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Demographic heterogeneity, cohort selection, and population growth

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Abstract. Demographic heterogeneity—variation among individuals in survival and reproduction—is ubiquitous in natural populations. Structured population models address heterogeneity due to age, size, or major developmental stages. However, other important sources of demographic heterogeneity, such as genetic variation, spatial heterogeneity in the environment, maternal effects, and differential exposure to stressors, are often not easily measured and hence are modeled as stochasticity. Recent research has elucidated the role of demographic heterogeneity in changing the magnitude of demographic stochasticity in small populations. Here we demonstrate a previously unrecognized effect: heterogeneous survival in long-lived species can increase the long-term growth rate in populations of any size. We illustrate this result using simple models in which each individual's annual survival rate is independent of age but survival may differ among individuals within a cohort. Similar models, but with nonoverlapping generations, have been extensively studied by demographers, who showed that, because the more "frail" individuals are more likely to die at a young age, the average survival rate of the cohort increases with age. Within ecology and evolution, this phenomenon of "cohort selection" is increasingly appreciated as a confounding factor in studies of senescence. We show that, when placed in a population model with overlapping generations, this heterogeneity also causes the asymptotic population growth rate λ to increase, relative to a homogeneous population with the same mean survival rate at birth. The increase occurs because, even integrating over all the cohorts in the population, the population becomes increasingly dominated by the more robust individuals. The growth rate increases monotonically with the variance in survival rates, and the effect can be substantial, easily doubling the growth rate of slow-growing populations. Correlations between parent and offspring phenotype change the magnitude of the increase in λ, but the increase occurs even for negative parent–offspring correlations. The effect of heterogeneity in reproductive rate on λ is quite different: growth rate increases with reproductive heterogeneity for positive parent–offspring correlation but decreases for negative parent–offspring correlation. These effects of demographic heterogeneity on λ have important implications for population dynamics, population viability analysis, and evolution.

Key words: cohort selection; demographic heterogeneity; frailty; population dynamics; structured population model; survival.

INTRODUCTION

As a cohort ages, the most frail individuals (those with high intrinsic mortality risks) tend to die earliest. This "cohort selection" means that if each individual's relative frailty remains constant throughout its lifetime then the cohort as a whole becomes steadily less frail (Vaupel and Yashin 1983, 1985). More than a theoretical curiosity, cohort selection has been rigorously demonstrated in experimental laboratory populations (Manton et al. 1981, Carey et al. 1992, Vaupel and Carey 1993). The underlying frailty heterogeneity has been documented in a variety of species, including

crocodiles (Isberg et al. 2006), baboons (Bronikowski et al. 2002), birds (Wintrebert et al. 2005, Fox et al. 2006), wild plants (Beckage and Clark 2003, Landis et al. 2005), domestic animals (Ducrocq et al. 2000, Casellas et al. 2004), humans (Yashin et al. 1999, Garibotti et al. 2006), and British aristocrats (Doblhammer and Oeppen 2003).

The taxonomic breadth of this list suggests that cohort selection may be a very common ecological phenomenon. Another reason to think so is that a number of common processes can generate persistent heterogeneity in both frailty and reproduction among individuals in a cohort. These include fine-scale spatial habitat heterogeneity (e.g., Gates and Gysel 1978, Boulding and Van Alstyne 1993, Menge et al. 1994, Winter et al. 2000, Franklin et al. 2000, Manolis et al.
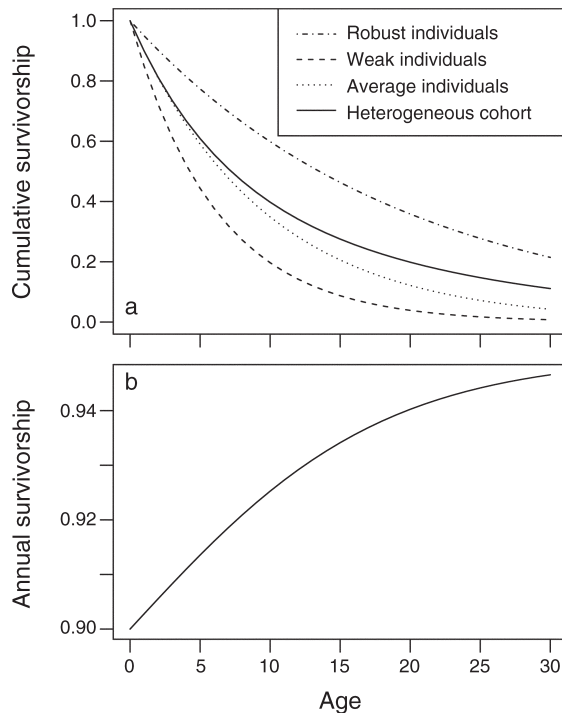
Fig. 1. Patterns of survival in a heterogeneous cohort, illustrating cohort selection and the frailty effect. Suppose that at birth, a cohort is made up of equal parts "strong" individuals (with constant annual survivorship of 0.95) and "weak" individuals (with constant annual survivorship of 0.85). The strong and weak classes within the cohort have differing survival curves (panel a). Frail individuals selectively die as the cohort ages, causing the cohort to become progressively more dominated by robust individuals, and so the average survival rate of the surviving individuals increases with cohort age (panel b). As a consequence, the survivorship curve of the heterogeneous cohort is always higher than that of a homogeneous cohort with the "average" annual survivorship of 0.9 (panel a).

