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Journal

Ecology, 83(7)

Authors

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Publication Date

2002

DOI

10.1890/0012-9658(2002)083[1928:DSATVR]2.0.CO;2

Peer reviewed

DEMOGRAPHIC STOCHASTICITY AND THE VARIANCE REDUCTION EFFECT

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Abstract. Demographic stochasticity is almost universally modeled as sampling variance in a homogeneous population, although it is defined as arising from random variation among individuals. This can lead to serious misestimation of the extinction risk in small populations. Here, we derive analytical expressions showing that the misestimation for each demographic parameter is exactly (in the case of survival) or approximately (in the case of fecundity) proportional to the among-individual variance in that parameter. We also show why this misestimation depends on systematic variation among individuals, rather than random variation. These results indicate that correctly assessing the importance of demographic stochasticity requires (1) an estimate of the variance in each demographic parameter; (2) information on the qualitative shape (convex or concave) of the mean–variance relationship; and (3) information on the mechanisms generating among-individual variation. An important consequence is that almost all population viability analyses (PVAs) overestimate the importance of demographic stochasticity and, therefore, the risk of extinction.

Key words: conservation biology; demography; extinction risk; homogeneous populations; population viability analysis; stochastic demography; variance reduction effect.

INTRODUCTION

The idea of demographic stochasticity, variation in vital rates due to chance variation in how individuals realize their propensities to survive and reproduce, has been used in population ecology for three (MacArthur and Wilson 1967, Levins 1969) or four decades (Leslie 1958), and in probability theory for well over a century (Watson and Galton 1874, Lotka 1931, Feller 1939, Kendall 1949), although early workers did not use the term ''demographic stochasticity.'' Nevertheless, population ecologists continue to grapple with how to define, estimate, and assess the importance of demographic stochasticity (Richter-Dyn and Goel 1972, Gilpin and Soulé 1986, Shaffer 1987, 1990, Menges 1992, Lande 1993, Engen et al. 1998, Kendall 1998). Demographic stochasticity plays an important role in both theoretical and applied demography (Pollard 1973, Gabriel and Bürger 1982, Nisbet and Gurney 1982, Tuljapurkar 1990, Tuljapurkar and Caswell 1997). Our understandings of extinction processes (Legendre et al. 1999) and of life history evolution (Fox 1993, Benton and Grant 1999) thus depend, to some degree, on how well we understand demographic stochasticity.

Kendall (1998) pointed out that demographic stochasticity is usually modeled incorrectly, as sampling error in a population that is stochastically uniform (i.e., every individual in a stage class has the same chance of surviving and reproducing), rather than having dif-

fering chances. This model simplification is not necessarily benign (contra Kendall 1998). *Individual heterogeneity* (variation in demographic traits that each individual retains through its lifetime) reduces the extinction risk of small populations of iteroparous organisms (Conner and White 1999). Engen et al. (1998) suggested that if the *demographic covariance* (a description of how an individual's fate depends on the fates of others in the population) is negative, it would effectively raise the magnitude of environmental stochasticity. However, Kendall and Fox (2002) showed that this non-independence actually lowers the magnitude of demographic stochasticity.

More generally, Kendall and Fox (2002) showed that any sort of structured variation among individuals means that modeling populations as homogeneous can cause overestimation of the importance of demographic stochasticity for survival. By structured variation, we mean any factor that imposes systematic demographic variation. Kendall and Fox (2002) list a number of examples, including variation in maternal provisioning among offspring, some cases of genetic variation, and differences in territory quality. In a nutshell, a population with individual variation in survival probabilities has reduced variation in the number of survivors (and hence reduced mean survival probability) when compared with a homogeneous population. This is a consequence of Jensen's inequality, which states that for a concave function of a variable argument, the mean of the function is less than the function of the mean (Jensen 1906; see Ruel and Ayres 1999).

Survival over a time interval is a binomial process

(see the Appendix for a discussion of human demography models using a Poisson approximation to the binomial): an individual either survives or it doesn't. The function relating the variance in the number surviving to the survival probability is a quadratic with negative second derivative. Thus, if survival probabilities vary within a population, then the variance in survival probability for the population as a whole must be less than the variance in a population homogeneous for the mean survival probability (Kendall and Fox 2002). We refer to this reduced variance in the demographic outcome as the *demographic variance reduction effect.* Kendall and Fox (2002) demonstrated that this qualitative effect is general and included some worked examples, but failed to find a formulation for the magnitude of this effect. We discuss some issues concerning the binomial model in the *Discussion*.

