

## THE GENESIS OF SIZE VARIABILITY IN PLANTS AND ANIMALS

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**Abstract.** The changes in the size structure of a cohort of individuals through time can have important population-level consequences, but the determinants of patterns of variation in individual size are not well understood. We use an individual-based simulation model to determine what factors can lead to changes in the variability in size among individuals through time, with attention paid to those factors that increase variability. A pattern of increasing size variability over time (i.e., growth depensation) has been documented in a variety of taxa. Size-dependent growth has generally been advanced as the explanation for its occurrence. We evaluated the independent and interactive effects of size-dependent growth factors, positive temporal correlates in growth (termed “growth autocorrelation”), and environmental stochasticity in generating growth depensation. In contrast to expectation, our results indicate that size-dependent growth is not the major contributor to changing variability patterns. Instead, our simulations indicate that growth autocorrelation is often the major determinant causing growth depensation. Data collected from the kelp *Alaria nana* and the whelk *Nucella canaliculata* showed evidence for positive correlations in growth (independent of size), while the tidepool sculpin *Oligocottus maculosus* does not. The changes in size variability in these three species (increasing size variability through time in *A. nana* only) were consistent with model predictions. Our results emphasize a need to disentangle size-related processes from other correlates of growth in field populations and to document how these phenomena are affected by environmental stressors and intraspecific interactions.

**Key words:** *Alaria nana*; cohort model; growth autocorrelation; growth depensation; individual-based model; *Nucella canaliculata*; *Oligocottus maculosus*; population modeling; size variability; stochasticity.

### INTRODUCTION

One of the challenges of understanding the development of population structure is to understand how the distribution of the age and size of a population changes through time. Whereas age structure changes are easily followed and projected, changes in size structure are less easily predicted due to the tremendous plasticity in growth that many organisms experience (Sebens 1987, Weiner 1988). Because some organisms are aged with great difficulty, and because size can be both an excellent predictor of fitness (e.g., Samson and Werk 1986) and an important state variable in population models (e.g., Caswell 2001), understanding the genesis of size structure in a population is important for both evolutionary and ecological analyses. Population size structure can also greatly influence the dynamics of the population (e.g., Ebenman and Persson 1988). When plants and animals are harvested with size-based criteria, accurate predictions of the mean and variance of future size distributions are needed. In instances of environmental stress, variability among individuals may provide a metric with which to detect

stress (Forbes and Depledge 1996). Additionally, recent studies point to the importance of variability in size in mediating intraspecific interactions, including cannibalism (DeAngelis et al. 1979, Maret and Collins 1994, Brunkow and Collins 1996, 1998) and interspecific interactions such as predation (Wootton 1992, 1993, Crowder et al. 1992, Chase 1999). For any new cohort of organisms, the variability among individuals in size either decreases through time, remains the same, or increases.

Changes in the size variability of individuals through time have been referred to as “growth depensation” if the variability increases through time, and “growth compensation” if the variability decreases through time (Ricker 1958). Growth depensation has been attributed to positive correlations through time in the change in size (Ricker 1958). However, these positive correlations in size could be the result of either positive size-dependent growth, positive temporal correlations in growth that are independent of size, or both. In the first case, if growth is positively size-dependent (e.g., many plants, Harper 1977), then a positive feedback might occur and individuals might diverge in size (DeAngelis and Huston 1987, DeAngelis et al. 1993). Variability that is independent of size might be either equally distributed among all individuals at every time step. Alternatively, variability among individuals might be distributed in such a way that some individ-

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uals were always better growers while others were always poor growers. In the latter instance, growth among individuals would be correlated positively through time, a scenario we refer to as "growth autocorrelation." What is unknown is the extent to which size-dependent growth vs. correlations with previous growth rates, or some combination of both, generates growth depensation. In order to accurately predict the structure of a population in the future, we need a better understanding of the processes that drive the patterns of size that we see in field populations.

Despite the different factors that might lead to changes in the size structure of a population through time, early definitions of growth depensation were vague about the process or processes that generated it (e.g., Ricker 1958) and the difficulty in determining the causal factors was noted (Magnuson 1962). An impediment to determining which factors contributed to growth depensation in previous empirical studies was the lack of distinction between size-dependent processes and other correlates of growth (e.g., Weiner 1988). In order to disentangle the two processes, *individuals* must be identifiable; simply following a cohort or the size structure of a population through time provides insufficient information.

We envision three mechanisms that might change the variability among individuals. The first, size-dependent factors, has been studied in various organisms (Kaufmann 1981, Ebert 1982, Wethey 1983, Samson and Werk 1986, Schmitt et al. 1987, Karlson 1988, Pfister 1996). Examples of size-dependent factors include light capture, foraging ability or success, and risk avoidance. A second mechanism is one where resources are consistently distributed unevenly among individuals, resulting in positive correlations in growth through time (growth autocorrelation). A number of empirical studies suggest that growth autocorrelation may be present in a wide variety of taxa. In some cases, especially with plants, this may exacerbate size-dependent effects. For example plant studies often suggest that there are "dominant" and "suppressed" individuals in populations (Harper 1977, Cousens and Hutchings 1983, Weiner 1985, Schmitt et al. 1986, 1987, Dean et al. 1989, Reed 1990). Other factors that might explain growth autocorrelation in natural populations are the differential specialization and performance of animals on prey (Palmer 1983, West 1986), the potential for associative learning about resources (Dukas and Bernays 2000), and the outcome of light competition in plant species (Schmitt et al. 1986). Growth autocorrelation might result from variation in traits that are possessed by individuals in a population (such as trait variation in how individuals garner or assimilate resources). Alternatively, or additionally, factors extrinsic to individuals, such as environmental heterogeneity, could underlie growth autocorrelation. Thus, the processes underlying positive temporal correlations in individual performance may be diverse and

may result from any variety of mechanisms that lead to unequal resource use among individuals. Finally, stochastic variation is a third mechanism that produces differences among individuals.

Modeling studies have provided some insight into the contribution of size-dependent processes to growth depensation. Cohort models described with partial differential equations show that positive size-dependent growth in tandem with some variability in growth can lead to increasing variance in size through time (DeAngelis and Huston 1987, DeAngelis et al. 1993). Additionally, when growth variability that was correlated through time was used in a simulation model without size-dependent growth, the distribution of individual size could change greatly (DeAngelis et al. 1993), suggesting that growth autocorrelation alone could greatly influence size distributions through time. As with the empirical data, however, we know of no instances where the separate effects of growth-autocorrelation terms and size-dependent growth on growth depensation have been investigated.

The density and distribution of conspecifics are environmental factors that influence the degree of variability among individuals and the propensity for growth depensation (Rubenstein 1981, Ziemba and Collins 1999, Ziemba et al. 2000). In an experiment with pygmy sunfish, Rubenstein (1981) found increasing size differences with increasing density. His data also suggested that positive correlations in growth may be important to generating variability: some fish were good competitors at high densities while others consistently grew little. Although a number of studies with plants and animals support the pattern of increased variability or skew in size with density or resource limitation (e.g., Wilbur and Collins 1973, Harper 1977, Weiner and Thomas 1986, Schmitt et al. 1987, Peckarsky and Cowan 1991, Thomas and Bazzaz 1993, Ziemba and Collins 1999), there is some evidence for the opposite pattern. In a grass species, Turner and Rabinowitz (1983) found that decreasing density led to increased size variability through time, a possible outcome of either the spatial distribution of competitors (Huston 1986) or the result of releasing individual plants from growth inhibition (Lomnicki 1988). Despite the differences in empirical results, these studies all suggest that attention to density effects and competitive interactions are critical to understanding the generation of the size patterns among individuals in a population.

