded). Plants in the open circles comprised generation *t*. When generation *t* plants were flowering, pots with bare soil were placed in positions shown by slashed circles. After the generation *t* plants set seed, the pots for generation *t* were removed and demographic data recorded. When the generation *t+1* plants (now in pots marked by slashed circles) flowered, positions shown by open circles were replaced with bare soil for generation *t+2*, *etc.* A.) homogeneous shallow soil, B.) homogeneous deep soil, C.) coarse-grained heterogeneous soil, D.) fine-grained heterogeneous soil.

homogeneous deep - all pots filled with deep soil, (2) *homogeneous shallow* - all pots filled with shallow soil, (3) *coarse-grained heterogeneous* - one end of the population entirely deep soil and the other end entirely shallow soil, and (4) *fine-grained heterogeneous* - deep and shallow soil in alternate pots (Figure 1). Our initial goal was to compare the dynamics of populations in above- and below-ground limited environments and to investigate the evolutionary responses to the different treatments. However, similarities across environments were much greater than differences (see Results), and in this paper we focus on these similarities. (A discussion of the differences between environmental treatments and the evolutionary responses of populations to environments will be presented elsewhere.) Although the environmental treatments were not necessary for this study, we present data from all twelve populations to emphasize general patterns. However, we only show statistical analyses of data from homogeneous environments (homogeneous deep and homogeneous shallow soil). This is because we do not know that dynamics were identical in deep and shallow soil pots, so analyses of populations in heterogeneous environments were complicated by spatial environmental heterogeneity.

At the end of each generation, demographic data were collected from the populations. For each pot in each population the number of flowering plants and the total number of plants were recorded. For the first ten generations, a random sample of 25 pots of each soil type was selected from among the entire set of pots in each generation. For each plant within those pots, height, number of siliques (seed pods) and dry biomass were recorded. Relationships between plant size and fecundity and plant density were used to test for local density-dependent responses of plants in each environment (deep or shallow soil).

The dynamics of the numbers of flowering adults and the total numbers of individuals in each population were characterized using autocorrelation functions (ACF;

Nisbet and Gurney 1982, Chatfield 1989). The population size (N) for each population (i) in each generation (t) was log transformed ($L_{i,t} = \log N_{i,t}$), and the ACF was estimated by calculating the correlation coefficient between $L_{i,t-t}$ and $L_{i,t}$, where t is the number of generations lag. Single short time series generally require large autocorrelations ($r > 0.5$ for two generation lags in a 15-point series) to statistically distinguish correlations from noise. However, we were also able to estimate statistical confidence by comparing ACFs from replicate populations in each environment. We considered correlations significant by this criterion if the 95% confidence-interval around the mean correlation of the three time series did not overlap zero. Note that if oscillations are random, autocorrelations averaged across replicates should be zero; if oscillations are similar in shape (but not necessarily synchronous) across replicates, then the average autocorrelation across replicates should be nonzero at some temporal lags. Like ACF analyses of single time series, this detects periodicity but does not distinguish between externally-driven and internally-generated periodic oscillations because the experimental populations were initiated at the same time with similar population sizes. We obtained qualitatively similar results from analyses of both total population density and adult plant density, so we report only data from total numbers of flowering adults. We feel that these data are more reliable than data for total number of plants because seeds dispersed into the current generation's pots may have germinated before the harvest, and the generation to which small seedlings belonged could not be identified unambiguously.

Curve-fitting analyses

The results of fitting population regulation models to time series data are often highly sensitive to the choice of models and curve-fitting methods (Morris 1990, Turchin 1990, Berryman 1992). Thus, we characterized population dynamics by fitting a number

Table 1. Population regulation models fit to data from the population cage experiment. For all models, N_t = the number of individuals in generation t, r = density independent growth rate, k = equilibrium population size. Estimates of density independent growth rates (± standard error) are from models fit to pooled data from replicate time series using the SAS NLIN procedure. Where models could be linearized and fit using the SAS GLM procedure, F-statistics are shown for qualitative comparison of model fits. For additional parameter estimates and model comparisons, see Appendix.

Model:	Soil Type	F (model, error DF)	Density-independent growth rate ± std err.
Ricker: $N_{t+1} = N_t \exp(r(1 - N_t/k))$	deep:	13.5 (1,26)	e^r = 4.7±1.6
Ricker (1954)	shallow:		e^r = 3.0±0.8
discrete logistic: $N_{t+1} = N_t (r(1 - N_t/k))$	deep:	18.7 (2,28)	r= 2.8±0.5
May and Oster (1976)	shallow:	40.4 (2,28)	r= 2.6±0.3
Hassell: $N_{t+1} = N_t (r(1 - N_t/k)^\theta)$	deep:	-	r= 4.4±2.3
Hassell *et al.* (1976)	shallow:	-	r= 2.3±0.7
lagged Ricker: $N_{t+1} = N_t \exp(r(1 - ((1-p)N_t - pN_{t-1})/k))$	deep:	10.4 (2,25)	e^r = 4.3±1.3
Ricker (1954), Turchin (1990)	shallow:	13.7 (2,27)	e^r = 5.4±1.4
Turchin and Taylor: $N_{t+1} = N_t \exp(r + a_1 N_t^{\theta 1} + a_2 N_{t-1}^{\theta 2} + a_{11} (N_t^{\theta 1})^2 + a_{22} (N_{t-1}^{\theta 2})^2 + a_{12} N_t^{\theta 1} N_{t-1}^{\theta 2})$	deep:	-	e^r = 0.1±0.1
Turchin and Taylor (1992)	shallow:	-	e^r = 2.1±1.3

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Crone, E. 1995, Causes and consequences of complex population dynamics in an annual plant, *Cardamine pensylvanica*. Dissertation. Department of Botany, Duke University, Durham, North Carolina, USA.

of discrete-generation models to density transitions ($N_t \rightarrow N_{t+1}$ or $N_{t-1} \rightarrow N_t \rightarrow N_{t+1}$) from the time series data from experimental populations (Table 1): the non-lagged Ricker, Hassell, and discrete logistic models, a lagged Ricker model with one- and two-generation delays, and the one- and two- generation delay model suggested by Turchin and Taylor (1992). All models included a one-generation feedback term because our demographic data indicated direct density dependence (see Results). We chose to add the second lag at two generations based on ACF patterns (Berryman 1992). A single set of parameter estimates for each model was calculated for all of the pooled observations from three replicate populations in each environment type. As mentioned above, in this paper we only present analyses of populations in homogeneous deep and homogeneous shallow soil environments. We repeated analyses of heterogeneous environments using a number of assumptions about spatial environmental heterogeneity and the results of all these analyses were nearly identical to those for homogeneous environments. The analyses of heterogeneous environmental treatments are therefore much more laborious to explain without giving any additional information about the nature of population dynamics.

For the first four models, curves were fitted to data by (1) using the SAS NLIN procedure (SAS Institute 1987), (2) using a downhill simplex function minimization algorithm (Press *et al.* 1989) to find maximum likelihood estimates of parameters assuming Poisson-distributed (rather than normally-distributed) errors, (3) fitting equivalent models using the SAS GLM procedure (SAS Institute 1987) where possible (*i.e.* linear regressions of ln-transformed replacement rates for Ricker-variant models and polynomial regressions for the discrete logistic) and (4) fitting maximum likelihood models to individual population data sets within each environment type and averaging parameter estimates. Although each of these methods makes different assumptions

about the source and distribution of error, the results of the four analyses were virtually identical, and we report only parameters fitted using the SAS NLIN procedure. Due to the large number of parameters in Turchin and Taylor's lagged model, we fit this model only by using linear regressions of ln-transformed replacement rates.

Prediction of dynamics from population growth rates

We also characterized population dynamics by estimating the rate of population increase at low density. In simple nonlagged population growth models (*e.g.* Ricker and discrete logistic), asymptotic stability depends only on the value of this parameter (r), and stability decreases monotonically with increasing r. Our intention was not to obtain an exact value for r, but to determine whether or not this value was high enough to potentially generate complex dynamics.

A single plant (N_t) was placed in the center of an array identical to the arrays used for a population in the population cage experiment. When this plant set seed, additional pots were added to receive seed for the next (N_{t+1}) generation. N_{t+1} plants were left in the arrays until they set seed, and then numbers of flowering adults were censused to determine the rate of increase. Transitions were measured for six populations with the source plant (N_t) in deep soil, and six in shallow soil. Pots for the N_{t+1} generation were filled with deep soil on one side of each plant and with shallow soil on the other side of each plant. Thus, we estimated recruitment from $N_t(deep)$ to $N_{t+1}(deep)$ as twice the number of adult plants in the N_{t+1} generation in the deep half of the population, and recruitment from $N_t(deep)$ to $N_{t+1}(shallow)$ as twice the number of adult plants in the N_{t+1} generation in the other half of the population.

Results

All of the experimental populations exhibited complex numerical dynamics over fifteen generations, with most populations having three or four distinct peaks during the experiment (Figure 2). The autocorrelation functions from the time series indicated cyclic behavior through significant negative autocorrelations after two and/or three generations and oscillation between positive and negative autocorrelations (Figure 2). Thus, the populations appear to cycle with a period of approximately four or five generations.

Data from the census of the individual pots demonstrate density-dependent reproduction in both environments (Figure 3). In both environments fecundity increased with increasing biomass, but extremely small plants were able to set seed (Figure 4), suggesting that the direct effects of density-dependent reproduction would not necessarily have caused oscillatory dynamics (Rees and Crawley 1989, but see Discussion). Total plant density was lower and surviving plants were larger and more fecund in pots with shallow soil (Table 2), but a smaller proportion of survivors flowered in deep soil, reflecting the persistence of smaller plants in that environment (Table 2).

When fit to the time series data, all the population growth models explained significant amounts of variation in the data (Table 1), and quadratic terms (density-dependence *sensu* Turchin 1990) were significant in SAS PROC GLM curve fits (Type III sums of squares, $p < 0.05$). However, statistical significance is based on the null hypothesis that there is no relationship between density in one generation and density in the next, rather than the null hypothesis that density dependent feedback is present, but not strong enough to generate cyclical dynamics. This, combined with the lack of established procedures for estimating statistical confidence when using iterative curve-fitting methods, makes interpretation of statistical significance problematic. Only models with time lags reproduced the qualitative dynamics. In some cases, deterministic

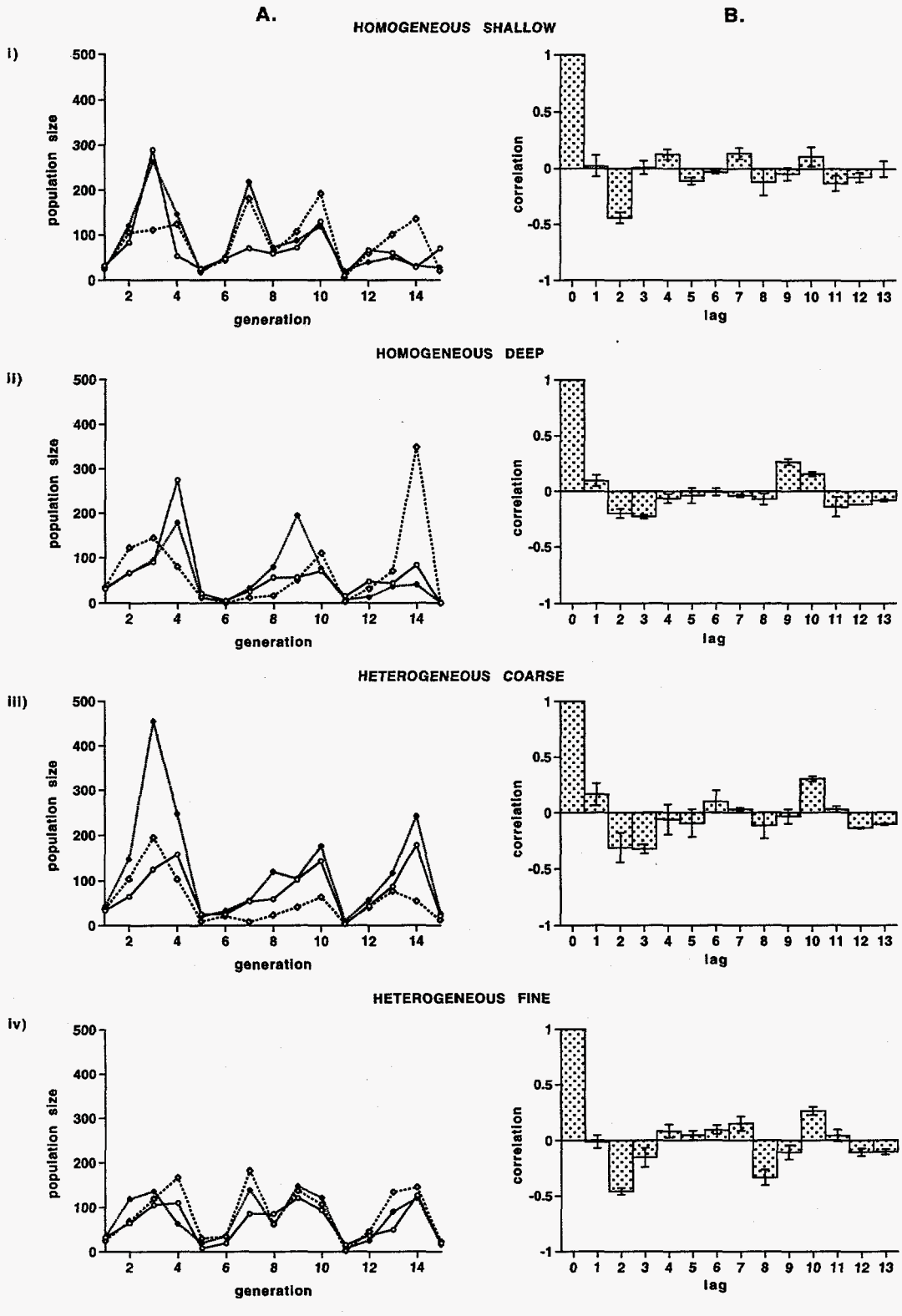

Figure 2. A.) Dynamics of experimental populations over fifteen generations for three replicate populations in four environments: i) homogeneous shallow soil, ii) homogeneous deep soil, iii) coarse-grained heterogeneous, iv) fine-grained heterogeneous. B.) Associated autocorrelation function for each treatment; bars are mean correlations of three replicate populations at each lag ± standard error.

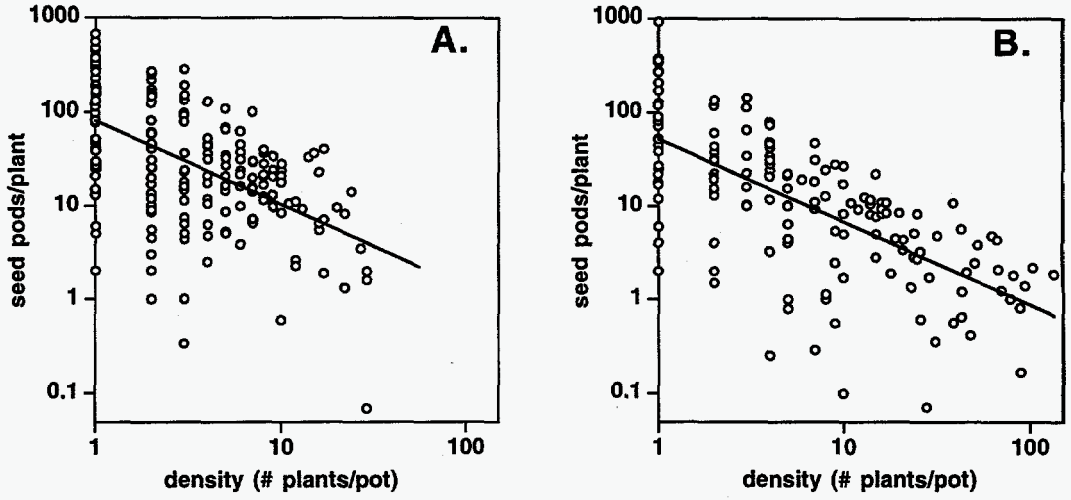

Figure 3. Fecundity *vs.* density for individual plants from randomly selected pots in the experimental populations: A.) shallow soil, B.) deep soil. Linear regressions of ln-transformed variables calculated using the SAS GLM procedure were: $y = 4.42(\pm 0.16) - 0.912(\pm 0.105)^*x$ in shallow soil ($R^2 = 0.292$, $p < 0.0001$, $N = 237$) and $y = 3.78(\pm 0.18) - 0.825(\pm 0.081)^*x$ in deep soil ($R^2 = 0.413$, $p < 0.0001$, $N = 151$), with standard errors of parameter estimates given in parentheses.

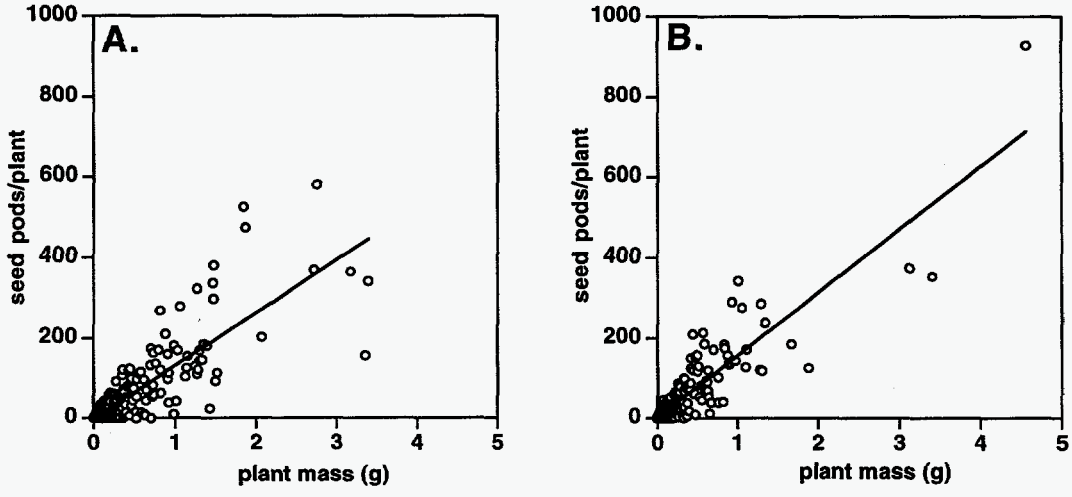

Figure 4. Fecundity *vs.* size for randomly selected plants in the experimental populations. A.) shallow soil, B.) deep soil. Linear regressions calculated using the SAS GLM procedure were: $y = -0.937(\pm 1.060) + 131.5(\pm 2.7)^*x$ in shallow soil ($R^2 = 0.737$, $p < 0.0001$, $N = 817$) and $y = -1.480(\pm 0.283) + 106.8(\pm 1.3)^*x$ in deep soil ($R^2 = 0.783$, $p < 0.0001$, $N = 1785$), with standard errors of parameter estimates given in parentheses.

Table 2. Characteristics of the plants from individual pot harvest data. N = sample size, standard errors are given in parentheses.

Trait:	Deep Soil mean	N	Shallow Soil mean	N
mass (g)	0.052(±0.005)	1796	0.140(±0.012)	841
fecundity (# siliques)	6.58(±0.631)	2743	27.67(±2.084)	1161
height (cm)	4.84(±0.125)	2743	12.98(±0.315)	1161
density (plants per pot)	11.50(±1.676)	146	5.05(±0.570)	152
percent flowering	23.3	640	61.7	716

Table 3. Recruitment of seedlings and adult plants from an isolated target plant into an array of 36 pots. N = 6 plants/environment for seedling recruitment data and N = 5 plants/environment for adult recruitment. Standard errors are given in parentheses.

Progeny Plant Type/ Progeny Environment	Parent Plant Environment	
	Deep Soil	Shallow Soil
a) mean number of seeds germinating in:		
deep soil	83 (± 61)	150 (± 68)
shallow soil	933 (± 477)	1236 (± 397)
b) mean number of plants flowering in:		
deep soil	12 (± 9)	13 (± 5)
shallow soil	45 (± 16)	41 (± 10)

iteration of the lagged models produced dynamics nearly identical to our data (fits of lagged Ricker model to shallow soil and Turchin and Taylor's model to deep soil, Figure 5C and 5D). In others, dynamics were asymptotically stable but with transient cycles (fits of Turchin and Taylor's model to shallow soil and lagged Ricker to deep soil, Figure 5C and 5D). With stochastic noise, the latter also realistically reproduce cycles similar to the actual dynamics, unlike the nonlagged models which rapidly return to equilibrium population size, even in the presence of stochastic noise (E. Crone, unpublished).

Since the time series data only used census data from flowering plants, the maximum potential population growth rate is estimated as the total number of flowering plants produced (at the time of the census) by an isolated flowering plant in an experimental population (Table 3). The experimental estimates of the rate of density-independent population increase are noisy, but mean values are considerably larger than values estimated from the time series data. Independent estimates of maximum growth rates are 12.0 and 41.0 in deep and shallow soil environments, while the maximum fitted estimates of these parameters are 4.7 and 5.4 respectively (Tables 1 and 3). The dynamics predicted from simple models using independent estimates of the parameters predicted complex dynamics (Figure 6), but did not give the qualitatively close fit of the fitted lagged models (Figure 5).