An immediate conclusion flowing from our result (Kendall and Fox 2002) is that population viability analyses (PVAs), which universally model survival by using the binomial sampling variance, have overestimated the importance of demographic stochasticity and, thus, the risk of extinction of small populations. This result may help to explain a recent empirical perception that PVAs tend to generate predictions that are overly bleak (Beissinger and Westphal 1998, Belovsky et al. 1999, Mann and Plummer 1999).

Brook et al. (2000) applied five PVA models to 21 long-term demographic data sets and concluded that the "PVA predictions were surprisingly accurate." However, examination of their "supplementary material" reveals that in fully 12 out of the 21 analyses, all of the PVA models had the same bias (either all negative or all positive) in their prediction of final population size; the bias was negative in eight of these cases. This is far more often than would be expected if the model errors were independent of one another, suggesting that many populations have important demographic structure that all of the existing PVA models overlook.

Here we present further results on the nature of the reduction in survival variance in heterogeneous populations. First, we show how to calculate the magnitude of the variance reduction, both for theoretical cases with continuous variation and for empirical cases with finite populations. Then we provide a simple model that helps to explain why it is that population structure, but not random assignment of survival probabilities, causes the variance reduction effect.

How Much Variance Reduction is There?

The key result is, in words: when there is variation among individuals in survival probability, then the variance in the number of survivors is proportional to the sampling variance in a population of "average individuals" minus the variance in individual survival probability. In the section on *Variance reduction and random assignment of survival probabilities,* we delimit exactly what we mean by "variation among individuals in survival probability."

Define the total variance in the number of survivors in a population of size N as $\text{Var}(S)$. Using p for individual survival probabilities, define among-individual variance in survival probabilities as $\text{Var}(p)$, with $E[p]$ as the expected value of p across populations. Define the sampling demographic variance (V_D) associated with a particular value of p as $V_D(p)$. Then the verbal result above can be written as

$$\text{Var}(S) = N\{V_D(E[p]) - \text{Var}(p)\}. \qquad (1)$$

Infinite populations

The derivation of this result is simple. The sampling variance for survival probability p is given by the binomial variance $p(1 - p)$. To get the variance for the number surviving in the whole population, simply multiply this binomial variance by the population size N and the frequency distribution of p, $f(p)$, and integrate over all possible values of p. Recalling a basic result from probability theory, that for a random variable p, $\text{Var}(p) = E[p^2] - E[p]^2$, this integration gives

$$\begin{aligned}
\text{Var}(S) &= N \int_0^1 p(1 - p)f(p)\,dp \\
&= N\left\{\int_0^1 pf(p)\,dp - \int_0^1 p^2f(p)\,dp\right\} \\
&= N\{E[p] - E[p^2]\} \\
&= N\{E[p] - [\text{Var}(p) + E[p]^2]\} \\
&= N\{E[p](1 - E[p]) - \text{Var}(p)\} \\
&= N\{V_D(E[p]) - \text{Var}(p)\} \qquad (2)
\end{aligned}$$

which is the result we asserted previously.

This result (Eq. 2) is entirely general. As an illustration, consider the case in which variation in p is described by the beta distribution, which is often used in survival models. Integrating Eq. 2 with the frequency of p described by the beta probability density function (pdf) (with parameters a and b) gives $N(a + b)\text{Var}(p)$. By $\text{Var}(p)$ here, we mean the variance under the beta distribution, $ab/(a + b)^2(1 + a + b)$. If p is fixed at its mean ($= a/(a + b)$ under the beta), the result is $N(a + b + 1)\text{Var}(p)$. Thus the variable population has a variance in survival that is smaller than that of the homogeneous population by exactly $N\text{Var}(p)$. In this example, as for any frequency distribution of p, the total variance in number surviving is proportionally reduced by the variance in the survival probabilities (Eq. 2).

Finite populations

Demographic stochasticity is important mainly in small populations. The results just presented are couched in terms of continuous variation in p, but in small populations, variation is likely to be discrete:

each individual i may have a unique survival probability p_i.

