

INDIVIDUAL VARIATION AND ENVIRONMENTAL STOCHASTICITY: IMPLICATIONS FOR MATRIX MODEL PREDICTIONS

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Abstract. Populations are characterized by variability among individuals, a result of both intrinsic differences among individuals and environmental effects on individual performance. Despite the ubiquity of individual variation, its implications for population model choice are not fully understood. Population models that use state variables representing individual features such as age or size assume that these state variables can predict the population trajectory. However, state variables are often chosen based on convenience or necessity; only rarely are they tested for importance and accuracy when individuals vary. We examined whether matrix projection models, a common choice in population modeling, provide accurate predictions when individuals vary in a population. With both density-dependent and density-independent formulations, we tested whether matrix projection models that used size as a state variable captured the dynamics of populations projected with an individual-based simulation (or *i*-state configuration model). We varied the initial size distribution of individuals, the degree to which individual growth was size dependent, the tendency for positive correlations in growth through time, and the amount of stochasticity in growth, and we asked what conditions affect the accuracy of matrix models. Stochasticity alone did not compromise the predictions of matrix models; rather, only populations with individual variation generated by strong size-dependent growth and growth correlations were poorly described by matrix models. Otherwise, matrix models captured the trajectories of populations with a fair degree of accuracy. Overall, our results provide guidance as to when and how individual variation must be included in population projections, and when a simple matrix model framework is inadequate.

Key words: *cohort model; environmental stochasticity; individual-based model; individual variation; mathematical model; matrix projection model; population modeling; size variability; stochasticity.*

INTRODUCTION

A fundamental question for population biologists is what level of detail to include in population models that seek to estimate the abundance of individuals through time. Variation among individuals, sometimes exacerbated by environmental stochasticity, is a ubiquitous feature of populations. Because matrix projection models can use one or two simple indications of state, such as size, age, or location, they are a common tool used to model populations of heterogeneous individuals (Wootton and Bell 1992, Doak et al. 1994, Caswell 2001). Matrix model analyses have provided much insight into population dynamics, including how conservation efforts to increase population growth should be focused (Crouse et al. 1987, Lande 1988, Menges 1990, Crowder et al. 1994, Doak et al. 1994, Heppell et al. 2000). They have also revealed persistent patterns in the demography of organisms across many taxa (Silvertown et al. 1993, Pfister 1998, Heppell et

al. 2000, Sæther and Bakke 2000). The state or states that are chosen to be the basis for a specific matrix model are, at best, based on correlations between demographic rates and size, age, or the spatial location of the individuals in the population. At worst, a state variable such as size is chosen for necessity, because it is the only state of the organism that is known. However, it is rarely the case that a particular state variable is tested to determine its importance to explaining the data and to the predictions of the model (but see Law and Edley 1990, Cochran and Ellner 1992, Smith et al. 1997).

Two possible problems arise when discrete state variables such as size and age are used in a matrix model. First, a usually continuous indicator of fate is broken down into discrete categories. This problem has received attention, including how parameter estimation and model predictions are affected (Easterling et al. 2000). Analytical models that assume continuous functions, such as partial differential equation models (e.g., de Roos et al. 1992, McCauley et al. 1996) and integral projection models (Easterling et al. 2000), can reduce this problem. Algorithms for choosing category size seek to minimize the effect of forming discrete categories (Vandermeer 1978, Moloney 1986).

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A second problem arises when any state variable is an incomplete predictor of individual fate and individuals with very different potential are grouped. This is a problem common to any analytical model with one or few state variables. Populations are characterized by variability among individuals, a result of both intrinsic differences among individuals and environmental effects on individual performance. In plants, variability in size can be a critical determinant of the distribution of resources and the effects of biological processes (e.g., Weiner 1988), whereas in animals, variability in size can alter an individual's risk of predation as well as its trophic biology (DeAngelis et al. 1979, Maret and Collins 1994). There is also ample evidence that age is an important state variable in a multitude of species (e.g., Deevey 1947, Caswell 2001). However, there are processes, important among individuals, that are independent of size or age. For example, variability in many aspects of behavior, including activity levels and foraging strategies, can affect growth and mortality rates (Arnold 1981, Palmer 1984, West 1986, Werner 1992). In marine organisms as diverse as a kelp and a predatory snail, growth is dependent not only on size, but also on that individual's previous growth, a term referred to as growth autocorrelation (Pfister and Stevens 2002). Although the mechanisms underlying growth correlations may be diverse, the net result is that individuals in the population have markedly different size trajectories through time. Positive correlations in performance through time violate one assumption of matrix model projections, that of a one-step Markov process (Caswell 2001). They also bear on the methods used to determine size categories (e.g., Vandermeer 1978, Moloney 1986), which assume that individuals within a category will share similar fates. Although architects of analytical population models have emphasized the importance of the condition that individuals characterized by one or few state variables experience the same environment (Metz and Diekmann 1986, Caswell 2001), rarely is the condition tested. As a result, we have little guidance as to how differences among individuals will affect the accuracy of analytical models with one or a few state variables.

Here, we address when and if variation among individuals leads to scenarios in which a common state variable such as size is an incomplete predictor of population change. We ask if there are particular life history and population characteristics that will weaken the importance of size as a state variable in size-structured populations described with matrix models. In contrast to a state-based analytical model, a modeling approach that allows many states to be modeled simultaneously is an individual-based model (IBM or i-state configuration model; Caswell and John 1992), a simulation model in which all individuals in the population are tracked. Because IBMs do not necessitate grouping individuals into larger categories with identical probabilities of different fates, they have been suggested to

be a superior modeling approach when individuals vary (Huston et al. 1988, Judson 1994). Thus, we ask if matrix models will be poor predictors of population change when there are particular features of individual variation.

We focus on several types of variability, including (1) differences in the initial size variability of individuals, (2) stochastic variation among individuals in growth, and (3) variation that is the result of the positive temporal correlations in performance discussed previously. It has been suggested that IBMs are a superior model choice when stochasticity is relatively large, because it is not easily incorporated into many of the analytical models and its inclusion can change model predictions (DeAngelis and Rose 1992). However, Wilson (1998) successfully incorporated stochastic variation into deterministic population models (integro-differential equations) and found close congruence with a similar IBM. Variation among individuals in which the variation is either correlated among individuals or through time can also be a difficult feature to include in analytical models (Rice et al. 1993). Despite these important constraints on analytical models, we still have little guidance as to whether stochasticity will always lead to inaccurate predictions by analytical models, or whether there are features of populations that will lessen the importance of stochasticity. Because there is evidence that "growth autocorrelation" does occur in natural populations and that it can be important to patterns of size variability (Pfister and Stevens 2002), and because these correlations have been shown to be important to generating differences among partial differential equation and simulation models (DeAngelis et al. 1993), we include it here. We thus examine the effect of these three types of variation, in both density-independent and density-dependent matrix models, and over a spectrum of relationships between size and growth.

METHODS

To understand how individual variation could affect matrix model accuracy, we simulated population change with an IBM (individual-based model) under different scenarios of individual variability and then asked how accurately matrix models reflected the change.

An individual-based model for population change

A large number of organism attributes can be used in an IBM to reflect possible individual differences in all aspects of performance. However, our goal was to minimize the complexity of the IBM, including only key features of individuals that we assumed would be tractable in natural populations. We focused on using IBMs to model size-structured populations in which individuals of the same age can vary in size due to growth variability (e.g., Pfister 1996). In all cases, we used an IBM to generate populations with differing

amounts and types of variability in size and growth and then “sampled” those populations to parameterize a size-based matrix model. In all of our comparisons, the IBMs generated population change with a cohort model. Although we assumed that differences generated in the size structure of individuals within a generation would propagate among generations, due to the strong relationship between size and reproductive investment in size-structured populations, we also estimated how well matrix models predicted the number of offspring produced in a population.