Discussion

To the best of our knowledge, this is the longest-term (*i.e.* over most generations) time series data set available for plant populations. Contrary to theoretical expectations, we observed strong cyclic variation in population density over time. Although our experimental populations were maintained in controlled-environment arrays, dispersal, germination, competition and recruitment occurred naturally.

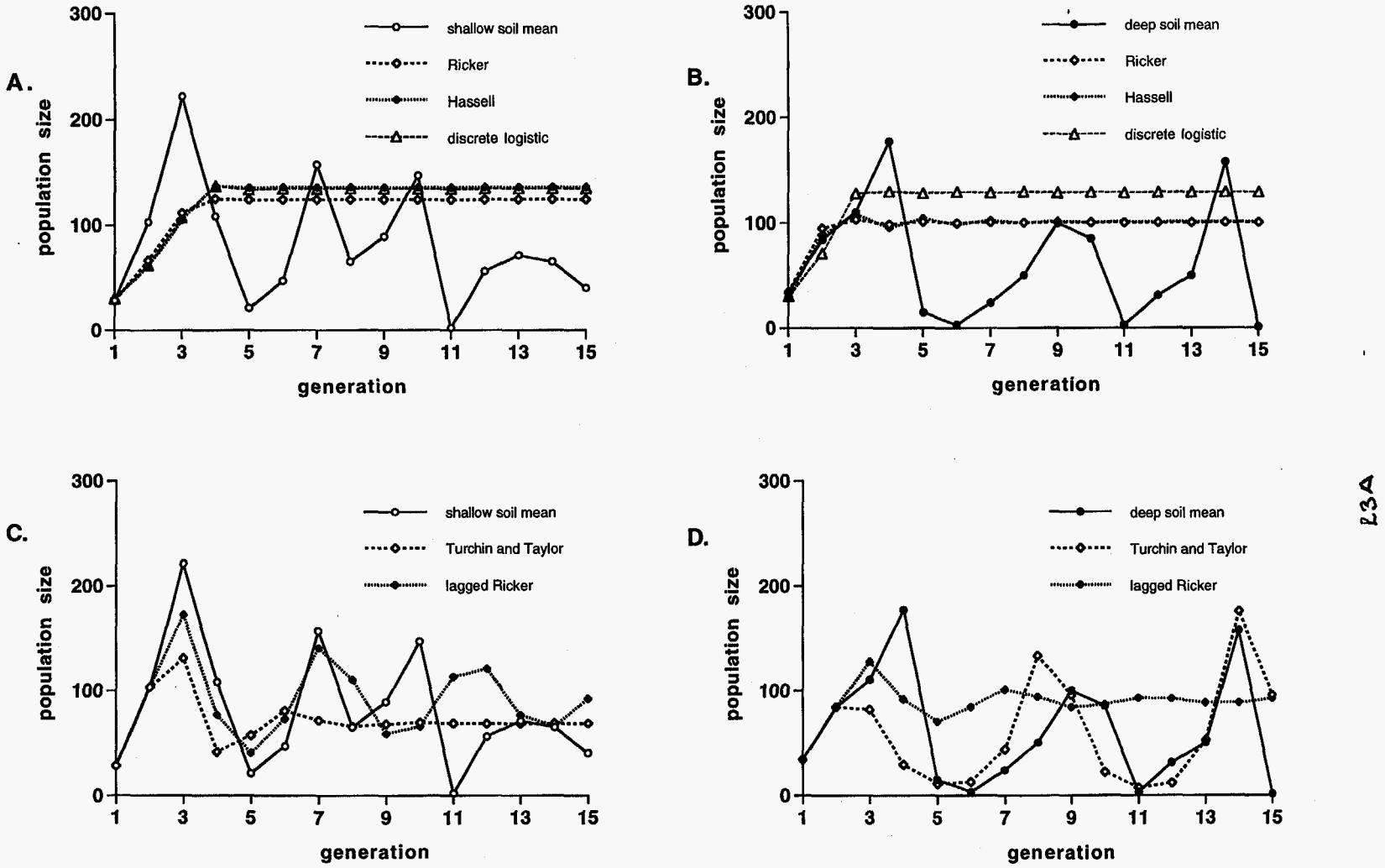

Figure 5. Fit of population regulation models to the time series data. A.) nonlagged models fit to homogeneous shallow soil data, B.) nonlagged models fit to homogeneous deep soil data, C.) lagged models fit to homogeneous shallow soil data, D.) lagged models fit to homogeneous deep soil data. Mean data from three replicate populations in each treatment are shown for comparison.

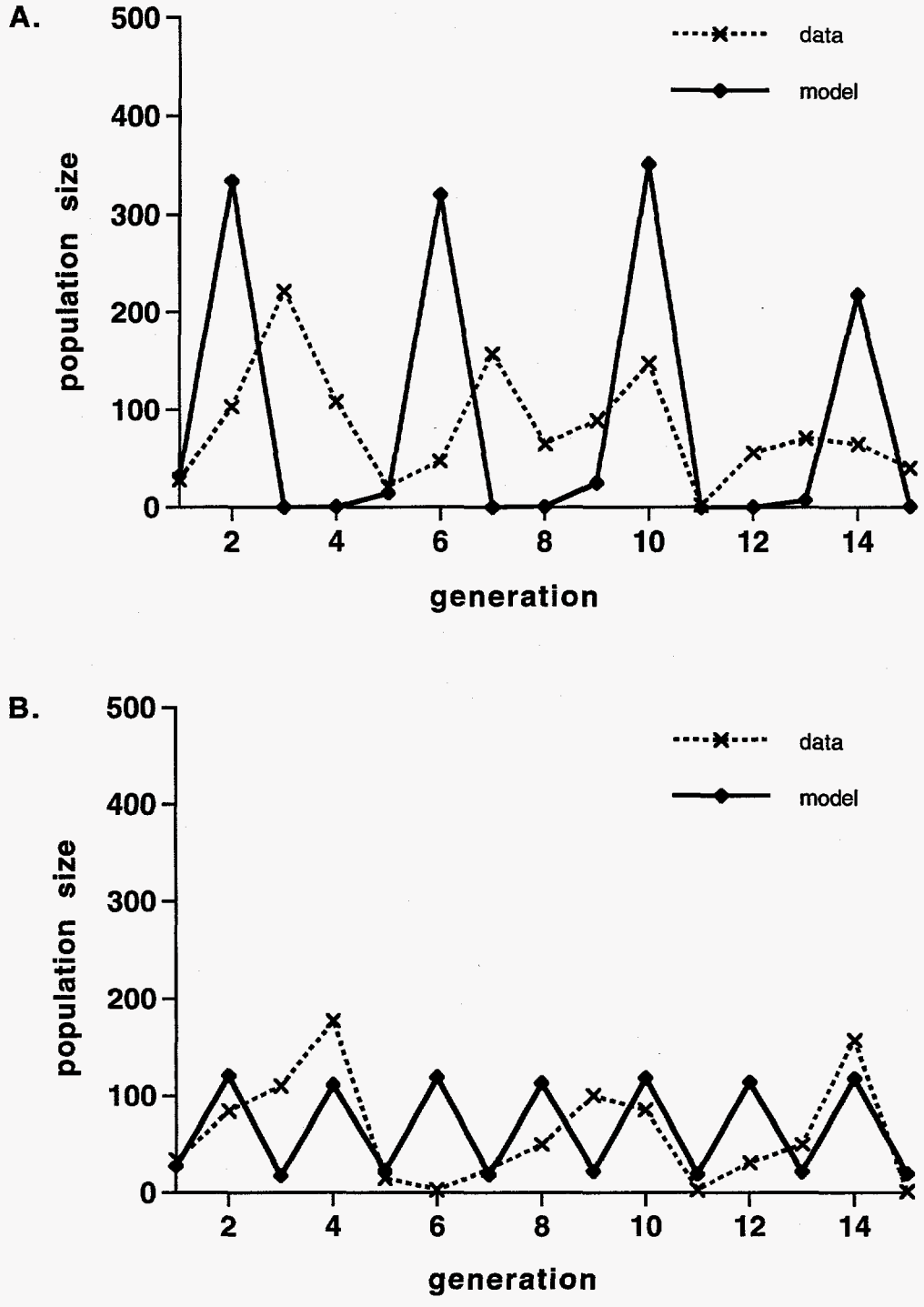

Figure 6. Plot of predicted dynamics using the Ricker model and experimental estimates of the density-independent growth rate (r) for A.) homogeneous shallow soil and B.) homogeneous deep soil. Mean data from the three replicate populations in each treatment are shown for comparison.

Dispersal into current-generation (as opposed to next generation) pots is roughly analogous to landing in unsafe sites in natural environments, and limited recruitment periods are analogous to seasons in natural populations of annual plants. Furthermore, our populations captured many of the key stabilizing features of plant populations described by Rees and Crawley (Rees and Crawley 1989, Crawley 1990). For example, density rapidly became variable in space (E. Crone, personal observation). Because competition took place primarily within individual pots but seeds dispersed large distances through explosively dehiscent siliques, the effects of crowding in one part of the population should have been lessened by recruitment from other less dense areas in the population. Similarly, our demographic data indicate that, although reproductive output decreases with decreasing size, extremely small plants are able to reproduce (Figure 4). The dynamics of natural populations of *C. pensylvanica* could be stabilized by recruitment from seed banks, which were completely absent in our experimental arrays. Nonetheless, our data suggest that complex dynamics are at least plausible in natural plant populations, even when potentially stabilizing factors are present.

In fitting population models to the data we see the same pattern when the studies of Hassell *et al.* (1976) and Turchin and Taylor (1992) are compared. Nonlagged models predict stable dynamics but models incorporating time lags extract complex dynamics. There is a degree of circularity in fitting recruitment models to time series data and then iterating these models to "post-dict" dynamics, *i.e.* models are tested only by determining how accurately they reproduce the temporal dynamics of the data set from which they were derived. Thus, it is not surprising that the lagged models were able to reproduce the observed dynamics. Rather, the striking feature of our curve-fitting analysis is the huge discrepancy between the post-dictions of nonlagged models and the actual dynamics. Our data support the growing body of literature (Turchin 1990,

Berryman 1992, Turchin and Taylor 1992, Dennis and Taper 1994) suggesting that density-dependent population regulation takes place at many temporal scales, and that inclusion of delayed density dependence is a minimum requirement for extracting dynamics from time series data. Unlike the natural populations analyzed in the above studies, our populations were maintained in growth chambers, so it seems unlikely that oscillations were driven by cyclic fluctuations in climate or weather.

In this study, a number of factors could be responsible for delayed density feedback. The first is feedback through direct maternal effects. Specifically, density may affect maternal provisioning (*e.g.* seed size) in one generation, offspring quality in the next generation, and density in subsequent generations. The potential importance of offspring quality on population dynamics has been emphasized recently by Ginzburg (1992), and density effects on seed size and seed size effects on plant biomass and seed set are common in plants (Roach and Wulff 1987, Bazzaz *et al.* 1992). Time-lagged feedback could also be due to the effects of herbivores and pathogens interacting with the plant populations (Turchin and Millstein 1993). This is quite plausible in our study, even in a controlled environment chamber. Mild aphid and gnat infestations were common, and though there were some noticeable fluctuations in insect abundance, we did not quantify them. In retrospect, it would have been very interesting to do so. Insects were free to disperse between populations within the growth chamber so the remarkable synchrony of the replicate populations may, in fact, be due to chamber-wide delayed regulation *via* insect interactions. For example, there was a noticeable crash across populations in generation 11 (Figure 2), and there was a noticeable aphid infestation that generation. Finally, Tilman and Wedin (1992) attributed chaotic dynamics in perennial grasses to delayed feedback through accumulation of dead plants from previous years' growth which gradually changed site quality. Changes in site

quality are unlikely in our experimental arrays because soil conditions were reset when new pots were added each generation. However, seed in offspring-generation pots often germinated before parent-generation pots were removed. Plant quality could have been affected by shading from the previous generation, and the amount of shading varied with density (E. Crone, personal observation).

Another interpretation of the failure of the population growth models to describe the complex dynamics of *C. pensylvanica* is that population dynamics were altered by the enforcement of discrete generations. This has been found in population cage experiments with *Drosophila* species. Development rates in *Drosophila* are slower at high density, causing fewer individuals to reproduce at high density under constant generation length, whereas more individuals might reproduce at high density if generation length were increased to span the reproduction of all individuals (M. Gilpin and T. Philippi, personal communication). In our populations of *C. pensylvanica*, the population cycles appeared not to be sensitive to the amount of time available for recruitment (see Methods). We believe that this reflects a difference in the mechanisms of density-dependent competition in *Drosophila* and *Cardamine*. In *Drosophila*, crowded conditions result in slower development because less food is available to each larva (Gilpin *et al.*1986). In *Cardamine*, only the first seeds to germinate in each pot survive to adulthood; later germinants are shaded by adults (E. Crone, personal observation). Thus, as seed density increases, more individuals survive (because more seeds germinate simultaneously), but these individuals are only from among the earliest recruits. In direct contrast to *Drosophila*, *C. pensylvanica* generations were actually shorter at high density (E. Crone, unpublished data).

Another possible explanation of the results of our curve-fitting analyses is that total population censuses average over among-pot spatial variation in density and

genetic heterogeneity among individuals, and this could also mask the causes of population dynamics (spatial: Pacala and Silander 1985, Hastings 1993; genetic: May and Anderson 1983, and see Chapter 3). However, the ACF pattern in our data (strong autocorrelation at two-generation lags and no autocorrelation at one-generation lags) is typical of lagged density feedback and is only infrequently generated by nonlagged models, even those including spatial, genetic, and/or stochastic variation (see Chapters 2 and 3). In fact, spatially discrete metapopulation models of population dynamics usually generate either stable equilibria or stable two-point cycles in population size (Hastings 1993, Molofsky 1994), and genetic diversity increases the probability of asymptotic stability (see Chapter 3). Neither factor is likely to cause the four-generation cycles we observed and neither spatially explicit models nor explicit modeling of clonal dynamics accurately reproduced the observed dynamics (E. Crone, unpublished). Thus, we favor the hypothesis of delayed density feedback.

Of course, without direct experimental evidence for delayed feedback, we cannot prove the causes of complex dynamics in *C. pensylvanica*. However, our analyses bring an important point to light: the theoretical reasons why plants should display stable dynamics (Rees and Crawley 1989, Crawley 1990) are based on the assumption that density feedback is through direct density dependence. For example, direct density dependence stabilizes plant population dynamics when there is extreme plasticity in reproductive output of plants. However, if extremely small plants set seed but produce offspring that cannot set seed or are more susceptible to predators or pathogens, this could actually decrease dynamical stability by increasing the time lag between growth and density feedback. Similarly, plant population dynamics would not be stabilized by additional recruitment from a seed bank or from other sites if delayed feedback occurs through changes in site quality which inhibit seed germination or seedling growth (as in

Tilman and Wedin 1992). Thus, until we obtain data on the dynamics of natural plant populations and the factors which cause delayed density feedback in plant populations, the complexity of plant population dynamics remains an open question.

Appendix

The set of possible models that could potentially describe population dynamics is the set of models that: (1) explain significant amounts of the variance among paired time series observations ((N_t, N_{t+1}) or (N_{t-1}, N_t, N_{t+1})), and (2) predict population dynamics which do not differ significantly from the dynamics used to estimate parameters in (1). Because there are many established statistical procedures for fitting models to data and testing (1), I simply tried several methods for fitting possible models to data (see Methods). Because the results of these analyses were redundant, I did not list them all in the text. However, for those interested, the exact results of several of these analyses are given in Tables A1-A4.

In spite of a noticeable amount of variation among parameter estimates derived by different methods of parameter estimation, there was a clear distinction between the predictions of models that did and did not include delayed density dependence. Models without delayed density dependence predicted stable population dynamics with mean population sizes much higher than we observed, while models with time delays predicted dynamics with damped or apparently stable oscillations in the general range of the data. Thus, I visually rejected models without delayed density dependence as possible causes of the observed cycles, and retained models with delayed density dependence. As described in Chapters 2 and 3, the difference between the predictions of these classes of models reflects the difference between populations driven by flip vs.

Hopf bifurcations. Flip bifurcations can never generate the ACF pattern we observed in *C. pensylvanica* populations, while Hopf bifurcations can.

A third step is to ask which of the models that meet criteria (1) and (2) provides the best description of the data. When two or more models are able to predict dynamics that capture the key features (mean and ACF) of time series data, I advocate estimating the probability of parameters that reproduce these features being the true parameter estimates, given paired observations from the time series. Although I have not fully pursued this issue with these data, I have tested the significance of individual parameters in a simplified (6-parameter) version of Turchin and Taylor's 8-parameter model, and compared the Ricker and lagged Ricker models (Tables A5 and A6). These analyses show that Turchin and Taylor's model is overparameterized for this data set, and that the time delay parameter in the lagged Ricker model, in addition to predicting more realistic dynamics than the Ricker model, significantly improves the fit of the model to paired time series observations.

Table A1. Summary statistics for models fit to data using the SAS NLIN procedure. Because the parameters in these models were not identical to the parameters in Table 1, the relationships between the two parameterizations of the models are given.

		shallow soil		deep soil	
model	parameter	estimate	standard error	estimate	standard error
Ricker	$a = e^r$	3.05	0.82	4.72	1.59
	$b = r/k$	0.0090	0.0030	0.0155	0.0058
discrete logistic	$a = r$	2.36	0.34	2.78	0.50
	$b = r/k$	0.0043	0.0005	0.0050	0.0007
Hassell	$a = r$	2.31	0.71	4.41	2.33
	$b = r/k$	0.0044	0.0014	0.0021	0.0085
	$c = \theta$	0.92	0.98	6.26	31.07
lagged Ricker	$a = e^r$	5.36	1.42	4.31	1.35
	$b = r(1-p)/k$	0.0087	0.0022	0.0098	0.0041
	$c = rp/k$	0.0096	0.0039	0.0065	0.0073

Table A2. Summary statistics for models fitted using the SAS GLM procedure. DF are model degrees of freedom for each parameter, F-statistics are from Type III sums of squares, and p-values are based on T-statistics testing the null hypothesis that the parameter value is zero. In these models, all terms involving direct or delayed density dependence are statistically significant.

parameter	DF	F	p	estimate	standard error
1. shallow soil (30 total DF)					
a. Ricker: $\ln(N_{t+1}/N_t) = a - bN_t$					
a	0	n/a	0.0027	0.829	0.252
b	1	17.49	0.0003	0.0092	0.0022
b. discrete logistic: $N_{t+1} = aN_t - bN_t^2$					
a	1	34.83	< 0.0001	1.76	0.30
b	1	15.05	0.0006	0.0057	0.0015
c. lagged Ricker: $\ln(N_{t+1}/N_t) = a - bN_t - cN_{t-1}$					
a	0	n/a	< 0.0001	1.25	0.28
b	1	20.11	< 0.0001	0.0090	0.0020
c	1	6.51	0.0167	0.0050	0.0020
2. deep soil (28 total DF)					
a. Ricker: $\ln(N_{t+1}/N_t) = a - bN_t$					
a	0	n/a	0.1419	0.672	0.444
b	1	13.46	0.0011	0.0144	0.0039
b. discrete logistic: $N_{t+1} = aN_t - bN_t^2$					
a	1	28.88	< 0.0001	1.77	0.33
b	1	19.34	0.0002	0.0056	0.0013

c. lagged Ricker: $\ln(N_{t+1}/N_t) = a - bN_t - cN_{t-1}$

a	0	n/a	0.0156	1.27	0.49
b	1	13.47	0.0012	0.0135	0.0037
c	1	5.13	0.0325	0.0115	0.0051

Table A3. Summary statistics for parameters r and k (as defined in Table 1) fitted to individual time series using the simplex algorithm (Press et al 1989), assuming Poisson-distributed error. (Poisson-distributed error is expected if differences in recruitment are due to demographic stochasticity, rather then environmental variance.) Parameters were fit to each replicate population and variance among replicate populations was used to estimate the distribution of maximum likelihood estimates.

	replicate population				
	1	2	3	mean	standard
parameter					error
1. shallow soil					
a. Ricker					
r	1.08	2.05	0.97	1.37	0.28
k	96.6	102.2	120.3	106.4	5.8
b. lagged Ricker					
r	1.32	2.24	1.24	1.60	0.15
k	88.1	99.4	105.6	97.7	4.7
p	0.26	0.10	0.40	0.25	0.07
2. deep soil					
a. Ricker					
r	1.00	1.55	1.05	1.20	0.14
k	83.8	102.7	91.1	92.5	4.5
b. lagged Ricker					
r	1.09	1.72	1.31	1.37	0.15
k	79.7	83.3	64.6	75.9	4.7
p	0.13	0.41	0.73		0.14

Table A4. Parameters fitted using Turchin and Taylor's Response Surface Methodology, as originally proposed (Turchin and Taylor 1992). Note that in the intervening years, Turchin has greatly modified and improved this technique (*e.g.* Turchin and Millstein 1993, Ellner and Turchin 1995).

parameter	shallow soil	deep soil
a_0	0.76	-3.25
a_1	-0.0018	0.00000015
a_2	-5.12	23.8
a_{11}	0.00000020	0
a_{12}	337.0	29.6
a_{22}	0.024	0.00000097
θ_1	1.5	3.0
θ_2	-1.0	-0.5

Table A5. Models of population regulation calculated using stepwise multiple regression of parameters a_0-a_{22} in a simplified 5-parameter version of Turchin and Taylor's model with $\theta_1 = \theta_2 = 1$. Model selection began with all 5 parameters and eliminated the parameter at each step that explained the least variation, until all variables were significant at a liberal $p < 0.15$ level.

parameter	estimate	standard error	F	p
1. shallow soil				
a_0	0.81	0.23	12.37	0.0016
a_1	-0.0042	0.0027	2.37	0.1357
a_{12}	-0.00053	0.00002	6.95	0.0137
2. deep soil				
a_0	0.73	0.42	3.02	0.0946
a_2	-0.011	0.005	5.11	0.0328
a_{11}	-0.000043	0.000011	15.29	0.0006

Table A6. Likelihood ratio (LLR) statistics for comparison of the Ricker model of recruitment vs. the lagged Ricker model. Because the models differ by 1 parameter, this ratio is distributed as χ^2 with one degree of freedom. In all populations, delayed density dependence significantly improves model fits.

population	shallow soil		deep soil	
	-2 LLR ($\sim\chi^2$)	p	-2 LLR ($\sim\chi^2$)	p
1	48.26	< 0.005	6.82	< 0.01
2	19.54	< 0.005	95.96	< 0.005
3	120.10	< 0.005	193.14	< 0.005

Acknowledgments

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CHAPTER 2.