The results just presented extend to finite populations. In a population of N individuals, each with survival probability p_i, the total variance in number of survivors is given by

$$\text{Var}(S) = \sum_{i=1}^{N} p_i(1 - p_i) = \sum_{i=1}^{N} p_i - \sum_{i=1}^{N} p_i^2$$

$$= N\overline{p} - N\overline{p_i^2} = N\overline{p} - N[\text{Var}(p) + \overline{p}^2]$$

$$= N\{V_D(\overline{p}) - \text{Var}(p)\}. \qquad (3)$$

The result for finite populations (Eq. 3) is exactly analogous to that for continuous variation (Eq. 2): the total variance in number of survivors is proportionally reduced by the variance in the survival parameter p. This means that one can apply our results on variance reduction in empirical settings (in particular, in PVAs) when the population's survival rates are only poorly approximated by a continuous model.

EXTENDING THE RESULTS TO REPRODUCTION

Demographic stochasticity occurs in reproduction as well as survival: some individuals are more fecund than others. Unfortunately, there is no simple stochastic model like the binomial for variance in reproductive output. We observed (Kendall and Fox 2002) that reproduction is often modeled with the Poisson distribution, but without biological justification.

If one could justify the use of the Poisson model, the variance for number of offspring *would* equal the sampling variance in a population of "average individuals." Because the mean equals the variance under the Poisson, the variance as a function of the mean is neither concave nor convex, and Jensen's (1906) inequality would have no effect. On the other hand, if the variance is a convex function of the mean (that is, the opposite of survival), individual variation in number of offspring would actually *increase* the total variance in number of offspring.

There is no a priori basis for predicting the shape of the mean–variance relationship in fecundity, and we suspect that the shape of the mean–variance relationship may vary among taxa. Here we propose a way of estimating the direction and magnitude of the changes in variance induced by demographic stochasticity in fecundity.

This method uses a Taylor series approximation of the sampling variance of offspring number. Expanding the sampling variance for the per capita offspring number m around its expectation $E[m]$ gives

$$V_D(m) = V_D(E[m]) + (m - E[m])V_D'(E[m])$$

$$+ \frac{1}{2}(m - E[m])^2 V_D''(E[m]) + \cdots \qquad (4)$$

where the primes represent differentiation with respect

to m. As long as the third derivative of the mean–variance relationship is small, one can approximate the sampling variance for a given value of m using just the first three terms that we have spelled out. If the approximation is a good one, we can find the total variance for the population by integrating Eq. 4 over all possible values of m to get

$$\text{Var}(F) = N \int_0^{\infty} V_D(m) f(m) \, dm$$

$$\approx N\left\{ V_D(E[m]) \int_0^{\infty} f(m) \, dm \right.$$

$$+ V_D'(E[m]) \int_0^{\infty} (m - E[m]) f(m) \, dm$$

$$\left. + \frac{1}{2} V_D''(E[m]) \int_0^{\infty} (m - E[m])^2 f(m) \, dm \right\}$$

$$= N\left\{ V_D(E[m]) + V_D'(E[m])E[m - E[m]] \right.$$

$$\left. + \frac{1}{2} V_D''(E[m])E[m - E[m]]^2 \right\}$$

$$= N\left\{ V_D(E[m]) + \frac{1}{2} V_D''(E[m])\text{Var}(m) \right\} \qquad (5)$$

where $\text{Var}(m)$ is the among-individual variance of m and $\text{Var}(F)$ is the variance in the total number of offspring. Clearly Eq. 5 is analogous to the results for survival (Eqs. 2–3; for survival, $V_D'' = -2$ always), and can be restated in words: the total variance in reproduction is approximately given by the sampling variance in reproduction of a population of "average individuals" plus the variance in individual fecundity times half of the second derivative of the mean–variance relationship. Variance in individual fecundity can thus either reduce or inflate the total variance for the population, depending on whether the mean–variance relationship is concave or convex. Thus the second derivative term in Eq. 5 provides a first-order approximation for the magnitude (and direction) of the effect of individual variation on total variance in reproduction.

Inspection of Eq. 5 reveals that, had we kept higher order terms in the Taylor expansion, they would have been multiplied by the corresponding higher moments of m (skew, kurtosis, etc.). Thus if m is normally distributed, Eq. 5 is exact. Any skew in the per capita offspring number is likely to be positive (long positive tail), so if the variance–mean relationship were sigmoidal (negative third derivative), the variance in total offspring number would be reduced further. It is easy to imagine plausible distributions of m that have either positive (e.g., uniform) or negative (e.g., bimodal) kurtosis, so there is no way to generalize about this term. However, an empirical estimate of the fourth derivative

of the variance–mean relationship will typically be possible only if there is a known parametric form for the distribution of m.