2002, Bollinger and Gavin 2004, Landis et al. 2005), unequal allocation of parental care (e.g., Manser and Avey 2000, Johnstone 2004), maternal family effect (e.g., Fox et al. 2006), conditions during early development, including birth order effects (e.g., Lindström 1999), persistent social rank (e.g., von Holst et al. 2002), and genetics (e.g., Yashin et al. 1999, Ducrocq et al. 2000, Gerdes et al. 2000, Casellas et al. 2004, Isberg et al. 2006). Thus, we would expect cohort selection to be quite common in nature. What are its ecological effects?

Ecological studies of cohort selection have examined two general phenomena. First, frailty heterogeneity and the associated selection can mask individual senescence (e.g., McDonald et al. 1996, Pletcher and Curtsinger 1998, Service 2000, Nussey et al. 2008), complicating studies of the evolutionary ecology of aging. Second, correlations between survival and fecundity in long-lived organisms provide evidence of trade-offs or heterogeneity in overall quality, both of which have implications for life-history evolution (e.g., Bérubé et al. 1999, Cam

et al. 2002, Reid et al. 2003). In both cases, the focus has been on cohort selection's effects on the dynamics of cohorts, and ultimately in uncovering individual-level demographic characteristics that might be subject to natural selection. In contrast, the effects of cohort selection on population dynamics have not been studied.

An independent research effort has analyzed the effects of "demographic heterogeneity" (both persistent and transient differences among individuals in their expected demographic rates) on population dynamics (e.g., Johnson et al. 1986, Engen et al. 1998, Conner and White 1999, Melbourne and Hastings 2008). This research focuses on the effects of heterogeneity on demographic stochasticity (Kendall and Fox 2002, 2003, Fox and Kendall 2002, Vindenes et al. 2008) and extinction risk (Conner and White 1999, Robert et al. 2003, Lloyd-Smith et al. 2005) in small populations. In at least two of these studies (Conner and White 1999, Vindenes et al. 2008) the models were structured in such a way that cohort selection might operate, but the potential impact of this phenomenon on the population dynamic outcomes was either unrecognized or poorly understood.

Cohort selection tends to increase the average survival at older ages, relative to a homogeneous population (Fig. 1). As with any process that increases survival rates, intuition suggests that (all else being equal) cohort selection should thereby increase population growth rates. If this simple intuition is correct, it has a number of important ecological, evolutionary, and management implications because cohort selection could have a substantial impact on population dynamics. For example, since spatial environmental heterogeneity is an important source of frailty heterogeneity in plants and sessile animals, cohort selection will affect how such populations respond to changes in spatial heterogeneity. As a second example, note that variation in conditions and resources during early development can cause frailty heterogeneity. In many cases parents actively provide their offspring with unequal resources; if cohort selection provides a way for a mother to increase her long-term fitness, it can allow selection for variation in offspring provisioning even in a constant environment (in contrast to bet-hedging mechanisms, which require variable environments). Finally, by influencing the average population growth rate, cohort selection could be a means by which demographic heterogeneity influences extinction risk that is far more important than the effects on demographic stochasticity that have been previously studied, and could be equally important for understanding the growth and spread of invasive species.