Density-independent model.—We first developed an IBM that reflected simple rules of survivorship and individual growth that were unrelated to population density. All simulations were initialized with 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. Our decision to run the model for 24 time steps was based on comparing the output from 12 to 40 time steps. Although the patterns of model concordance were similar across this time horizon, increasing the simulation beyond 24 time steps resulted in an ever-declining number of individuals with which to make comparisons.

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 $1 \text{ SD} = 25$. Both distributions were truncated such that 1% of the distribution in the tails was not available, preventing negative or extremely large draws. Although normal distributions of organism size are often observed in nature (Uchmanski 1985), lognormal distributions, where there are many small individuals and few large ones, have also been observed in a variety of organisms such as plants (e.g., Stoll et al. 1994) and amphibians (Wilbur and Collins 1973). The use of the two size distributions allowed us to explore whether there were any model predictions that differed solely due to the initial size structure of the population.

Individuals in our population grew in accordance with a linear equation that described how growth at time t (the difference between size at time t and size at $t + 1$) was a function of individual size at time t (S_t), the previous growth rate of the individual (G_{t-1}), a growth rate parameter (C_g), and a term for error (ε_g):

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

In this way, we were able to see the effect of changes in α , β , or ε_g while other parameters were held constant or eliminated. Individual size was thus projected through time by adding growth (G_t) at each t .

Size dependency in growth, represented in our model by α , has been observed to be both 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 if α were either weakly or strongly negative or positive, and we used α values of 0.03, 0.005, -0.03 , and -0.005 . Here, β is a one-step autocorrelation in growth rate that controls individual differences in growth that are independent of size. The observation that a perfect positive temporal correlation in growth (correlation = 1.0) favors the use of IBMs (DeAngelis et al. 1993) prompted us to explore the effects of a range of correlations. We modeled no growth correlation ($\beta = 0.0$), weak growth correlation ($\beta = 0.4$) and strong growth correlation ($\beta = 0.8$). We explored only positive growth correlations because negative terms, although possible for short intervals during the life span of an individual, eventually lead 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 rescaled C_g depending upon the values of α and β . It 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, and \bar{G} was 2.0. Thus, Eq. 2 insures that the smallest individuals in the population will not suffer negative growth. This allowed us to construct model scenarios in which all individuals were certain to grow, but the strength of the contribution of different factors to growth varied among model runs.

The error term ε_g represents the variation in G_t that is unexplained by size and previous growth. We set ε_g to be a normally distributed random error with a mean of 0 and a standard deviation of 0.5, 1.0, or 2.0. Thus, increasing the standard deviation of ε_g increased the stochasticity in growth and, ultimately, size. Thus, our alterations of ε_g allowed us to examine how increasing stochasticity affects the concordance of the two model types.

Many theoretical analyses of the effects of size on growth have used a more complicated equation that expresses both the increase in growth due to assimilation and the loss due to respiration (e.g., Uchmanski 2000). We chose to use this relatively simple formulation for growth instead because of the ease with which parameters could be estimated in the field. Our analyses of individual growth in a species of kelp (*Alaria nana*), a gastropod mollusc (*Nucella canaliculata*), and a marine fish (*Oligocottus maculosus*) all show that the formulation has some generality, that the linearity assumption is valid, and that α and β estimates were in the range used here (Pfister and Stevens 2002).

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

Parameter	Biological meaning	Values used
α	relationship between size and growth	0.03, 0.005, -0.005 , -0.03
β	growth autocorrelation	0, 0.4, 0.8
ε_g	stochasticity in growth	mean of 0; SD of 0.5, 1.0, 2.0, 3.0
C_g	growth rate parameter	0.061 to 2.339
γ	strength of density dependence in growth	-5.0×10^{-5}
C_p	survivorship constant	0.95 or 0.97
δ	relationship between size and survivorship	0, -0.0005 , 0.0005
ε_p	stochasticity in survivorship	mean of 0; SD of 0.01
σ	strength of density dependence in survivorship	-5.0×10^{-6}

The survivorship probability of each individual at each time step (P_t) had a maximum value of 0.970 and changed with a slope (δ) with increasing size (S_t). 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), we report the results of using both positive and negative relationships. However, for most simulations we used a negative relationship between size and survivorship, with a δ value of -0.0005 . We introduced variation in survivorship using a normally distributed error term (ε_p) with a mean of 0 and a standard deviation of 0.01. Thus, survivorship could be described as

$$P_t = 0.97 + \delta S_t + \varepsilon_p. \quad (3)$$

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 the 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. Thus, our iterations added growth to existing size in the following way:

$$S_{t+1} = S_t + G_t. \quad (4)$$

Because we used two initial size distributions, four estimates for the relationship between size and growth (α), three values for the strength of correlations with previous growth (β), and three different values for the amount of unexplained variation in growth (ε), we ended up with 72 unique parameter combinations with respect to growth. Of these, we eliminated models with $\alpha = -0.03$ and $\beta = 0.8$, because they yielded individuals that continually shrunk in size. This reduced the number of IBM variants that we did to 66. We ran each model variant 50 times in order to generate the range of possibilities associated with each model. Parameters that we varied in the IBMs are summarized in Table 1.

A density-dependent individual-based model.—We introduced negative density dependence into the survivorship term to determine whether density depen-

dence alters the concordance between individual-based simulation models and matrix projection models. To do this, we simply added another term to the survivorship equation such that the probability that an individual survived (P_t) each time interval of the model was also related linearly to population size at time t (N_t):

$$P_t = C_p + \delta S_t + \sigma N_t + \varepsilon_p \quad (5)$$

where the survivorship constant (C_p) was generally 0.97, δ determined the relationship between size and survivorship, σ determined the strength of density dependence and was set at -5.0×10^{-6} , and ε_p was a normally distributed error term with a mean of 0 and a standard deviation of 0.01.

Negative density dependence was added in a similar way to the growth rate of an individual. A term for density dependence (γN_t) was added to Eq. 1, where γ was the coefficient for density dependence and was usually set at -5.0×10^{-5} :

$$G_t = C_g + \alpha S_t + \beta G_{t-1} - \gamma N_t + \varepsilon_g. \quad (6)$$

Thus, increasing population density resulted in decreased individual growth rates. We ran the model with density dependence in survivorship only, and with density dependence in both survivorship and growth.

Parameterizing a size-based matrix projection model

In order to compare the ability of matrix models to represent our individual-based dynamics, we needed to parameterize a matrix model for each IBM variant. Size-based matrix projection models describe the n size classes of an organism in a $n \times n$ matrix, where the matrix elements are either transition probabilities for one size to the next or reproductive rates (Lefkovich 1965, Caswell 2001). One determination made by every empiricist using a matrix model is the number of size classes that adequately describe their population. Sometimes the number of size classes is determined from biological information (such as size-related changes in survivorship and fecundity, e.g., Crouse et al. [1987]), while quantitative algorithms exist that minimize the error associated with size class boundaries (Vandermeer 1978, Moloney 1986). These algorithms estimate sampling error (the increasing error associated with a decreasing number of individuals in

each category) and distribution error (the error that results from a nonuniform distribution of individuals in each size class) as a function of size class boundary. Although, in theory, the size class boundary is chosen where each of these errors is minimized, in practice we found that this determination is hampered by error functions that do not monotonically change with size class. In other words, we found that each error estimate could have more than one minimum. Additionally, each type of error could indicate different size class boundaries, making it difficult to objectively determine size class boundaries. Given this difficulty, we chose size class boundaries that equalized the number of individuals in each size class. However, in order to see if we were systematically introducing any types of error, we estimated the sampling and distribution error for each of our size categories using the methods of Vandermeer (1978).