Although inequalities in growth can be a result of either size-dependent phenomena, variability independent of size, or both, there has been little systematic examination of the causes underlying changes in the variance among individuals. We first present demographic data from three marine species (a kelp, a snail, and a fish) to explore the relative contribution of size vs. growth autocorrelation to growth. We then use these data to provide realistic parameter estimates for a simulation model designed to explore the effects of size-

dependent growth processes and different types and amounts of individual growth variability on the patterns of size variation. Our model is individual based so that we can readily include several state variables to ask how they affect variation among individuals. In addition to size and previous performance, population density is also included in our models because empirical studies point to the importance of density in generating size-distribution patterns. For all simulations and for the three field populations, we asked what set of growth conditions give rise to growth depensation.

#### METHODS

##### *Growth and size variability in natural populations*

We used data from three marine species to determine whether growth was significantly size-dependent and whether growth autocorrelation was present. We then asked if changes in size variability through time were positive or negative by estimating the change in the cv for individuals from the beginning of the censusing to the end.

The three species chosen are taxonomically diverse and have different resource requirements. The kelp *Alaria nana* (Order Laminariales) is an annual seaweed that inhabits rocky intertidal habitats from Alaska to Oregon (Widdowson 1971). The predatory whelk *Nucella canaliculata* occupies rocky intertidal habitats from Alaska to central California (Harbo 1997) where it often occupies relatively new patches in mussel beds and preys upon small mussels and barnacles (Palmer 1983). The common tidepool sculpin *Oligocottus maculosus* is a ubiquitous member of tidepools from Alaska to California (Hart 1973). *A. nana* and *N. canaliculata* were studied in the rocky intertidal of Tatoosh Island in Washington state (48°24' N, 124°44' W), USA, while the common tidepool sculpin was studied at Third Beach (48°23' N, 124°35'), a locality 3 km east of Neah Bay, Washington along the Strait of Juan de Fuca. All three species are indeterminate growers (sensu Sebens 1987); individuals of all species were within a size range where we could expect to continue to see growth.

The dependence of growth on size and the correlation with previous growth was estimated in each of the three species. We distinguished size-dependent effects on growth from growth autocorrelation by first estimating the relationship between size at time  $t$  and growth from time  $t$  to  $t + 1$  at every interval with a linear regression. If that regression was significant (either with a positive or negative  $\alpha$ ), we asked whether growth at time  $t$  was a function of growth at time  $t - 1$  with another linear regression. A positive and significant regression coefficient indicated significant growth autocorrelation. A simple linear regression using size as the only independent variable places more importance on size in explaining the variation in growth. Thus, we also used multiple regression with both size at time  $t$  and growth

at time  $t - 1$  as independent variables to compare the relative significance of these coefficients.

We examined how variability in size changed through time by estimating the cv in size at every census. To eliminate any effects on cv due to selective mortality, the initial cv was estimated only for those individuals who were later resighted or recaptured.

*The kelp, Alaria nana.*—*Alaria nana* occupies mid-intertidal rocky intertidal sites, often colonizing patches of relatively bare space (Paine and Levin 1981). Sporophytes become macroscopic in the late winter or early spring, grow throughout the summer, and then are usually ripped from the substrate by fall and winter storms. Six patches with young-of-the-year *A. nana* on Tatoosh Island, containing 215 plants among them, were chosen for study in May 1997. Individual growth, survivorship, and size were followed until 15 September 1997 by vinyl numbered tags (Floy Tag and Manufacturing, Seattle, Washington, USA) tied on the base of the stipe with marine bailing twine. At 2-wk intervals, plant size was estimated as the product of frond length and width at their maximum dimensions. Although *Alaria* has specialized reproductive structures (sporophylls) that can contribute substantially to plant mass, the area of vegetative tissue is the primary determinant of frond growth (Pfister 1992). The linear growth rate was estimated by punching a small hole (with an office hole punch) every 2 wk at ~5 cm from the base of the frond in the meristematic region. The movement of the hole was measured at each census to estimate linear growth and multiplied by the width of the frond at the beginning of the interval to estimate growth in area. We estimated the dependence of growth on frond size, the importance of previous growth (independent of size), and the pattern of change in size variability (as cv) through time.

*The whelk, Nucella canaliculata.*—*Nucella canaliculata* is an abundant and conspicuous predatory snail along rocky shores in the northeast Pacific. Young hatch from egg cases in the intertidal and do not have a planktonic dispersal phase; adults can remain in a relatively restricted area. In July 1997, 261 *N. canaliculata* in four patches were measured and tagged with numbered bee tags adhered with Z-Spar marine epoxy. The maximum length of resighted snails was measured with calipers every 2 wk until 16 September. Growth was defined as any change in the total length. The 3-mo period of study was divided into two ~6-wk intervals to determine if a correlation with previous growth was present. We estimated the relationship between size and growth, tested for the presence of growth autocorrelation, and determined how variability among individuals changed from the first to the last census.

*The tidepool sculpin, Oligocottus maculosus.*—*Oligocottus maculosus* occupies tidepools over a range of tidal heights. Except for the planktonic larval stage, these fish spend their life cycle in the same intertidal

locale. Individuals display site fidelity, often returning to the same pool or cluster of pools over their lifetime (Green 1971, Pfister 1995, 1996). In June 1999 we marked 248 *O. maculosus*, ranging in size from 20 to 61 mm standard length (SL), in three tidepools. Individuals were marked with a unique combination of water-based paints (Delta, Whittier, California, USA) that were injected immediately below the skin on the ventral side with a 27 G1/2 needle (Becton Dickinson & Company, Franklin Lakes; New Jersey, USA). Because they could not be effectively marked, individuals <20-mm SL were not marked. Fish were caught by dipnetting, aided by draining the tidepool and temporarily removing habitat features (cobble, shells, etc.) to reveal fish for capture. When 10 min elapsed without any new fish sighted or caught, we assumed we had thoroughly censused the tidepool and the pools were refilled. The tidepools were sampled and fish were recaptured and measured every 2 wk through September. As with the kelp and snail species, we asked whether growth was related to size and previous growth. We also estimated how the pattern of size variance had changed through the census period. We divided the ~3-mo duration of the study into two 6-wk intervals to estimate whether growth autocorrelation was present.

#### *Individual-based simulations*

We used an individual-size-based simulation model (IBM) to ask what features of organism growth change the variance in size among individuals. Although there are a number of ways to model the change in a size distribution through time (Aikman and Watkinson 1980, Hara 1984, DeAngelis and Huston 1987, Huston and DeAngelis 1987, DeAngelis et al. 1993, Wyszomirski et al. 1999), we chose to use an individual-based approach to control the amount and type of variation among individuals. We focused on a cohort of individuals starting from a hypothetical "recruitment" phase to some time in the future after multiple time periods of growth and survivorship. A cohort model allowed us to see within generation dynamics of the type that might occur following a settlement event. Size-dependence in growth, random variation in growth, and correlations in growth through time (growth autocorrelation) were included in the model formulation.

*Density-independent simulation.*—We first used an IBM that modeled survivorship and individual growth as processes unrelated to population density. Our simulated populations consisted of a starting population of 10 000 individuals with a mean size of 50 units. Our decision to start with 10 000 individuals was based on a quantitative assessment of how population size affected the mean and variance of the final number and size of individuals after 24 time steps and 50 model runs. In models runs of 200 to 45 000 individuals, we found that the mean and confidence limits of runs with

as few as 10 000 individuals matched well the output from greater population sizes.