In this paper, we rigorously confirm the above qualitative intuition and use simple models to quantify the population dynamic impacts of cohort selection. Because we are studying the long-term multigenerational effects of cohort selection we examine how positive or negative parent–offspring correlations in demographic

traits affects the results. Finally, we show that these results are a consequence of cohort selection in particular, rather than demographic heterogeneity more generally, by demonstrating the very different effects of fecundity heterogeneity. We also show that this mechanism can cause selection for heterogeneity in offspring demography, entirely without environmental stochasticity.

GENERAL RESULTS

As a heterogeneous cohort ages, the less robust individuals die off more rapidly and the survivors are, on average, more robust. Thus, the average survival rate of the survivors increases (as has been exhaustively described by Vaupel and Yashin 1985; Fig. 1). Consider cumulative survivorship. Because survivorship to age x of a particular phenotype is a nonlinear function of the annual survival rate, we need to apply nonlinear averaging to find the average survivorship to age x, $\bar{l}_x$. Application of Jensen's inequality reveals that $\bar{l}_x \geq \tilde{l}_x$, where $\tilde{l}_x$ is the survivorship of an individual with the average phenotype, and equality occurs only when there is no heterogeneity or $x < 2$ (see Appendix A). We can generate a quantitative estimate of this effect using a Taylor expansion (Appendix A):

$$\bar{l}_x \approx \tilde{l}_x \left[1 + \frac{x(x-1)}{2} \frac{\sigma_P^2}{\bar{P}^2} \right] \quad (1)$$

where $\bar{P}$ and σ_P^2 are the mean and variance of the annual survival distribution at birth.

Unfortunately, we cannot use nonlinear averaging to directly calculate the effects of heterogeneity on the asymptotic growth rate, λ, or even on the the average annual survivorship at age x, $\tilde{P}_x$ (see Appendix B). However, we can use Euler's equation to draw rigorous conclusions about the qualitative effect on λ. Recall that, at the stable age distribution, λ satisfies $1 = \sum_{x=1}^{\infty} l_x m_x \lambda^{-x}$, where m_x is the fertility of age-x individuals. We have already seen that cohort selection acts to increase the average value of l_x for $x > 1$, and doesn't change it for $x = 1$; if m_x is unchanged (e.g., there is no heterogeneity in fertility), then the only way to maintain Euler's equation is to increase λ.

The only way to quantitatively estimate the impact of cohort selection on population growth is to explicitly model the demographic heterogeneity. Here we show this effect in a simple model of two distinct survival phenotypes; in Appendix C we also model a continuous distribution of phenotypes.

A SIMPLE MODEL

We model two classes of individuals, with differing annual survival rates, P_1 and P_2; survival is otherwise independent of age. Each individual reproduces at rate f, with half of each parent's offspring going into each class (there is no heritability of survival class). If offspring recruit into the adult population after one year, with survival P_0, then the model can be encompassed in a
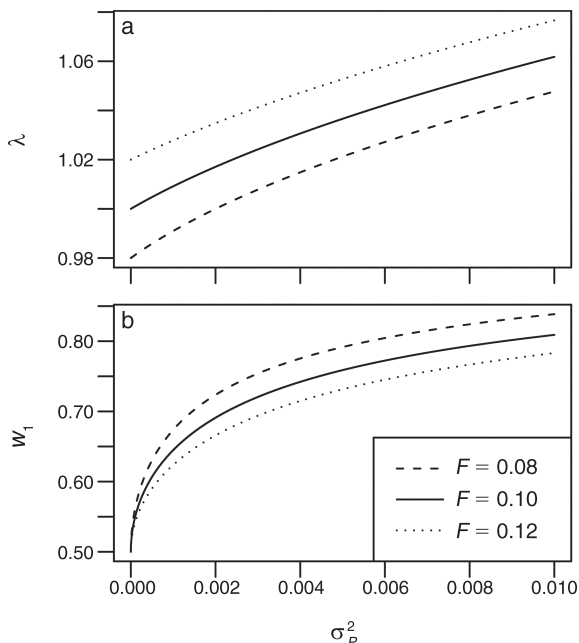

FIG. 2. (a) The asymptotic growth rate (λ) and (b) fraction of the stable stage structure in the high-survival class (w_1) as a function of the variance in survival heterogeneity (σ_P^2) for three values of the reproductive rate (F). Average survival is $\bar{P} = 0.9$ in all cases.

simple matrix projection model:

$$\mathbf{A}_1 = \begin{pmatrix} P_1 + F/2 & F/2 \\ F/2 & P_2 + F/2 \end{pmatrix} \quad (2)$$

where $F = fP_0$ is the net per-capita reproductive rate. If we let $\bar{P}$ be the mean survival of the two classes, we can write $P_1 = \bar{P} + \sigma_P$ and $P_2 = \bar{P} - \sigma_P$ and explore how the asymptotic growth rate λ (the growth rate when the population is at its stable phenotype distribution) depends on σ_P^2, the among-class variance in survival rates.

