Variation among Individuals and Reduced Demographic Stochasticity

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Abstract: *Population viability analysis (PVA) is a technique that employs stochastic demographic models to predict extinction risk. All else being equal, higher variance in a demographic rate leads to a greater extinction risk.* Demographic stochasticity *represents variance due to differences among individuals. Current implementations of PVAs, however, assume that the expected fates of all individuals are identical. For example, demographic stochasticity in survival is modeled as a random draw from a binomial distribution. We developed a simple conceptual model showing that if there is variation among individuals in expected survival, then existing PVA models overestimate the variance due to demographic stochasticity in survival. This is a consequence of Jensen's inequality and the fact that the binomial demographic variance is a concave function of mean survival. The effect of variation among individuals on demographic stochasticity in fecundity depends on the mean-variance relationship for individual reproductive success, which is not presently known. If fecundity patterns mirror those of survival, then variation among individuals will reduce the extinction risk of small populations.*

Variación entre Individuos y Estocasticidad Demográfica Reducida

Resumen: *El análisis de viabilidad poblacional (AVP) es un técnica que emplea modelos demográficos estocásticos para predecir el riesgo de extinción. Todo lo demás siendo igual, mayor variación en la tendencia demográfica conduce a un mayor riesgo de extinción. La* estocasticidad demográfica *representa variación debido a diferencias entre individuos. Sin embargo, los AVP actualmente asumen que el destino esperado para cada individuo es idéntico. Por ejemplo, la estocasticidad demográfica en la supervivencia es modelada como una muestra aleatoria de una distribución binomial. Desarrollamos un modelo conceptual simple que muestra que si hay variación entre individuos en la supervivencia esperada, entonces los modelos de AVP existentes sobrestiman la variación debida a la estocasticidad demográfica en la supervivencia. Esto es una consecuencia de la desigualdad de Jensen y del hecho de que la variación demográfica binomial es una función cóncava de la supervivencia promedio. El efecto de la variación entre individuos sobre la estocasticidad demográfica en la fecundidad depende de la relación media-varianza del éxito reproductivo individual, que actualmente es desconocida. Si los patrones de fecundidad son un reflejo de los de supervivencia, entonces la variación entre individuos reducirá el riesgo de extinción de poblaciones pequeñas.*

Introduction

Although population viability analysis (PVA) has attained a central role in conservation biology, serious questions remain about its effectiveness (Beissinger & Westphal 1998; Mann & Plummer 1999). In particular, scientists do not have much confidence in the detailed predictions of PVA models (Akçakaya & Raphael 1998). These apparent failures have been attributed largely to shortcomings in the data (e.g., Beissinger & Westphal 1998), but they may also reflect inadequacies in the way we model endangered species. We examined the process of demographic stochasticity, revealing that the way it is usually

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modeled may not match biological reality; hence, PVAs may often predict too much stochastic variation in population growth rate.

By demographic stochasticity, biologists mean fluctuations in population size and vital rates due to random variation among individuals (May 1973; Leigh 1981; Shaffer 1981, 1987; Lande 1993). Nevertheless, PVAs always model demographic stochasticity as though it were sampling variance: all individuals have identical expected fates, and the only variation is due to "sampling error" about this expected value arising from the small number of individuals observed (Levins 1969). The motivation for this assumption seems to be simplicity. Indeed, one of us (Kendall 1998) has argued that, lacking any information to the contrary, the most "parsimonious" assumption is that all individuals have identical survival probabilities, which leads to the sampling-error model.

At any given time in the real world, there can be substantial variation among individuals in traits that affect their expected fate. Genetic variation and spatial environmental variation (acting both on development and on current resources and conditions) lead to phenotypic variation among individuals. This, in turn, means that these individuals may vary in their intrinsic fecundity and vigor. Furthermore, different individuals face different suites of resources and environmental conditions, leading to additional variation in expected fecundity and survival.