We used two realistic initial size distributions, where sizes were either normal with a standard deviation of 15 or lognormal (skewed to the right) with 1 SD = 25. Normal distributions of organism size are often observed in nature (Uchmanski 1985), and there has been much interest in the processes that lead to the generation of nonnormal distributions, where there are many small individuals and few large ones (reviewed in Uchmanski [1985]), and observed in diverse taxa such as plants (e.g., Begon 1984, Stoll et al. 1994) and amphibians (Wilbur and Collins 1973). The use of both size distributions allowed us to examine how size variability was affected by the existing size structure of the population and the form of the distribution.

Individuals in our population grew in accordance with a linear equation that described how growth at time  $t + 1$  was a function of individual size at time  $t$  ( $S_t$ ), the previous growth rate of the individual ( $G_t$ ), a constant ( $C_g$ ) and a term for random variation (i.e., stochasticity in growth,  $\varepsilon_g$ ):

$$G_{t+1} = C_g + \alpha \cdot S_t + \beta \cdot G_t + \varepsilon_g. \quad (1)$$

In this way, we were able to see the effect of changes in  $\alpha$ ,  $\beta$ , or  $\varepsilon_g$  while other parameters were held constant or eliminated.

Size-dependency in growth, represented in our model by  $\alpha$ , has been observed to be negative (e.g., fishes: Pfister 1997, marine invertebrates: Spight 1981, Carlson and Pfister 1999) and positive (especially plants, Harper 1977). We were interested in how the model would change over a range of positive, negative, or zero values for  $\alpha$  (0.03, 0.005, 0, -0.03, and -0.005).

The observation that a perfect positive temporal correlation in growth (correlation = 1.0) favored the use of IBMs (DeAngelis et al. 1993) prompted us to explore the effects of a range of correlations. We considered this term to be "growth autocorrelation," where the amount of growth in a previous interval could affect the growth rate in subsequent intervals. We modeled no growth autocorrelation ( $\beta = 0.0$ ), and two levels of growth autocorrelation ( $\beta = 0.4$  and  $\beta = 0.8$ ). We include it simply as a single parameter correlation, although we recognize that growth autocorrelation is likely to be generated from mechanisms that might be either "inherent" to the individual or from environmental factors that are "imposed" (sensu Huston and DeAngelis 1987). For example, there may be genetic variation for differences in performance or resource use among individuals that are inherent in the organism (e.g., Arnold 1981). Alternatively, or collaboratively, heterogeneity in resources that are imposed by the environment can also yield positive temporal correlations in growth. Thus, although there are myriad mechanisms underlying the single parameter  $\beta$  in nature, we first explored the importance of  $\beta$  in affecting size variability. We explored only positive  $\beta$  terms because neg-

ative terms, although possible for short intervals during the life-span of an individual, eventually led to unrealistic decreases in organism size.

In order to prevent extremely large growth differences among different parameter combinations and also to prevent extreme negative growth, we did not set  $C_g$  at any value; rather,  $C_g$  had a unique value for each set of parameters.  $C_g$  was determined for each run by solving Eq. 1 for  $C_g$  at a constant average growth ( $\bar{G}$ ):

$$C_g = \bar{G}(1 - \beta) - \alpha \hat{S} \quad (2)$$

where  $\hat{S}$  was the smallest individual in the initial size distribution. Thus, Eq. 2 ensures that the smallest individuals in the population will not suffer negative growth, and that the mean individual size among different parameter sets will not be too disparate. This allowed us to construct model scenarios where all individuals were certain to grow, but the strength of the contribution of different factors to growth varied among model runs.

The  $\varepsilon_g$  term represents the stochasticity in growth, or the variation in  $G_{t+1}$  that is unexplained by size and previous growth. We set  $\varepsilon_g$  to be normally distributed with a mean of 0 and a standard deviation of either 0.5, 1.0, or 2.0. Increasing the standard deviation of  $\varepsilon_g$  increased the stochasticity in growth and, ultimately, size. Thus, our alterations of  $\varepsilon_g$  allowed us to examine how increasing stochasticity affects the size structure of the population and the generation of growth depensation.

We introduced mortality into our model in order to understand how size-related survivorship might affect the size structure of a population through time. The survivorship probability of each individual at each time step ( $P_i$ ) had a maximum value of 0.970 and changed with a slope ( $\delta$ ) with increasing size ( $S_i$ ). Because we might expect survivorship to have differing relationships with size (negative, if size is correlated with age and senescence occurs [Deevey 1947], or positive if increasing size reduces mortality due to disturbance or predators [e.g., Wethey 1983]), we report the results of using both positive and negative relationships between size and survivorship ( $\delta$ ). However, for most simulations we used a negative relationship between size and survivorship, with a  $\delta$  value of  $-0.0005$ . We introduced variation in survivorship using a normally distributed error term ( $\varepsilon_p$ ) with a mean of 0 and a standard deviation of 0.01. An individual died if a random number drawn between 0 and 1 exceeded the survivorship probability estimated for its size.

In sum, at each of 24 time steps our model determined whether each individual would continue to survive. If so, we assigned it a growth rate in accordance with Eq. 1 and computed a new size for that individual. Because we used two initial size distributions, five estimates for the relationship between size and growth ( $\alpha$ ), three values for the strength of correlations with previous growth ( $\beta$ ), and three different values for the

amount of unexplained variation in growth ( $\varepsilon$ ), we ended up with 90 unique parameter combinations. We eliminated models with  $\alpha = -0.03$  and  $\beta = 0.8$ , because they yielded individuals that continually decreased in size. This reduced the number of IBM variants that we performed to 84. We ran each model variant 50 times to generate the range of possibilities associated with each parameter configuration. Our decision to run the model for 24 time steps was based on comparing the output from 12 to 40 time steps. Although the predictions of the model were similar across this time horizon, increasing the simulation beyond 24 time steps resulted in an ever-decreasing number of individuals on which to base conclusions.

To assess how different factors affected the size distribution of a simulated population, we compared the mean coefficient of variation (CV) at the end of 50 projections with the mean CV at the start of 50 model runs using ANOVA. An increasing CV indicated growth depensation, while a decreasing CV indicated growth compensation. Although there are many estimators that can be used to describe inequality among individuals (Kokko et al. 1999), we were concerned here with an estimator of variance. We chose to use CV because the mean size of individuals increased through time as the cohort grew and CV provides an estimate normalized to the mean. Additionally, CV is a metric used previously in the description of growth depensation (Weiner 1988, Peckarsky and Cowan 1991, Ryer and Olla 1996, Wyszomirski et al. 1999).

We also used the density-independent model to ask whether the qualitative changes in the CV in size from the three field populations matched the predictions of the simulation model when estimated values of  $\alpha$  and  $\beta$  were used. For all species, we ran the model using a range of  $\varepsilon_g$  values (0.5, 1.0, and 2.0) and an initially normal size distribution. For *Alaria nana*, we used the full range of estimates that we had for  $\alpha$  and  $\beta$ .

*A density-dependent individual-based model.*—We introduced both linear and nonlinear negative density-dependence into the growth function of our individual-based model to assess how different forms of density-dependence might affect the occurrence of growth depensation. We added a term to the growth equation (Eq. 1) to reflect poorer performance at higher densities, using both linear and nonlinear forms of density-dependence.