As in any matrix population model, λ is the dominant eigenvalue of $\mathbf{A}_1$:

$$\lambda = \bar{P} + \frac{1}{2} \left(F + \sqrt{F^2 + 4\sigma_P^2} \right). \quad (3)$$

The asymptotic population growth rate increases more slowly than linearly with the variance in survival between the two phenotypes, although the curvature is not pronounced unless F is quite small (Fig. 2a). At the corresponding stable phenotype distribution, the fraction of individuals in the class with higher survival is

$$w_1 = \frac{F}{F - 2\sigma_P + \sqrt{F^2 + 4\sigma_P^2}} \quad (4)$$

(Fig. 2b). Cohort selection shifts the stable stage structure toward dominance by more robust individuals, with consequent increases to the asymptotic growth rate.
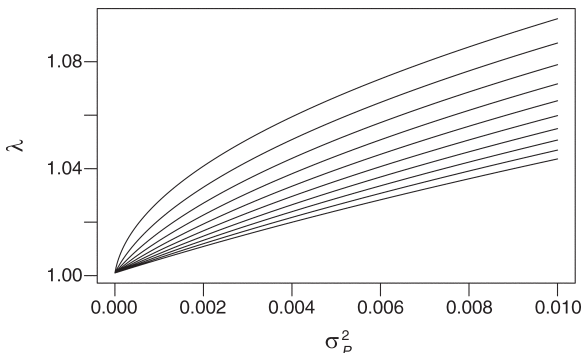
FIG. 3. The asymptotic growth rate (λ) as a function of the variance in survival heterogeneity (σ_P^2) and parent–offspring correlation (h). The correlation h increases monotonically from -0.9 to 0.9 (in increments of 0.2) from the lowest curve to the highest curve. Other parameters are $\bar{P} = 0.9$ and $F = 0.101$.

Asymptotically, the population growth is given by $\lambda = \bar{P} + F$, where $\bar{P}$ is the population average survival rate:

$$\bar{P} = w_1 P_1 + w_2 P_2 \qquad (5)$$

$$= \bar{P} + \frac{1}{2}\left(\sqrt{F^2 + 4\sigma_P^2} - F\right). \qquad (6)$$

This depends not only on the demographic variance (which controls the strength of cohort selection) but also on the reproductive rate, which controls the balance between young and old individuals in the (implicit) age structure of the population. Indeed, the intensity of the effect of survival heterogeneity on growth rate is

$$\frac{\partial \lambda}{\partial \sigma_P^2} = \frac{1}{\sqrt{F^2 + 4\sigma_P^2}} \qquad (7)$$

which decreases with the reproductive rate F. We can understand this with respect to the implicit age structure in the population: when F is large, there will be a high proportion of young individuals in the population. These individuals have experienced little cohort selection, bringing the population average survival closer to that in a homogeneous population.

These effects of survival heterogeneity on population growth rate are not unique to the simple model with only two phenotypes. In Appendix C, we show that similar effects arise in a model with a continuous distribution of survival phenotypes among offspring.

PARENT–OFFSPRING CORRELATION AND COHORT SELECTION

Eq. 2 assumes that a newborn's phenotype is independent of its parent's phenotype. Genetic heritability or shared environments can create a positive correlation between parent and offspring phenotypes. Negative parent–offspring correlations can also arise due to many different mechanisms in which parental traits affect the expression of the same trait in the offspring (Kirkpatrick and Lande 1989; a simple

example is that of a population of perennial organism in which favorable sites tend to remain occupied by successful parents, so that most offspring occupy poor sites). We can model both phenomena by incorporating a parent–offspring correlation, h (constrained to be between negative one and one), into the matrix model:

$$\mathbf{A}_2 = \begin{pmatrix} P_1 + \dfrac{1+h}{2}F & \dfrac{1-h}{2}F \\ \dfrac{1-h}{2}F & P_2 + \dfrac{1+h}{2}F \end{pmatrix}. \qquad (8)$$

The asymptotic growth rate for this model is

$$\lambda = \bar{P} + \frac{1}{2}\left[(1+h)F + \sqrt{(1-h)^2 F^2 + 4\sigma_P^2}\right] \qquad (9)$$

(Fig. 3). As one would expect, larger parent–offspring correlations mean that heterogeneity has a stronger positive impact on the population growth rate; but even with strongly negative correlations, heterogeneity causes λ to increase.