Engen et al. (1998) were the first to address the theoretical problem of variation in demographic performance within a population, introducing demographic covariance as a measure of the nonindependence of individual demographic fates. Their model, however, did not allow for variation among individuals in expected demographic fate. They suggested that intraspecific competition should lead to a negative demographic covariance but did not elaborate on the mechanism by which this would occur. In their model, a negative demographic covariance led to an increase in the apparent magnitude of demographic stochasticity and a decrease in the apparent magnitude of environmental stochasticity. This suggests that demographic covariance should reduce the extinction risk of a large population facing high environmental variability, but may actually increase the extinction risk of a small population.

Engen et al. (1998) modeled covariance among individuals at a given time. In contrast, Conner and White (1999) examined covariance within individuals across time. They began with the observation that, in many wildlife species, an individual's unusual demographic trait (e.g., higher than average vigor) derives from an inherent "fitness" retained through the individual's lifetime. They used the term *individual heterogeneity* to describe this pattern of variation among individuals retained through the individual lifetimes. Using simulations, they found that increasing individual heterogeneity increased the probability of persistence in small populations with or without environmental stochasticity. The effect was strongest in the populations with the smallest initial abundance. They suggested that in a heterogeneous population there are likely to be a few exceptionally "fit" individuals, with high survival probabilities, that are long-lived. These individuals prevent the population from reaching zero as long as they live. If these traits are heritable or persist due to spatial features in the environment, then persistence may be ensured for a long time. These results led the author of a recent review to suggest that "individual heterogeneity must be incorporated into a PVA if you don't want to underestimate viability."

A similar problem has been studied in human and insect demography. If individual organisms vary in their inherent tendency to die—referred to as "frailty"—then by following a cohort one might falsely conclude that mortality rates are decreasing over time. In reality, the most "frail" individuals tend to die earliest, so the observed mortality rate does decline—which does not imply that the risk of mortality is really decreasing for any individual (Vaupel et al. 1979, 1998; Keyfitz 1985; Lindsey 1993; Service et al. 1998).

How might these approaches be applied empirically? Conner and White (1999) are clear about how to estimate individual heterogeneity in a population: collect data on the lifetime individual performances of individuals, such as studies on lifetime reproductive success (e.g., Clutton-Brock 1988). Heterogeneity in survival is more difficult to estimate because an individual only dies once. Conner and White (1999) and White (2000) suggest looking for correlates of fitness, such as body size. On the other hand, it is not clear how demographic covariance should be estimated. Subsequent applications of the Engen et al. (1998) model have set this term to zero (e.g., Sæther et al. 1998, 2000; Diserud & Engen 2000).

We developed a conceptual framework that encompasses both nonindependence (following Engen et al. 1998) and variation among individuals (following Conner & White 1999). We show that nonrandom variation in survival among individuals always leads to a reduction in variance in survivorship due to demographic stochasticity. We also examined the biological processes that might lead to this "demographic determinism." Finally, we discuss how to determine whether variation among individuals in fecundity will increase or decrease the demographic variance.

Variation among Individuals in Survival

We developed a simple model of variation in survival among individuals to illustrate the potential errors asso-

ciated with ignoring this variation. Before presenting the full mathematical formalism, we introduce a simple heuristic example. Consider two identical individuals, each with a survival probability of 0.5 (hence, the population has a mean survival probability of 0.5). The number of survivors will be zero (extinction) with probability 0.25, one with probability 0.5, and two with probability 0.25. The variance in the number surviving is 0.5. Now consider a population with variability between individuals: one individual has a survival probability of 0 and the other has a survival probability of 1. The mean probability of survival is still 0.5, but the number of survivors is one with probability 1. The variance in the number surviving is zero (as is the extinction probability). This scenario is biologically implausible, of course, but more modest levels of variation among individuals also lead to reductions in demographic variance (Fig. 1; see Appendix for another example). This model can be generalized to show that increasing variability in demographic traits among individuals always decreases variability in the number surviving (Appendix).