To incorporate linear, negative density-dependence to the growth rate of an individual, a term for density-dependence ( $\gamma N_i$ ) was added to Eq. 1, where  $\gamma$  was the coefficient for density-dependence and was set at  $-5.0 \times 10^{-5}$ :

$$G_{t+1} = C_g + \alpha \cdot S_t + \beta \cdot G_t - \gamma \cdot N_t + \varepsilon_g. \quad (3)$$

Thus, increasing population density resulted in decreased individual growth rates.

Because growth rates may also be a nonlinear func-

TABLE 1. A list of parameters, their biological meaning, and the values used in the simulation model.

Parameter	Biological meaning	Values used
$\alpha$	relationship between size and growth	0.03, 0.005, -0.005, -0.03
$\beta$	growth autocorrelation	0, 0.4, 0.8
$\varepsilon_g$	stochasticity in growth	mean of 0; SD of 0.5, 1.0, 2.0, 3.0
$\gamma$	strength of linear density-dependence in growth	$-5.0 \times 10^{-5}$
$\mu$	coefficient for nonlinear density-dependence in growth	-0.025
$\sigma$	strength of nonlinear density-dependence in growth	0.0003
$\delta$	relationship between size and survivorship	0, -0.0005, 0.0005
$\varepsilon_P$	stochasticity in survivorship	mean of 0; SD of 0.01

tion of density (Rubenstein 1981), we also modeled growth using a nonlinear term, as follows:

$$G_{t+1} = C_g + \alpha \cdot S_t + \beta \cdot G_t - \mu \exp(\sigma \cdot N_t) + \varepsilon_g. \quad (4)$$

To make the range of growth rates comparable for linear and nonlinear density-dependence, we used  $\mu = -0.025$  and  $\sigma = 0.0003$ .

In all the density-independent and density-dependent runs, simulation protocols were identical and survivorship was modeled with negative size-dependence unless noted otherwise. Table 1 provides a summary of all the parameters used in the simulations.

## RESULTS

### *The kelp, Alaria nana*

Plant growth was always positively related to plant size (as frond area) during the study, with regression coefficients ranging from 0.003 to 0.018 (with a mean of 0.009, Table 2) over the nine 2-wk intervals that the plants were followed. Linear regressions were all statistically significant at  $P < 0.001$ ; the relationship between size and growth was linear (Fig. 1a), and the variation explained by size averaged 67.9%. Positive correlations in growth (independent of size) were found in every census interval; previous growth explained an average of 25.6% of the variance when residuals were examined and the regression coefficient was  $0.503 \pm 0.107$  (mean  $\pm 1$  SD). A representative example of growth autocorrelation is shown in Fig. 1b. When we used a multiple regression with size and previous growth as independent variables, previous growth usually explained more variation in growth than size. In 3 of the 9 intervals, size was not statistically significant, while previous growth was statistically significant ( $P \leq 0.001$ ) at every interval. The multiple-regression re-

TABLE 2. The relationship between size and growth ( $\alpha$ ) and the temporal correlation in growth (growth autocorrelation,  $\beta$ ) for the three marine species studied.

Species	$\alpha$	$\beta$
<i>Alaria nana</i>	0.009*	0.503*
<i>Nucella canaliculata</i>	-0.021*	0.438*
<i>Oligocottus maculosus</i>	-0.011*	0.143

Note: The estimates reported for *Alaria nana* and all  $\alpha$  are means.

\*  $P < 0.05$ .

sults demonstrate that variation in growth is often best explained by growth autocorrelation in *A. nana*.

The cv in frond size was 70.7 at the beginning of the study when plants were first established. The cv increased rapidly and remained in excess of 100 for the remainder of the growing season, with a net increase of 43.8 by the end of the growing season.

### *The whelk, Nucella canaliculata*

Growth was negatively and linearly related to size at both intervals where we summarized the data (e.g., Fig. 1c). In the first half of the censusing period, a linear regression showed that the coefficient for the effect of size on growth was  $-0.022$  ( $F_{1,71} = 16.37$ ,  $P < 0.001$ ,  $r^2 = 0.187$ , Table 3). In the second half of the censusing period, growth was again negatively related to the size of the individual and the coefficient was similar at  $-0.020$  ( $F_{1,71} = 9.76$ ,  $P = 0.003$ ,  $r^2 = 0.121$ ). Growth autocorrelation was indicated by the finding that the residuals from the first regression explained a significant amount of the variance in the residuals from the second regression ( $F_{1,71} = 6.86$ ,  $P = 0.011$ ,  $r^2 = 0.088$ ), with a regression coefficient of 0.438 (Fig. 1d). A multiple regression with size and previous growth as independent variables yielded nearly identical results (size coefficient =  $-0.017$ , previous growth coefficient = 0.502).

Variation in size among snails declined slightly throughout the study, starting with a cv of 20.6 and ending with a cv of 14.5 for a net decrease of 6.1.

### *The tidepool sculpin, Oligocottus maculosus*

Growth was negatively and linearly related to size at both intervals where we summarized the data (e.g., Fig. 1e). In the first half of the censusing period, a linear regression showed that the coefficient for the effect of size on growth was  $-0.011$  ( $F_{1,101} = 7.05$ ,  $P = 0.009$ ,  $r^2 = 0.065$ , Table 3). In the second half of the censusing period, growth was again negatively related to the size of the individual and the coefficient was identical ( $F_{1,101} = 8.36$ ,  $P = 0.005$ ,  $r^2 = 0.076$ ). There was little evidence for growth autocorrelation; a regression using the residuals was not significant ( $F_{1,101} = 2.62$ ,  $P = 0.109$ ,  $r^2 = 0.025$ ), indicating that the regression coefficient for previous growth (0.143) was not different from zero (Fig. 1f). A multiple regression using both size and previous growth as independent