HETEROGENEITY IN REPRODUCTION

We develop a simple two-type model analogous to Eq. 2. Here all individuals have the same survival rate P, but at birth acquire one of two reproductive phenotypes: $F_1 = \bar{F} + \sigma_F$ or $F_2 = \bar{F} - \sigma_F$. Note that while this will most likely reflect differences in fertility (f), it might also reflect differences in newborn survival (P_0) that are consistently associated with the parent (e.g., if the parent holds the same territory for life, and there is spatial heterogeneity in the risks to newborns). We also immediately introduce the parent–offspring correlation, h, so that the model is

$$\mathbf{A}_3 = \begin{pmatrix} P + \dfrac{1+h}{2}F_1 & \dfrac{1-h}{2}F_2 \\ \dfrac{1-h}{2}F_1 & P + \dfrac{1+h}{2}F_2 \end{pmatrix}. \qquad (10)$$

The asymptotic growth rate of the population, from the dominant eigenvalue of $\mathbf{A}_3$, is

$$\lambda = P + \frac{1}{2}\left[(1+h)\bar{F} + \sqrt{(1-h)^2 \bar{F}^2 + 4h\sigma_F^2}\right]. \qquad (11)$$

This is structurally nearly identical to Eq. 9, except that the heterogeneity term (σ_F^2) is multiplied by the parent–offspring correlation.

There are two key things to notice about this result. The first is that when $h = 0$ (there is no parent–offspring correlation), then $\lambda = P + \bar{F}$ for all values of σ_F: heterogeneity in reproduction has absolutely no effect on the population dynamics. This is because, without the parent–offspring correlation, reproductive heterogeneity has no impact on the stable stage distribution, which remains 50:50. Second, in the presence of positive parent–offspring correlations, heterogeneity in repro-

duction increases the asymptotic growth rate; the opposite is true with negative correlation. Qualitatively, this matches our intuition, although there is a marked asymmetry between the patterns with positive and negative h (Fig. 4).

COHORT SELECTION AND NATURAL SELECTION

Cohort selection has important evolutionary consequences. In a nutshell, it can cause natural selection for heterogeneous offspring, entirely in the absence of environmental variability. It can do so under both density-dependent and density-independent selection, as we now show.

First, consider selection on the trait "ability to produce heterogeneous offspring." In other words, assume that the offspring phenotypes (specifically, their values of F_i or P_i) do not vary genetically, but are determined by, say, positional effects on the mother (e.g., Silvertown 1984, Venable 1985, Venable and Búrquez M. 1990, Cowley and Atchley 1992) or birth order (e.g., Lindström 1999, Manser and Avey 2000, Johnstone 2004). Then the appropriate measure of long-term maternal fitness is λ, and the selection gradient is given by $d\lambda/d\sigma_P^2$ (Fisher 1958, Charlesworth 1994). The trait is favored in any population for which heterogeneity increases the growth rate, as given in Eqs. 9 and 11.

Now consider selection on multiple genotypes. All else being equal, do genotypes having higher σ_P^2 have higher fitness? In the simplest case, where σ_P^2 varies independently of $\bar{P}$ and $h = 0$, the selection gradient is given by Eq. 7, which is always positive (albeit a declining function of both F and σ_P^2). Again, heterogeneity is selected for even without environmental variability.

However, we can imagine that there might be a trade-off between σ_P^2 and $\bar{P}$. In fact, if $\bar{P} > 0.5$ such a trade-off seems unavoidable, as the variance is then a decreasing function of the mean. In this case, because $\partial\lambda/\partial\bar{P} = 1$, Eq. 7 sets the break-even point: the selection gradient for survival heterogeneity is positive as long as $\partial\bar{P}/\partial\sigma_P^2 > -\partial\lambda/\partial\sigma_P^2$. For some range of values, then, heterogeneity is favored within these populations even without environmental stochasticity; the range over which this holds depends on the specific model for the trade-off.