This result is a consequence of a mathematical relationship known as Jensen's inequality: when a function has a negative second derivative (concave down) and is applied to a variable argument, then the expectation of the function is less than the function of the expectation (e.g., Ruel & Ayres 1999). "Expectation" and "expected value" are to probability theory what "population mean" is to statistics: the true, underlying theoretical average (to be contrasted with the "sample mean," which is the average of a finite collection of objects). The expectation of a quantity x is written $\mathrm{E}[x]$, so the mathematical representation of Jensen's inequality is

$$\mathrm{E}[f(x)] < f(\mathrm{E}[x]) \text{ if } \frac{d^2 f}{dx^2} < 0.$$

Only if the second derivative is zero does the inequality become an equality. In the present context, f is the function relating the variance in an individual's fate (V_I) to its survival probability, p. This function is $V_I(p) = p(1 - p)$. The second derivative of this function is

$$\frac{d^2 V_I}{dp^2} = -2 \text{ for all } p.$$

Suppose there are N individuals in the population, each with a survival probability of p_i. The expected number of survivors (S) is $\mathrm{E}[S] = N\mathrm{E}[p]$. The variance in the number surviving is

$$\mathrm{var}(S) = \mathrm{E}\left[\sum_{i=1}^{N} V_I(p_i)\right] = N\mathrm{E}[V_I(p)].$$

If all individuals are identical, each having the expected survival probability ($p_i = \mathrm{E}[p]$), then the variance in the number surviving is the binomial variance, $\mathrm{var}(S) = N\mathrm{E}[p](1 - \mathrm{E}[p])$. In contrast, if the individuals differ in their survival probabilities (i.e., the p_i are not all equal), then Jensen's inequality applies:

$$\mathrm{var}(S) = N\mathrm{E}[V_I(p)] < NV_I(\mathrm{E}[p]) = N\mathrm{E}[p](1 - \mathrm{E}[p]).$$

These results are concordant with those of Conner and White (1999). Indeed, their "individual heterogeneity" is an important source of variation among individuals. Our results seem to contradict those of Engen et al. (1998), however, who found that negative demographic covariance reduces environmental stochasticity and increased demographic stochasticity. As we show in the appendix, their "demographic covariance" is proportional to $1/(N - 1)$. When we carried this effect of population size through the variance calculation, the apparent effect on environmental stochasticity disappeared, and the effect on demographic stochasticity changed sign, matching our result that individual variation reduces demographic stochasticity (see appendix for details).

The preceding analysis depends on individual survival probabilities being "predetermined." If individuals have different survival probabilities, but each individual draws its survival probability at random from a distribution and the random draws are independent and identically distributed among the individuals, then the variance-reduction effect is eliminated. Consider the two individuals again. Now suppose that a female has a survival probability of one and a male has a survival proba-

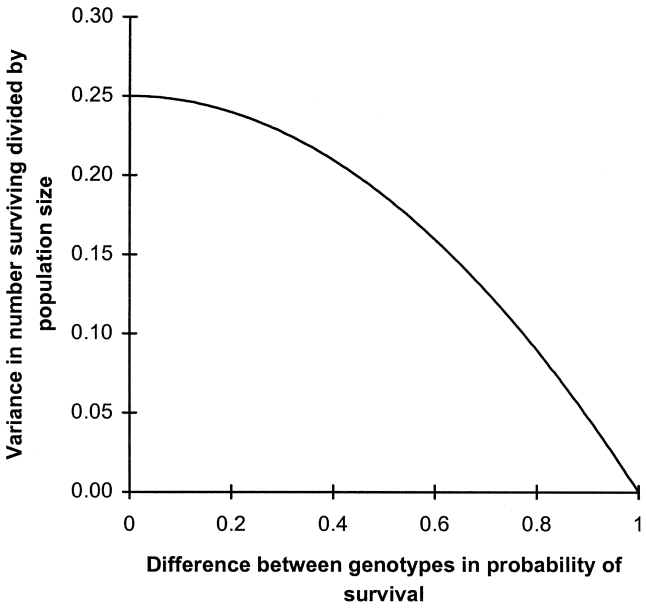

Figure 1. Variance in number surviving in a population made up of equal parts of two genotypes with differing survival probabilities as a function of the difference in survival probability. The population average survival is always 0.5. Because the variance is also proportional to population size, it has been rescaled, dividing by population size.