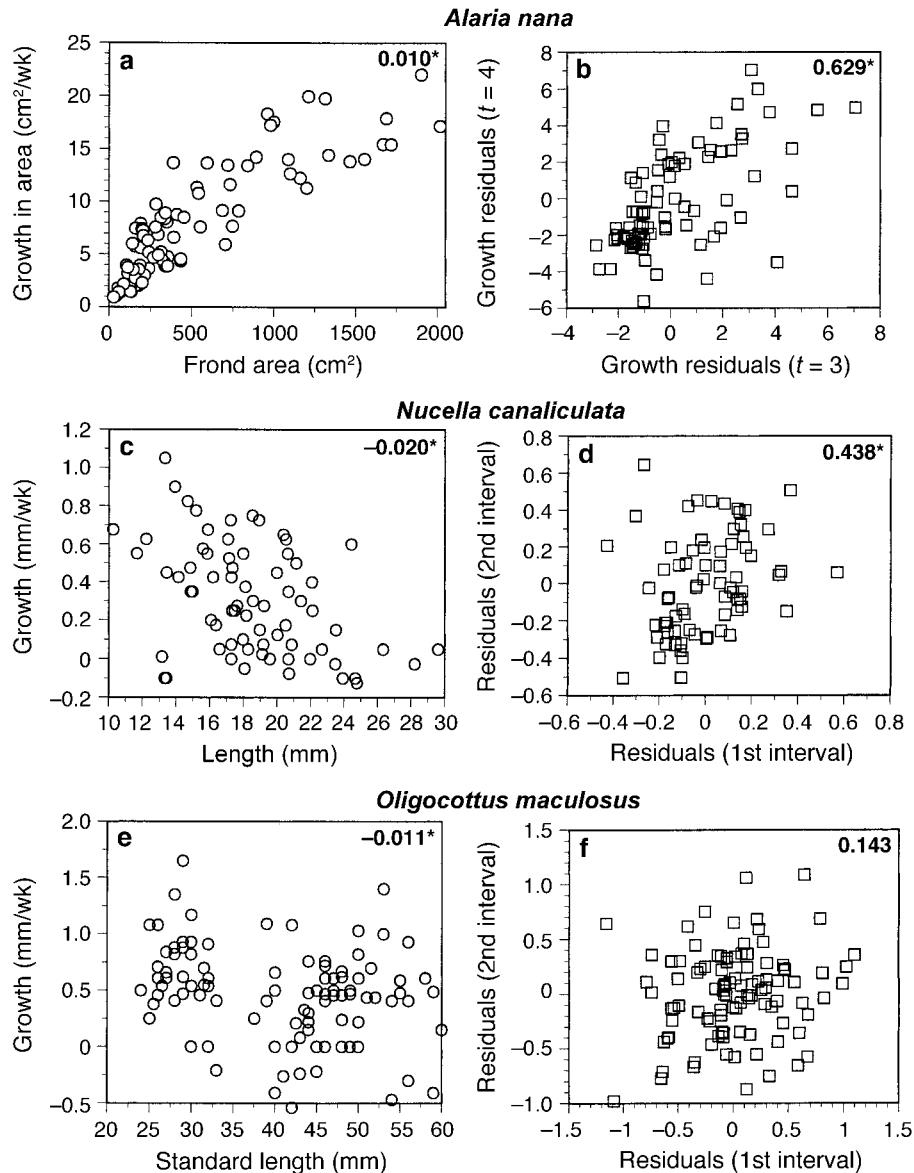


FIG. 1. The relationship between size and growth (a, c, and e) and the temporal correlation in growth (b, d, and f; as residuals) for the three marine species studied. The regression coefficients are given. The data shown for *Alaria nana* are for one of the nine 2-wk intervals during which its demography was studied.

\* Statistically significant ( $P < 0.05$ ).

variables showed similar results; the regression coefficient for growth was  $-0.011$  and statistically significant, while the regression coefficient for previous growth (0.140) was not significant.

Variation among individuals declined slightly throughout the study, starting with a cv of 27.4 and ending with a cv of 23.9 for a net decrease of 3.8. There were no censuses where the cv in size increased over the initial cv.

#### Individual-based simulations

In all density-independent simulations, growth depensation only occurred with strong growth-autocor-

relation terms ( $\beta = 0.8$ ) (Figs. 2 and 3). Positive size-dependent growth ( $\alpha = 0.03$  or  $\alpha = 0.005$ ) alone did not yield an increasing cv in size without a growth-autocorrelation term  $> 0.4$ . Over other parameter values, however, the variability in size could decline greatly or remain relatively unchanged. Populations with a strong growth-autocorrelation term ( $\beta = 0.8$ ), but negative size-dependent growth ( $\alpha = -0.005$ ) usually showed a decreasing variability in size through time. The initial distribution had a relatively small effect, although an initially normal size distribution had a greater tendency for individuals to increase in variability than one that started with a lognormal distri-

TABLE 3. Results of three-factor ANOVA for the effect of size-dependent growth ( $\alpha$ ), growth autocorrelation ( $\beta$ ), and stochasticity in growth ( $\epsilon_g$ ) on change in CV of size for density-independent models. (a) The initial size distribution was normal; results are given in Fig. 2. (b) The initial size distribution was lognormal; results are given in Fig. 3.

Source of variation	df	ss	F	Total variance explained (%)
a) Initially normal distribution				
Size-dependence, $\alpha$	3	10 443	13 679	5.5
Growth autocorrelation, $\beta$	2	88 258	173 416	46.2
Stochasticity, $\epsilon_g$	2	47 994	94 301	25.1
$\alpha \times \beta$	6	160	960	0.5
$\beta \times \epsilon_g$	4	38 892	38 207	20.4
$\alpha \times \epsilon_g$	6	1890	1237	1.0
$\alpha \times \beta \times \epsilon_g$	12	2124	696	1.1
Error	1764	448		
b) Initially lognormal distribution				
Size-dependence, $\alpha$	3	15 537	9500	12.1
Growth autocorrelation, $\beta$	2	56 128	51 479	43.6
Stochasticity, $\epsilon_g$	2	30 562	28 032	23.7
$\alpha \times \beta$	6	768	234	0.6
$\beta \times \epsilon_g$	4	23 692	10 865	18.4
$\alpha \times \epsilon_g$	6	534	164	0.4
$\alpha \times \beta \times \epsilon_g$	12	576	88	0.4
Error	1764	961		

Note: All analyses were based on 50 replicate model runs, and all factors and their interactions were significant at  $P < 0.001$ .

bution of individuals. In model runs with an initially lognormal distribution of individuals, growth depensation occurred only at strong size-dependent growth ( $\alpha = 0.03$ ) with strong growth autocorrelation (0.8) and high amounts of stochasticity in growth (Fig. 3c). In no case was positive size-dependent growth alone sufficient to result in growth depensation. However the amount of stochasticity in growth was important; all runs showed a greater tendency for growth depensation with increasing stochasticity (an increasing  $\epsilon_g$ ) (compare Fig. 2c with 2a and Fig. 3c with 3a). It appeared that an increased amount of random variation, in tandem with positive growth correlations, increased the range of possible growth rates.

We used three-factor ANOVA to estimate the contribution of size-dependent growth, growth autocorrelation, and stochasticity to the patterns of size-structure changes in Figs. 2 and 3 (Table 3). Although all factors and their interactions were statistically significant, there were large differences in the contributions of each factor to the overall sum of squares. With an initially normal distribution of sizes (Table 3a), growth autocorrelation alone accounted for almost half of the variation in changes in the CV, and the interaction between growth autocorrelation and stochasticity explained  $>20\%$ . Size-dependence in growth explained relatively little of the pattern in variability among individuals. The patterns for an initially lognormal distribution were very similar (Table 4b). When we examined either linear or nonlinear density-dependence (Table 4 a and b), growth autocorrelation had the greatest effect on the change in CV among individuals. In all models except those with linear density-dependence,

stochasticity and its interaction with growth autocorrelation also had a large influence. To prevent an analysis with an unbalanced ANOVA (because the parameter combination of  $\alpha = -0.03$  and  $\beta = 0.8$  was not used), the results in Tables 4 and 5 are based on model output without any model runs where  $\alpha = -0.03$  was used.

Model runs that used  $\alpha$  and  $\beta$  values estimated from the three field populations generated predictions about the change in the CV of size that were qualitatively similar to the results we obtained in the field (Table 5). The model predicted that only the kelp species would show growth depensation, and the snail and fish species would have a declining CV in size through time. The large positive change shown by *A. nana* in the field was in excess of that predicted by the model, a result that might be due to a lack of knowledge about other parameter values in the model (such as those related to survivorship), or to the fact that *A. nana* size can change as a result of factors that are unrelated to growth (e.g., herbivory and wave action).

Although stochasticity was always a significant factor in changing the CV in size (Tables 4 and 5), random variation itself did not generate growth depensation. When the stochasticity term ( $\epsilon_g$ ) was increased from 0.5 to 3.0, while size-dependent growth and growth-autocorrelation terms were set to 0, variability among individuals always decreased through time (Fig. 4). However, when growth was positively size-dependent ( $\alpha = 0.03$ ) and growth autocorrelation was strong ( $\beta = 0.8$ ), random variation in growth further enhanced the tendency for growth depensation (Fig. 4).