Parental environmental effects and cyclical dynamics in plant populations

Abstract

Parental environmental effects have been widely reported in plants, but never incorporated into models of plant population dynamics. Nonetheless, inclusion of effects of parental density on offspring quality fundamentally changes population dynamics models by making recruitment a function of population size in two previous generations ($N_{t+1} = f[N_t, N_{t-1}]$), rather than one ($N_{t+1} = f[N_t]$). In this study, I measured effects of parental and offspring density on offspring quality in an annual plant, *Cardamine pensylvanica*, by manipulating plant density independently in parent and offspring generations, and comparing the effects of parent and offspring density on offspring performance. Parental density effects were detectable, but were noticeably weaker than offspring density effects. Nonetheless, the parental effect was large enough to change the predictions of a model of population dynamics. Without parental effects, this model predicts sharp density-dependent crashes from high to low density (flip bifurcations). With parental effects, the model predicts gradual cycles in population size (Hopf bifurcations). Thus, parental effects may be a critical and overlooked component of the numerical dynamics of plant populations.

Introduction

Numerous studies have shown that a plant's environment can affect not only the traits of that plant but the traits of its offspring (reviewed in Roach and Wulff 1987, Lacey 1991). Variation in parental environmental conditions has been shown to affect aspects of offspring performance across wide ranges of experimental manipulations (nutrient availability, water availability, temperature, light quality, and plant density) and plant taxa. However, despite the fact that parental environmental conditions can affect demographically important traits (*e.g.* survivorship, size and fecundity), few, if any, studies have asked whether these "parental environmental effects" might influence long-term population dynamics.

Effects of parental density on offspring quality are particularly likely to alter population dynamics. Most theoretical models of plant population dynamics assume that population density in one generation is a declining function of population density in the previous generation (*e.g.* Watkinson 1980, Pacala and Silander 1985, Rees and Crawley 1989, Molofsky 1994). However, if plants whose parents were crowded are smaller and less fecund than plants whose parents were uncrowded, then population size in the third generation is affected by population size in two previous generations. Parent plant density has been shown to directly affect seed size, seed germination characteristics, and seedling size (*e.g.* Harper 1977, Roach and Wulff 1987, Mazer and Wolfe 1992, Platenkamp and Shaw 1993). Furthermore, many other studies have shown that plants whose parents grew in low-quality environments (low nutrients or low light) are smaller and less fecund than plants whose parents were in high-quality environments (high nutrients or high light; *e.g.* Roach and Wulff 1987, Aarssen and Burton 1990, Miao *et al.* 1991, Wulff and Bazzaz 1992). Because nutrient availability, light availability, and other differences in environmental quality are often mediated by plant density,

these parental effects could also make population density in the third generation a function of density in two previous generations.

It is not clear that predictions made by models with parental effects would be the same as those made by models without parental effects. For example, the potential importance of density feedback from multiple past generations has been demonstrated in recent studies of animal population dynamics. Time series data from several natural populations of insects and small mammals can be explained by models that include delayed density dependence, but not by models that only include direct density dependence (Turchin 1990, Berryman 1992, Turchin and Taylor 1992, Turchin 1993). In some cases, delayed density dependence in insect and small mammal populations has been shown to be caused by direct effects of parent density on offspring survivorship and/or fecundity, which are analogous to parental density effects in plants (Royama 1981, Rossiter 1994).

In this paper, I measure parental density effects in an annual plant, *Cardamine pensylvanica*, and ask whether these effects might be important in driving population dynamics. In an earlier study (Chapter 1), I observed that population size in *C. pensylvanica* cycled from high to low density over time, apparently due to density-dependent population regulation. This suggests that *C. pensylvanica* populations are exposed to high variation in population density and that discrete-generation models of density-dependent population regulation are appropriate for this species. To determine whether parental density effects might be an important component of population dynamics in *C. pensylvanica*, I use three approaches. First, I measure the effects of parent and offspring density on demographic traits in *C. pensylvanica*. I then extend these relationships to a model of population dynamics that includes parental effects, and explore the general stability properties of this model relative to an identical model that

does not include parental effects. Based on this analysis, I show that parental effects change population dynamics when they are fairly large relative to direct effects of offspring density effects. Thus, I use my experimental data to generate a probability distribution for the relative size of parent and offspring density effects, and ask whether these parameters predict population dynamics that differ from the predictions of models without parental effects.

A. Study species

Cardamine pensylvanica (Brassicaceae) (Muhl.) is an ephemeral plant of damp habitats (Al-Shebaz 1988) and is a widespread and noxious greenhouse weed (Cloutier *et al.* 1991, Whitcomb and Santlemann 1983). Plants from greenhouse populations of *C. pensylvanica* germinate and flower readily and are present throughout the year under a variety of environmental conditions. Seed from these populations can germinate within a week of reaching a suitably moist environment, and individuals begin to set seed approximately two months after germination. In the field, *C. pensylvanica* behaves as a winter annual, setting seed in the early spring before most other plants are established. The source population for this experiment was created by combining seeds from more than 100 individuals from each of three long-established greenhouse populations: the greenhouses at Duke University (Durham, NC) and North Carolina State University (Raleigh, NC), and a private greenhouse in Durham, NC. Each greenhouse was decades old, and in each greenhouse the staff recognized *C. pensylvanica* as a chronic pest. Thus, the experimental conditions were similar to the recent experience of populations in "natural" conditions.

B. Density feedback in *Cardamine pensylvanica*

Methods

To compare the effects of parent and offspring densities on offspring reproduction and subsequent recruitment into the third, grand-offspring, generation, I conducted two parallel experiments. In the first, I manipulated density in the first (parental) generation and examined effects of parental density on plants in the second (offspring) generation. In the second, I held parent density constant and varied offspring density.

1. *Parental effects* - To generate plants for this experiment, seeds from the source population (described above) were grown under high light, water and nutrient conditions in the Duke University greenhouse for one generation. Seeds from these plants were harvested and grown for one generation in the Duke University Phytotron at low density. Experimental conditions in phytotron chambers were identical to the "shallow soil" treatment described in Chapter 1. Seeds harvested from these plants were used for the first (parental) generation in the density experiment. In the parental generation, seeds were sown in 2.5 cm pots (RL-100 Conetainers) at each of three densities: 6, 100, and 800 seeds per pot (Figure 1). Twenty-four replicate pots were planted at each seed density. Densities were chosen based on preliminary experiments to generate a range of adult densities similar to those observed in population dynamics studies (1-25 adult plants/pot; see Chapter 1). To make above-ground plant density similar to within-pot density, the pots for each density treatment were grouped into four blocks, and the twelve blocks (3 densities x four blocks/density) were randomly located in a growth chamber in the Duke University Phytotron.

Seeds for the offspring generation were harvested from the parent-generation plants. In many plant species, early maturing seeds are larger and more robust than later seeds (Roach and Wulff 1987). Seed set in *C. pensylvanica* takes place over about

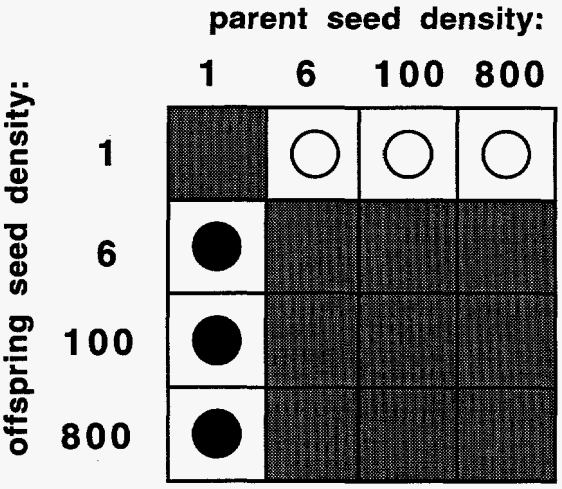

Figure 1. Design of experiments used to measure effects of parent (○) and offspring (●) plant densities. This range of seed densities led to a continuous range of plant densities (1-25 plants/pot; Figure 2). Shaded cells (■) were not included in this experiment (but see Methods).

two weeks, and seed pods dehisce explosively after seeds are mature. Thus, to ensure even collection of seeds across time and treatment, I collected seed from each pot weekly for a three-week period, beginning when about 20% of plants had begun to set seed, and ending after about 80% of plants had produced mature seed for two weeks and all plants that had flowered had dehisced several seed pods.

For the second generation, I randomly selected a single seed from each pot in the first generation. These seeds were planted in 2.5 cm pots at a constant density of 1 seed/pot (Figure 1). I planted second generation pots at a constant density to avoid confounding effects of parent and offspring densities (see below). I planted only one seed per parent generation pot to ensure independence of second-generation plants. This design yielded a total of 72 seeds planted in the second generation (24 from each of 3 parent generation seed densities).

To determine the potential effects of parental density on population dynamics, it is necessary to know whether differences in parental density will change offspring fecundity, and thus also affect recruitment into the third (grand-offspring) generation. In previous experiments (E. E. Crone, unpublished), ln-transformed seed density was proportional to ln-transformed adult density ($N = 59$, $R^2 = .87$, $p < .0001$), and ln-transformed mass of isolated adult plants was proportional to ln-transformed seed rain into an adjacent array of pots ($N = 5$, $R^2 = .89$, $p = .0052$). Thus, I used mass of offspring plants as an estimate of offspring fecundity and recruitment into the third (grand-offspring) generation.

Specifically, I fitted a linear model of parent density effects to ln-transformed offspring mass (SAS GLM procedure, SAS Institute 1987):

$$\ln[y_t] = a - bN_{t-1} + \varepsilon \qquad (1)$$

where y_t is the average mass of individual plants in the offspring generation, N_{t-1} is the density of plants in the parent generation, and ε is stochastic error. I repeated the analysis using both seed density (which was directly manipulated) and adult plant density (which is probably a better indicator of the competitive environment). Although I expected that adult density would be the best predictor of performance, it was not logistically possible to independently manipulate seed and adult density, to exactly manipulate adult density, or to census plants between the seed and adult stages (recall that, to achieve realistic adult densities, up to 800 seeds were planted in single 2.5 cm pots).

2. *Offspring density effects* - Effects of variation in offspring (second generation) density on recruitment into the third generation were measured by keeping parent generation density constant and varying offspring generation density. In the parent generation, seeds were planted at a constant density of 1 seed/pot (Figure 1). Plants were grown and seeds were collected from each plant in this generation as described above.

Seeds from randomly selected parent plants were planted at each of three offspring densities: 6, 100, and 800 seeds per pot (Figure 1). Twenty-four pots were planted at each density. Each pot contained seed from only one parent-generation plant, and each parent plant was used only once. Third generation recruitment was estimated from second-generation adult mass as described above, except that plant mass was related to current (rather than parental) density:

$$\ln[y_t] = a - cN_t + \varepsilon \qquad (2)$$

where y_t is the average mass of plants in each pot and N_t is the total plant density in that pot, and ε is error. As above, the analysis was repeated using seed density in each pot, and adult density at the time of harvest.

3. *Interactions between parent and offspring density* - My original goal was to test for interactions between parent and offspring effects by doing a full factorial experiment of four parent and offspring-generation seed densities (1, 6, 100, and 800 seeds/pot). However, this experiment was impossible to perform because none of the pots planted at parent densities of 100 or 800 seeds/pot yielded enough seed to plant pots for the next generation at a density of 800 seeds/pot, and few yielded enough seed to plant at 100 seeds/pot. This implies that transitions from high density to high density are highly unlikely if not impossible in *C. pensylvanica* populations, and is consistent with my earlier observation of cyclical population dynamics in *C. pensylvanica* (Chapter 1).

Nonetheless, as a preliminary test for interactions, I aggregated seeds from all parent generation plants at each density and planted 10-24 pots from each parent density treatment at each of the offspring densities (depending on the availability of seeds). I then tested for heterogeneity among slopes of mass *vs.* parent density for plants from different offspring seed densities and slopes of mass *vs.* offspring density for plants from different parent seed densities. Analyses of these data indicated that offspring effects were independent of parent density ($p = 0.84$). There was a weak interaction between parent effect and offspring density in which the effect of parent density declined at high offspring densities ($p = 0.06$). However, stability analysis of a model incorporating this interaction showed that this would not significantly alter the predicted population dynamics. Because numerous caveats apply to these data (including bias toward the more successful plants at high-high density transitions and

lack of strict independence of replicates) and the interaction did not change the general pattern of results, I will not discuss these results further in this paper.

Results

Both parent and offspring density affected recruitment, *i.e.* slopes of offspring mass *vs.* parent and offspring plant density were both negative and unlikely to be zero (Table 1, Figure 2). In both cases, adult plant density was a better predictor of performance than seed density. This is consistent with my observations of competition among *C. pensylvanica* seeds, seedlings and adult plants. For example, when 100 seeds are placed in a 2.5 cm pot, several seeds germinate at approximately the same time, and form a canopy that covers the pot. The majority of these individuals appear to survive to be adults, while subsequent germinants do not survive long or become large, and probably do not significantly affect resource availability. If this scenario is true, the available resources would be consumed almost exclusively by the first cohort of germinants, which are also the plants that survive to be reproductive adults..

Nonetheless, although this analysis suggests that parent density affects offspring fecundity, the effects of parent density are only marginally significant ($p = 0.08$), and are noticeably weaker than the effects of offspring density. Thus, it is not immediately clear whether parental effects are significant for population dynamics. It may be that parental effects would be overwhelmed by offspring density effects, or would simply add noise to predictions of density-dependent population dynamics based only on offspring density. To address the importance of parental effects, I will begin by using a mathematical model to explore when parental density effects change population dynamics.

Table 1. Statistics for effects of parent and offspring seed and plant density on plant biomass. ML estimate is the maximum likelihood estimate for each parameter, p and F have their traditional values. Note that seed density and adult density are highly correlated, so it is not surprising that the results of the two analyses are similar.

	slope estimate	standard error	p	
parent effect				
seed density	-0.0011	0.0008	0.1684	2.03
adult density	-0.0767	0.0431	0.0835	3.16
offspring effect				
seed density	-0.0024	0.0008	0.0037	9.32
adult density	-0.1483	0.0440	0.0015	11.34

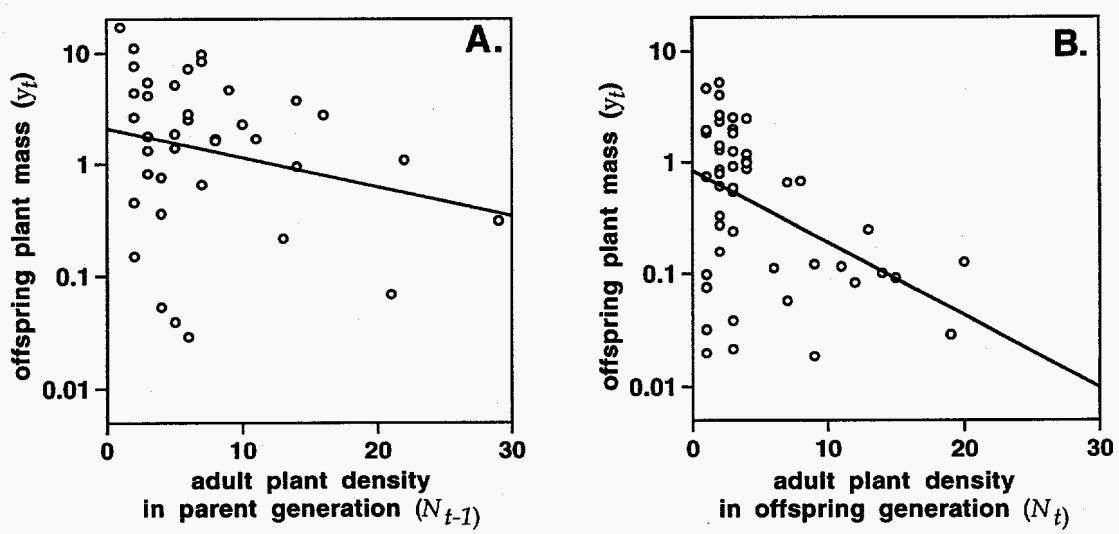

Figure 2. Relationships between A) parent and B) offspring plant density and average mass of plants in the offspring generation.

C. Model of population dynamics with parental effects

Given the absence of significant interactions between parent and offspring density effects, the models fitted to parent and offspring effects (equations 1 and 2) can be combined into a single model:

$$\ln[y_t] = a - bN_{t-1} - cN_t \tag{3}$$

where all parameters are as described above. This relationship suggests that a similar model can be used to describe population growth (see Appendix):

$$\ln\left[\frac{N_{t+1}}{N_t}\right] = a' - b' N_{t-1} - c' N_t, \tag{4}$$

This model can be reparameterized and written using traditional notation from models of population growth:

$$N_{t+1} = N_t \exp[r(1 - \frac{(1-g)N_t + gN_{t-1}}{k})] \tag{5}$$

where N_{t-1}, N_t, and N_{t+1} are population size in the parent, offspring and grand-offspring generations (respectively), r $(=a')$ is the density-independent population growth rate, k $(=a'/(b'+c'))$ is equilibrium population size (or carrying capacity), g $(=b'/(b'+c'))$ is the proportion of density feedback from parent density, and 1-g is the proportion of density feedback from offspring density. When parental effects are not present (g =0), this model collapses to the well-known Ricker model of population growth (Ricker 1954, May and Oster 1976, Edelstein-Keshet 1987):

$$N_{t+1} = N_t \exp[r(1 - \frac{N_t}{k})] \tag{6}$$

In addition to emphasizing its relationship to classical models of population dynamics, equation 5 has two advantages over equation 4: (1) asymptotic stability of equation 5 depends only on two parameters, r and g (see below), while analysis of equation 4

depends on 3 parameters, and (2) equation 5 allows qualitative patterns of population dynamics (see below) to be predicted from parent and offspring effects on biomass, which are much easier to measure then effects on actual recruitment into subsequent generations (see Appendix).

To explain the consequences of parental effects, I distinguish three possible patterns of variation in population size over time (Figure 3). The first is asymptotically stable dynamics, in which population size grows to a constant equilibrium density (carrying capacity) from any starting size (Figure 3A). This kind of population dynamics is probably the classical view of most plant population biologists, who have generally hypothesized that fluctuations in population size in plants are driven by variation in environmental conditions, rather than density dependent population regulation (Crawley 1990).

The second and third kinds of population dynamics describe two kinds of cycles in population size due to overcompensating density dependence. Although population cycles have been assumed to be rare in plant populations (Rees and Crawley 1989, Crawley 1990; but see Silvertown 1991, Molofsky 1994, Cousens 1995 and Discussion), they are clearly relevant here because *C. pensylvanica* populations cycle in the absence of environmental variation (Chapter 1). The first kind of population cycles is characterized by dynamics which alternately overshoot and undershoot carrying capacity in successive generations (hereafter called "flip bifurcations"; Thompson and Stewart 1986; Figure 3B). This is the familiar "period doubling" instability described by May and Oster (1976). Another notable feature of flip bifurcations is that population size always falls from the highest density to the lowest density in one generation, even when population dynamics are asymptotically chaotic, rather than cyclical.