We used the simulated population generated by the IBM as a population that could be "sampled" to estimate the transition probabilities in the matrix. Because the transition probabilities for the density-dependent matrix models needed to change as a function of the population density at each time t , the matrix entries were estimated differently from the density-independent and density-dependent individual-based models.

For the density-independent models, we assessed the growth of one-quarter of the population at times 1, 8, 16, and 24, such that no individuals were sampled twice. We used this "sampling" to divide the population into three size classes and to estimate the transition probabilities for a matrix model with three size classes:

$$\begin{bmatrix} s_1(1 - g_1) & 0 & 0 \\ s_1g_1 & s_2(1 - g_2) & 0 \\ 0 & s_2g_2 & s_3 \end{bmatrix} \quad (7)$$

where s_i is the estimated survivorship of size class i and g_i is the probability that an individual grows out of size class i . The survivorship in the matrix model, s_i , was equivalent to P_i from Eq. 3 (with no ε_p term) and was estimated from the S_i that corresponded to the midpoint of each size class. The choice of three size classes has no biological basis here; it merely reflects a common dimensionality in the literature. Using Eq. 7 and an initial population vector for the three size classes, we projected the population size for 24 time intervals. Of course, we might expect that matrices of ever higher dimensions would eventually converge on an IBM, and we tested this.

As in the IBM, our matrix model simulated the growth of a cohort only, with no fecundity. Nevertheless, we did estimate the consequences for population growth of each model variant. Because size-structured populations are usually characterized by positive relationships between size and fecundity (Bierzychudek

1982, Huenneke and Marks 1987, Doak et al. 1994, Horvitz and Schemske 1995), we estimated the reproductive output of each parameterized matrix model and compared it with the reproductive output of our populations simulated with an IBM. We assumed that only size class 3 was fecund and that each individual in size class 3 produced offspring in direct proportion to its size at the end of the simulation. Biologically, this is analogous to a population that reproduces at the end of a growing season. For the matrix model estimation, the number of offspring was estimated as the mean size of size class 3 multiplied by the number of individuals within that size class. Thus, the number of offspring produced by a population parameterized with a matrix model could be compared with the number simulated with an IBM.

For the density-dependent models, matrix entries were a linear function of density. To determine the slope and intercept of the linear function, we ran the individual-based models at 17 different starting densities from 6000 to 22 000 individuals and used linear regression analyses to estimate how each matrix entry depended upon the current density of individuals. Matrix entries were estimated from the growth rate and survivorship of individuals at four disparate time intervals during the simulation, such that no individual was sampled more than once. Regression analyses then provided entries for the matrix that were dynamically dependent upon population size. In other words, every matrix entry was a function of current population size (e.g., DeAngelis et al. 1980, Levin and Goodyear 1980); in our analyses, this function was linear. Thus, our parameterization of the matrix projection model was analogous to what might be used empirically: establishing or observing populations at different densities to determine how transition probabilities are related to density.

Assessing matrix model performance

We compared how well matrix models predicted the individual-based simulation using two types of model output. First, we estimated the similarity in the predicted total number of surviving individuals at the end of the projection, by comparing the percentage by which the matrix model prediction differed from the IBM output. We asked if the matrix model prediction was greater than the differences that could be generated among the 49 other repeated IBM runs. Thus, we were able to ask whether the prediction made by the matrix model was a statistical outlier relative to the output generated from the IBM runs. The second type of model output that we examined was the concordance between the predictions for the number of individuals in each of the three size classes. We summed the square of the differences (sum of squares) in each size class for the matrix model and the mean of the 50 IBM runs. By comparing the size-structured output, we were also able to gain insight into why the matrix model could differ.

Thus, we were able to look at the relative difference between models as a function of the changes that we made to each of the parameters.

To test whether our results were robust over a variety of matrix dimensions, we compared the performance of matrix models when the number of size classes varied from three to 15. Again, we estimated the similarity in the predicted total number of individuals at the end of the projection, as well as the sum of the squares of the differences in each size class.

For all matrix model assessments, we estimated parameters from the individual-based simulations. These “populations” had stochastic inputs (ϵ_g and ϵ_p) and size-dependent survivorship. Thus, matrix model estimates from our sampling methodology might be expected to have some deviation from the individual-based simulations. Although any deviations in matrix predictions due to this sampling methodology would be present for all model runs, we tested how large this effect was by running a comparable IBM-to-IBM comparison. We estimated parameters for the IBM with the same sampling methodology used in the matrix model estimations. We then used these estimates to run the IBM again, and compared the final outputs of the original IBM with the reestimated IBM.

The effects of stochasticity on matrix model performance

We isolated the effects of stochasticity in growth (ϵ_g) on model concordance by setting α and β to zero in the density-independent model of Eq. 1. We increased ϵ_g from 0.5 to 3.0 and quantified model divergence.

RESULTS

Density-independent models

Depending on the parameter combination, after 24 time intervals there were 497–2585 individuals remaining in the IBM simulations and 1464–2667 individuals in the corresponding matrix model projections. This range was almost certainly due to the size disparity that could be generated by differences in growth, allowing size-dependent survivorship to affect total population size. The total final population size projected by matrix models deviated from a minimum of 3.0% to a maximum of 193.0% compared with the IBMs. Over many combinations of α and β , matrix models deviated relatively little (Fig. 1). However, when growth was both highly positively size dependent ($\alpha = 0.03$) and correlated strongly with growth in the previous interval ($\beta = 0.8$), matrix models consistently showed large differences, indicating that the matrix models predicted a greater number of individuals than was seen in the simulations after 24 time intervals. There were only four instances in Fig. 1 in which the deviation by the matrix model was greater than any difference that arose among IBM runs ($\alpha = 0.03$ and all β , and $\alpha = 0.005$, $\beta = 0.8$). Thus, for an initially