When growth was modeled as a negative density-

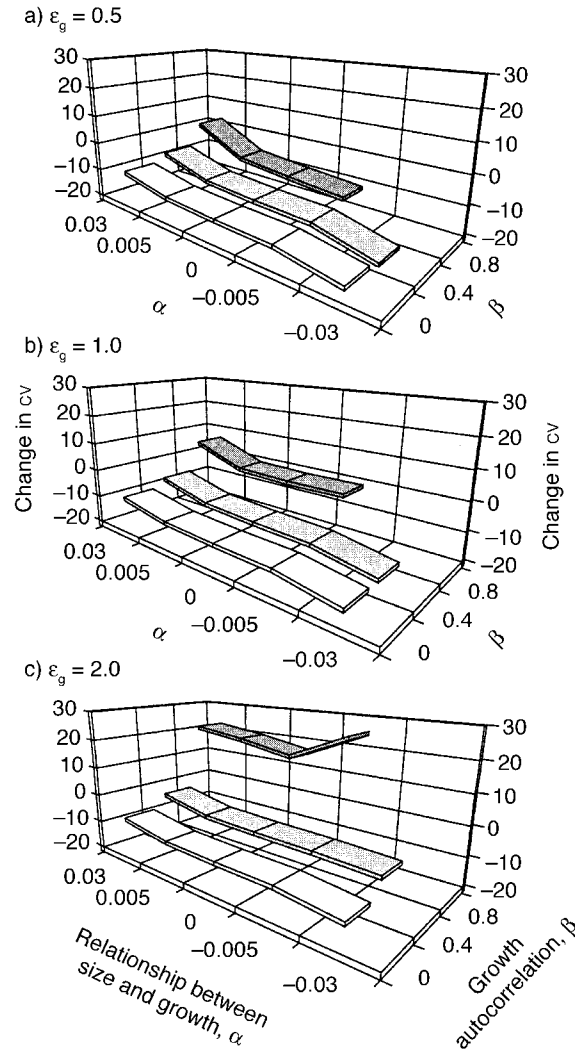


FIG. 2. The change in the CV of size from the end of the simulations to the beginning for a population modeled with a density-independent individual-based model. A positive value indicates an increasing CV and growth depensation. The initial size distribution was normally distributed with a mean of 50.0 and 1 SD = 15.0. The stochasticity in growth,  $\epsilon_g$ , increases from a to c. ANOVA results for the effect of each of the three factors are shown in Table 3a.

dependent process, the results were similar to those in the density-independent case. Linear density-dependence in growth resulted in growth depensation, or an increasing CV in size through time, only when a strong growth-autocorrelation term ( $\beta = 0.8$ ) was present (Fig. 5). Because the results were similar whether a cohort started with a normal or a lognormal distribution, we show results only for the initially normal distribution. Model runs with the combination of strong positive size-dependent growth ( $\alpha = 0.03$ ) and strong growth autocorrelation ( $\beta = 0.8$ ) could show a decline in size variability as many individuals became very large.

Nonlinear density-dependence in growth had similar net effects to linear density-dependence, although the generation of growth depensation was not as pronounced (Fig. 6). Again, because an initially lognormal distribution generated similar results, we do not show them here. As with previous results, growth depensation was most likely where there was higher stochasticity and growth autocorrelation. However, the addition of a nonlinear density-dependent term in growth was associated with a lack of growth depensation over some parameter values. In some cases, even strong growth autocorrelation did not result in an increased variability in size through time (Fig. 6a), indicating that although strong growth autocorrelation can result in growth depensation, it may not always do so.

DISCUSSION

The three species studied showed a range of growth patterns. The tidepool sculpin had a negative relation-

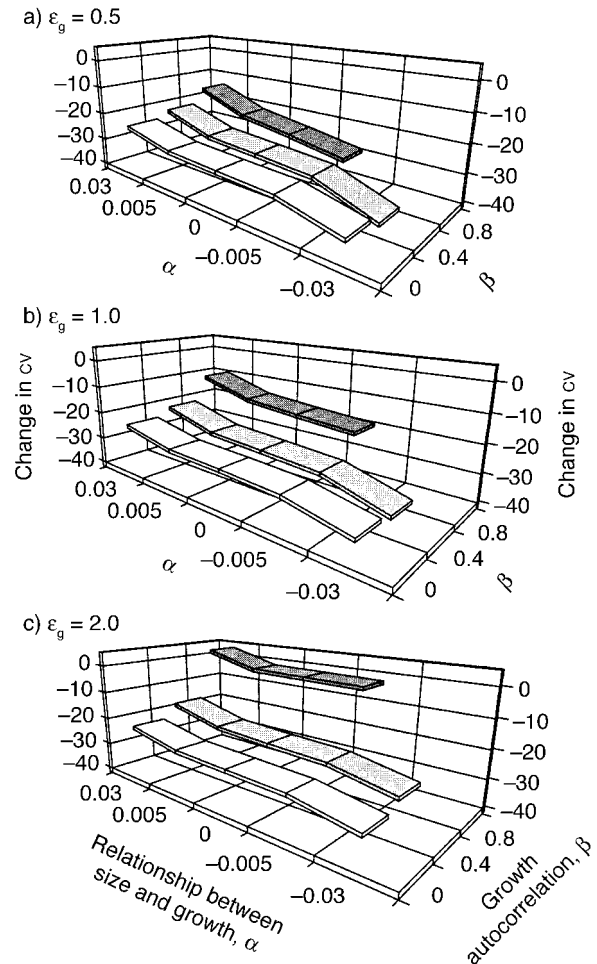


FIG. 3. The change in the CV of size as in Fig. 2, except that the initial size distribution was lognormally distributed, with a mean of 50.0 and 1 SD = 25.0. ANOVA results for the effect of each of the three factors are shown in Table 3b.

TABLE 4. Results of three-factor ANOVA for the effect of size-dependent growth ( $\alpha$ ), growth autocorrelation ( $\beta$ ), and stochasticity in growth ( $\epsilon_g$ ) on change in CV of size for density-dependent models with an initially normal distribution. (a) Growth was linearly density-dependent; results are given in Fig. 5. (b) Growth was a nonlinear function of density; results are given in Fig. 6.

Effect	df	ss	F	Total variance explained (%)
a) Linear density-dependence				
Size-dependence, $\alpha$	3	273	124	<0.01
Growth autocorrelation, $\beta$	2	814 920	556 449	96.0
Stochasticity, $\epsilon_g$	2	5760	3932	0.7
$\alpha \times \beta$	6	19 752	4496	2.3
$\beta \times \epsilon_g$	4	3836	1309	< 0.1
$\alpha \times \epsilon_g$	6	348	80	< 0.1
$\alpha \times \beta \times \epsilon_g$	12	3132	357	< 0.1
Error	1764	1291		
b) Nonlinear density-dependence				
Size-dependence, $\alpha$	3	11 901	4155	5.7
Growth autocorrelation, $\beta$	2	126 060	66 011	60.2
Stochasticity, $\epsilon$	2	39 184	20 518	18.7
$\alpha \times \beta$	6	606	106	0.3
$\beta \times \epsilon_g$	4	26 096	6832	12.5
$\alpha \times \epsilon_g$	6	1956	341	0.9
$\alpha \times \beta \times \epsilon_g$	12	1836	160	0.9
Error	1764	1684		

Note: All analyses were based on 50 replicate model runs and all factors and their interactions were significant at  $P < 0.001$ .

ship between size and growth and no evidence for growth autocorrelation, while the whelk also had a negative relationship between size and growth, but showed growth autocorrelation (Fig. 1). The kelp had positive size-dependent growth and growth autocorrelation. Based on the models presented here, we predicted that these patterns of growth should generate different patterns of variability among individuals. Model predictions were consistent with the data from these field populations. The two species with negative size-related growth and either no growth autocorrelation (tidepool sculpin), or “weak” growth autocorrelation (whelk, 0.438, Table 3), showed relatively stable or declining CV in size. In contrast, the kelp, which had relatively strong positive correlations in growth (a mean  $r$  of 0.503) as well as strong positive size-dependent growth, had an increasing CV in size through time.