bility of zero. If the sex ratio is 50:50 on average, but each zygote has a 50% chance of being male, then both individuals are male with probability 0.25 (zero survivors), one is male and one is female with probability 0.5 (one survivor), and both individuals are female with probability 0.25 (two survivors). Thus, the outcome that results from random assignment of survival probability exactly matches the outcome that results when both individuals have the same survival probability. A more detailed example is given in the appendix. This sort of thought experiment has probably contributed to the lack of recognition of the importance of variation among individuals (for example, some years ago B. E. K. used the above calculation to satisfy himself of the adequacy of the sampling model of Kendall 1998).

Causes of Variation among Individuals

A reduction of demographic variance occurs when the distribution of survival probabilities among individuals is not "independent and identically distributed" (iid). In other words, there needs to be some systematic structure in the population (i.e., individual survival probabilities are not identical; e.g., exactly half of the individuals are male). Alternatively, an individual's demographic properties could depend on the properties of other individuals in the population (i.e., individual survival probabilities are not independent of one another; e.g., the probability that one individual gets a favorable territory depends on who else is in the population). Common biological mechanisms that will cause a reduction of demographic variance are contest competition (including territoriality), long-lived individuals with lifetime demographic traits ("individual heterogeneity"; sensu Conner & White 1999), maternally imposed variation, and certain genetic traits. Age and stage structure also introduce variability among individuals, but this form of structure is usually recognized and modeled explicitly. The other forms of structure could also be modeled explicitly if they could be recognized and quantified.

The demographic fates of individuals are often not independent of one another. Intraspecific interactions, such as competition, can sometimes assure that this is the case. Scramble competition results in all individuals having reduced fitness, but contest competition produces losers and winners with differing demographic traits. The proportion of losers depends on the size of the population. This mechanism was recognized by Engen et al. (1998) as leading to negative demographic covariance. In territorial species, for example, the number of territories is often fairly constant, and there are usually more reproductively mature individuals than territories. Those individuals that fail to obtain territories must subsist in suboptimal habitats or become helpers in social species. These floaters have no reproductive success (extra-pair copulations by floater males should have little effect on the fecundity of the territorial females), and they often have a lower survival probability (e.g., Sandercock et al. 2000). Thus, in any given year with N individuals there will be T territorial individuals with "good" demographic traits and $N - T$ floaters with "poor" traits. The number of floaters depends on the number of territories and the total number of individuals. Although this source of structure could be modeled explicitly (as could any of the examples we discuss here), it is less commonly recognized than age- or stage-structured populations.

If individuals are long-lived and have lasting traits that affect their survival or fecundity, demographic structure is likely to persist from year to year. In essence, the demographic structure of the current year will be at least partly determined by the demographic structure of the previous year. Conner and White (1999) called this phenomenon "individual heterogeneity" and both provided a review of relevant empirical work and developed a model. Their interpretation of the phenomenon focuses on survival and is analogous to frailty (Vaupel et al. 1979), but a similar effect also could arise from individual heterogeneity in fecundity. It may be valuable to also consider an individual's "extended phenotype," such as the territory it inhabits. Territory quality, which is predictable from year to year, will affect the survival and fecundity of the resident individuals (e.g., Koenig & Mumme 1987). Even if a territory is not held by the same individual from year to year, its demographic qualities may persist.

In many species, mothers allocate resources unequally among their offspring. Birds often feed large chicks more than small ones, which reinforces the size differences and may lead to differential survival rates after fledging. Legume seeds that are proximal to the plant have different characteristics than do those more distal (Silvertown 1984). Seeds from disk florets in Asteraceae often have quite different demographic characteristics than those from ray florets (Venable & Búrquez M. 1990). In all of these cases, each sib group varies systematically within itself, which will lead to a reduction in demographic stochasticity.