VARIANCE REDUCTION AND RANDOM ASSIGNMENT OF SURVIVAL PROBABILITIES

Kendall and Fox (2002) observed that the reduction in demographic stochasticity due to individual variation requires that survival probabilities of individuals not be randomly assigned. More technically, the requirement is that the survival probabilities not be independently and identically distributed (i.i.d.). This seems puzzling at first. To gain intuition on this point, consider a simple model of a random seed rain onto sites that are "good" or "bad." The probability of landing on a good site is ϕ; in good sites survival probabilities are p_g, while in bad sites survival probabilities are p_b. Thus, the mean survival probability in the population is

$$\bar{p} = (1 - \phi)p_b + p_g\phi. \tag{6}$$

Following two seeds through their lives, the probabilities of two, one, and zero survivors are

$$P_2 = (1 - \phi)^2 p_b^2 + 2\phi(1 - \phi)p_b p_g + \phi^2 p_g^2$$

$$P_1 = (1 - \phi)^2(1 - p_b)p_b$$
$$\qquad + \phi(1 - \phi)[p_b(1 - p_g) + (1 - p_b)p_g]$$
$$\qquad + \phi^2(1 - p_g)p_g$$

$$P_0 = (1 - \phi)^2(1 - p_b)^2 + 2\phi(1 - \phi)(1 - p_b)(1 - p_g)$$
$$\qquad + \phi^2(1 - p_g)^2. \tag{7}$$

These are exactly the probabilities of survival obtained by using the mean survival probability. For example, squaring Eq. 6, the average probability of survival, gives the probability of two survivors. This result is identical to P_2. The probabilities of 1 and 0 survivors are also identical to those obtained if the population were composed of individuals with the mean survival probability. The variances in survival are thus also identical.

Why does this occur? In a basic sense, all individuals *do* have the same survival probability. Prior to dispersal, every seed has the same chance of falling in a good spot and the same chance of surviving if it does. Thus assignment of survival probabilities, when it is i.i.d., leads to exactly the same fraction of survivors as well as the same variance in survival.

More generally, random assignment of survival probabilities always leads to a different result than systematic structure because the variance is calculated differently in the two cases. When survival probabilities are i.i.d., the total variance in survival is calculated as the average squared deviation from $E[p]$. However, when survival probabilities vary in some systematic fashion, the total variance in survival is calculated as the sum of the squared deviations from each of the p_i's.

For the sake of brevity, we do not present an analogous model for reproduction, especially because, as we have suggested, there is no simple, general model. However, it is clear that the same logic holds as for survival: variance reduction (or inflation) requires that fecundity not be i.i.d. among individuals in a population.

DISCUSSION

Most empirical demographic studies have focused on average behavior, assuming a homogeneous population. This can lead one to seriously misestimate extinction risks. Our results show that total variance of survival is reduced by exactly the variance in survival probabilities. Our results for reproduction show that variance in offspring number depends (approximately) linearly on the variance in fecundity; the direction and magnitude of the change depends on the curvature of the mean–variance relationship.

These results underline the importance of acquiring data from real populations on variation among individuals for demographic parameters. Correctly assessing the effects of demographic stochasticity, crucial in theoretical demography (Pollard 1973, Nisbet and Gurney 1982, Tuljapurkar 1990, Tuljapurkar and Caswell 1997), life history evolution (Fox 1993, Benton and Grant 1999), and applied (e.g., pest management and conservation) settings, hinges on knowing both the means and variances of demographic parameters, as well as the shape of the mean–variance relationship for fecundity.

Estimating vital rates and their variances

Previously (Kendall and Fox 2002), we commented that accounting for variance reduction requires more intensive study but not longer data sets. The present results clarify this: for any given demographic parameter, what is needed is an estimate of the among-individual variance in the parameter, quantitative information as to whether the mean–variance relationship is concave or convex, and a determination as to whether the parameters are i.i.d. Although this is more work than estimating the mean for the parameter, it generally will not involve orders of magnitude more work, and in many cases, the required individual-level demographic data probably already exist in field notebooks.