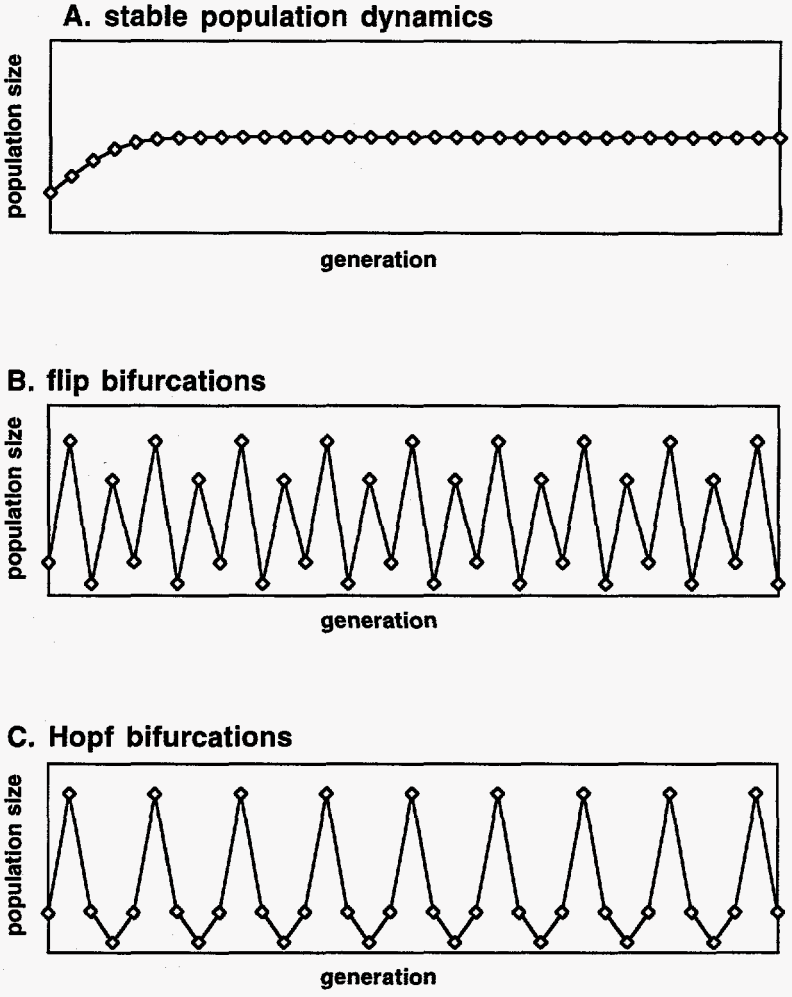

Figure 3. Typical time sereis of three kinds of population dynamics. A.) stable dynamics; B.) flip bifurcations: C.) Hopf bifurcations. Note the contrast between 4-point cycles generated by flip and Hopf bifurcations.

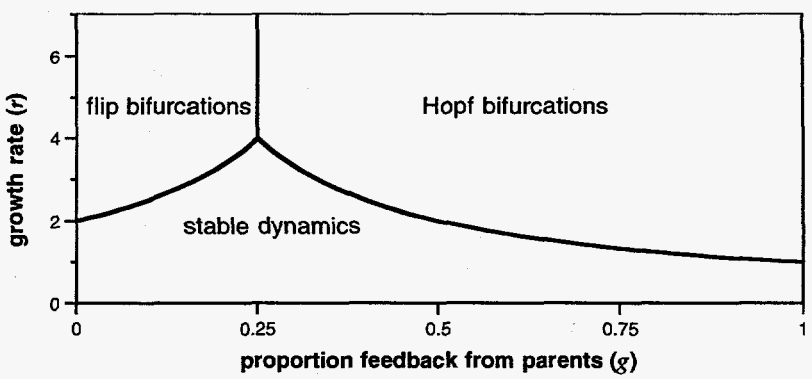

Figure 4. Stability regions for a model of population dynamics including parental density effects. The region below the lines is stable, the region in the upper left is unstable with flip bifurcations, and the region in the upper right is unstable with population cycles determined by Hopf bifurcations (see text).

This contrasts directly with the second possible form of cycles which are characterized by population cycles which rise and fall over several generations (hereafter called "Hopf bifurcations"; Thompson and Stewart 1986; Figure 3C). In this case, population size can slowly fall from high to low density, rather than "crashing" in a single generation. The distinction between these two kinds of cycles has a well-established theoretical basis (instability to real *vs.* complex eigenvalues, Nisbet and Gurney 1982). However, unlike the distinction between stable and cyclical dynamics, the distinction between population cycles caused by flip and Hopf bifurcations has never been made in discussions of plant population dynamics.

Standard techniques for stability analysis of equation 4 (May 1973), show that all three kinds of population dynamics (stable dynamics, flip bifurcations, and Hopf bifurcations) are possible in this model (Figure 4) (see Chapter 3), depending on the values of the population growth rate, r, and the time delay parameter, g. If there are no parental effects ($g = 0$), then population dynamics are stable if $r \leq 2.0$, and unstable due to flip bifurcations if $r > 2.0$ (May and Oster 1976, Figure 4). If $g \leq 0.25$, then population dynamics are unstable if

$$r > \frac{2}{1-2g}. \tag{7}$$

This instability is due to negative real eigenvalues of the Jacobian stability matrix, which cause flip bifurcations. In other words, when $g \leq 0.25$, the presence of parental effects changes population dynamics by increasing the minimum growth rate that leads to cyclical population dynamics, but does not change the shape of population cycles. On the other hand, if $g > 0.25$, then population dynamics become unstable through complex eigenvalues, which cause Hopf bifurcations (Figure 4). In this case population dynamics are unstable if

$$r > \frac{1}{g}. \tag{8}$$

This means that when parental effects cause 25% or more of the total density-dependent variation in demographic parameters, population dynamics can be radically different from the dynamics predicted by models without parental effects (contrast Figure 3B with Figure 3C).

In other words, the shape of population cycles is determined only by the proportion of density feedback from parental density effects (g). For parental effects to allow kinds of population dynamics that would never be predicted by models without parent effects, g, the proportion of density feedback from parents *vs.* offspring, must be greater than 0.25. Thus, to ask whether parental effects in *C. pensylvanica* might be an important factor in population dynamics, I will use the experimental data to estimate the value of g.

D. Empirical estimate of g in *C. pensylvanicaa*

As shown in the Appendix, the parameter g can be directly estimated from parent and offspring density effects as follows:

$$g = \frac{b}{b+c} \tag{5}$$

where b and c are the effects of adult plant density in the parent and offspring generations, respectively, on ln-transformed offspring mass (equations 1-5; see Appendix). Thus, it is possible to calculate a maximum likelihood estimate of g simply from the slope estimates presented in Table 1 (g = 0.77/(0.77+0.148) = 0.35). For any fixed nonzero value of c (offspring effect), the probability that g is zero is equal to the probability that b (the parental effect) is zero. Because b is different from zero with marginal statistical significance (p = 0.08), g is also different from zero with the same

"significance". However, this analysis does not give any information about our confidence in the distribution of g, particularly around the value of $g = 0.25$, which is the critical value for population dynamics.

To understand the distribution of g, I adopt a Bayesian perspective and treat the experimental data as fixed and the estimates of parent and offspring effects (b and c) as random variables, with probability distributions based on the data. (See Walters and Ludwig (1994) for a discussion of Bayesian reasoning with applications to ecological parameter estimation.) Given the assumptions of an uninformative prior distribution and normally distributed errors, both of which are appropriate for these data, Bayesian posterior distributions for the parameters b and c are normally distributed with mean and variance identical to those of parameters estimated by least-squares methods (Table 1; Box and Tiao 1973). Because b and c are independent random variables, the probability distributions of functions of these variables (such as g) can be calculated from their joint distributions (Box and Tiao 1973). I found this distribution numerically by taking all values of b and c with posterior probabilities > 0.001. Values of g were calculated for each b, c pair. The probability of any given b, c pair is the product of their two independent probabilities, and the probability of obtaining for any given value of g is the sum of the probabilities of all pairs of slopes which generate that value of g.

Based on this analysis, g is likely to be large enough to affect population dynamics in *C. pensylvanica*. 69% of the area of the posterior probability distribution of g fell in the Hopf bifurcation region ($.25 < g \leq 1$), where population cycles are very different from cycles predicted by models without parental effects, while only 25% was in the flip bifurcation region of parameter space ($0 \leq g \leq .25$), where parental effects could be statistically significant, but not large enough to affect population dynamics (Figure 5). The remaining 6% of the distribution yielded values of g that were less than 0

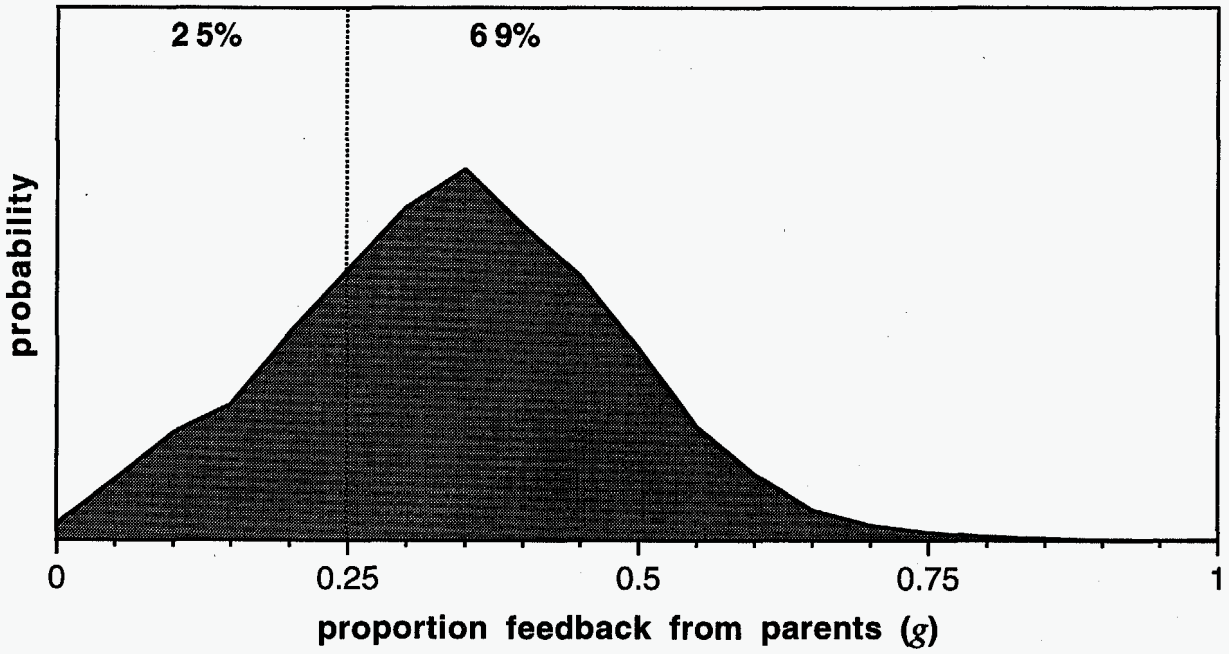

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Figure 5. Probability distribution for the parameter g fitted to experimental data. The horizontal line divides values of g which predict flip and Hopf bifurcations (see Figures 2-4 and text). Numbers at the top of each section are fraction of the probability density that falls in that section. These numbers do not sum to one because there is some probability of that g is greater than 0 or less than 1.

or greater than 1 (which would not be consistent with this model of population dynamics).

Discussion

Parental density effects change the predictions of plant population dynamics models by predicting a completely different kind of population cycles (cycles caused by Hopf bifurcations) from models without parental effects. This is significant because earlier models of density-dependent population regulation in plants have only predicted flip bifurcations or stable dynamics (Figures 3B and 3A). It is important to recognize that analyses based on these models only ask whether cycles characteristic of flip bifurcations are possible and would attribute cycles caused by Hopf bifurcations (Figure 3C) to "noise". In fact, of the three published studies which show cyclical dynamics in plant populations, one (Symonides *et al.* 1987) shows cycles consistent with flip bifurcations, while the other two (Tilman and Wedin 1991, Chapter 1) show cycles that are only consistent with Hopf bifurcations.

For example, in an earlier study (Chapter 1), I monitored population dynamics and characterized the shape of population cycles using autocorrelation functions (ACFs, Chatfield 1989; Figures 6 and 7). ACFs are correlations between time series observations separated by different temporal lags. In other words, the autocorrelation between observations separated by a lag of one generation is the correlation between parent and offspring density, and the autocorrelation at a lag of two generations is the correlation between parent and grand-offspring densities. ACFs of unstable population dynamics caused by flip bifurcations are characterized by negative correlations at one-generation lags (parents *vs.* offspring), and positive (cyclical dynamics) or weaker negative (chaotic dynamics) autocorrelations at lags of two generations (parents *vs.*

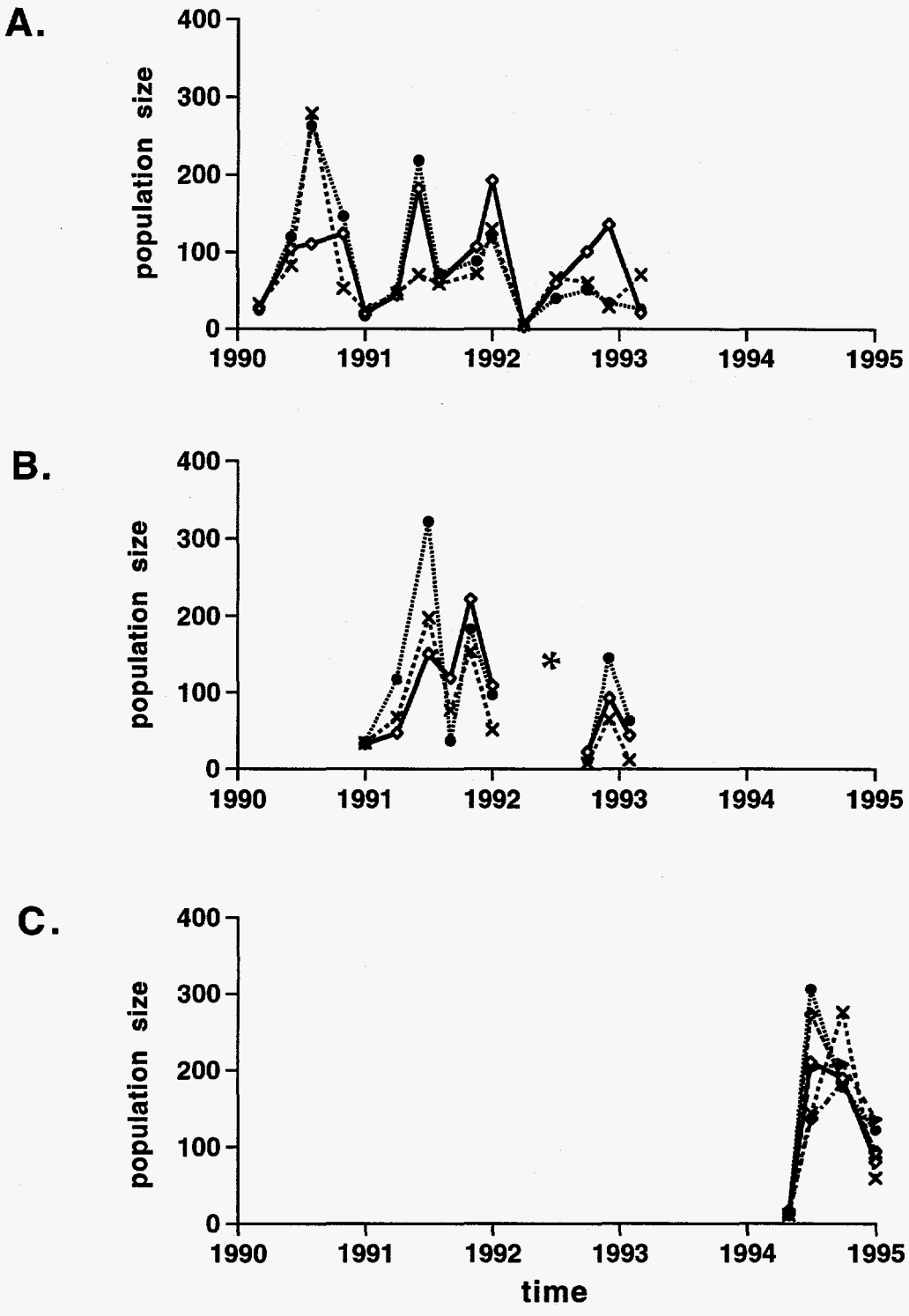

Figure 6. Population dynamics in experimental populations of Cardamine pensylvanica in 3 separate experiments (A,B,C). * indicates that no data were collected for this generation. Data in A are from Crone and Taylor (1996).

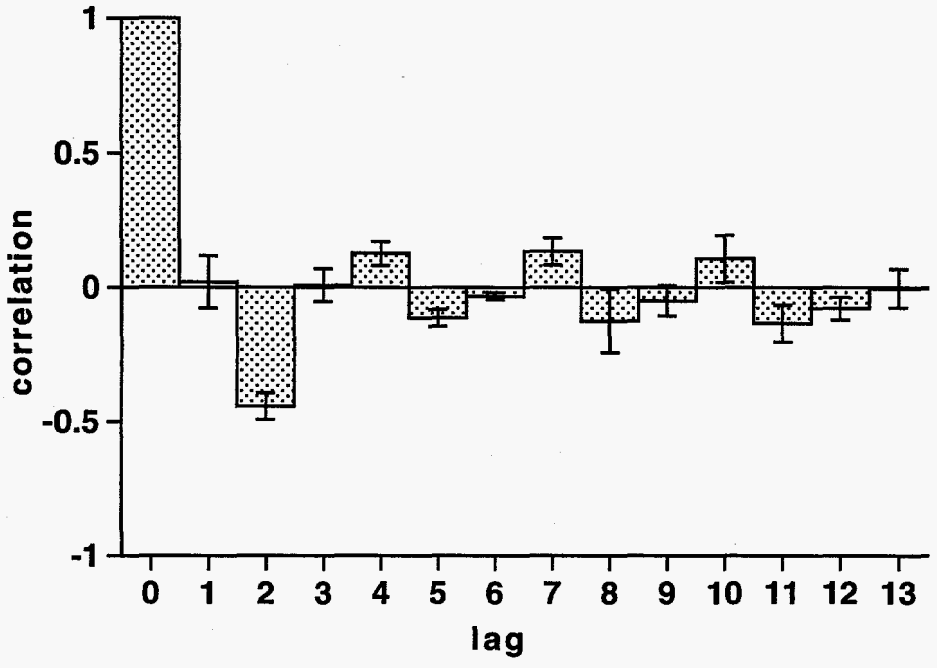

Figure 7. Autocorrelation function of time series data in Figure 5A. Correlations are means of ACFs of three replicate populations ± standard error. Significant correlations at lags of two generations are consistent with Hopf bifurcations. Lack of significant correlations at one-generation lags show that cycles were not caused by flip bifurcations. (Figure from Crone and Taylor (1996).)

grand-offspring; Nisbet and Gurney 1982). ACFs of cycles caused by Hopf bifurcations have strongest negative correlations at lags of two or three generations (parents *vs.* grand-offspring or great-grand-offspring; Berryman 1992). Because the ACFs of population cycles we observed in *C. pensylvanica* had largest negative correlations at lags of two generations and no significant correlations at one-generation lags (Figure 7), these cycles were only consistent with Hopf bifurcations.

The significance of the parental feedback for population dynamics in *C. pensylvanica* can be illustrated by comparing the time series data and ACFs to predictions of population dynamics models that do and do not include maternal effects, when parameterized with experimental estimates of demographic parameters r (as measured in Chapter 1) and g (Figure 8). Without parental feedback (Figure 8A and 8B) population dynamics are cyclical, but are always governed by flip bifurcations and therefore cannot reproduce the actual dynamics, even in the presence of environmental noise. Models including parental feedback predict longer cycles, and are more similar to the observed dynamics (Figure 8C and 8D). When demographic parameters are modeled as random variables (with distributions based on Bayesian posterior distributions), simulations of population dynamics with parental effects (Figure 8D) can be strikingly similar to the observed dynamics (Figure 6).