Some types of genetic variation can have similar consequences for variation in demographic performance. Simple additive polymorphisms that lead to either high or low survival will not persist in the population (unless differences in reproductive success compensate). Overdominance or linkage can make such polymorphisms more likely. More important, such polymorphisms are more likely if genotypes vary in their response to the environment, so that there are "year-type specialists" or "patch-type specialists," and if mating tends to occur among specialists (i.e., positive assortative mating). Classical analyses of models in which individuals in different types of environments mate randomly show that polymorphisms are difficult to maintain without overdominance (Haldane & Jayakar 1963; Hoekstra et al. 1985).

But polymorphism is easy to maintain in more realistic versions of these models. For example, polymorphism can be maintained when individuals mate preferentially with others in the same patch (Wilson & Turelli 1986) or with others who respond genetically to the same germination cue (G. A. F. & A. S. Evans, unpublished data). In cases like this, genetically driven demographic structure may persist. It has not been possible to find general conditions assuring such persistence because these vary with underlying genetics, mating system, and migration patterns. It seems clear, however, that limited dispersal and nonrandom mating pools—the case for many taxa—can make such structure more likely. The point is that this kind of genetic/demographic structure is biologically reasonable, and it leads to a reduction in demographic stochasticity because, in a given year, a predictable fraction of the population will have each genotype.

Implications for Population Viability Analysis

Given this potential source of error in current PVA models, what can be done to improve the models? Many sources of variation among individuals that lead to a reduction in demographic stochasticity may be more or less cryptic forms of population structure. Ideally, this structure needs to be quantified and incorporated into models.

Identifying and Quantifying Sources of Variation among Individuals

To account for the negative covariance in territorial organisms requires that territory holders and floaters be modeled separately. Generally this doubles the amount of demographic data required. The recognition and quantification of variation in expected fate among individuals can be more difficult. For example, quantification of individual heterogeneity requires lifelong observations of known individuals (Conner & White 1999). To fully understand maternally induced variation, individuals need to be identified to sib group, so that within-group variation can be compared with among-group variation. It may be possible to identify certain taxa (such as birds) that may be particularly prone to this phenomenon. For such species, the PVA could explore the consequences of different levels of maternally induced variation.

Genetic variation is the most difficult to identify of the structuring mechanisms we have discussed. Quantitative genetic studies of traits of demographic importance can help quantify this source of variability within populations. In contrast to maternally induced variation, genetic structure should lead to low variability within sib groups.

All of these population structures require additional demographic data, either in the form of parameter estimates for each group or in the form of among-individual variances. The good news is that, although empirically quantifying these various sources of population structure requires more intensive study, it does not generally require more years of study; the one exception to this is individual heterogeneity in long-lived organisms. Because effort can be bought and time cannot, this means that incorporating information about variation among individuals into a PVA may be feasible, at least where there is opportunity for further data collection.

If a species is rare or difficult to observe, however, it may never be possible to obtain a sample size large enough to detect or adequately quantify variation in demographic traits among individuals. Under such circumstances, it may be advisable to perform a sensitivity analysis: how sensitive are the projections of the PVA to a plausible magnitude of individual variability? If the sensitivity is high, then it will be important to present that uncertainty and redouble efforts to quantify the individual variation. Identifying "plausible magnitude" requires studies of similar but more abundant species. Many existing intensive demographic studies contain information that would allow an investigation of individual variation; we urge a reanalysis of these data with this goal in mind.

Variation in Fecundity among Individuals

So far, our discussion has been about survival. What are the effects of variation among individuals in fecundity? The answer depends on how one describes "sampling variation" in fecundity. Many PVA models assume that fecundity is Poisson distributed (e.g., Akçakaya 1999). The variance of the Poisson distribution is proportional to the mean, so the mean-variance relationship is neither concave up nor concave down. This condition transforms Jensen's inequality into an equality, so under the Poisson assumption, variation among individuals has no effect on demographic variance.