Although our study does not elucidate the mechanistic basis of these growth patterns, we have hypoth-

eses about the underlying cause of these patterns. The terminology used by Huston and DeAngelis (1987) to describe the mechanistic basis of bimodality in size distributions is also applicable here. Positive correlations in performance among individuals might be the result of a mechanism that is “inherent” to the individual, such as genetic variation in performance or foraging behavior. Prey selection can have a genetic basis (Arnold 1981) and this might generate persistent growth differences. Alternatively, correlations in growth might be the result of “imposed” factors, such

TABLE 5. The change in the CV of size for the three species studied.

Species	Change in CV of size	
	Observed	Predicted
<i>Alaria nana</i>	43.8	-12.7 to 3.0
<i>Nucella canaliculata</i>	-6.1	-18.3 to -7.0
<i>Oligocottus maculosus</i>	-3.8	-15.7 to -11.1

Notes: The observed value is the net change through the study period, while the predicted values are based on the density-independent individual-based simulation with field estimates of  $\alpha$  and  $\beta$  and a range of  $\epsilon_g$  values. In the case of *Alaria nana*, the predicted values are based on the range of values we found for  $\alpha$  and  $\beta$ .

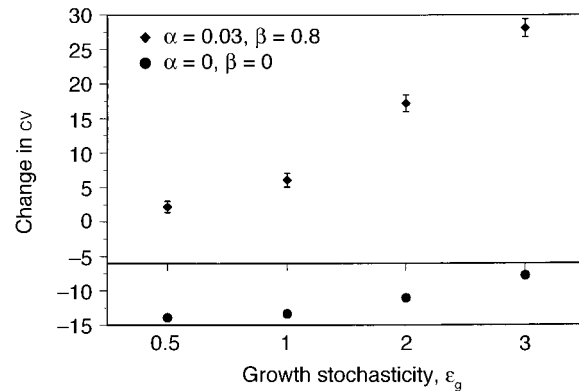


FIG. 4. The effects of increasing stochasticity in growth ( $\epsilon_g$ ) on the variability among individuals. The initial distribution of individual sizes was normal, and dynamics were density-independent. Data shown are means  $\pm 1$  SD for 50 replicate runs, when other determinants of growth rate ( $\alpha$ ,  $\beta$ ) were either set to zero or  $\alpha = 0.03$  and  $\beta = 0.8$ . The error bars are smaller than the symbol size for the case where  $\alpha = 0$  and  $\beta = 0$ .

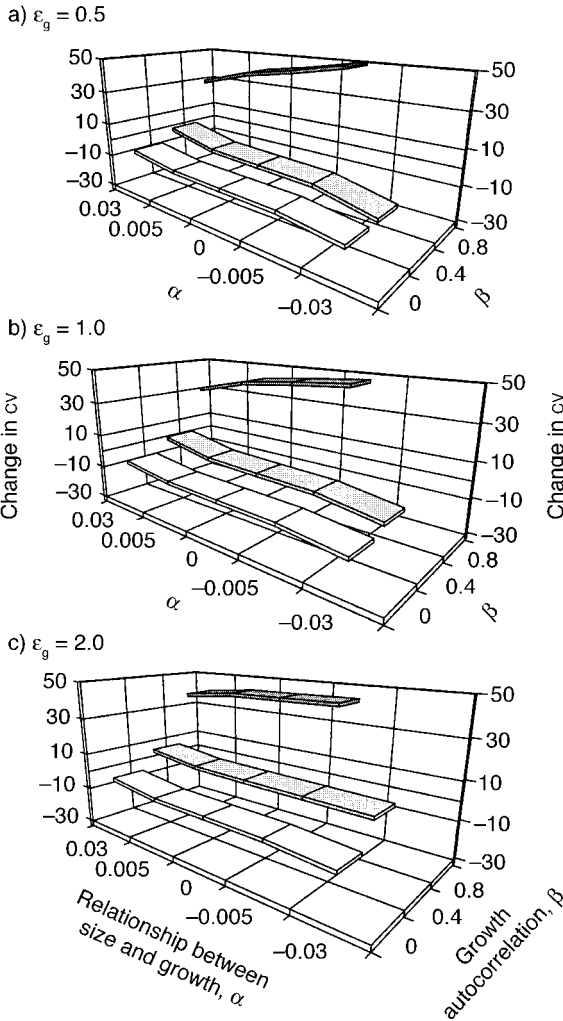


FIG. 5. The change in cv from the end of the simulations to the beginning for a model with linear density-dependence. A positive value indicates an increasing cv and growth depensation. The initial size distribution of  $50.0 \pm 15.0$  (mean  $\pm 1$  SD) was normally distributed. The stochasticity in growth,  $\epsilon_g$ , increases from a to c. ANOVA results for the effect of each of the three factors in shown in Table 4a.

as temporal and spatial heterogeneity in resources or mortality risks that impose predictable costs. For example, growth autocorrelation in the performance of individuals might be the outcome of unequal distribution of resources among individuals, resulting in a spectrum of performance among individuals. The correlations in growth may persist through time as a form of inertia in individual performance. In the case of the kelp species, inertia in growth might be explained by the interaction of individual plants with their light resource. Some plants might consistently have had better access to light while others consistently had less access. The feeding ecology of the whelk suggests that growth autocorrelation may be a result of individual interactions with prey (Palmer 1983). *Nucella canaliculata*

showed reduced growth when fed a diet of the barnacle *Semibalanus cariosus*, compared with a diet of other mussel or barnacle species. Additionally, *N. canaliculata* performance differed on the two mussel species it encountered. Conditioning and learning appear important in the prey choices made by individual *N. canaliculata* (Palmer 1984). Individual differences in foraging have also been shown in a related species, *N. emarginata* (West 1986). Thus, it is possible that temporally correlated variation in growth among individuals is due to spatial patterns of prey abundance, although the factors that affect gastropod growth are numerous (e.g., Spight 1981).

Tidepool fish showed no evidence of growth autocorrelation, suggesting that variation in performance is independent over each interval where it was estimated. Perhaps resources are distributed more equitably among these fish when compared with *Alaria* or *Nu-*

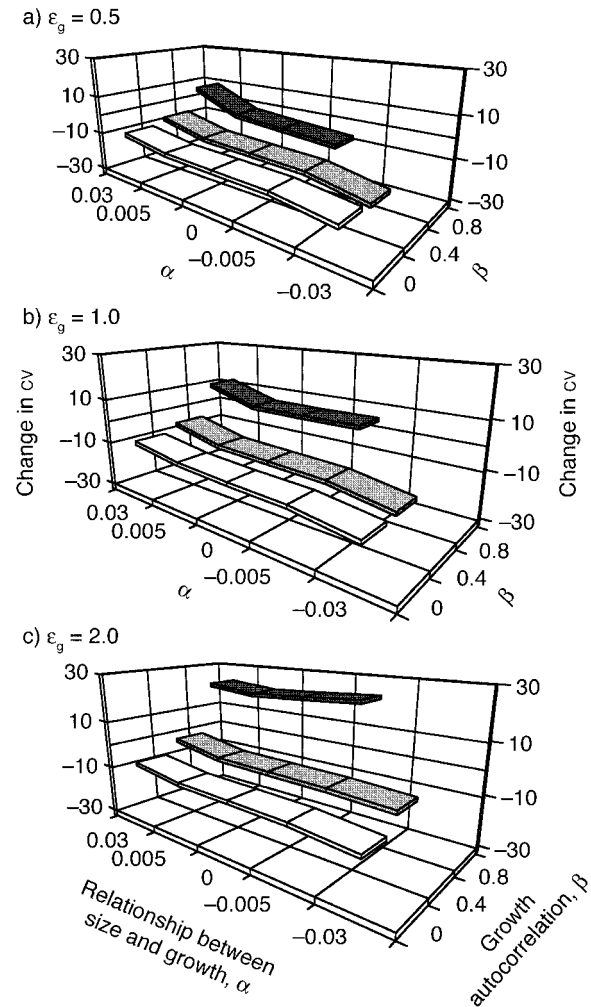


FIG. 6. The change in cv from the end of the simulations to the beginning for a model with nonlinear density-dependence. All else is as in Fig. 5 legend. ANOVA results for the effect of each of the three factors are shown in Table 4b.