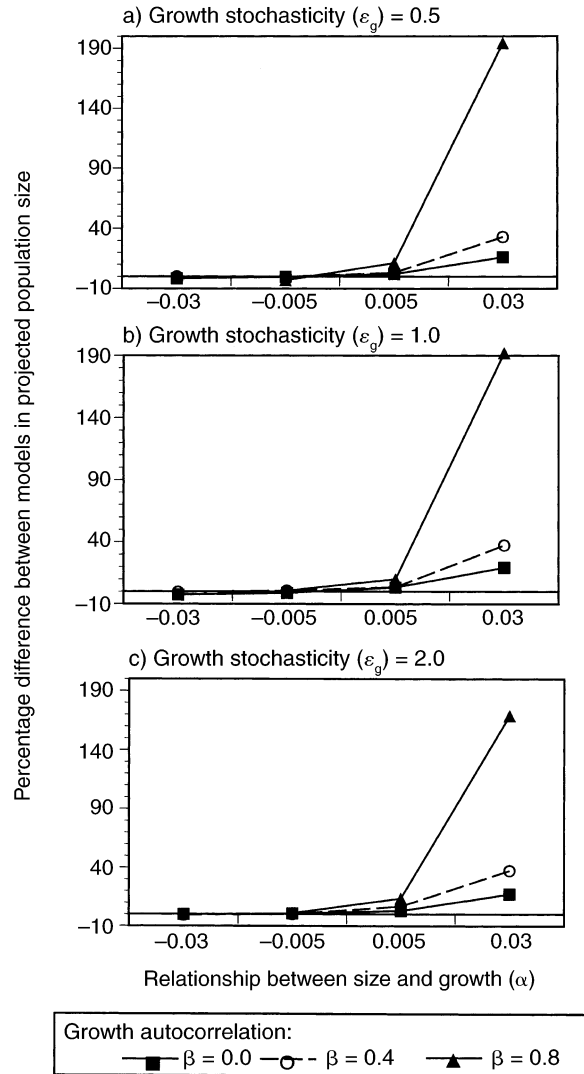


FIG. 1. The percentage difference in the population size projected with a matrix model compared with an individual-based simulation over a combination of model parameters ($\alpha = -0.03, -0.005, 0.005, 0.03$; $\beta = 0, 0.4, 0.8$; and $\epsilon_g = 0.5, 1.0, 2.0$) for an initially normal distribution of sizes and density-independent dynamics. The stochasticity in growth increases from (a) to (c). Positive values indicate that matrix models have a greater number of individuals at the end of the projection. The deviation by the matrix model is greater than any difference that arose among IBM runs in four instances ($\alpha = 0.03$ and all β ; and $\alpha = 0.005$, $\beta = 0.8$). The lines are included for clarity but are not meant to imply a continuous relationship. In this and subsequent figures, there is no result for $\alpha = -0.03$ and $\beta = 0.8$, because this parameter combination was eliminated due to extreme negative growth of individuals.

normal distribution, matrix models were poor predictors only in populations with either strong positive size-dependent growth ($\alpha = 0.03$) or weak size-dependent growth ($\alpha = 0.005$) with strong autocorrelation in growth (Fig. 1). Fig. 1 also shows that increasing the amount of stochastic variation in growth (ϵ_g) had little

effect on how matrix models captured the dynamics. Although Fig. 1 shows only the results for an initially normal distribution, the results were unchanged for an initially lognormal distribution. Thus, variation among individuals in initial size did not seem to change the accuracy of matrix models.

The large difference between models with positive α and β values persisted when we examined the number of individuals predicted to be in each size class (Fig. 2). Matrix model results matched the simulated populations well over many parameter values, with the obvious exception of the case in which growth is both strongly positively size dependent ($\alpha = 0.03$) and correlated strongly with growth in the previous interval ($\beta = 0.8$). Over all other α , β , and ε_g values and increasing levels of stochasticity (increasing ε_g), the estimated sum of squares was relatively low. However, when we contrasted population size differences, we found that, over all parameter values, the sum of squares values for a matrix model vs. its corresponding individual-based simulation was always greater than the sum of squares values among IBMs. Clearly, however, deviations for parameter values other than $\alpha = 0.03$ and $\beta = 0.8$ are relatively small and correspond to some instances in which total population size was predicted well by matrix models (Fig. 1).

In many instances, matrix models accurately predicted the total population size (Fig. 1); however, they consistently estimated a higher number of individuals in the first two size classes and underestimated the number that reached the third size class. The exceptions to this were the instances in which growth was strongly positively size dependent ($\alpha = 0.03$) and growth autocorrelation was present ($\beta = 0.4, 0.8$); in these cases, the number of individuals in each size class was always overestimated in the matrix model. Because the accuracy of the matrix models was relatively unaffected by the initial distribution (normal vs. lognormal) of those individuals, the lognormal results are not shown.

Increasing the amount of stochasticity in growth (ε_g) could either increase or decrease the concordance between models in all parameter combinations, regardless of the initial size distribution (Fig. 2). However, the changes in sum of squares values were relatively small, and the overall patterns in the sum of squares data remained similar as ε_g values were increased. When coefficients from determinants of growth were set to zero (Eq. 1) and the stochasticity term alone affected growth rate, increasing stochasticity (ε_g) resulted in little pattern in model concordance (Fig. 3). The sum of squares values in Fig. 3 are directly comparable to those in Fig. 2, and suggest that even very large stochasticity terms do not decrease the accuracy of matrix models.

Because we used a sampling methodology to estimate matrix models parameters that might lead to deviations due simply to the inclusion of stochasticity in our individual-based simulations, we estimated how

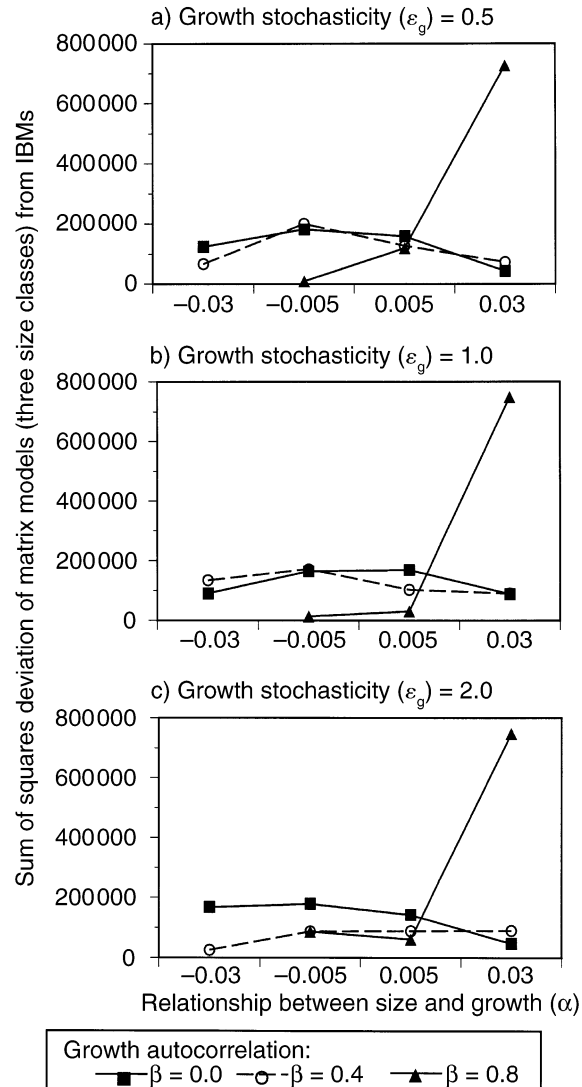


FIG. 2. The deviation (in sum of squares) of three-size-class matrix models from individual-based simulations with an initially normal size distribution and density-independent dynamics. The stochasticity in growth increases from (a) to (c).

similar two IBM model runs were when the second run was based on parameters estimated from sampling the first. We did this for all combinations of α and β , an ε_g of 0.5, and an initially normal distribution. These results are thus directly comparable to those in Figs. 1a and 2a. We found that the average sum of squares in these comparisons was only 50 072 and the average difference in the final population size was 6.6%. The combination of α and β in the model had no effect on the magnitude of deviation. Thus, although the stochasticity included in the simulations meant that parameters such as α , β , δ and regression intercepts could not be estimated precisely, the small differences in their estimations led to relatively small differences in the projected population.