We know, however, of no biological justification for using the Poisson distribution. We speculate that many populations are likely to depart substantially from the Poisson. For example, a population of competing plants is likely to contain many individuals with low fecundity and few with high fecundity. Likewise, distributions of vertebrate fecundity are often bimodal, with one peak at zero—reproductive failure—and the other at a higher value (e.g., Hoogland 1995). Many studies have collected data on variation in fecundity, especially for vertebrate taxa, but the frequency distributions are published only rarely. It would be valuable to reanalyze these data to look for empirically supported models of fecundity variation in various taxonomic groups.

We suspect that the biologically appropriate model for demographic stochasticity in fecundity will differ from species to species, and there has been no rigorous treatment of this problem. Without further data, we can

make only a general statement: if the variance is a decelerating function of the mean, then variation among individuals will reduce demographic stochasticity in fecundity. The flip side of Jensen's inequality is that if the variance is an accelerating function of the mean, then variation among individuals will increase the demographic variance. Thus, it is critical to develop at least a qualitative understanding of the variance-mean relationship in different taxonomic groups. For example, Conner and White (1999) modeled reproduction as a binomial process: each individual could have at most one offspring per year. Thus, we expect individual heterogeneity in fecundity to reduce demographic variance. But if the fecundity process were such that the second derivative of the variance-mean relationship were positive, then individual heterogeneity in fecundity would increase demographic variance, counteracting the effects of heterogeneity in survival.

Variation among Individuals and Extinction Risk

How does variation among individuals affect extinction risk? Variance in the per capita growth rate due to demographic stochasticity increases the extinction risk of a population (Lewontin & Cohen 1969), and the effect is most important in small populations (MacArthur & Wilson 1967). If variation among individuals substantially reduces this variance, then the extinction risk faced by small populations will be lower than projected by current PVA models. This is similar to the effects of "double dipping" (Brook 2000), in which negative effects such as catastrophes and demographic stochasticity contribute to the base parameter estimates but are also put into the models as additional factors. If the mean-variance relationship for fecundity is concave down (as in the mean-variance relationship for survival), ignoring variation among individuals is guaranteed to produce overly dire estimates of extinction risk. At its extreme, variation among individuals can completely eliminate demographic stochasticity, removing the extra risk faced by small populations. This is unlikely, however, and we do not know how the magnitude of this effect is likely to compare with other common errors in PVAs (Brook 2000). If the mean-variance relationship for fecundity is concave up, the errors may to some degree cancel one another out. Thus, the net effect of variation among individuals on overall extinction risk remains an important unknown, requiring investigation of the magnitude of variation among individuals, the nature of the mean-variance relationship for fecundity, and analysis of the resulting models.

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Appendix

Model Examples, Detailed Calculations, and Relation to Demographic Covariance

EFFECT OF VARIATION AMONG INDIVIDUALS

To demonstrate that our results are not peculiar to the simple example in the text, we provide a more realistic example and work through the calculations in detail. Consider a population of two individuals with survival probabilities p_1 and p_2 and expected survival rate ($\mathrm{E}[p] = (p_1 + p_2)/2$) equal to 0.3. If there is no variation among the individuals ($p_1 = p_2 = \mathrm{E}[p]$), then the probability distribution of the number surviving, S, is given by

$$\begin{aligned}\Pr[S=0] &= (1-p_1)(1-p_2) = 0.7\times 0.7 = 0.49\\ \Pr[S=1] &= (1-p_1)p_2 + p_1(1-p_2) = 0.7\times 0.3 + 0.3\times 0.7 = 0.42\\ \Pr[S=2] &= p_1p_2 = 0.3\times 0.3 = 0.09.\end{aligned} \tag{1}$$

The expected value of S is $\mathrm{E}[S] = 2\,\mathrm{E}[p]$, so the variance of S is

$$\begin{aligned}\mathrm{var}(S) &= \Pr[S=0](0-\mathrm{E}[S])^2 + \Pr[S=1](1-\mathrm{E}[S])^2 \\ &\quad + \Pr[S=2](2-\mathrm{E}[S])^2\\ &= 0.49\times 0.6^2 + 0.42\times 0.4^2 + 0.09\times 1.4^2\\ &= 0.1764 + 0.0672 + 0.1764\\ &= 0.42,\end{aligned} \tag{2}$$

which is $2\,\mathrm{E}[p](1 - \mathrm{E}[p])$, as expected from binomial sampling theory.