Generally speaking, parental effects in *C. pensylvanica* are similar to parental effects in other species: statistically detectable, but smaller than direct effects of offspring environment (analogous to $0 > g > 0.5$). Because other studies of parental environmental effects in plants have emphasized changes in individual phenotype, rather than population dynamics, it is necessary to revisit these studies to compare their results to the data presented here. Unfortunately, although previous studies have reported effects of parent density on seed and seedling performance (Roach and Wulff

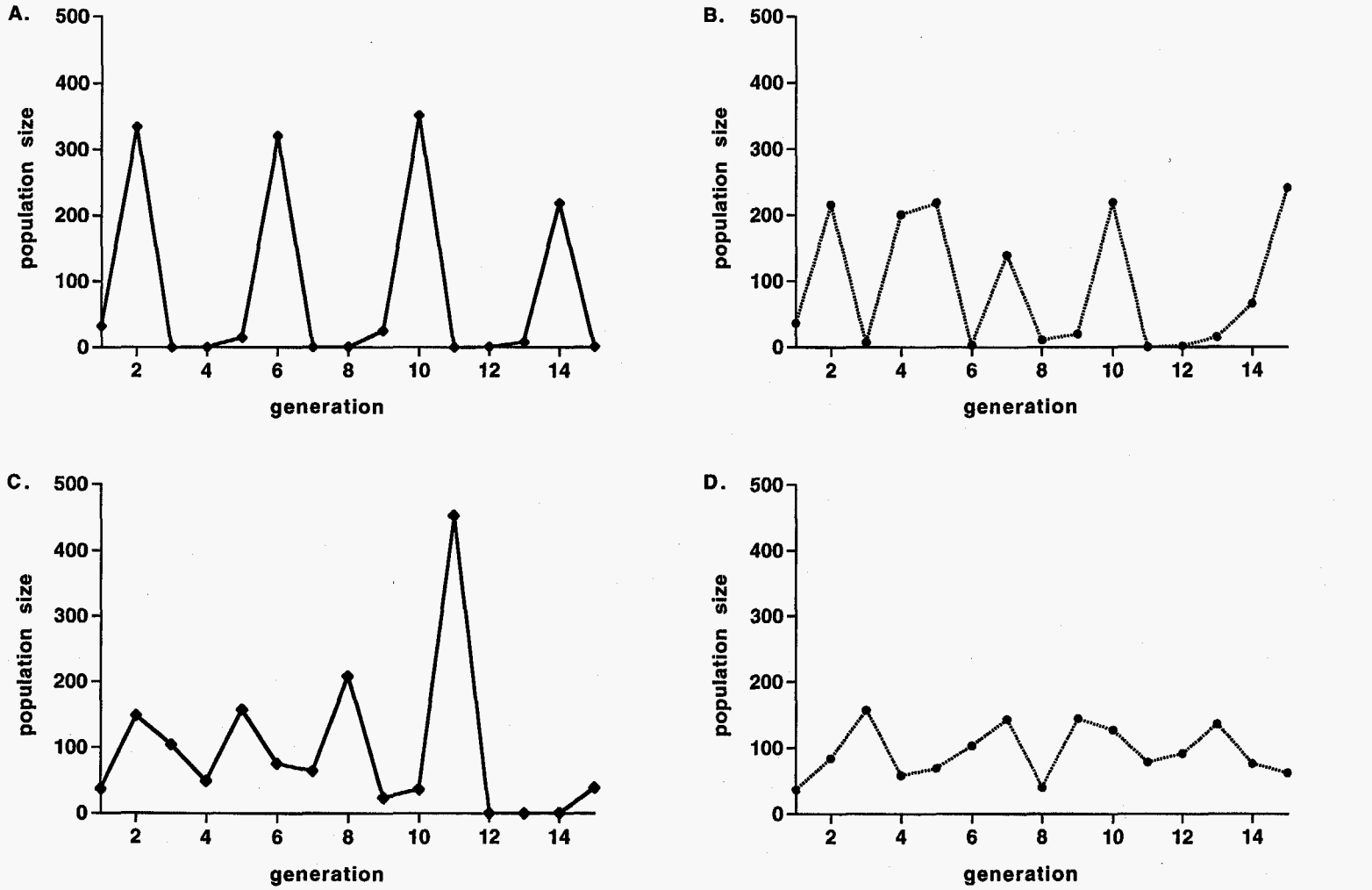

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Figure 8. Predicted time series from models with and without parental effects. In deterministic simulations (A and C), the nonlagged Ricker model (Ricker 1954) and the parental effects model (equation 5) were simulated using maximum likelihood estimates of r and g obtained in independent experiments. In stochastic simulations (B and D), values for parameters were drawn each generation from the probability distribution for each parameter. Shown is the best (least absolute deviation from time series data) of 20 stochastic simulations. Visual inspection of many other simulations indicated that these were typical "good" matches of each model to the data.

1987, Mazer and Wolfe 1992, Platenkamp and Shaw 1993), I have not found any published studies that report effects of parental density on the adult mass or fecundity of offspring. Nonetheless, there a few studies of the effects of nutrient availability on annual plants from which it is possible to estimate the relative magnitudes of the effects of parental and offspring-generation environmental conditions on offspring plant size (Table 2). Of the six experiments for which I was able to compare parental and offspring nutrient effects, two report nonsignificant parental effects, two report significant effects which do not translate into relative values that would change population dynamics ("g" < 0.25, see Table 2), and two have significant effects with relative magnitudes high enough to change predictions of population dynamics ("g" > 0.25). Obviously, these analyses should be interpreted with caution, because the studies were not designed for this purpose, and different estimates of size were often used in estimating parental *vs.* offspring effects. Nonetheless, they show that the magnitude of parental density effects in *C. pensylvanica* is not atypical of parental nutrient effects in other species. This suggests that parental effects might be an important component of population dynamics in numerous plant species.

In extending these results to other species, it is also important to note that the qualitative shift in population dynamics that results from parental density effects can also result from other ways in which parent density can influence offspring performance. For example, litter from plants in previous generations can accumulate and suppress plant germination and fecundity in future generations (Bergelson 1990). In addition, theoretical investigations have shown population cycles due to age-structured demographic rates and plant-pathogen interactions (May and Anderson 1983, Wilkan 1994). Although the specific functional forms of models that describe these processes are different from the model of parental effects presented here, they lead to Hopf

Table 2. Parental and offspring nutrient effects reported in other studies. Slopes were estimated by least squares regressions when more than two treatment levels were used, and by taking the difference between size at low nutrients and high nutrients when only two were used. To scale parameters, estimates were divided by plant size at low nutrients. Relative values were calculated from these using equation (5). Note that in many cases the same set of plants was used to measure both parent and offspring effects, that experiments were designed to test the null hypothesis that parent and offspring effects were zero, rather than estimating parameters, and that, given the published data, I was usually unable to calculate confidence intervals for these slopes. Nonetheless, the parental density effect reported in *C. pensylvanica* falls well within the range of values reported in other studies. * denotes effects which were statistically significant ($p < 0.05$) in the analysis reported in the original publication, and [1] denotes effects for which I estimated values from graphically presented data. In Stratton (1989), C and NC refer to offspring grown in competitive and noncompetitive conditions.

	species	# levels	offspring nutrient effect		parental nutrient effect		relative values ($\propto g$)
			trait measured	slope	trait measured	slope	
Miao and Bazzaz 1990, Miao, Bazzaz and Primack, 1991	*Plantago major*	2	biomass (g) @ 145 days	-0.36*	biomass (g) @ 8 months[1]	-0.09*	0.20
	Plantago rugelii	2	biomass (g) @ 173 days	-0.63*	biomass (g) @ 9 months[1]	0.09	-0.17
Wulff and Bazzaz 1992	*Abutilon theophrastii*	2	biomass (mg) @ 56 days[1]	-0.86*	biomass (mg) @ 56 days[1]	-0.14*	0.14
Stratton 1989	*Erigeron annuus* (C)	2	basal stem diameter (mm)	-0.23*	biomass (g) @ 8 weeks	-0.30*	0.57
	Erigeron annuus (NC)	2	basal stem diameter (mm)	-0.23*	biomass (g) @ 11 weeks[1]	-0.07	0.23
Aarssen and Burton 1990	*Senecio vulgaris*	3	biomass (g) @ 12 weeks	-0.34*	biomass (g) @ 7.5 weeks	-0.20*	0.37

bifurcations, and therefore can allow both long cycles in population size and cycles at relatively low population growth rates (see Turchin and Millstein 1993). Thus, although the parental effects model analyzed here describes an annual plant with discrete generations, other forms of delayed density dependence (litter accumulation and age structure) show that cycles can occur in perennial, as well as annual, species. Because of the difference between cycles driven by flip *vs.* Hopf bifurcations, cycles caused by these mechanisms would neither be predicted by simple discrete generation models nor detected by statistical tests derived from these models. I do not know whether there are extant data sets of dynamics of natural plant populations over several generations that could be examined for population cycles consistent with Hopf bifurcations. (To date, I have found very few.) Nonetheless, the potential for cyclical dynamics in plant populations is clearly worthy of further consideration in light of the potential for cycles due to Hopf bifurcations.

This is the first study to relate parental environmental effects to population dynamics in plants. Parental density effects in *C. pensylvanica* populations not only changed the phenotype of offspring plants, but were large enough to cause a qualitative shift in patterns of long-term population dynamics. Thus, in addition for their long-recognized potential for changing the evolutionary dynamics of plant populations, parental environmental effects may be a significant factor in numerical population dynamics.

Appendix

In this section, I show that estimates of the relative effects of parental and offspring density on offspring biomass (b and c in equation 3) can be used to calculate

the time delay parameter (g in equation 5). My goal is not to prove that this is the best possible model of plant population dynamics. In fact, several different assumptions about the relationship between biomass and fecundity in one generation and the number of adult plants in the next generation fit the data almost as well and make similar predictions about population dynamics, but that is beyond the scope of this paper. In this appendix, I simply show that these steps are consistent with empirically estimated functional relationships.

Based on preliminary experiments (see Methods), I know that ln-transformed mass of single isolated plants (y_t) is proportional to ln-transformed seed rain (s_{t+1}):

$$s_{t+1} = p y_t^{\,z} \tag{A1}$$

and that ln-transformed seed rain is proportional to ln-transformed recruitment of adult plants into that generation (N_{t+1}):

$$N_{t+1} = s_{t+1}^{\,w}. \tag{A2}$$

In order to relate effects of density (N_t) on ln-transformed average mass on single plants (y_t from equation 3) to ln-transformed replacement rates (N_{t+1}/N_t from equation 4), the following relationship must also hold:

$$\ln\left[\frac{N_{t+1}}{N_t}\right] = u - v\ln[y_t] \tag{A3}$$

so that

$$\ln\frac{N_{t+1}}{N_t} = u - v\left(a - bN_{t-1} - cN_t\right) = uva - vbN_{t-1} - vcN_t \tag{A4}$$

and

$$\frac{b'}{b'+c'} = \frac{vb}{vb+vc} = \frac{b}{b+c}. \tag{A5}$$

Taken together, equations A2 and A3 imply the following relationship between density, biomass, and seed rain:

$$\ln[s_{t+1}] = k - l\ln[N_t] + m\ln[y_t] \qquad \text{(A6)}$$

It is not possible to test whether this relationship is consistent with seed rain data from the experiment used to generate A1, because all of the plants in that experiment were planted as single plants per pot ($N_t = 1$). However, in an earlier experiment (E. Crone, unpublished), I measured plant density, plant mass, and the number of seed pods per plant (which is likely to be proportional to seed rain) in *C. pensylvanica*. I calculated a regression of the form described by A6, and found that ln-transformed fecundity was linearly related to ln-transformed density and ln-transformed average plant mass (SAS GLM procedure; N=242 pots of plants, $p < .0001$ for both factors (Type III SS), $R^2 = 0.656$ for the full model). Although this does not test whether A6 is the best possible description of the relationship between density, mass, and fecundity in *C. pensylvanica*, it shows that these relationships are not inconsistent with A6.

Acknowledgments

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CHAPTER 3.

Delayed density dependence and the stability of interacting populations and subpopulations

Abstract

Theoretical investigation of the dynamics of populations with discrete generations have traditionally been based on simple models of the form $N_{t+1} = f[N_t]$. However, recent studies of the dynamics of natural populations indicate that density dependent population regulation probably takes place over many generations ($N_{t+1} = f[N_t, N_{t-1}, ...]$). In this paper, I contrast the stability properties of discrete-generation models of population growth which do and do not include delayed density dependence. Relative to non-delayed models, inclusion of delayed density dependence changes the shape of population cycles (flip *vs.* Hopf bifurcations) and decreases the range of parameters which predict stable equilibria. I also explore extensions of these models that include interspecific competition and coupling of spatially isolated patches. In both cases, delayed density dependence significantly changes the way in which demographic parameters scale to overall dynamics. For example, when delayed density dependence does not differ between two species, the asymptotic stability of both species is determined by a weighted average of the population growth rates of the two species. However, when species differ in time delay, some pairs of species that would both exhibit cyclical or chaotic dynamics in isolation can stably coexist. Analogous conclusions hold for the effects of deterministic spatial environmental variation among coupled patches. This implies that inclusion of delayed density dependence in investigations of population dynamics can dramatically change the inferences we draw from mathematical models, and that further investigations of the effects of delayed density dependence are warranted.

Introduction

In recent years, our understanding of the processes controlling the dynamics of natural populations has been considerably changed by the recognition that density-dependent population regulation can take place at a variety of temporal scales. Ecologists have demonstrated that past generations can affect population growth rates through a number of time delay mechanisms, including density-mediated changes in habitat quality, maternal (or parental) effects of population density on offspring quality, age structure in populations with overlapping generations, and population regulation through predator-prey or host-pathogen interactions (Royama 1981, Roach and Wulff 1987, Mousseau and Dingle 1991, Bergelson 1990, Turchin and Taylor 1992, Hornfeldt 1994, Rossiter 1994). Furthermore, first-order discrete-generation population models ($N_{t+1} = f[N_t]$) cannot reproduce the dynamics of most natural populations which cycle in size (*e.g.* Hassell *et al.* 1976). However, models including density feedback from more than one past generation ($N_{t+1} = f[N_t, N_{t-1}, ...]$) can often explain cycles in natural populations (Turchin and Taylor 1992, Turchin 1993, Ellner and Turchin 1995). These results do not prove that delayed density dependence causes cycles in natural populations. However, they do show that population cycles due to direct density dependence are probably unusual in natural populations, but cycles due to delayed density dependence may be relatively common.

Nonetheless, the majority of theoretical studies of discrete-generation models of population dynamics have included only direct density dependence. Thus, the goal of this paper is to point out some of the consequences of variation in the temporal scale of population regulation. Specifically, I investigate three simple models which include delayed density dependence: a model of single species population growth, a model of two competing species, and a coupled-patch model of population dynamics. In the first

analysis, I show how the predictions of a model with delayed density dependence differ from the predictions of a model with only direct density dependence. In the second and third analyses, I investigate how the presence of differences in the time scale of regulation (across species and habitat patches) scale to overall population dynamics, and contrast this with the effects of differences in other demographic parameters (such as population growth rates). In general, I emphasize simple aspects of population dynamics, such as predictions of stable or unstable dynamics. I make no attempt to explore all the dynamical behaviors of these models, and I do not distinguish chaotic dynamics from high period or quasiperiodic cycles. Rather, my emphasis is a relatively straightforward difference between the qualitative dynamics of models with and without delayed density dependence, and how this result changes the effects of coupling between spatial patches and interacting species.

Models and results

1. single species population dynamics

Single-species population dynamics can be described by a simple time-delayed extension of a discrete exponential model of population growth (*e.g.* Turchin 1990):

$$A_{t+1} = A_t \exp[r(1 - \frac{(1-g)A_t + gA_{t-1}}{k})] \qquad (1)$$

where A_t is the density of single species at time t, r is the density independent population growth rate, k is the equilibrium population size (or carrying capacity), and g is the proportional feedback on population growth from past-generation density (making $(1 - g)$ the proportion of feedback from current density). At a very abstract level, the parameter g is a simple phenomenological way to incorporate variable time delays into population growth while allowing r and k to retain their traditional ecological values.

I have chosen to investigate this model because of its similarity to commonly analyzed models of direct density dependence. Furthermore, in earlier studies, I found that this model was sufficient to explain population cycles I had observed in annual plant populations (Chapter 1), due to effects of parental density on offspring performance (Chapter 2). Similar parental effects have been widely observed in plant and insect populations (Roach and Wulff 1987, Mousseau and Dingle 1991). Other mechanisms generating delayed density dependence give slightly different forms to time delayed population growth equations, but the qualitative behavior of these equations is similar (Berryman 1992, Turchin and Taylor 1992, Turchin and Millstein 1993, but see Ginzberg and Taneyhill 1994).

Like the non-lagged version of (1), there is a single non-trivial equilibrium population size at $A^* = k$. To determine whether this equilibrium is stable, it is necessary to rewrite the equations as a pair of equations with a dummy variable ($\tilde{A}_t$) to represent the time delay (Turchin and Millstein 1993):

$$\begin{aligned} A_{t+1} &= A_t \exp[r(1-\frac{(1-g)A_t + g\tilde{A}_t}{k})] \\ \tilde{A}_{t+1} &= A_t \end{aligned} \tag{2}$$

This pair of equations can then be analyzed for asymptotic stability at equilibrium using standard techniques for stability analysis (May 1973). The nontrivial equilibrium is asymptotically stable if

$$\left| \frac{1-r+gr \pm \sqrt{-4gr+(-1+r-gr)^2}}{2} \right| < 1, \tag{3}$$

so the dynamics of the model are affected by the relationship between the growth rate r and time delay g. Like many similar models, this model has two criteria for stability (Figure 1). If $g < 0.25$, then population dynamics are unstable if

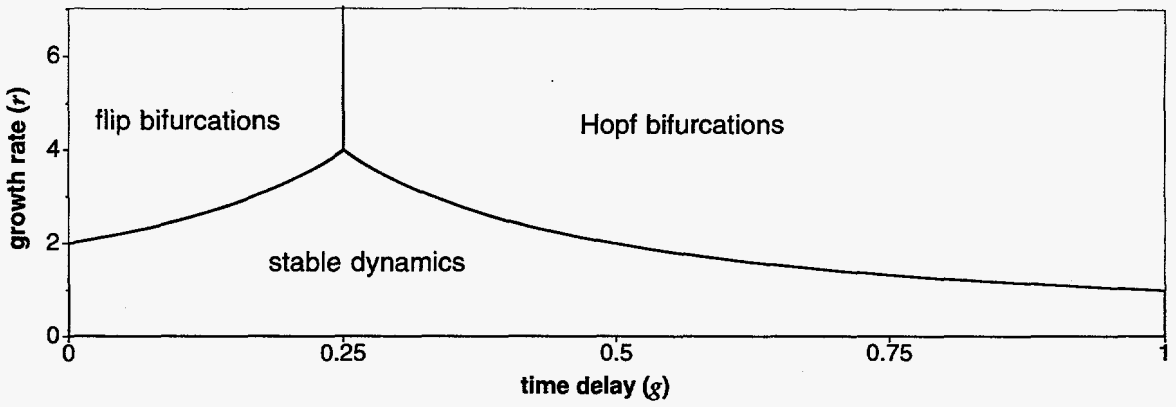

Figure 1. Stability of single-species delayed Ricker equation as a function of population growth rate (r) and time delay (g).

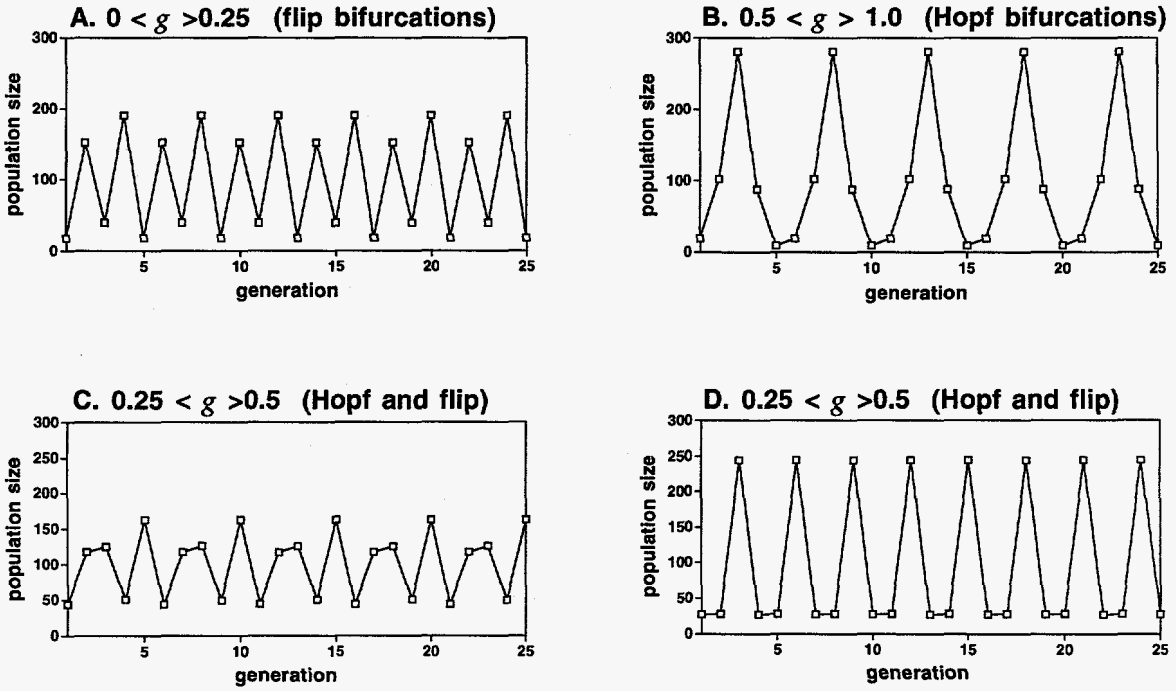

Figure 2. Simulations demonstrating typical population cycles from regions of parameter space with (A) flip bifurcations, (B) Hopf bifurcations, (C and D) solutions for both flip and Hopf criteria.

$$r > \frac{2}{1-2g}. \tag{4}$$

This instability is due to real negative leading eigenvalues of the Jacobian stability matrix with magnitude greater than one, and leads to the familiar "period doubling" route from stability to chaos (flip bifurcations; Thompson and Stewart 1986). If $g > 0.25$, then population dynamics are unstable if

$$r > \frac{1}{g}. \tag{5}$$

This instability is due to complex leading eigenvalues, which cause slow, often quasiperiodic oscillations from high to low density (Hopf bifurcations; Thompson and Stewart 1986).