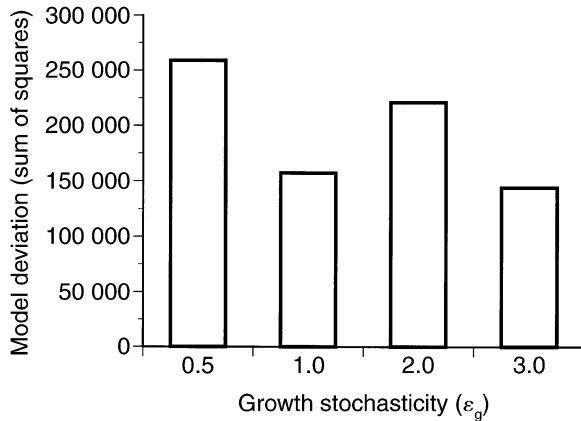


FIG. 3. The effects of increasing stochasticity in growth (ϵ_g) on the accuracy of matrix model projections shown as the deviations (in sum of squares) between matrix models and individual-based projections as growth stochasticity increases. All other determinants of growth rate (α , β) are set to zero. The initial distribution of individual size is normal and dynamics are density independent.

To determine if the population structure generated within one cohort could have multigenerational effects, we estimated the reproductive output by estimating the number of offspring produced in each simulated population. When the number of offspring produced was directly proportional to the size of individuals in the largest size class, we again saw that matrix model performance was poor when growth was strongly positively size dependent and correlated through time (Fig. 4). The deviation between the matrix model and IBMs was greater than any difference that arose among IBM runs, except in the case of parameter combinations $\alpha = -0.005$ and $\beta = 0.8$ and $\alpha = 0.03$ and $\beta = 0.0$. The largest disparity was, of course, for the scenario in which $\alpha = 0.03$ and $\beta = 0.8$; the matrix model predicted 62.2% more offspring after one generation than the corresponding individual-based simulations. Thus, the matrix model disparities in population size (Fig. 1) and population structure (Fig. 2) are likely to have strong multigenerational effects through their effect on the estimated number of offspring produced (Fig. 3).

Because algorithms for size class selection seek to minimize sampling and distribution errors (Vandermeer 1978, Moloney 1986), we estimated the sampling and distribution error for each size class in all matrix models used to generate the results in Figs. 1 and 2. To simplify our analyses, we focused our attention on the errors in the middle size class. Although the sampling error was very low (<0.13) for all parameter values, the distribution error could be much greater, ranging from 0.26 to 0.86. Distribution errors decreased as growth became increasing negatively related to size (decreasing α). Because we were concerned that our size class boundaries might result in sampling or distribution errors that caused differences in model comparison, we asked if there were a correlation between

either of the error types and the sum of squares values in Fig. 2. We found no significant relationship between distribution error and model deviations ($r = 0.089$, $P = 0.476$, $n = 66$), but a positive relationship between sampling error and the sum of squares values ($r = 0.465$, $P < 0.001$, $n = 66$). However, the relationship between sampling error and model deviation switched in sign when the values associated with $\alpha = 0.03$ and $\beta = 0.8$ were removed. Thus, it appeared that extremely large deviations by matrix models were associated with greater sampling errors, although in no case did the sampling error exceed 0.13. However, over most parameter values, there was little evidence that errors associated with size class boundaries were also associated with model deviations. Although we used absolute growth rates, it is possible that the use of specific growth rates would result in increasing skewness in size (Hara 1984) and greater error in parameterizing matrix models.

The nature of the relationship between survivorship and size (the parameter δ ; negative, positive, or unrelated) resulted in somewhat predictable deviations between the two model types. Fig. 5 illustrates a comparison of the projection of each model when we varied size dependence in survivorship. In the case of negative size-dependent survivorship previously reported, strongly positive size-dependent growth ($\alpha = 0.03$), in combination with growth autocorrelation ($\beta = 0.4$ and 0.8), resulted in individuals getting large and suffering higher mortality. In these cases, matrix models deviated because they overestimated the number of individuals

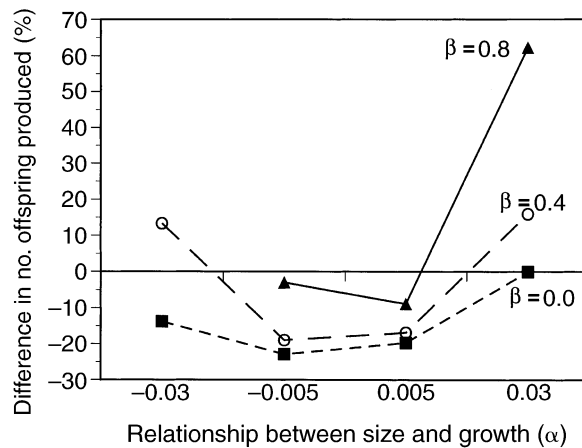


FIG. 4. The percentage difference in the number of offspring produced by a population described with a matrix model vs. an individual-based projection; only the case in which stochasticity in growth (ϵ_g) is 1.0 is shown. The strength of growth autocorrelation is denoted by β . Positive values indicate that matrix models had a greater number of offspring at the end of the projection. The deviation between the matrix model and IBMs is greater than any difference that could result among IBMs, except in the case of parameter combinations $\alpha = -0.005$, $\beta = 0.8$ and $\alpha = 0.03$, $\beta = 0.0$. The initial distribution of individual size is normal, and dynamics are density independent.