Now suppose there is nonrandom variation among the individuals, so that $p_1 = 0.1$ and $p_2 = 0.5$, retaining $\mathrm{E}[p] = 0.3$. This gives a probability distribution of S given by

$$\begin{aligned}\Pr[S=0] &= (1-p_1)(1-p_2) = 0.9\times 0.5 = 0.45\\ \Pr[S=1] &= (1-p_1)p_2 + p_1(1-p_2) = 0.9\times 0.5 + 0.1\times 0.5 = 0.5\\ \Pr[S=2] &= p_1p_2 = 0.1\times 0.5 = 0.05.\end{aligned} \tag{3}$$

The resulting variance of S is

$$\begin{aligned}\mathrm{var}(S) &= \Pr[S=0](0-\mathrm{E}[S])^2 + \Pr[S=1](1-\mathrm{E}[S])^2 \\ &\quad + \Pr[S=2](2-\mathrm{E}[S])^2\\ &= 0.45\times 0.6^2 + 0.5\times 0.4^2 + 0.05\times 1.4^2\\ &= 0.162 + 0.08 + 0.098\\ &= 0.34,\end{aligned} \tag{4}$$

which is substantially less than $2\,\mathrm{E}[p](1 - \mathrm{E}[p])$. More generally, the variance of S is the sum of the binomial sampling variances of each individual, so for any p_1 and p_2

$$\begin{aligned}\mathrm{var}(S) &= p_1(1-p_1) + p_2(1-p_2)\\ &= p_1 + p_2 - (p_1^2 + p_2^2)\\ &= 2\mathrm{E}[p] - (p_1^2 + 2p_1p_2 + p_2^2)/2 - (p_1^2 - 2p_1p_2 + p_2^2)/2\\ &= 2\mathrm{E}[p] - 2\mathrm{E}[p]^2 - (p_1 - p_2)^2/2\\ &= 2\mathrm{E}[p](1-\mathrm{E}[p]) - (p_1 - p_2)^2/2.\end{aligned} \tag{5}$$

Thus, reduction in the survivorship variance is half the squared difference between the individuals.

EFFECT OF RANDOM ASSIGNMENT

Now suppose each individual has either survival probability p_1 or survival probability p_2, but that each individual's survival probability is assigned at random, with the two possibilities being equally likely. In our population of two individuals, there are three possible demographic structures in a given year. With probability 0.25, both individuals have survival probability p_1. With probability 0.25, both individuals have survival probability p_2. With probability 0.5, one individual has survival probability p_1 and the other p_2. The distribution of S is then given by

$$\begin{aligned}\Pr[S=0] &= 0.25(1-p_1)^2 + 0.25(1-p_2)^2 + 0.5(1-p_1)(1-p_2)\\ &= 0.25[(1-p_1) + (1-p_2)]^2\\ &= (1-\mathrm{E}[p])^2\end{aligned}$$

$$\begin{aligned}\Pr[S=1] &= 0.25[(1-p_1)p_1 + p_1(1-p_1)]\\ &\quad + 0.25[(1-p_2)p_2 + p_2(1-p_2)]\\ &\quad + 0.5[(1-p_1)p_2 + p_1(1-p_2)]\\ &= \mathrm{E}[p] - 0.5(p_1^2 + p_2^2) + \mathrm{E}[p] - p_1p_2\\ &= 2\mathrm{E}[p](1-\mathrm{E}[p])\end{aligned}$$

$$\begin{aligned}\Pr[S=2] &= 0.25p_1^2 + 0.25p_2^2 + 0.5p_1p_2\\ &= 0.25(p_1 + p_2)^2\\ &= \mathrm{E}[p]^2,\end{aligned} \tag{6}$$

which is exactly as expected from a monomorphic population with survival probability $\mathrm{E}[p]$.