An essential feature of the necessary data is that they be recorded as individual data rather than population aggregates. As an example of what not to do, consider a standard approach to estimating plant fecundity for matrix models: divide the number of seedlings by the number of adults, perhaps weighted by adult size. This does estimate the mean fecundity, but does not lead to an estimate of individual variance. To get the latter, we need data from individuals; admittedly, this is difficult in some taxa.

To estimate vital rates and their variances, one must

select an underlying model. This can be done either from a priori biological considerations (Lindsey 1995, Fox 2001) or by fitting data to a number of distributions and asking whether they differ in a measure of goodness-of-fit such as their likelihoods. Demographic data strongly violate normality: survival probabilities are constrained to be between 0 and 1, and individual fecundities are constrained to be positive integers. As we have described, survivorship can be modeled as a binomial distribution (also see the Appendix). Elucidating plausible models for demographic variation in fecundity and growth remains an important empirical and theoretical challenge.

To account for additional population structure, there are essentially two approaches, which are not mutually exclusive. The first involves hypotheses about particular factors that structure populations, whereas the second entails estimating the effect of underlying and unmeasured inherent differences in vital rates.

The first approach is probably more familiar to ecologists. One asks how the vital rates are affected by membership in a particular stratum such as gender, family within a population, or possession of a genetic marker. A familiar example is the use of logistic regression (Floyd 2001) or categorical modeling approaches to ask whether groups differ in their risk of death. The procedure directly provides estimates of mortality rates for the different strata in the population, as well as their variances. If the fraction of the population in each stratum can be estimated from these or other data, then one can directly infer the variance of the mortality rates for the population as a whole. Data on the dates of death also can be used to estimate mortality rates and their variance. There are several statistical models designed for estimating the effects of different strata on time-to-event data (Fox 2001). The model's parameter estimates can then be used to calculate the cumulative chance of dying in the interval of interest (for examples using SAS, see Allison 1995). Similar approaches can be used for fecundity data. For example, one can use categorical models to estimate the probabilities of individuals in different strata having a particular number of offspring. Depending on how the data are distributed, especially for fecundity data, there may be no standard regression models for examining the effect of strata on the vital rates. However, programmable statistical packages like S+, SAS, and GLIM can accommodate almost any underlying distribution.

The second approach is to use "frailty" or "heterogeneity" models (Vaupel et al. 1979, Keyfitz 1985, Lindsey 1993, Fox 2001). These assume that individuals vary in an unobserved quality that can be thought of as the chance that an individual dies, relative to a "standard" individual (with a frailty of 1). In a typical frailty model, the risk of mortality $\mu(t)$ at time t is multiplied for each individual i by a frailty z_i, so that individuals with $z_i = 2$ face twice the risk of a standard individual.

Clearly, an individual's frailty can be directly estimated if one has repeated measurements of the event (this is done in industrial reliability applications), but for better or worse, one only dies once. Perhaps surprisingly, one can estimate the variance in frailty from a sample even though no individual's frailty can be estimated. For example, Manton et al. (1981) estimated the variance in frailty within both genders of two human populations. Similarly, Service et al. (1998) estimated the variance in frailty for a *Drosophila* population. In both of these studies, models including frailty terms fit the data better than do models assuming that the population was homogeneous. Frailty models have been severely criticized (Mueller et al. 2000) as being ill-defined (because individual heterogeneity can occur through any parameter in any model) and as making it possible to always propose post hoc frailty models (which by definition model unmeasured variables) to fit data. Analogous criticisms, of course, can be applied to many hypotheses.

How does one estimate the shape of a mean–variance curve for fecundity? A completely empirical approach is to plot observed among-individual variances against a series of observed population means and ask whether the curve is convex or concave. Desirable as such analyses are, it is clear that we will not often be able to conduct them. An alternative relies on parametric statistical models. Given data on individual fecundities, one will already have used a model to estimate the variance of fecundities. For example, the data might be best fit by a generalized Poisson distribution (B. Kendall and K. Keith, *unpublished manuscript*). If there are no further data from other populations, one could proceed by assuming that the same distribution (with different parameters) is likely to give a reasonable description of variation in fecundity for a range of conditions. Analysis of the distribution and estimated parameters will usually provide the needed insight: for example, in the Poisson distribution, the mean–variance function is a straight line, whereas in the generalized Poisson distribution, the variance can be either a concave or a convex function of the mean.