Visual inspection of simulated time series suggests that these two criteria divide parameter space into three regions with qualitatively different kinds of population cycles. When $g < 0.25$, population dynamics are typical of flip bifurcations (Figure 2A). When $g > 0.50$, population dynamics are typical of Hopf bifurcations (Figure 2B). When $0.25 < g < 0.50$ (*i.e.* when the Hopf criterion (4) determines the initial bifurcation but nonnegative solutions still exist for the flip criterion (5)), population dynamics appear to combine aspects of both the sharp crashes from high to low density that typify flip bifurcations and the quasiperiodic cycles of Hopf bifurcations (Figures 2C and 2D).

2. two-species competition

Two-species competition can be described by a simple extension of the single-species model (1):

$$A_{t+1} = A_t \exp[r_A(1 - \frac{(1-g_A)(A_t + \alpha_A B_t) + g_A(A_{t-1} + \alpha_A B_{t-1})}{k_A})]$$
$$B_{t+1} = B_t \exp[r_B(1 - \frac{(1-g_B)(\alpha_B A_t + B_t) + g_B(\alpha_B A_{t-1} + B_{t-1})}{k_B})] \quad (6)$$

In this model A_t and B_t are densities of two species at time t, r_A and r_B are density independent growth rates for species A and B (respectively), g_A and g_B are the respective density feedback coefficients, and α_A and α_B are the Lotka-Volterra competition coefficients (the strength of interspecific competition relative to intraspecific competition). As in the single species model, there is a single nontrivial equilibrium at

$$A^* = \frac{k_A - \alpha_A k_B}{1 - \alpha_A \alpha_B}$$
$$B^* = \frac{k_B - \alpha_B k_A}{1 - \alpha_A \alpha_B} \quad (7)$$

A^* and B^* are both positive when

$$\frac{1}{\alpha_A} > \frac{k_B}{k_A} > \alpha_B \quad (8)$$

where A now arbitrarily designates the species with the smaller α, *i.e.* the better competitor. When criterion (6) is met both species will also increase at low density, so conditions for existence of a nontrivial equilibrium and protected coexistence are both met.

To simplify stability analysis of equations (6) to include only species pairs that coexist, I reparameterized equations (6) to be dimensionless as follows: $A'_t = A_t/k_A$, $\alpha_A' = \alpha_A k_B/k_A$, $B'_t = B_t/k_B$, $\alpha_B' = \alpha_B k_A /k_B$. In this model, species will coexist when

$$\frac{1}{\alpha_A'} > 1 > \alpha_B'. \quad (9)$$

Thus, all coexisting species pairs are mathematically equivalent to a species pair in equation (6) with $k_A = k_B = 1$, and $\alpha_A \leq \alpha_B \leq 1$. These criteria were used to limit the range of parameter space explored in numerical analyses (below).

Following Hastings (1993), stability of equations (7) was solved for species pairs using a numerical program in Mathematica (Wolfram 1991) in which eigenvalues of the Jacobian stability matrix were solved iteratively for species pairs that differ in demographic parameters. I present the results of these analyses graphically in diagrams outlining regions of stability in r_A / r_B parameter space for given values of $g_{A,B}$ and $\alpha_{A,B}$. Using maximum stable r values from the single species model (1) (Figure 1), r_A / r_B space can be divided into three regions (Figure 3): (I) the region in which both species are asymptotically stable in the absence of other species, (II) the region where one species is stable and the other unstable, (III) the region where neither species is stable in isolation. To evaluate the effects of competition on stability, I compare single-species population growth rates for which the two-species community is stable to the dynamics predicted by those growth rates for isolated species. For example, if the temporal variance in population size of the mixture is in any way an average of the variance of its component species in isolation, then species pairs in regions (I) and (III) should be stable and unstable, respectively. Conversely, if two stable species form an unstable mixture (unstable points in region I) or two unstable species form a stable mixture (stable points in region III), then the dynamics of the mixture are clearly outside the range of qualitative dynamics of species in isolation.

When competing species differ only in density independent growth rate (r), the dynamics of the mixture are intermediate between the dynamics of species in isolation (graphs along the diagonal of Figure 4); all stable points fall in regions I and II. However, when species vary in time delay, the shape of the stability region becomes highly

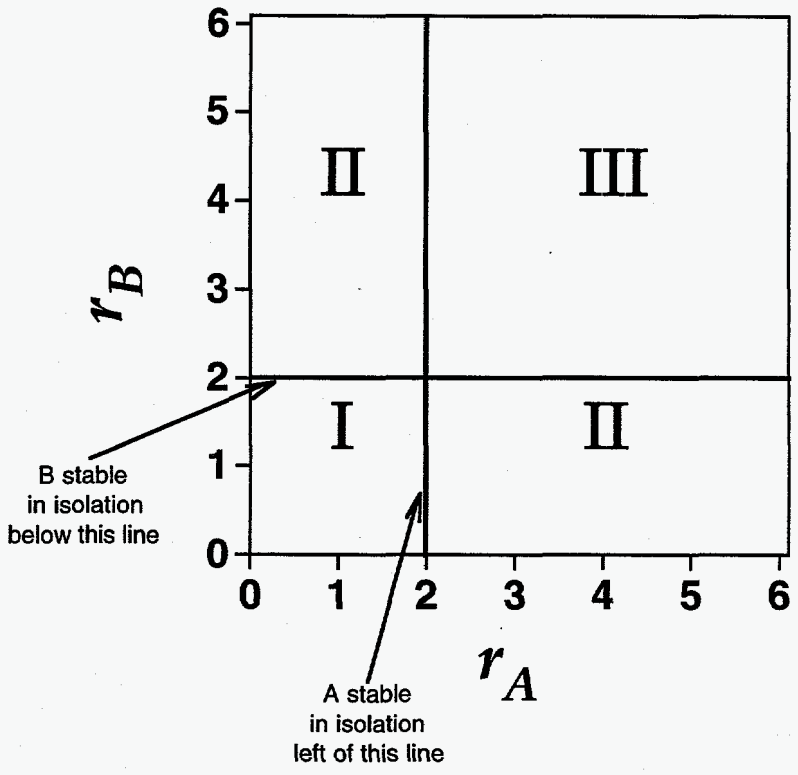

Figure 3. Stability diagram for two-species competition. The vertical line divides parameter space into regions in which species A is stable (left) or unstable (right) in isolation, and the horizontal line divides stable (lower) from unstable (upper) regions for species B in isolation. In this example, $g_A = g_B = 0$, so the maximum stable r for each species is 2 (see Figure 1). In the lower left corner of parameter space ($r_A < 2$ and $r_B < 2$; region I), both species would be stable in isolation. In the upper right corner ($r_A > 2$ and $r_B > 2$; region III), both species would be unstable in isolation. In the other two corners (region II), one species is stable and the other species unstable in isolation.

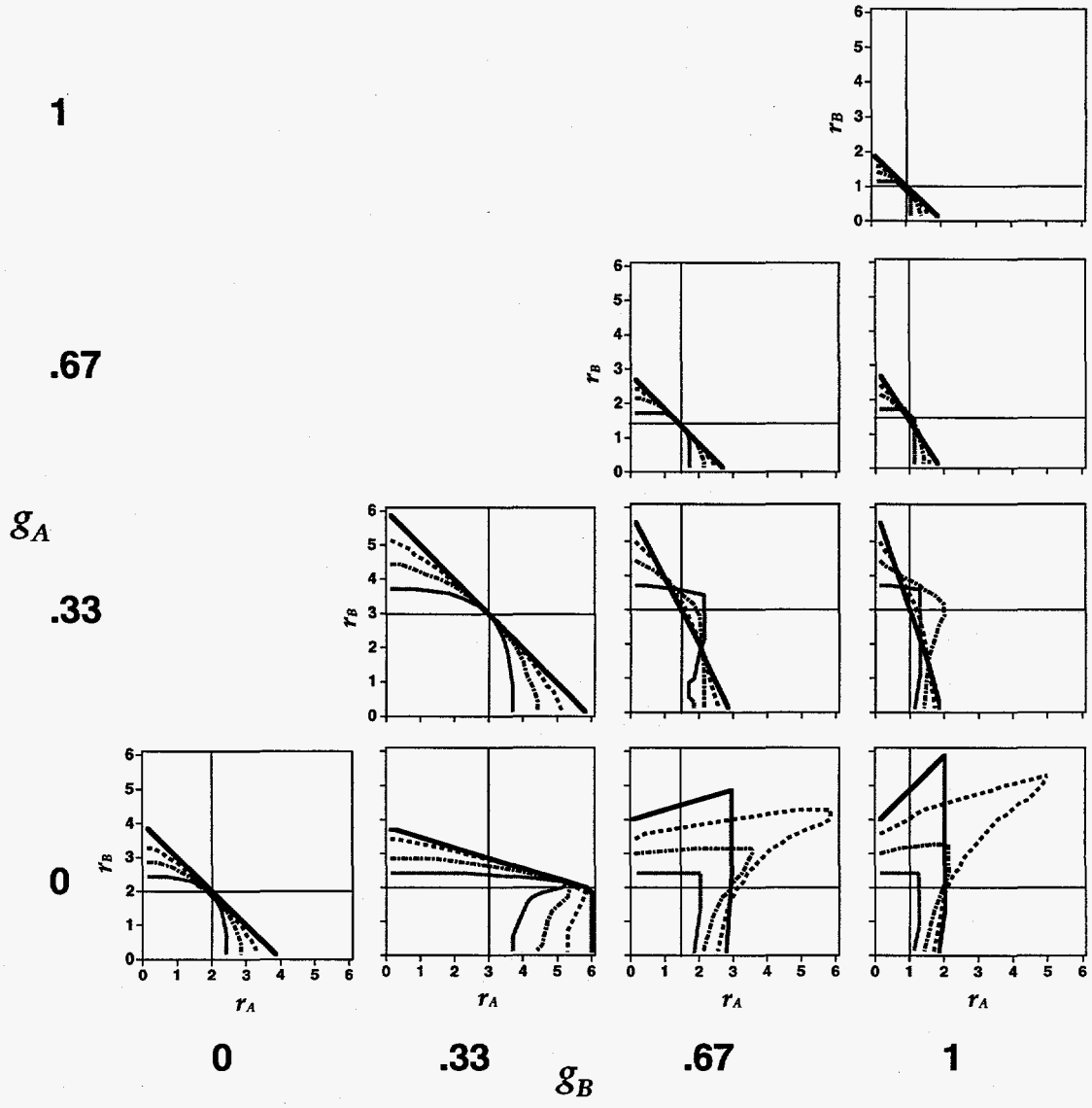

Figure 4. Stability regions for mixtures of species which vary in g and r. Eigenvalues were numerically solved at intervals of $\Delta r = 1/7$. Stability regions are outlined as follows: ——= isolated species (see Figure 2), ——= mixtures with $\alpha_A = \alpha_B = 0.25$, ·····= mixtures with $\alpha_A = \alpha_B = 0.5$, ·····= mixtures with $\alpha_A = \alpha_B = 0.75$, ——= mixtures with $\alpha_A = \alpha_B = 0.99$. Communities are stable in the lower left and unstable in the upper right.

irregular, and in many cases pairs of otherwise unstable species can stably coexist. The dynamics of the two species are always linked (*i.e.* both stable or both oscillatory) because of the density feedback between species. However, the magnitudes of density fluctuations in the species in unstable communities can vary greatly (see Figure 8 and Discussion).

When species pairs differ in competition coefficient and growth rate but not time delay, there are no stable combinations of species that would be unstable in isolation (Figure 5). In fact, there are only a few combinations of species in which the inferior competitor (higher α) can stabilize the dynamics of the superior competitor (points in upper left part of region II). On the other hand, stable dynamics in the superior competitor can easily stabilize the dynamics of the inferior competitor (points in lower right part of region II). When both g and in α vary, asymmetric competition does not change the qualitative effects of variation in time delay, but it does increase the ability of the superior competitor to stabilize population dynamics and decrease the same ability in the inferior competitor (Figure 6).

3. Coupled patch model

In the same way that demographic parameters can differ between species, demographic parameters can vary within a single species due to spatial environmental variation. For example, a species could be spread over patches in which a predator is and is not present (Hassell *et al.* 1991). Similarly, the importance of maternal effects has been shown to vary with offspring environmental conditions (Miao *et al.* 1992), which could differ between patches for numerous reasons. Thus, single species population growth (equation (1)) can be extended to a model of population dynamics across patches in which demographic parameters differ as follows:

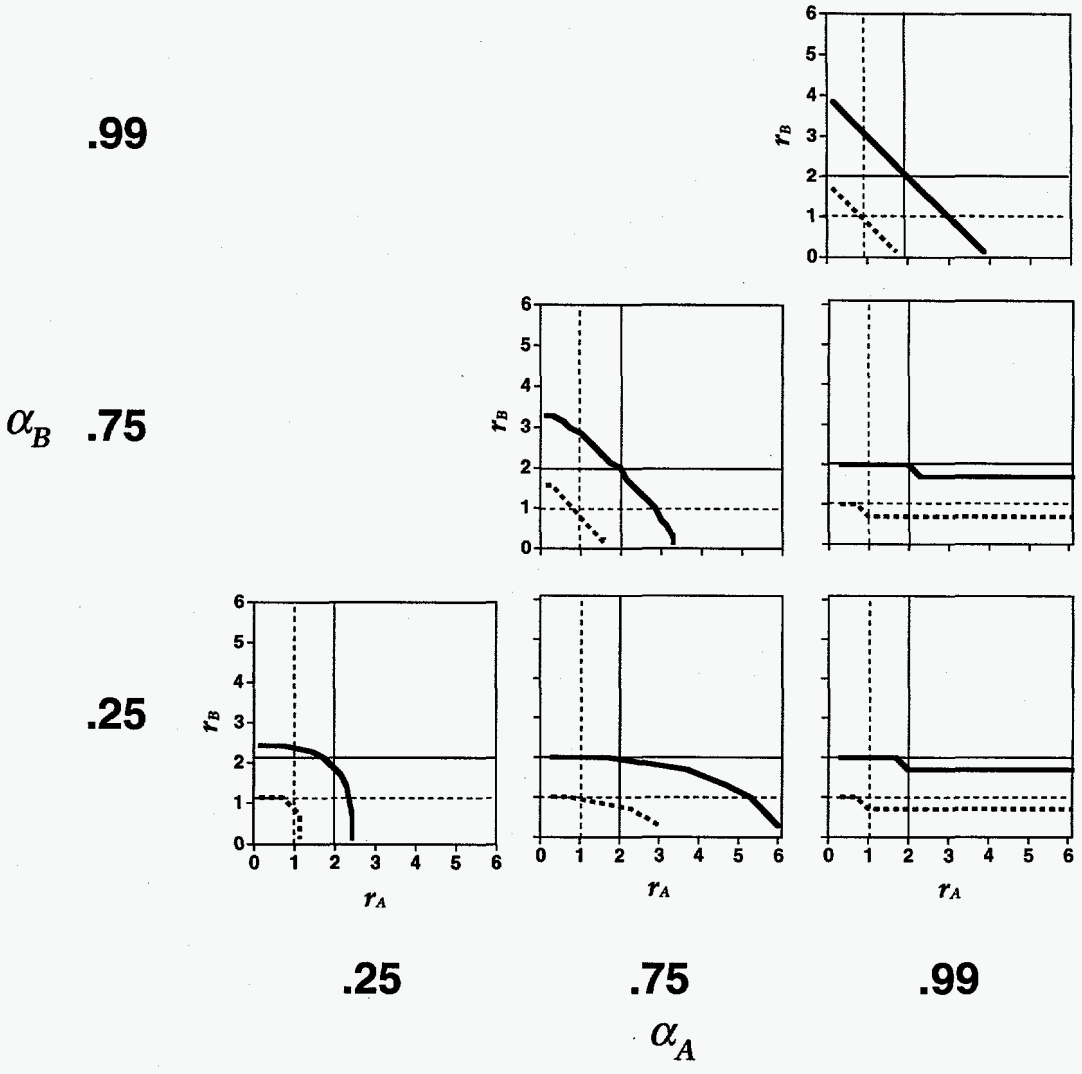

Figure 5. Stability regions for mixtures of species which vary in a and r. Eigenvalues were numerically solved at intervals of $\Delta r = 1/3$. Stability regions were solved for species pairs with $g = 0$ (——) and $g = 1$ (·····). Light lines indicate stable regions of isolated species and heavy lines indicate stability of two-species communities. Communities are stable in the lower left and unstable in the upper right.

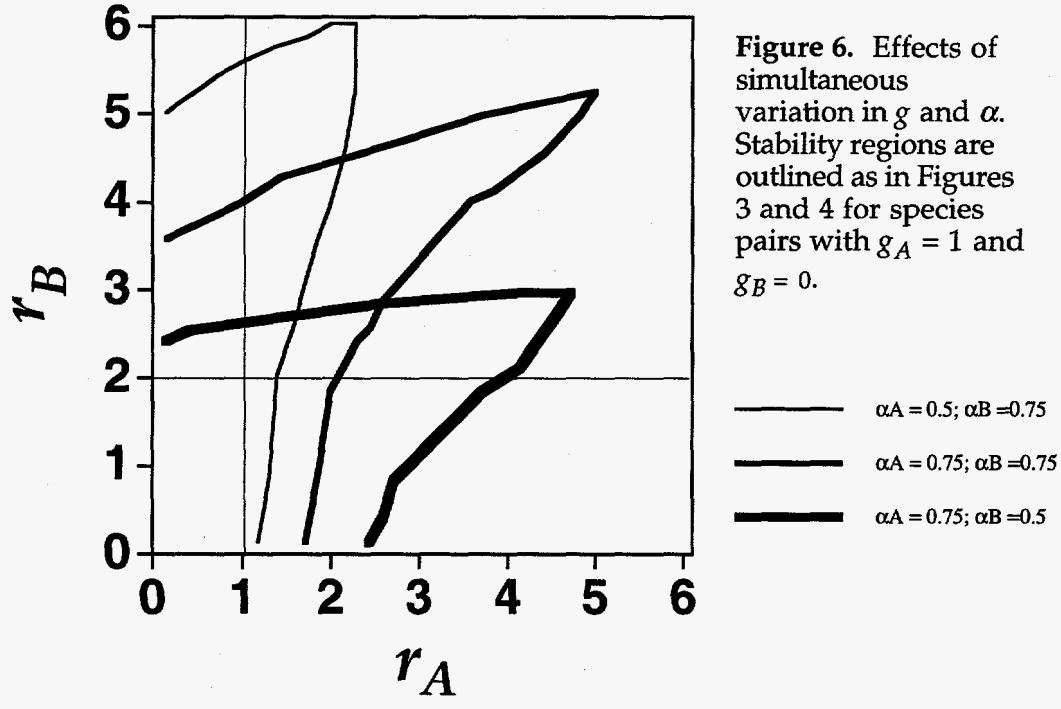

Figure 6. Effects of simultaneous variation in g and α. Stability regions are outlined as in Figures 3 and 4 for species pairs with $g_A = 1$ and $g_B = 0$.

$$A_{t+1} = (1-D)A_t \exp[r_A(1-\frac{(1-g_A)A_t + g_A A_t)}{k_A})] + DB_t \exp[r_B(1-\frac{(1-g_B)B_t + g_B B_{t-1})}{k_B})]$$

$$B_{t+1} = (1-D)B_t \exp[r_B(1-\frac{(1-g_B)B_t + g_B B_{t-1})}{k_B})] + DA_t \exp[r_A(1-\frac{(1-g_A)A_t + g_A A_t)}{k_A})]$$

(10)

where A_t and B_t are now population densities in patch types A and B, D is the rate of dispersal between patches, r_A and r_B are population growth rates in patches A and B, and g_A and g_B are the time delay parameters for each patch. If there is no delayed density dependence ($g_A = g_B = 0$), this model is equivalent to one of the models analyzed by Hastings (1993) and similar to one of the models analyzed by Gyllenberg *et al.* (1993).