THE DEMOGRAPHIC COVARIANCE

What is the demographic covariance, and how does it relate to the effect of variation among individuals described in the paper? Engen et al. (1998) define it as $\tau = \mathrm{cov}(d_i, d_j)$, $i \neq j$, where d_i is the difference between individual i's actual fate and its expected fate (in the formalism of the present paper, d_i is either $\mathrm{E}[p]$ if the individual dies or $1 - \mathrm{E}[p]$ if the individual survives). This definition is unclear, but we deduced

from usage elsewhere in the paper that Engen et al. (1998) meant that

$$\tau = \mathrm{E}\left[\frac{1}{N(N-1)}\sum_{i=1}^{N}\sum_{\substack{j=1\\ j\neq i}}^{N} d_i d_j\right], \tag{7}$$

where N is the population size and

$$\sum_{\substack{j=1\\ j\neq i}}^{N}$$

means "sum over all j from 1 to N except when j equals i." The demographic covariance is zero when individual fates are independent, and the sorts of demographic structure we describe here result in $\tau < 0$. Because we are concerned only with demographic stochasticity, we set the environmental variable e of Engen et al. (1998) equal to zero. From equation 3 of Engen et al. (1998), the variance in the number surviving is

$$\mathrm{var}(S) = N(\sigma_d^2 - \tau) + N^2\tau, \tag{8}$$

where $N\sigma_d^2$ is the variance in a monomorphic population

$$(\sigma_d^2 = \mathrm{var}(d_i) = \mathrm{E}[p](1 - \mathrm{E}[p])).$$

The term in N^2 is characteristic of environmental stochasticity (the per capita variance is independent of population size), and the term in N is characteristic of demographic stochasticity (the per capita variance is inversely proportional to population size). Because τ is negative for the sorts of demographic structure we describe here, "environmental stochasticity" appears to become negative, and the effect on demographic stochasticity appears to go in the opposite direction from what we suggest is the case.

Let us return to the extreme example from the text, where half the individuals are guaranteed to die and half are guaranteed to live, and calculate the demographic covariance. Because $\mathrm{E}[p] = 0.5$, each d_i is either 0.5 or -0.5. For any individual i, there are $N/2$ individuals j with the opposite fate (hence $d_i d_j = -0.25$) and $N/2 - 1$ individuals j with the same fate (hence $d_i d_j = 0.25$). Thus,

$$\sum_{\substack{j=1\\ j\neq i}}^{N} d_i d_j = -0.25$$

for each i, so the demographic covariance is

$$\tau = \mathrm{E}\left[\frac{1}{N(N-1)}\sum_{i=1}^{N}(-0.25)\right] = -\frac{0.25}{N-1}. \tag{9}$$

Inserting this result into equation 8 reveals that

$$\begin{aligned}\mathrm{var}(S) &= N\left(\sigma_d^2 + \frac{0.25}{N-1}\right) - N^2\frac{0.25}{N-1}\\ &= N\sigma_d^2 - N(N-1)\frac{0.25}{N-1}\\ &= N(\sigma_d^2 - 0.25)\end{aligned} \tag{10}$$

which is exactly what our approach would yield. The relationship between the demographic covariance and $1/(N-1)$ seems to hold for most forms of population structure. We prefer to define a per capita variance reduction (v), which is independent of population size. The variance reduction is related to the demographic covariance as $v = -(N-1)\tau$, so the variance in the number surviving is

$$\mathrm{var}(S) = N(\sigma_d^2 - v). \tag{11}$$

This seems less open to misinterpretation than equation 8.