How can we determine that variation among individuals in survival probability or fecundity is not i.i.d.? Rarely will there be sufficient data to test this statistically. Instead, we must examine the natural history of the organism to determine that an individual's membership in a particular demographic stratum is not completely random, or that an individual's fate is not entirely independent of its neighbor's. Kendall and Fox (2002) gave a number of biologically reasonable examples in which survival probabilities of individuals are not i.i.d.

Variance reduction or model misspecification?

We have shown that the variance reduction effect is caused by systematic population structure. Does this imply that models, such as those used in many PVAs, are simply misspecified? Should all that structure simply be included in the model?

Consider the case of genetic variation. At best, it is difficult to model quantities like genetic variance for survival or fecundity; there is no organism for which the genetic architecture of this kind of variation is well understood. The errors are large for parameter estimates in quantitative genetics (Shaw 1991), and in the case of endangered species, can easily require studies larger than the number of individuals in a population. Without knowing the detailed genetic architecture of such traits, as well as the details of the mating system, it is impossible to predict how parameters like heritabilities and genetic covariances will change over time (Turelli 1988), even if there were no selection on the traits. Indeed, our approach of finding second-order terms to simulate the effects of the unmodeled structure is analogous to the use of quantitative genetics in modeling traits that are affected by many (potentially interacting) loci.

Similar arguments can be made regarding many of the causes of population structure. The sad truth is that there are a large number of factors that structure populations, and they may have interactive effects on one another. Even with very large populations and long careers, we are unlikely to be able to specify models incorporating more than a very few of these factors. The remainder must be treated as random variation.

The future may be better (or worse) than we thought

A biased method can be useful if the direction of bias is known. In particular, our results can be used to place bounds on the importance of demographic stochasticity. For example, a simple PVA of a homogeneous population always gives an overestimate of the variance of the number surviving due to demographic stochasticity, and therefore overestimates the extinction probability when the population is small. By the same token, if it is known whether the mean–variance relationship for fecundity is concave or convex, one can say that a model of a homogeneous population gives an overestimate (if concave) or underestimate (if convex) of the variance in fecundity. Even if models are able to describe some of the population structure, they will still tend to err in the same direction, because it is not possible to fully account for all of the factors causing population structure.

This sort of logic may suggest an optimistic view of our results: we know how models are biased, and extinction risks are often lower than had been thought. We believe, however, that such optimism must be strongly tempered. First, an upper bound for the importance of demographic stochasticity is not a particularly useful quantity if it is greatly overestimated. Second, variation among individuals in survival probability and fecundity may act in opposite directions; without additional information on the sizes of the individual effects (and their covariances) the net direction of bias for a given PVA is unknown. Third, and perhaps most important, this uncertainty is made worse by the way

most PVA applications implement environmental and demographic stochasticity. They estimate environmental stochasticity by the observed variation (which incorporates demographic stochasticity) and then add on "demographic stochasticity" as sampling error (Kendall 1998). This procedure can cause substantial errors in estimating quantities like variance in population size, minimum viable population size, and extinction risk (see also Ludwig 1998, Brook 2000, Fieberg and Ellner 2000) in unknown directions.

Thus, it is not clear whether to be optimistic or pessimistic about extinction risks and the net direction of bias: they may be better or worse than PVAs have suggested. What is clear, however, is what we need to know to answer that question. We need estimates of the among-individual variances in vital rates. In the case of fecundity, we also need to know the shape of the mean–variance curve. For both survival and fecundity, we need to estimate the effects of factors suspected to make populations heterogeneous.

In the last decade or so, there has been considerable progress in understanding and modeling the effects of population structure caused by differences in size, developmental stage, and age (Tuljapurkar and Caswell 1997, Caswell 2001). We now need to add estimates of factors that cause heterogeneity in vital rates with stage, size, and age classes. In some cases, this can be done by analyzing the effects of observable factors (like family, genetic markers, birth order or position, etc.) using statistical methods like those just discussed (see *Estimating vital rates and their variances*). However, there is also a multitude of factors that we are unable to observe, much less model, that can also contribute to heterogeneity among individuals. Heterogeneous populations have properties that are very different from homogeneous populations; although the heterogeneity can be challenging to quantify, doing so will improve the predictive accuracy of population models.

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APPENDIX

A description of binomial and other survival models is available in ESA's Electronic Data Archive: *Ecological Archives* E083-033-A1.