As above, I analyzed this model by numerically solving for the eigenvalues of the Jacobian stability matrix. However, because non-delayed versions of this model have been extensively explored elsewhere, I simplified this analysis by using a relatively coarse-grained exploration of parameter space (intervals of $\Delta r = 1/3$), and solving for cases with regulation through entirely direct or entirely delayed density dependence ($g_A = 0, g_B = 0$; $g_A = 0, g_B = 1$; and $g_A = 1, g_B = 1$). Nonetheless, the results of this analysis are sufficient to demonstrate that spatial differences in the importance of delayed density dependence could significantly alter the ways in which dispersal affects population dynamics. As was true for interspecific differences, spatial environmental variation in growth rates causes population stability to be determined by a weighted average of growth rates in the two patch types, and the tendency for one patch to stabilize overall dynamics increases as the connectivity (dispersal fraction) increases (Figure 7) (see Gyllenberg *et al.* 1993 for further discussion). However, when a population is spread across habitat patches which differ in the time scale of population

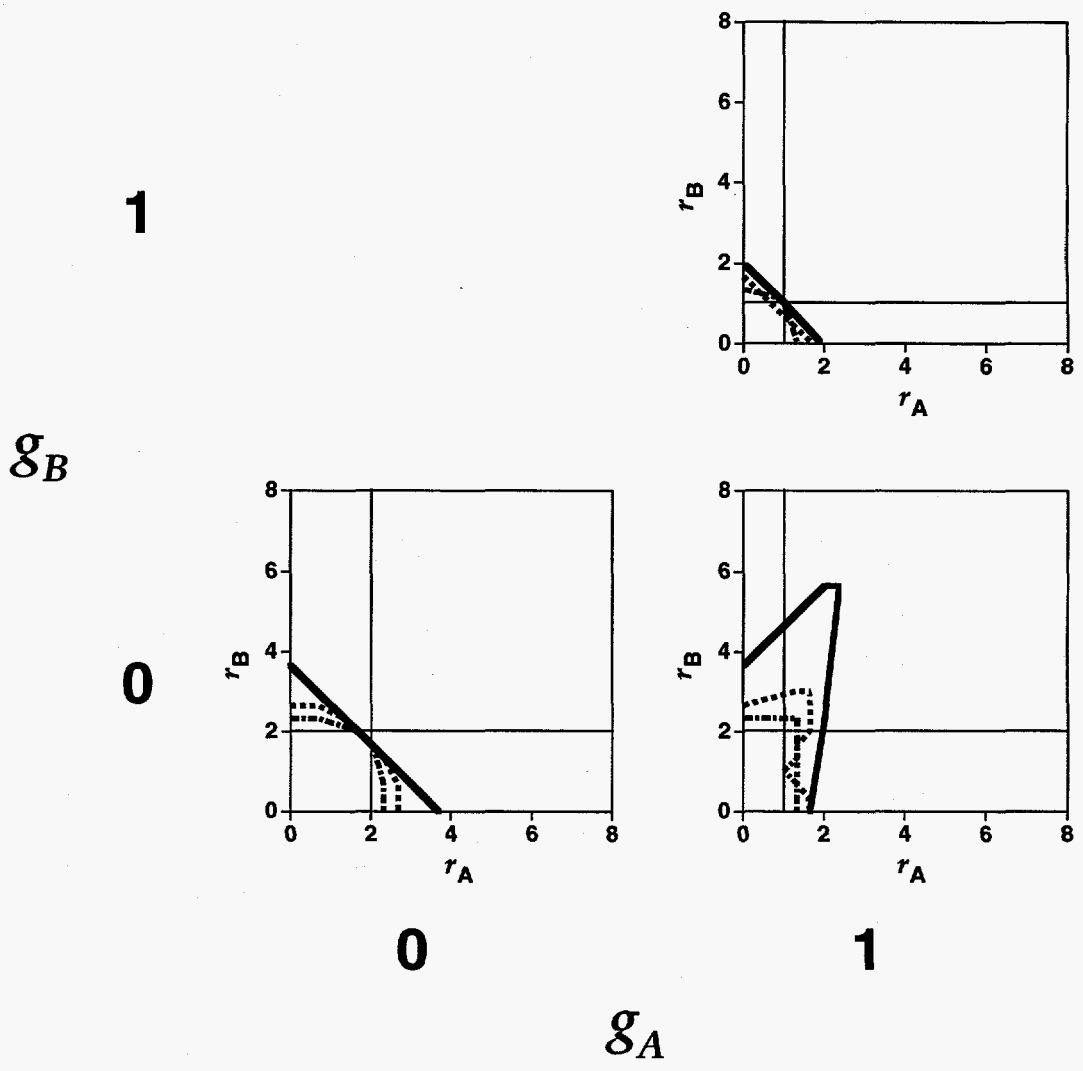

Figure 7. Effects of spatial variation in growth rate (r) and time delay (g) Eigenvalues were numerically solved at intervals of $\Delta r = 1/3$. Patch models without time delay ($g_A = g_B = 0$) are similar to models analyzed by Gyllenberg et al 1993 and Hastings 1993, but, like species diversity, environmental differences in time delay ($g_A = 1, g_B = 0$) allows unstable species to stably coexist . Dispersal fractions: $D = .495$ (——); $D = .375$ (- - - - -); $D =$ (·····).

regulation, dispersal among patches can stabilize population dynamics in some combinations of internally unstable patches (Figure 7).

Discussion

Population dynamics are affected by delayed density feedback. As the strength of delayed density dependence increases, population size cycles slowly over many generations, rather than jumping from high to low density in successive generations, and the range of population growth rates which lead to stable equilibria decreases. Visual inspection of the time series data analyzed by Hassell *et al.* (1976) and Turchin and Taylor (1992) suggests that it is this change in the shape of population cycles that differentiates the ability of models that do and do not include delayed density dependence to explain the dynamics of natural populations. The difference between the qualitative effects of different forms of density dependence has long been recognized among mathematical biologists (*e.g.* Nisbet and Gurney 1982). However, its utility in discriminating among possible causes of dynamics of natural populations (*e.g.* Chapter 2) has not been adequately emphasized in these studies.

In addition to affecting the shape of population cycles, delayed population regulation has a dramatic effect on the way in which differences in demographic parameters scale to overall population dynamics. In the absence of differences in the importance of delayed density dependence, the asymptotic stability or instability of both species is apparently determined by a weighted average of the demographic parameters of interacting species. However, when there are differences in time delay between species, these differences can significantly stabilize population dynamics, and individually unstable species can stably coexist. The effects of differences in time delay can be clarified through simulations of population dynamics of species pairs that go

from being independent to being linked by competition (Figure 8). When two competitors differ only in r, they both experience the same density at any given time; when one species is above its equilibrium population size, the other is also above, and both fall to lower density (Figure 8A). When species differ in competition coefficient (α) as well as r, species are responding to different densities, but because the difference in α changes the equilibrium population size as well, species still rise and fall synchronously (Figure 8B). When species differ in time delay, however, the density of the two species can be the result of two very different past densities, depending on the difference between population size in successive generations. It is then (and only then) possible for one species to increase while the other is decreasing. This allows some combinations of species that would be unstable in isolation to stabilize each other (Figure 8C).

The majority of previous studies of competition have looked at the effects of interspecific differences on species coexistence, rather than dynamical stability of coexisting species. Thus, a second counterintuitive effect of species diversity is also worthy of further explanation: when species coexist, the dynamics of an intrinsically unstable competitor can be stabilized by the presence of a stable superior competitor (Figure 5). In fact, if the asymmetry of competition is high enough, even an inferior competitor with wildly chaotic dynamics in isolation can stably persist, albeit at low density. This is because the stable superior competitor is relatively unaffected if the inferior competitor overshoots equilibrium and crashes, so the superior competitor approaches its equilibrium population size more or less as it would in isolation. As the superior competitor approaches its equilibrium, population growth in the inferior competitor is suppressed, and does not overshoot and undershoot its equilibrium. Because the probability of population extinction declines as variance in population size declines (Lande 1993), this implies that, in some cases, the presence of a stable superior

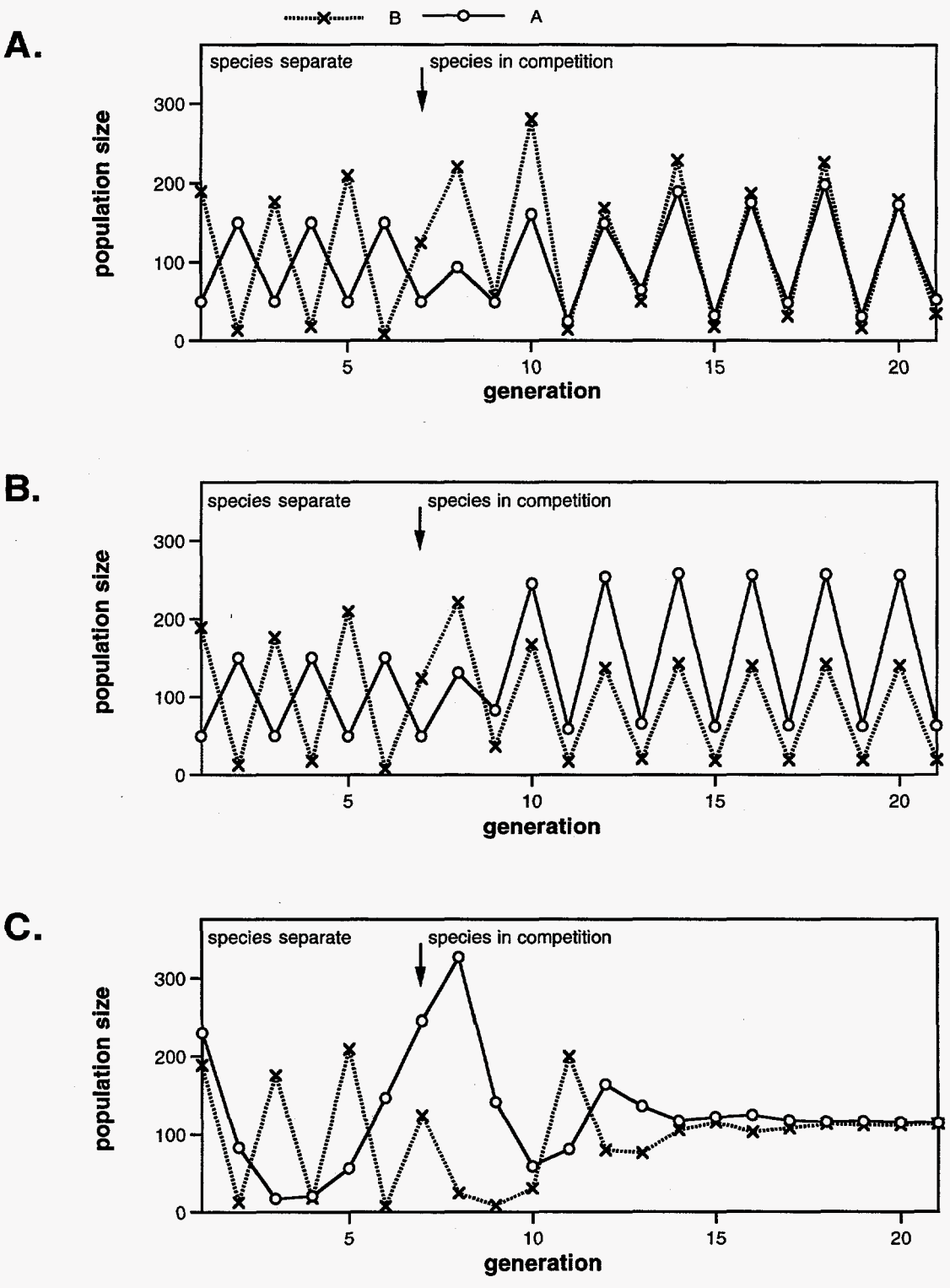

Figure 8. Demonstration of the effects of the introduction of a competitor on population dynamics. A) differences only in r ($r_A = 2.2, g_A = 0.0, \alpha_A = 0.75, r_B = 3.0, g_B = 0.0, \alpha_B = 0.75$); B) differences in r and a ($r_A = 2.2, g_A = 0.0, \alpha_A = 0.5, r_B = 3.0, g_B = 0.0$, aB = 0.75); C) differences in r and g ($r_A = 1.2, g_A = 1.0, \alpha_A = 0.75, r_B = 3.0, g_B = 0.0, \alpha_B = 0.75$). See text for further discussion.

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competitor could increase the persistence of an inferior competitor that would be unstable in isolation.

Mathematical models of two-species competition and are extremely similar to models of connected patches (equations 6 and 10). Thus, it is not surprising that the results of comparing stability of single patches to coupled-patch dynamics (Gyllenberg *et al.* 1993, Hastings 1993) are virtually identical to those of single-species *vs.* two-species competition (Figures 5 and 8). The effects of spatial environmental differences in time delay appear to be very similar to those of interspecific differences in time delay; when a population is spread across habitat patches which have different effects on delayed population regulation, dispersal among patches can stabilize dynamics across patches that would both be unstable in the absence of dispersal. This result is not equivalent to earlier results in which global stability is achieved through local patches fluctuating asynchronously (*e.g.* Reeve 1990, Hastings 1993). Although such behavior is also possible in this model, the analysis here defines population dynamics as stable only when dynamics are both locally and globally stable.

It would be intriguing to investigate the effects of delayed density dependence on aspects of population dynamics for which coupled patches and competing species differ. For example, an interesting difference between spatial and interspecific differences in demographic parameters in the absence of delayed density dependence is that patches linked by dispersal have stable two-point cycles over an extremely wide range of parameter values compared to single patch dynamics (Hastings 1993). This is because migration from less crowded patches can rescue population crashes and thus prevent chaotic dynamics. This kind of rescue effect is not caused by species diversity because the presence of a competing species can only prevent population crashes by suppressing reproduction, not rescue extinct populations by providing additional

recruits (Hassell and Comins 1976). Interestingly, similar stable two-point cycles do result from some explicit genetic models with recombination (Doebeli and Koella 1995), in which recombination allows recruitment from one genotype to other genotypes. However, extensions of these analyses to include delayed density dependence are beyond the scope of this paper.

Taken together, the analyses presented here show that inclusion of delayed density dependence can dramatically affect the inferences we draw from models of interacting populations or subpopulations. However, these analyses only begin to show the extent to which greater emphasis on the time scale of population regulation may change our theoretical understanding of population dynamics. Given the empirical evidence that delayed density dependence is important in natural populations, inclusion of this aspect of population regulation in further studies is clearly warranted.

Acknowledgments

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CHAPTER 4.

Population dynamics and evolution in experimental populations of an annual plant, *Cardamine pensylvanica*

Introduction

Numerous theoretical studies have pointed out the potential for interaction between demographic, environmental and evolutionary factors in determining the behavior of populations (*e.g.* Nicholson 1954, King and Anderson 1971, Roughgarden 1971, Asmussen 1979, Turelli and Petry 1980, Holt 1990, Endler 1992, Lynch and Lande 1993, Saloniemi 1993, Doebeli and Koella 1995, Hartt and Haefner 1995, Abrams in press). For example, reduction in population size may increase the importance of genetic drift, and thereby reduce the ability of populations to respond to selection pressure (Hedrick 1985, Hartt and Haefner 1995). Similarly, genetic changes in a population may alter demographic parameters, and shift the dynamics of that population (King and Anderson 1971, Pease *et al.* 1991). Nonetheless, remarkably few studies have simultaneously monitored the demographic and evolutionary dynamics of populations (Antonovics and Levin 1980, Schemske *et al.* 1994).

We present results from an experiment designed to monitor phenotypic evolution in homogeneous and heterogeneous environments. Our initial goal was to compare the rates and directions of adaptation to different environmental treatments at differing levels of connectedness between habitat patches. Although many theoretical studies have discussed possible effects of migration between patches on evolution (*e.g.* Levene 1953, Levins and MacArthur 1966, Gillespie 1973, Hedrick 1973, Christensen 1975, Slatkin and Lande 1976, Via and Lande 1985, Gillespie and Turelli 1988, van Tienderen 1991; and see reviews in Felsenstein 1976, Hedrick 1986), few studies have experimentally manipulated habitat and then followed evolutionary responses of populations (but see Hedrick 1986). However, our ability to make inferences about evolutionary responses was confounded by an unexpected result of this experiment. Throughout the experiment, populations cycled from high to low density over time due to density-dependent feedback from prior generations (Chapters 1 and 2). Exploration

of the evolutionary response of these populations highlights ways in which population dynamics can influence evolution, and the importance of considering the ecological context of evolutionary predictions.

Methods

1. Study species

Cardamine pensylvanica (Muhl.) (Brassicaceae) is an annual plant of damp habitats (Al-Shebaz 1988) which often invades greenhouses as an ephemeral weed (Cloutier *et al.* 1991). *C. pensylvanica* individuals are highly self-fertile; isolated plants may set thousands of seed in the greenhouse and siliques often form before flowers open (pers. obs.) The seed source for this experiment was a composite of lines from three long-established greenhouse populations: the greenhouses at Duke University (Durham, NC) and North Carolina State University (Raleigh, NC), and a private greenhouse in Durham, NC. The histories of these populations were not known specifically except that each greenhouse was decades old, and in each greenhouse the staff recognized *C. pensylvanica* as a chronic pest. Plants from these naturalized greenhouse populations have no specific germination or flowering requirements and flowering adults are present throughout the year (pers. obs.). Moreover, the "natural" conditions which these plants experience in greenhouses are similar to the conditions under which experimental populations were maintained.

Fifty non-flowering plants from each greenhouse source were planted in a single flat in the Duke University greenhouse until they flowered and set seed. Progeny of these plants showed considerable among- population variation for numerous traits, including height, fecundity, and plant size (Table 1); thus, the aggregate mixture of plants was genetically variable for the response variables measured below. Seeds from

Table 1. Phenotypic differences among populations used to generate the ancestral seed mixture. Duke, NCSU, and DECI refer to populations from the Duke University , North Carolina State University, and Durham Exchange Club Industries greenhouses. There were two model degrees of freedom (73 total df) for all F-tests. Superscripts mark populations that were not significantly different ($p > 0.05$) using Tukey's multiple range test (SAS GLM Procedure).

character	F	p	rank order
# leaves at 4 weeks	7.18	0.001	Duke^{a} > DECI^{a} > NCSU^{b}
height at 4 weeks	3.66	0.031	DECI^{a} > Duke^{ab} > NCSU^{b}
stem diameter at 4 weeks	2.70	0.074	DECI^{a} > Duke^{a} > NCSU^{a}
# leaves at 7 weeks	2.97	0.058	Duke^{a} > DECI^{a} > NSCU^{a}
height at 7 weeks	5.06	0.009	NCSU^{a} > DECI^{ab} > Duke^{b}
stem diameter at 7 weeks	1.41	0.250	Duke^{a} > DECI^{a} > NCSU^{a}
# inflorescences at 7 weeks	2.52	0.088	Duke^{a} > NCSU^{a} > DECI^{a}
width of terminal leaflet	3.43	0.038	Duke^{a} > NCSU^{a} > DECI^{a}
days to flowering	4.90	0.010	NCSU^{a} > DECI^{b} > Duke^{b}

these plants were collected and used for the first generation of plants in population cages. Additional seeds from these plants were saved for use in later experiments.

2. Population cage maintenance

The experimental populations were maintained for fifteen generations in a growth chamber in the Duke University Phytotron with a 16-hour photoperiod and a constant temperature of 27°C. Each population consisted of seventy-two 2.5 cm diameter by 10 cm deep tubular pots (RL-200 Conetainers™, Stuewe & Sons, Corvallis, OR) arranged in a long narrow array (2 pots x 36 pots). The populations were watered from below by filling a system of interconnected tanks with dilute Hoagland's solution until the pots were saturated, then draining the system. Populations were isolated from each other within the growth chamber by clear plastic sheeting suspended from the ceiling. The location of the populations within the chamber was randomized twice each generation.

Experimental populations had combinations of two different environments: pots completely filled with fine vermiculite (deep soil) and pots half-filled with fine vermiculite (shallow soil). Because arrays were watered from below and lit from above, plants in deep soil experienced high light and low water availability, and plants in shallow soil experienced the reverse. Each population had one of four environment types (see Figure 1, Chapter 1): (1) homogeneous deep - all pots filled with deep soil, (2) homogeneous shallow - all pots filled with shallow soil, (3) coarse-grained heterogeneous - one end of the population entirely deep soil and the other end entirely shallow soil, and (4) fine-grained heterogeneous - deep and shallow soil types interdigitated. Three replicate populations were maintained for each of the four environment types. Recruitment occurred from one generation to the next when plants in one generation dehisced seeds into an adjacent interspersed array of pots for the next generation (see

Figure 1 in Chapter 1). Thus, although discrete generations were enforced, competition, growth and dispersal were not manipulated (see Chapter 1).

3. Phenotypic evolution in experimental populations

At the end of each of the first ten generations, the phenotype of plants was measured by subsampling 25 pots of each soil type, randomly selected from among the entire set of pots in each generation. For each plant within those pots, we recorded height and number of siliques (seed pods). For the first eight generations we also measured mass of all plants. These data were collected to monitor the performance of plants in the experimental populations over time, rather than to test any specific statistical hypotheses. Nonetheless, in order to quantify trends in these data, we present results of two analyses.

In the first, our goal is to qualitatively estimate how individual performance was influenced by plant density within each pot, soil depth, and environmental grain (homogeneous, coarse, or fine), and evolution (as measured by changes in plant phenotype) on individual performance. To do this, we present Type III F-statistics (SAS GLM procedure, SAS 1987) for these factors and all interactions as if this were a full factorial experiment. However, because this was not a factorial experiment, we do not use this analysis to test specific hypotheses about any of these effects. Because the variance of all response variables was highly skewed, we ln-transformed all variables prior to analysis, which normalized residuals.