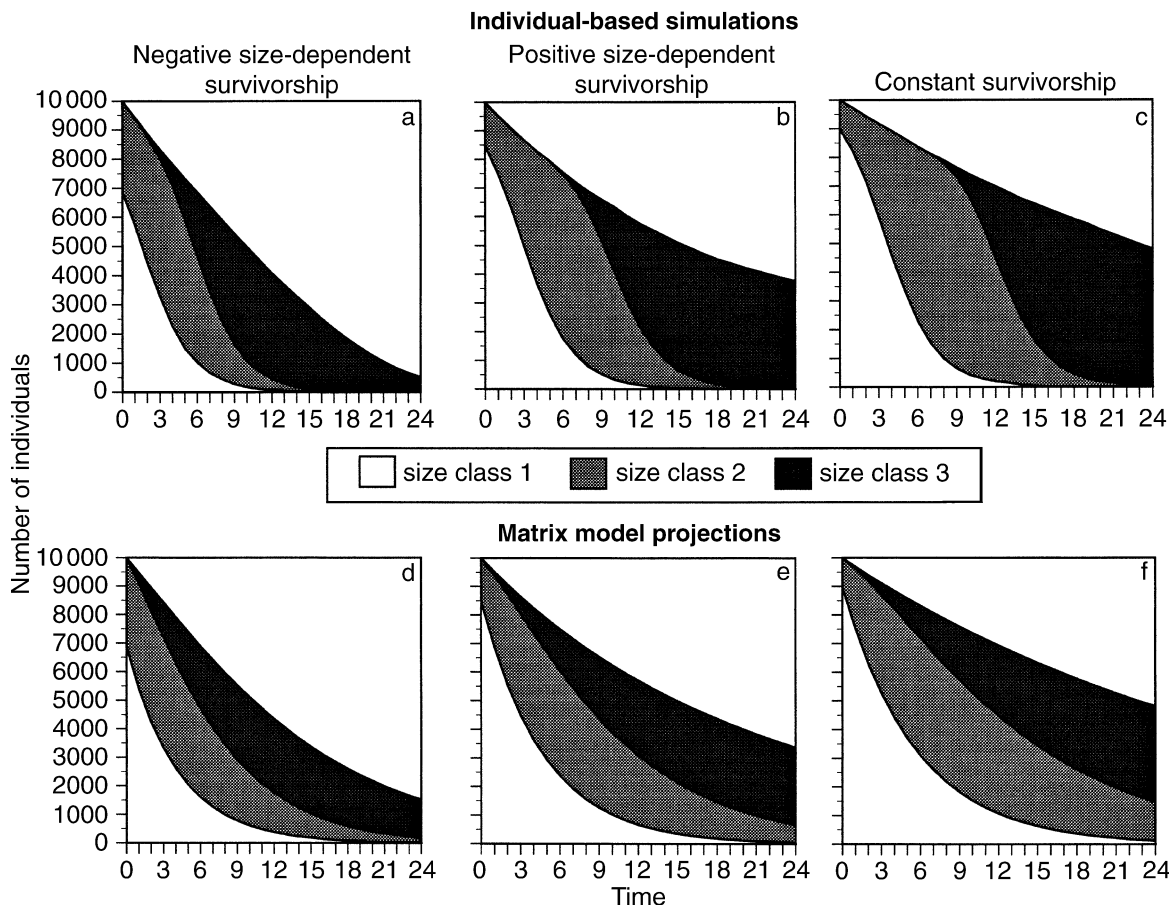


FIG. 5. The difference in the predicted size class composition through time for matrix model projections compared with an individual-based simulation with three different scenarios for survivorship. In the left-hand column, survivorship is negatively related to size, whereas the middle column shows the results when survivorship is positively related to size. Survivorship is unrelated to size in the right-hand column. Results are for the density-independent model when $\alpha = 0.03$ and $\beta = 0.8$.

in the largest size class (contrast Fig. 5d and a). In comparison, positive size-dependent survivorship resulted in nearly identical patterns in sum of squares and total population size, with matrix models underestimating the number of individuals in the larger size class (compare Fig. 5e and b). We explored how large α and β alone can generate deviations in the model when survivorship is unrelated to size, and we found that even in the absence of size-dependent survivorship, matrix models underestimated the number of individuals in the largest size classes, while generally overestimating the number in the smaller size classes (compare Fig. 5f and c). Despite the deviations seen in the distribution of individuals among size classes, the total number of individuals predicted by both models was highly concordant and differed by <1%. Thus, variability in growth alone (via α , β , and ϵ) can generate size class differences and large sum of squares, while total population size remains similar in both models. If, however, size-dependent survivorship is acting in tandem with processes that generate large size

differences among individuals (as positive values of β do), then matrix models that use mean values for each size class are increasingly unable to predict either the distribution of individuals among size classes or the total population size.

An important consideration for these model comparisons is whether the results from a matrix model based on three size classes is representative of matrices with increasing dimensionality. Fig. 6 shows comparisons of the effect of increasing the number of size classes for two cases: single where model deviation was great ($\alpha = 0.03$, $\beta = 0.8$) and where matrix models matched IBMs well ($\alpha = 0.005$, $\beta = 0$). Although the matrix model better predicted the final population size as dimensionality increased, the final population size estimate still differed by 63% when positive size-dependent growth and growth autocorrelation were present. Not surprisingly, the already accurate predictions of the case where $\alpha = 0.005$, $\beta = 0$ were little changed by increasing from three size classes to 15. Thus, although increasing the number of size classes could

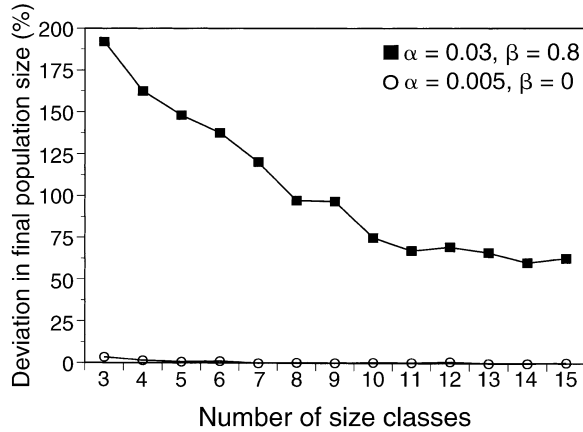


FIG. 6. The percentage difference in the projected population size for a matrix model as the number of size classes is increased from 3 to 15. Two scenarios are shown, one with positive size dependence and strong growth autocorrelation ($\alpha = 0.03$, $\beta = 0.8$), and one without ($\alpha = 0.005$, $\beta = 0$). The initial distribution of individual size is normal, and dynamics are density independent. The stochasticity in growth (ϵ_g) is 1.0.

change the disparity of matrix models, the trends were similar and still indicate that matrix models perform poorly when growth correlations are coupled with strong positive size dependence in growth.

Density-dependent models

The performance of density-dependent matrix models was similar to the density-independent scenarios when negative density dependence was included in either survivorship or both survivorship and growth. As with the density-independent models, matrix models matched individual-based simulations poorly when growth was strongly size dependent ($\alpha = 0.03$) and growth correlations were high ($\beta = 0.8$). Fig. 7 shows model differences (as the percentage difference in final population size) for a selected group of model runs with negative density dependence in survivorship (Fig. 7a) and both survivorship and growth (Fig. 7b); these scenarios are directly comparable to Fig. 1b. Predicted population sizes could differ as much as 34% between matrix and individual-based models when density dependence was included, but the extremely large deviations ($>100\%$) were again restricted to $\alpha = 0.03$, $\beta = 0.8$ (Fig. 7). The deviation between the matrix model and IBMs was greater than any difference that could result among IBMs for all models with $\alpha = 0.03$ and all models with $\beta = 0.8$ in Fig. 7a, and in all parameter combinations in Fig. 7b. As in the density-independent results, although some differences below 10% could represent significant deviations, the extremely large deviations were restricted to instances of strong positive size-dependent growth and growth autocorrelations. The percentage differences in predicted population size were very similar, whether density dependence was included (Fig. 7a, b) or ignored (Fig. 1b).

When we examined model concordance with sum of the squared deviations in the three size classes, the inclusion of density dependence (Fig. 8a, b) led to similar patterns reported for the density-independent models (Fig. 2b). It is important to note that the decreased sum of squares estimates for the density-dependent models indicate only that fewer individuals survived until the end of the model run (due to the detrimental effect of density dependence). As with the density-independent models, large model deviations were restricted to $\alpha = 0.03$, $\beta = 0.8$, regardless of whether negative density dependence affected survivorship (Fig. 8a) or both survivorship and growth (Fig. 8b). As reported for the density-independent models with negative size-dependent survivorship, differences in the number of individuals in each size class resulted when some individuals became large (positive α and

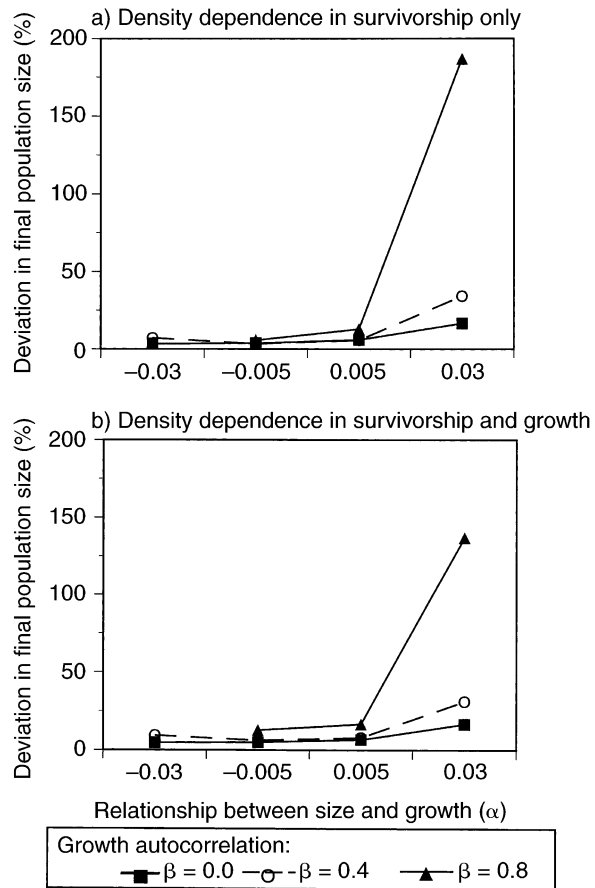


FIG. 7. The percentage difference in the projected population size for matrix models compared with individual-based simulations using density-dependent models. There is an initially normal distribution and (a) density dependence in survivorship only or (b) density dependence in both survivorship and growth. The stochasticity in growth (ϵ_g) is 1.0. The deviation between the matrix model and IBMs is greater than any difference that could result among IBMs for all models with $\alpha = 0.03$ and all models with $\beta = 0.8$ in (a) and in all parameter combinations in (b).