Similar conclusions hold for density-dependent population growth. In density-dependent settings, the fitness criterion is no longer the effect on low-density population growth rate, but the effect on equilibrium population size (Fisher 1958, Charlesworth 1994). A recent study analyzes the effects of demographic haterogeneity in a continuous-time density-dependent model, and shows that under cohort selection, the equilibrium population size increases linearly with σ_P^2 (Stover et al. 2011). Although that paper only examines density dependence in reproduction, it is straightforward to show that a qualitatively similar result occurs with density-dependent survival, at least as long as the interaction coefficients are homogeneous across phenotypes; and because the result depends on cohort selection
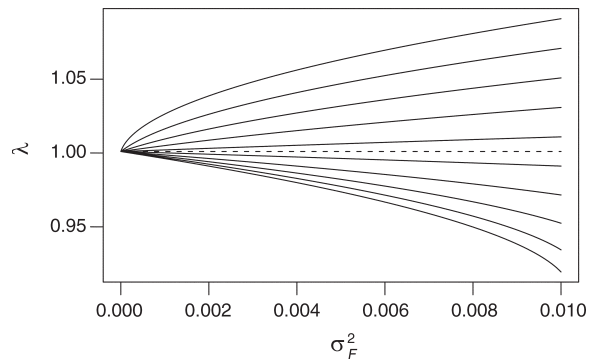

FIG. 4.   The asymptotic growth rate (λ) as a function of the variance in reproductive heterogeneity (σ_F^2) and parent–offspring correlation (h). The correlation h increases monotonically from −0.9 to 0.9 (in increments of 0.2) from the lowest to the highest solid curve; the dashed line indicates $h = 0$. Other parameters are $P = 0.9$ and $\bar{F} = 0.101$.

in ways analogous to the results reported here, we expect qualitatively similar results in a discrete-time density-dependent model. Although not explicit in Stover et al.'s model, it is easy to show that the equilibrium population size is also an increasing function of the mean survival rate. As with the density-independent case, trade-offs may limit the circumstances under which heterogeneity increases the equilibrium population size, but this depends on the specific model for the trade-off.

DISCUSSION

We have shown that cohort selection, a common demographic phenomenon that has been well studied within individual cohorts (in the context of aging and senescence), has substantial impacts on the dynamics of populations with overlapping generations. In particular, we have shown that in a simple density-independent population model, increasing the variance of non-heritable but persistent heterogeneity in individual survival rates increases the asymptotic population growth rate. In contrast, heterogeneity in reproductive rates has no effect on the asymptotic growth rate. This fundamental difference reveals that cohort selection, rather than demographic heterogeneity per se, is the driver of the effects of survival heterogeneity on population growth, and the result highlights an important interaction between demography and population ecology.

Heterogeneity in survival impacts population dynamics even without any correlation between parent and offspring phenotypes. As expected, introducing a positive parent–offspring correlation in either survival or reproduction increases the population growth rate relative to the baseline case of zero correlation; negative correlation has the the opposite effect. Notably, however, heterogeneity in survival increases the population growth rate somewhat even in the presence of strong negative correlations.

Most populations probably have some amount of survival heterogeneity; does this mean that the many existing demographic models and life table analyses that have ignored heterogeneity have systematically underestimated λ? Not necessarily. Imagine that a heterogeneous population governed by Eq. 2 is at its stable phenotype distribution, and one estimates the mean survival of the all the individuals found in the population, independent of age, at a given time. This average survival will be the quantity in Eq. 5, which when added to the per capita reproductive rate gives exactly the asymptotic growth rate of the heterogeneous population. Alternatively, if individuals can be aged, then one could estimate the apparent age-dependent survival (as illustrated in Fig. 1b) and put that into a Leslie matrix model. This assumes homogeneity within each age class, but that survivorship increases with age. We have found, both through simulations and by writing down the apparent age-specific survival rates and solving the resulting Euler equation, that the value of λ resulting from this (incorrect) description of the population is also identical to the asymptotic growth rate of the heterogeneous population. These two results hold even if the parent–offspring correlation is not zero.