*cella*. Thus, the three species studied here have a range of growth autocorrelation estimates from none (*Oligocottus maculosus*) to significant (*Alaria nana* and *N. canaliculata*). The biology of these species suggests that inherent and imposed mechanisms might be present simultaneously. For example, the ability to move is an inherent factor that interacts with heterogeneity of local resources. The stationary *Alaria* individuals, in an environment of heterogeneity in light resources, may readily develop correlations in performance. In contrast, the combination of mobility in fish and constant resource renewal by tidal action may serve to equalize resource use in *O. maculosus*. We expect, however, that growth autocorrelation may not always be absent in fish species based on previous work on fish growth. Using otolith increments in the larvae of bluefish (*Pomatomus saltatrix*) to estimate growth, Hare and Cowen (1997) found positive correlations, suggesting persistent performance differences in the early life history of these fish.

The unifying feature among all simulations, either density-independent or density-dependent, is the overwhelming role that growth-autocorrelation terms played in generating growth depensation, or an increased variation in size through time. Based on the parameter estimates from field populations reported here, parameter values used in Eq. 1 are very realistic and bracket a wide variety of taxa. We did, however, explore how increases in the strength of size-dependent growth ( $\alpha$ ) affected the generation of growth depensation and found that increases of an order of magnitude above those found in field populations were needed to result in an increased variability in size through time. Although a number of studies have provided important insight into the role of size-related processes in generating size distributions (DeAngelis and Huston 1987, Huston and DeAngelis 1987, Schmitt et al. 1987, Wyszomirski et al. 1999, Ziemba and Collins 1999, Ziemba et al. 2000), we know of no examples where growth autocorrelation was disentangled from size-related processes. Resolution of the relative contribution of growth autocorrelation vs. very strong positive size-dependence to the patterns of individual differences in size can only come from field studies where individual demography is followed through time and the strength of size-dependence is quantified.

Our models showed little change in the tendency for growth depensation with the inclusion of negative density-dependence. We modeled negative density-dependence as an additive term, assuming that increasing density would decrease the growth rate that all individuals realize in either a linear (Fig. 5) or nonlinear (Fig. 6) way. What is needed empirically, is an indication of how density affects the performance of individuals. If individuals are affected differently as density changes, so that size-dependent growth (our  $\alpha$  term) or growth autocorrelation (our  $\beta$  term) is changed, then the pattern of size variance may change

greatly as density changes. There is some suggestion that increasing density can change size-dependent growth in plants (Schmitt et al. 1987, Thomas and Bazzaz 1993), and a number of other studies describe how the distribution of sizes within a population changes when density is varied. Most studies document increased variability among individuals and increased skewness as density and intraspecific competition increases (Wilbur and Collins 1973, Harper 1977, Rubenstein 1981, Weiner 1985, 1988, Schmitt et al. 1987, Thomas and Bazzaz 1993, Ziemba and Collins 1999; reviewed for plants by Weiner and Thomas [1986]). Ziemba and Collins (1999) show this pattern in salamander larvae results from slowed growth of small individuals only. However, Turner and Rabinowitz (1983) showed that the variability in the size of isolated plants decreased through time, reflecting a lack of dominance-suppression interactions. Lomnicki (1988) attributed this to a decreased inhibition of isolated plants. However, Huston (1986) points out the importance of the distribution of individuals; random distributions may allow competitive asymmetries and dominance-suppression, while regular distributions may promote more symmetric interactions. Thus, it is possible to increase or decrease the tendency for growth depensation with density, demonstrating that it is important to determine how density affects individual attributes and the distribution of individuals, including how performance is correlated among individuals through time.

The importance of growth patterns that are independent of size in generating variability among individuals prompts interest in the mechanistic basis of autocorrelation in growth. Clearly, a nonuniform distribution of resources among individuals could generate differences among individuals. As recognized by Lomnicki (1988), intraspecific competition plays a likely role in generating increased variability through time, a result supported by his review of several analytical and simulation models. Additionally, Huston's (1986) study suggests that a regular distribution of plants will promote symmetric competition and dampen variability among individuals, whereas a random distribution may promote the phenomenon of dominance and suppression. Intraspecific competition is only a subset of the processes that will generate persistent differences among individuals. Environmental factors that are "imposed" on populations (sensu Huston and DeAngelis 1987) may also play a role in generating growth autocorrelation and variability among individuals. For example, the distribution of prey may be very important. As prey were increasingly unevenly distributed and spatially concentrated, Medaka fish showed greater size variation (Magnuson 1962); an uneven distribution of food also generated growth depensation in a laboratory study of coho salmon (Ryer and Olla 1996).

Increased skewness in size distributions, often associated with growth depensation, has also been attributed to asymmetric competition (Aikman and Wat-

kinson 1980, Begon 1984), although a model by Wyszomirski et al. (1999) suggests that asymmetric competition will not always lead to increasing variability in size. Simulation models suggest that asymmetric interactions may need to be of a critical strength and some initial variability in size may need to be present to allow competitive asymmetries to be enhanced (Wyszomirski et al. 1999). The importance of random variability in growth in enhancing growth depensation is also seen in our simulated populations; as random variation in growth ( $\epsilon_g$ ) increases, the magnitude of growth depensation also increases, an effect that is especially noticeable when growth is positively size-dependent and growth autocorrelation is strong (Fig. 4). Although asymmetries may be unrelated to size so that individuals that are better competitors are affected less than the "average individuals" (sensu Rubenstein 1981), size-related processes may compound asymmetries and thus correlations in growth through time (Magnuson 1962, Maret and Collins 1994). An extreme example of the latter is cannibalism, where the size structure of the population can be highly variable as some individuals achieve high growth by consuming conspecifics (DeAngelis et al. 1979, Maret and Collins 1994) while the performance of small individuals is stunted (Ziemba and Collins 1999). In plant populations, the presence of dominant and suppressed individuals (Obeid et al. 1967, Harper 1977) may be the result of both size-related competition for light and growth autocorrelation.

Our model and data from field populations demonstrate the need to disentangle size-related processes in individuals from temporal correlations in performance that are unrelated to size. Although theoretical studies emphasize the role of size in growth dynamics, our results suggest that growth autocorrelation will be important in some populations. Despite the empirical difficulties of gathering individual-based data, assays for the existence of growth autocorrelation will inform us about the propensity for growth depensation in a population and the future dynamics of that population. Attention to size-based processes and other correlates of performance in field populations, and how they may change as a function of environmental stressors or conspecific density, will improve our understanding of the dynamics of structured populations.

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