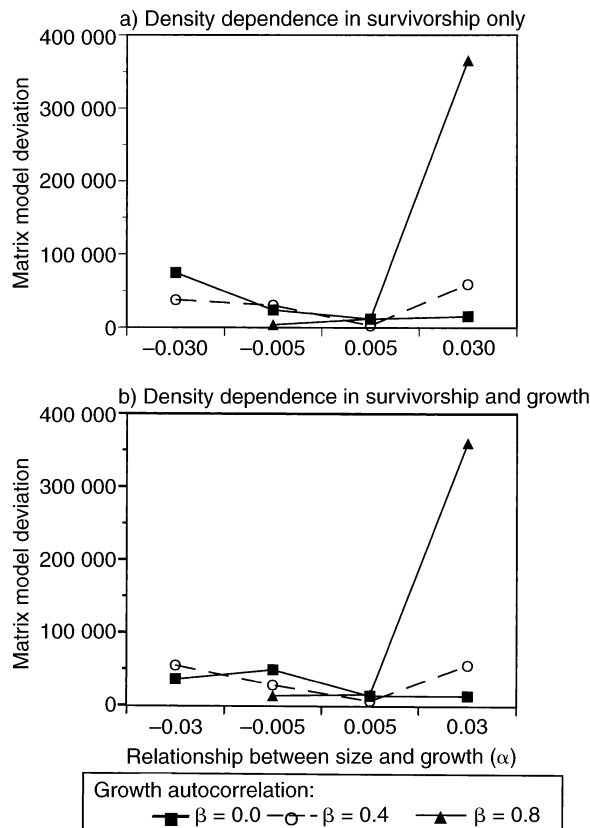


FIG. 8. The deviation (in sum of squares) of three-size-class matrix models from individual-based simulations using density-dependent models. Populations were initiated with a normal size distribution and (a) density dependence in survivorship only or (b) density dependence in both survivorship and growth. The stochasticity in growth (ϵ_g) was 1.0.

β values) and then suffered higher mortality in the IBM than in the matrix model (Fig. 5a). Thus, the matrix model again overestimated the number of individuals in the largest size class.

Because the role that stochasticity in growth (ϵ_g) played in density-dependent models was also similar to that seen in density-independent models, we show only the density-dependent model output for an initially normal distribution of sizes and an intermediate level of stochasticity ($\epsilon_g = 1.0$). As stochasticity increased, the matrix model predictions changed little, suggesting that matrix models can provide an accurate descriptor when stochastic variation in growth is present.

DISCUSSION

Over a number of parameter combinations, matrix models could capture the trajectories of simulated populations. However, we found that size could be an inadequate state variable for describing population changes in the cases in which growth was related positively to size ($\alpha = 0.03$) and variation among individuals was correlated through time (Figs. 1, 2, and 4). These patterns resulted whether survivorship was neg-

atively or positively related to size. Only when there was no relationship between size and survivorship could matrix models accurately project the number of individuals. Even in these cases, however, matrix models still incorrectly predicted the distributions of individuals among size classes (Fig. 5f vs. c).

The addition of size-related survivorship has a predictable effect on matrix model performance. When survivorship was negatively related to size, positive α values and β values ≥ 0.4 resulted in some individuals that became large and susceptible to mortality. As a result, IBMs predicted fewer individuals in size class 3 than did the corresponding matrix model, which used a mean survivorship estimate for the entire size class (Fig. 5e). Similarly, when survivorship was positively related to size, larger individuals had low susceptibility to mortality, and matrix models underestimated their abundance (Fig. 5b). However, even when there was no size dependence in survivorship, size and growth autocorrelations continued to contribute greatly to allowing rapid growth in some individuals. The net result was that matrix models were unable to capture these inequities and they underestimated the transition of individuals to larger size classes (Fig. 5f).

The greatest accuracy of matrix models occurred when growth was not positively size dependent and growth autocorrelation was absent. Comparisons of both projected population sizes (Figs. 1 and 7) and distributions of individuals among three size classes (Figs. 2 and 8) yielded similar results. Although we expected that IBMs and matrix models should converge in their results as more size classes were added to the matrix projections, the consistent use of three size classes indicated the individual features that cause model divergence. Although model concordance improved as the number of size classes increased from three to 15 (Fig. 6), large differences in the predicted population size remained when growth autocorrelation was strong and growth was positively size dependent. Where matrix models performed well with the use of only three size classes (weaker size dependence and no correlations in growth), increasing the number of size classes did not change concordance (Fig. 6). In field studies in which the number of individuals that can be studied is limited and individuals are often unevenly distributed among sizes or stages, increased sampling and distribution errors are an inevitable consequence of increasing the number of size categories (Vandermeer 1978, Moloney 1986).

When DeAngelis et al. (1993) compared an IBM and a partial differential equation model of a cohort of fishes, they found that the models could generate similar size frequencies in simulated populations, even when stochasticity in growth was added. However, when they used the IBM to model a population with positive temporal correlations in growth (e.g., our β term), they found that the predicted size frequency distribution could diverge markedly. Although they did not incor-

porate growth correlations in a partial differential equation, the authors inferred that the partial differential equation model would not accurately describe a population with these correlations in growth. Our results show that the effects of positive temporal correlations are likely to cause matrix models to deviate primarily when they are combined with positive size-dependent growth.

Importantly, positive temporal correlations in growth are a pattern seen in some natural populations. Pfister and Stevens (2002) show that strong growth correlates are present in the kelp *Alaria nana* and the predatory gastropod *Nucella canaliculata*, whereas the common tidepool sculpin *Oligocottus maculosus* and the sea star *Crossaster papposus* (Carlson and Pfister 1999) do not show these correlates. Positive correlations in performance through time could be the result of traits intrinsic to an organism, such as inherent or acquired foraging differences (Arnold 1981, Palmer 1984, West 1986, Werner 1992, Dukas and Bernays 2000). Alternatively, extrinsic factors such as environmental heterogeneity could also generate persistent differences in resource availability among individuals. For example, plant populations residing in heterogeneous soil conditions or environments that vary in the amount of light available will have some individuals with necessary resources and others that are limited. The sedentary nature of plants and some animals may make their populations especially prone to autocorrelations in growth. Simultaneously, the size-structured nature of plant populations also means that they are often described with matrix projection models (e.g., Bierzychudek 1982, Menges 1990, Silvertown et al. 1993, Horvitz and Schemske 1995) and are thus prone to inaccurate projections.