The second analysis is designed to test whether there was directional change in the mean phenotype of individuals over time within each patch type in each environment type. However, the effects of density were large, and could potentially confound temporal patterns, particularly because density varied over time. Thus, we fitted density functions to ln-transformed data from each patch type in each

environment (ln[performance] = a + b[density]). We then fitted a linear model to density residuals over time (ln[performance residual] = c + d[generation]). Thus, if the slope of this relationship is positive, plants became larger or more fecund for a given density over the course of the experiment. If the slope of this relationship is negative, plants became smaller or less fecund over time for a given density. Because of the potential confounding effects of parental environment in first-generation plants, we repeated this analysis twice, once including first generation plants and once excluding first generation plants.

4. Genotypic changes in reciprocal transplant experiments

We tested for genetic differentiation among the populations by growing seeds from family lines derived from each experimental population and from the seed mixture used to initiate the populations (hereafter designated the ancestral population) in reciprocal transplant experiments at two time points during the population cage experiment (after generations 8 and 14). Because the results were similar at both times, we present results from the final comparison.

To control for effects of maternal environment and seed age, seeds from plants in the fourteenth generation and from the ancestral source population were planted in the Duke University greenhouse. From these plants, seeds were collected from nine randomly chosen plants to generate maternal family lines. Because *C. pensylvanica* is a highly selfing species, these "family lines" probably represent highly related individuals. Seeds harvested from each maternal plant were germinated on wet filter paper in petri plates, and transplanted to deep or shallow soil in the phytotron at the two cotyledon stage. In this experiment, eleven or twelve plants from each maternal family were planted in each environment and plants were randomly placed in three phytotron chambers. The total size of the experiment (2,736 plants) was determined by the

amount of space in three phytotron growth chambers. Plants were harvested just after the first set of fruits matured, about eight weeks after transplanting. Plant height, mass, and fecundity (number of seed pods) were scored for each plant.

To test for overall differentiation of experimental populations, we used a multivariate analysis of variance (MANOVA option in SAS GLM procedure, SAS Institute 1987) to compare overall differences in phenotype among populations. To ensure that phenotypic differences were due to genetic differences, we analyzed family means for each trait as response variables. Families with fewer than three surviving individuals were eliminated from the analysis. To normalize residuals, variables were ln-transformed prior to analysis. Because replicate populations within each selection environment differentiated strongly over the course of the experiment (see Results), we present the results of both ANOVA and mixed-model ANOVA analyses (SAS GLM procedure, SAS Institute 1987; see Table 3). This is The mixed model analysis tests the hypothesis that selection environment effects and interactions were large relative to variance among replicates, rather than large relative to total variance in the data set.

Our initial goal was to conduct further analyses to estimate the genetic covariance structure of performance in the two environments and to investigate the effects of environmental heterogeneity on rates and direction of evolution. However, the results of the above analyses made interpretation of further analyses problematic.

Results

In the experimental populations, the effects of density on plant mass and fecundity were bigger than the effects of either patch type or generation (Table 2). Plant height, however, was affected almost as much by soil depth as by density (Table 2). Plants in homogeneous environments were taller and thinner in shallow soil (low light environments) and shorter and bushier in deep soil (low water environments). This is

Table 2. Analysis of plant phenotype in individual pots harvested each generation during the population cage experiment. Effects are: density = decline of mean performance with density of plants in each pot; depth = effect of deep or shallow soil; grain = effect of whether individual pots were located in homogeneous, coarse-grained or fine-grained heterogeneous environments; time = temporal trends; and all interactions except those including both continuous variables (density and time). F statistics are for Type III sums of squares (SAS GLM procedure, SAS 1987; 17 model df, 328 error df for height and seed pods; 17 model df, 225 error df for mass), but see Methods for interpretation of statistical significance.

effect	**height**	**mass**	**# seed pods**
density	28.39	73.14	31.83
time	89.50	15.43	0.03
depth	11.07	0.35	0.08
grain	4.41	2.82	1.80
density*depth	1.82	2.00	0.68
density*grain	3.34	1.78	0.32
depth*grain	3.87	1.68	0.40
depth*time	0.32	1.67	1.58
grain*time	1.70	3.01	1.25
density*depth*grain	0.56	1.07	1.20
depth*grain*time	3.51	6.98	3.10

the classic response of plants to above- *vs.* below-ground resource limitation. Interestingly, in heterogeneous environments, where plants were able to interact across patch types, plants in deep soil were smaller as well as shorter than plants in shallow soil (Figure 1).

Plant height and mass increased over time in both homogeneous selection environments (Table 2, Figure 2). These trends were accompanied by statistically insignificant trends toward increasing fecundity. However, trends in plant mass and fecundity in shallow soil became insignificant ($p > 0.20$) when the first generation plants were excluded from this analysis. This implies either that plants were so strongly selected in the second generation that heritable variation was immediately exhausted, or that first generation plants were different due to parental environmental effects. In heterogeneous selection environments, changes in the phenotype of plants in shallow soil were statistically indistinguishable from selection response in homogeneous environments, whether or not first generation plants were included in analyses. However, changes in phenotype of plants in deep soil differed markedly in homogeneous *vs.* heterogeneous environments. In heterogeneous environments, mean plant height increased over time, but at a slower rate than in homogeneous environments. Mean plant mass increased at a slower rate in coarse-grained heterogeneous environments than in homogeneous environments, and actually decreased over time in fine-grained heterogeneous environments. The fecundity (# of seed pods) of plants in deep soil patches of heterogeneous environments also decreased over time. This pattern was also apparent in analyses without first-generation plants.

In all analyses from the reciprocal transplant experiment, there was evidence for divergence of replicate populations within each selection treatment (pop(selenv) effect, Table 3). These effects were similar to or greater in magnitude than treatment effects and selenv by test environment interactions. Thus, the effects of selection environment

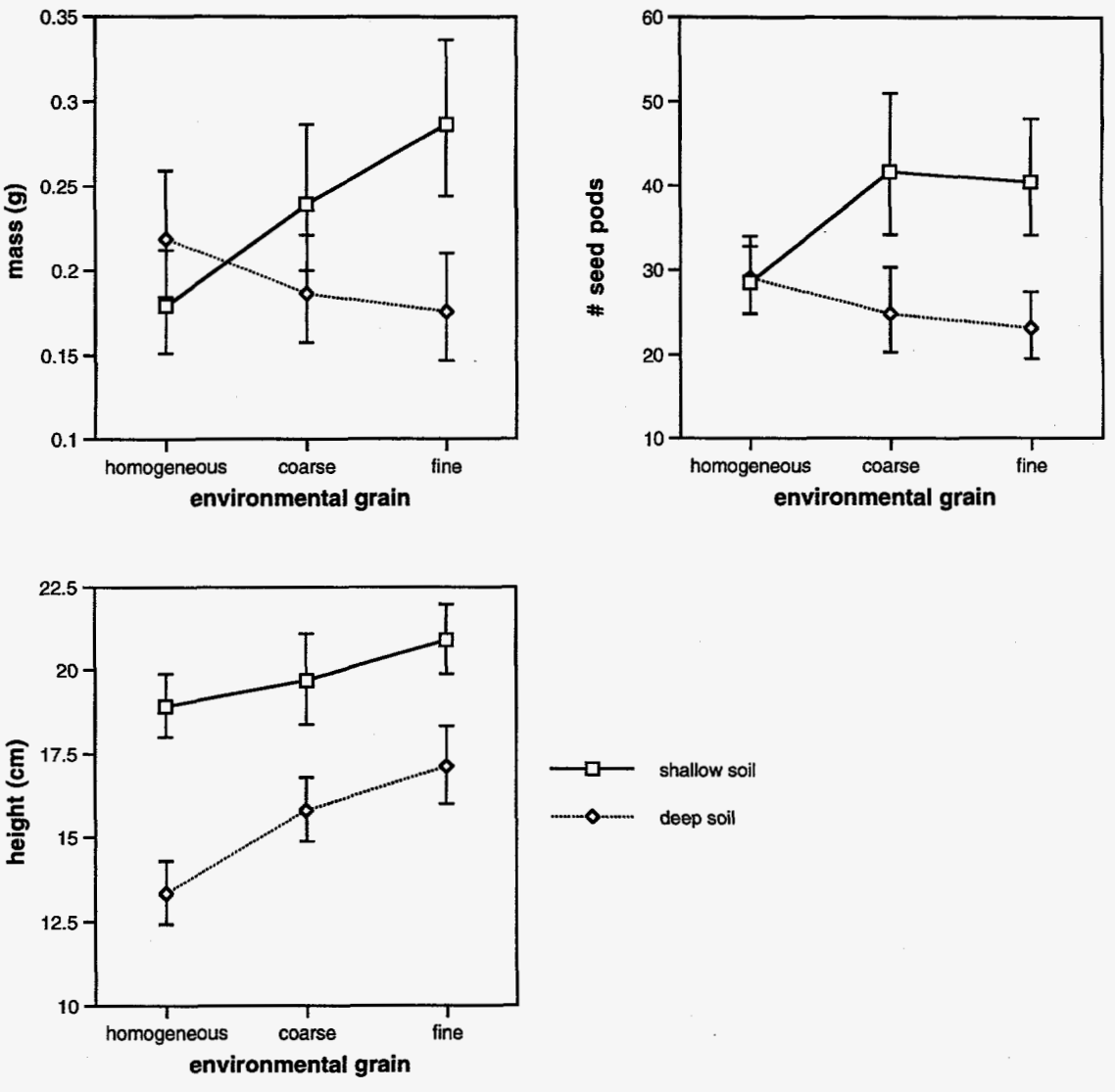

Figure 1. Effects of soil depth on plant phenotype ± standard errors, based on individual pot harvest data during the population cage experiment.

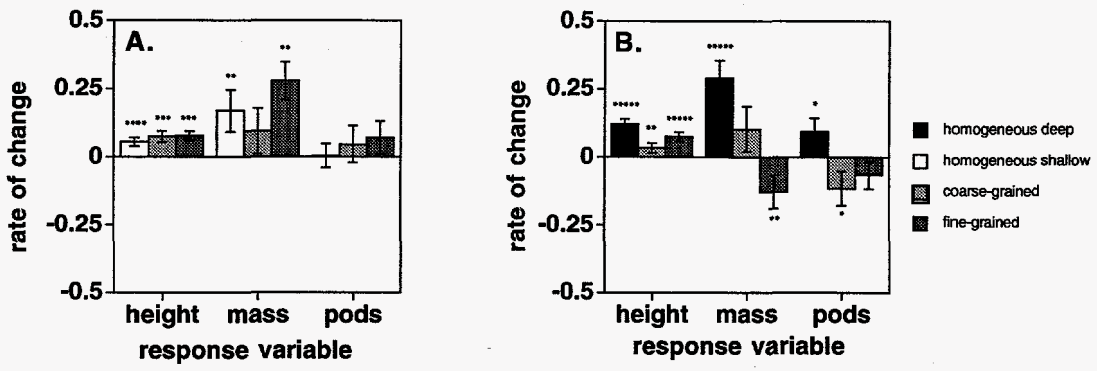

Figure 2. Phenotypic change over time in the experimental populations, ± standard errors of fitted slopes. Data shown are changes in the residuals of phenotype/density relationships over time, based on individual pot harvests in generation 1-10 (see Methods). Positive slopes indicate that plants in early generations were smaller than average for a given density, and plants in later generations were larger than average for a given density. Negative slopes indicate the reverse. A.) response of plants in shallow soil patches; B.) response of plants in deep soil patches. Similar results were obtained in analyses that excluded first-generation plants (see Methods), except that trends in the mass of plants in shallow soil patches became statistically insignificant (see Results). *.10 > p > .05, **.05 > p > .01, ***.01 > p > .001, ****.001 > p > .0001, *****.0001 > p

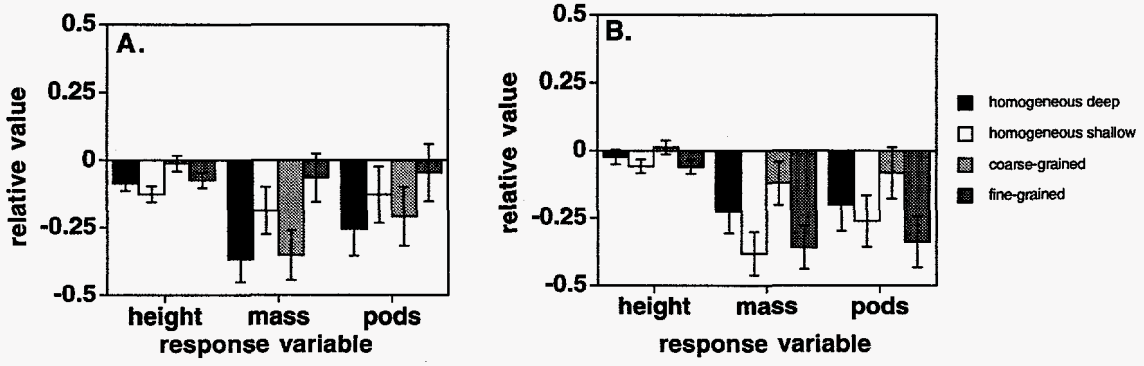

Figure 3. Differences between means of ancestral and selected populations. Data are graphed as deviations from the least-squares mean of family lines from the ancestral source population ± standard errors of least-sqaures treatment means. I chose this scale to emphasize the contrast between the results of this experiment and the population cage experiment. If these two experiments reflected performance in the same selective environments, the shapes of the two figures should be congruent, which is clearly not true. A.) phenotype in shallow soil test environment; B.) phenotype in deep soil test environment.

Table 3. Test results for overall differentiation of populations in different environmental treatments. Effects are: testenv = test environment of deep or shallow soil; selenv = populations from homogeneous deep, homogeneous shallow, coarse-grained, or fine-grained heterogeneous selection environments or the ancestral population; pop = replicate populations within each selection environment. Type III F statistics (SAS GLM procedure, SAS 1987) are reported for individual response variables, and Wilks' Lambda F statistic (MANOVA option in SAS GLM procedure) is reported for overall effects. A.) Comparison of all populations, B.) comparison including only homogeneous deep soil and homogeneous shallow soil populations. In the mixed model ANOVA, effects involving replicate populations were treated as random effects and all other effects were treated as fixed effects. In the mixed model, the following error terms were used (effect, error): (testenv, fam*pop*treat*soil), (selenv, pop(treat)), (pop(selenv), fam*pop*treat), (testenv*selenv, pop(treat)*selenv), where fam = mean of replicate family lines.

	ANOVA				mixed model ANOVA			
effect	**height**	**mass**	**pods**	**MANOVA**	**height**	**mass**	**pods**	**MANOVA**
A. *overall comparison*								
testenv	194.94	101.79	64.53	71.88*****	225.86	147.60	87.87	83.98*****
selenv	3.63	1.90	0.77	2.46***	0.82	0.46	0.21	1.23
pop(selenv)	5.09	4.75	4.47	2.38****	4.32	3.35	3.34	1.96**
testenv*selenv	0.46	3.39	1.32	2.14**	0.37	2.85	0.83	1.09
B. *populations in homogeneous deep vs. shallow soil selection environments*								
testenv	115.80	47.14	33.63	42.47*****	144.02	69.75	42.51	51.64*****
selenv	1.57	0.02	0.10	1.57	0.27	0.00	0.00	1.06
pop(selenv)	8.12	7.43	7.53	3.17****	6.00	5.02	5.75	2.26**
testenv*selenv	0.00	3.46	0.83	2.30*	0.06	2.85	0.34	1.12

*.10 > p > .05, **.05 > p > .01, ***.01 > p > .001, ****.001 > p > .0001, *****.0001 > p

were significant relative to the total variance in this experiment, but not relative to the variance among replicate populations (Table 3). Furthermore, the direction of this differences among populations from different selection environments is quite surprising (Figure 3). Plants tended to be more robust (bigger, taller, more seed pods) in the "away" environment than in their "home" environment. There was also an overall difference between ancestral and evolved populations. Plants from evolved populations were generally smaller (shorter, less mass, fewer seed pods) than their unselected ancestors (Figure 3, Table 3A). In addition to being unexpected, this is the exact opposite of the phenotypic changes observed in the experimental populations (Figure 2).

Discussion

In general, plants became taller, larger, and more fecund over time, and this trend was strongest in homogeneous environments, intermediate in coarse-grained heterogeneous environments, and weakest in fine-grained heterogeneous environments. Although we cannot predict (or even "post-dict") evolutionary trajectories in the absence of data on genetic correlations for performance in the two patches and the strength of selection in these patches, decreases in mass and fecundity in deep soil patches of heterogeneous environments are not inconsistent with theoretical models of evolution in heterogeneous environments. For example, in the presence of negative genetic correlations between performance in deep and shallow soil, there are many evolutionary scenarios under which performance in deep soil patches in heterogeneous environments would at least temporarily decrease (*e.g.* Via and Lande 1985).

However, there was virtually no congruence between phenotypic changes over the course of the population cage experiment and differences between selected and ancestral family lines in the reciprocal transplant experiment (compare Figures 2 and 3). In retrospect, this is not surprising, given our experimental design. In the reciprocal

transplant experiment, seedlings were individually planted in pots. In the population cage experiment, typical seed rains were on the order of hundreds of seeds per one-inch pot. Thus, despite the fact that the two experiments used the same carefully controlled abiotic conditions, conditions experienced by seedlings were vastly different. We had initially anticipated that effects of soil depth independent of density would be large with respect to density by environment interactions, and that inferences about these effects could be made from the reciprocal transplant experiment. Given the discrepancy between these results, this seems unlikely. It is possible to reconcile these results by hypothesizing that the phenotypic changes were not really genetic or that changes in performance in common-garden conditions are not correlated with changes in the same traits in competitive conditions. However, either hypothesis makes the significance of evolutionary changes in common garden conditions unclear.

A second unexpected result of this experiment is that replicate populations within each environmental treatment differentiated more than the means of populations in different treatments (Table 3). The most obvious explanation of this result is some form of genetic drift. We had initially expected random drift effects to be small for several reasons. First of all, the time frame of the experiment was relatively short (15 generations). Secondly, because *C. pensylvanica* reproduces largely through selfing and no pollinators were introduced into the growth chambers, we expected that evolution would occur through shifts in genotypic frequencies, rather than creation of new genetic combinations. Finally, we had not anticipated severe numerical bottlenecks due to density-dependent population regulation. It is likely that these bottlenecks significantly accelerated the importance of genetic drift in our experiments. In fact, given that evolutionary responses to selection are not significantly greater than random differentiation (Table 3), it is possible that apparent responses to selection (selenv effects in Table 3) are largely an artifact of random differentiation. Although the

possibility for interactions between cyclical population dynamics and genetic drift has been noted in theoretical studies (*e.g.* Hartl and Clark 1989, Hartt and Haefner 1995), it has not been widely publicized.

A final striking feature of this experiment is the fact that it would be extremely difficult to avoid confounding effects of population density in *C. pensylvanica.* It is interesting that the high average density and cyclical population dynamics in the population cage experiment resulted from the fact that we did not manipulate competition and recruitment. In other words, we could not have maintained populations at constant low or high density without artificially manipulating recruitment under these conditions.

Plants are notorious for hugely plastic density responses (Harper 1977, Rees and Crawley 1989, Crawley 1990), and a growing number of studies report interactions between plant density and genotype-phenotype relationships (*e.g.* Stratton 1989, Mazer and Wolfe 1992). However, asymptotically stable population dynamics have long been considered the rule, rather than the exception, particularly in plant populations (Crawley 1990, Cousens 1995), and most studies have implicitly assumed that constant-density common garden experiments can be used to infer performance as a function of environment. In many cases, this is probably true. However, several recent ecological studies indicate that population cycles due to density dependence may be far more common than earlier studies had indicated (Symonides *et al.* 1987, Turchin 1990, Silvertown 1991, Tilman and Wedin 1991, Turchin and Taylor 1992, Ellner and Turchin 1995). Furthermore, although the textbook example of density-dependent population cycles describes species with discrete generations and completely deterministic dynamics, the majority of cycles observed in natural populations are not consistent with this model. Instead, cycles in natural populations are more often consistent with models of populations which have overlapping generations, age or size structure, and noticeable

amounts of environmental noise (Turchin and Taylor 1992, Ellner and Turchin 1995, and see Chapter 2).

The results of our experiments highlight the importance of competition in determining the rank order of fitness of different lines, and the possibility that population cycles could dramatically reduce effective population size. The potential for both interactions has been noted in earlier studies (Roughgarden 1971, Hartl and Clark 1989, Mazer and Wulff 1992, Hartt and Haefner 1995). Nonetheless, we had not anticipated that the effects of competitive interactions among individuals would thoroughly overwhelm the effects of external environmental variation in determining patterns of evolution. Given the potential importance of density-dependent population cycles in natural populations, this kind of interaction deserves far wider recognition.

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