Regardless of the underlying mechanism, variation among individuals that is correlated through time and is unrelated to the usual states included in analytical models (e.g., age and size) can result in matrix model inaccuracies. However, the mechanism underlying these correlations may require different modeling approaches. If growth autocorrelation is identified as the result of traits intrinsic to the organism, then an analytical model with another state variable might be used to reflect this feature. For example, physiologically structured models using partial differential equations might be appropriate (e.g., Metz and Diekmann 1986, de Roos et al. 1992, de Roos 1997). Alternatively, correlations that result from environmental heterogeneity clearly violate the condition that all individuals described by a state variable such as size have identical experiences in the environment (e.g., Metz and Diekmann 1986, Caswell 2001); individual-based simulations may be the appropriate alternative.

Although none of the models used here included multigenerational dynamics, our estimates of offspring production suggest that model estimation errors in a single generation will have marked effects on the es-

timination of reproduction for the population and, hence, future dynamics. If reproduction is related to size, as we have assumed here and as demonstrated for myriad plant and animal species that have indeterminate growth (e.g., Samson and Werk 1986, Sebens 1987), then the size structure generated by any model will have important effects on the dynamics of future generations. For example, the divergence in the predicted numbers of individuals in each size class shown in Fig. 5d shows that the number of individuals in the largest size class is overestimated. As expected, the matrix model also overestimates the number of offspring produced in that population (Fig. 4); logically, we would expect matrix model projections to continue to diverge with every subsequent generation.

An interesting consideration for populations in which performance correlations can occur in reproductive success is whether autocorrelation can alter demographic stochasticity (sensu Fox and Kendall 2002, Kendall and Fox 2002). Jensen's inequality (Ruel and Ayres 1999) relates a response variable to variability in a driving factor. Depending upon the shape of the relationship between the variable factor and the response variable, variance in the response can be unchanged, depressed, or amplified (Lomnicki and Symonides 1990, Ruel and Ayres 1999). In populations characterized by persistent differences among individuals, long-term dynamics may be affected, stressing the importance of understanding the nature of variation among individuals.

Stochasticity and matrix model performance

Because of the difficulty of incorporating stochasticity into analytical models, IBMs have been suggested as a superior model choice when stochasticity is high (DeAngelis and Rose 1992). Indeed, it is the stochasticity incorporated in IBMs that has been suggested to cause deviations from analytical models; when stochasticity was included in integrodifferential population models for predator and prey, there was greater congruence with somewhat comparable IBMs (Wilson 1998). Surprisingly, however, when we examined whether increasing stochasticity in growth (increasing ϵ_g) resulted in greater disparity of matrix models, we found that there was no pattern associated with increasing ϵ_g , either in population size (Fig. 1) or sum of squares deviations in size classes (Fig. 2). Instead, the dependence of growth on size (α) and growth autocorrelation (β) influenced model results much more strongly, with great effects when growth was both positively size dependent and growth correlations were strong. When α and β were set to zero so that all variation in growth was a result of stochasticity, there was little evidence that matrix model performance declined (Fig. 3).

Rather than stochasticity or individual variability, per se, as a determinant of matrix model performance, our results suggest that the nature of the variation

among individuals is the key feature that dictates model choice. For example, although adding stochasticity in growth that is random and normally distributed among individuals changes matrix model performance little (Fig. 3), variability among individuals that is a result of size-dependent growth or growth autocorrelation can cause noticeable differences in model results (Figs. 1, 2, 4, 7, and 8). When we looked at the coefficient of variation (CV) in growth for individuals with these parameter combinations, we found that high stochasticity ($\varepsilon_g = 2.0$) with α and β set at zero had a CV of growth of 101.6, whereas when stochasticity was low ($\varepsilon_g = 0.5$) and positive size-dependent growth ($\alpha = 0.03$) and growth autocorrelations ($\beta = 0.8$) were present, CV was comparable at 94.7. Additionally, stochasticity that is not normally distributed may also affect model outcome differently. Thus, the variability among individuals alone is not a good indicator of the performance of matrix models; rather, the nature of the variation and whether it results in a spectrum of consistently good or bad performers appears to be the most important predictor. Our results point to the need to understand the source of variation among individuals, not just a metric of the amount of variability.

The inclusion of density dependence

The inclusion of linear negative density dependence showed results similar to those of the density-independent case (Figs. 7 and 8 compared with Figs. 1 and 2). Despite the added step of estimating the matrix transition probabilities as a dynamic function of density, matrix models were remarkably similar to IBMs over a variety of parameter combinations. As with the density-independent case, matrix models are poor predictors with positive α and large β values. Thus, negative density dependence alone does not change our conclusions about matrix model performance.

Where density dependence is likely to play a large role in model selection is in how density influences the dependence of growth on size (α) and on growth autocorrelation (β). Although experimental studies of density effects rarely report how patterns of individual growth are affected (but see Rubenstein 1981), some inferences can be made from published studies. For example, increased experimental density in plants, fishes, and amphibians can result in size distributions of individuals that become increasingly non-normal, often with some large individuals and many small and stunted individuals (Wilbur and Collins 1973, Harper 1977; but see Turner and Rabinowitz 1983) or great size inequalities (Weiner 1988, Maret and Collins 1994). The diverse mechanisms generating differences in individual growth and size are discussed in Lomnicki (1988, 1999). In the case of terrestrial plants for which size differences are the result of asymmetric competition mediated by shading (e.g., Weiner and Thomas 1986), some individuals are consistently fast growers, whereas others remain slow growers, a situation mimicked by

a high β value. Thus, our models indicate that density dependence may determine model choice only where it will significantly alter α or β or both via individual growth patterns. Unfortunately, density effects are usually reported only at the population level in empirical studies. To better determine model choice, we need to empirically assess the pattern of individual response to aspects of the environment (sensu Lomnicki 1999), including density, to understand whether α or β are context dependent.

IBMs have also been advocated when there is a need to include local interactions among individuals (Huston et al. 1988, Caswell and John 1992). Interestingly, asymmetric local interactions are usually associated with an unequal distribution of resources and the potential for some individuals to continually be resource deprived while others are resource rich (Post et al. 1997). In the case of plants, nearest neighbor analyses are often motivated by the fact that only nearby individuals affect focal plant performance (Pacala and Silander 1985, Weiner 1985, Bonan 1988). For example, shading can result in some continually stunted plants, while others are fast growing and dominate the canopy (Weiner 1985, Weiner and Thomas 1986), a phenomenon especially common at increasing density (Weiner 1985, Rice 1990) and possible even when competition is symmetric (Miller and Weiner 1989). Asymmetric interactions are also present in mobile vertebrates and can lead to differences among individuals that eventually result in cannibalism (e.g., DeAngelis et al. 1979, Maret and Collins 1994, Ziemba et al. 2000). In the case of cannibalism, the relative size differences, not the absolute differences, drive these important asymmetries (e.g., Maret and Collins 1994). Matrix models do not deal easily with asymmetric competitive interactions in which performance correlations may be important. Thus, strong local interactions may justify the use of an IBM (Huston et al. 1988, Caswell and John 1992), especially when those interactions are asymmetric and performance is positively size dependent.

CONCLUSIONS

In summary, we found that matrix models were robust to stochastic variation in growth rates of individuals, but they became inaccurate when individual variation was a result of performance correlations through time, especially when this autocorrelation was combined with positive size-dependent growth. Although variability among individuals is ubiquitous in natural populations, our results focus empirical analyses. Because of its importance to model choice, we need to know the strength of size-related processes in individuals and the extent to which individual performance is correlated temporally or spatially.

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