Thus, despite the likely ubiquity of demographic heterogeneity, failing to recognize it in the field may not compromise our ability to estimate long-run growth rates. This is reassuring, given all the empirical quantitative demography that has ignored heterogeneity! However, because the average death rate in any structured population is sensitive to population structure (e.g., Cohen 1986), these homogeneous models would fail to predict the population growth rate if the phenotypic distribution were perturbed away from its stable structure, say by stochastic forces that unduly affected a certain age class or that affected the phenotype distribution of newborns. This is directly analogous to the problem of trying to describe an age-structured population with an unstructured model: it is possible to replicate the asymptotic growth rate, but transient dynamics are not captured, nor is it possible to understand the elasticities of the growth rate to underlying biological parameters. Furthermore, we have found that some "natural" ways of characterizing the average survival cannot correctly reconstruct the asymptotic growth rate of the heterogeneous population. In particular, using the mean life span to estimate the average survival rate (e.g., Pereira and Daily 2006) will give incorrect population growth rates if survival is heterogeneous (see Appendix D).

Loop analysis is a technique for comparing the elasticities of λ to various life history pathways (van Groenendael et al. 1994, Wardle 1998). Our simple model has three loops: self-loops representing survival and self-reproduction of each of the phenotypes, and a loop that links the two phenotypes through alternate-type reproduction (Appendix E). Increasing survival heterogeneity increases the elasticity of the high-survival self-loop (Fig. E.1), as does increasing the parent–offspring correlation (Fig. E.2). These reinforce the intuitive understanding that as h or P_1 approach one, the demography of type-1 individuals dominates the dynamics. Interestingly, the high-reproduction self-loop increases with increasing reproductive heterogeneity, even when $h = 0$ (Fig. E.3), even though the reproductive heterogeneity has no effect on the asymptotic population growth rate or phenotype structure.

These effects of survival heterogeneity can have important evolutionary consequences. In particular, our results suggest that natural selection can favor traits that allow mothers to induce heterogeneity in the annual survival rates of their offspring, as long as the heterogeneity does not excessively reduce the mean offspring survivorship. Cohort selection can favor demographic heterogeneity entirely in the absence of environmental variability, and as such is a mechanism not previously understood by population biologists as providing selection for demographic heterogeneity.

This type of selection should not be confused with bet-hedging (e.g., Gillespie 1975, Marshall et al. 2008), which is an adaptation to environmental unpredictability. On the other hand, these two mechanisms are not mutually exclusive. In a variable environment, selection may occur both by bet-hedging and by cohort selection; we speculate that this may make it somewhat easier for heterogeneity to evolve by selection.

Given that models are now often central to empirical inference in ecology, "getting the model right" is of fundamental ecological importance. Modeling studies that deliberately or inadvertently introduce survival heterogeneity tend to use the mean phenotype at birth as the baseline for comparison, failing to recognize that cohort selection raises the mean survival rate in a heterogeneous population. For example, Conner and White (1999) analyzed an individual-based model that had persistent heterogeneity in both birth and survival rates, as well as demographic stochasticity, environmental stochasticity, and age structure. They found that increasing individual heterogeneity in survival rates, while holding the mean demographic rate at birth constant, reduced the likelihood of extinction. They attribute this result to the existence of "a few ...exceptionally 'fit' animals [that] are unlikely to die and be removed from the population [and] can contribute to births year after year." Our analysis reveals that the "exceptionally fit" individuals actually come to dominate the population. Furthermore, it appears that the authors did not recognize that increasing demographic heterogeneity would increase the deterministic population growth rate (and they see their "exceptionally fit" individuals living in small, rather than growing, populations). Indeed, it is likely that much of the reduction in extinction risk that they observed can be explained by the increase in the average population growth rate due to the selection effect described here (although in the smallest populations

there may also be a role for the effects of heterogeneity on demographic stochasticity; Kendall and Fox 2002).

In their study of the effects of demographic heterogeneity on demographic stochasticity, Vindenes et al. (2008) state that they held the deterministic growth rate constant, but they do not describe how they do this. We surmise that they must have reduced the mean demographic rates in order to counteract the effects of cohort selection that we have demonstrated here. However, the mean demographic rates have a direct impact on the demographic variance (their response variable of interest), so this does not represent a benign control for the effects of survival heterogeneity on mean population growth rates.

In many long-lived species, an individual's survival rate is lower as a juvenile and as a very old individual than as a prime-aged adult. Our models do not incorporate this age-dependence; but since cohort selection acts even when individual mortality varies with age (Vaupel and Yashin 1983, 1985), we expect a qualitatively similar effect of survival heterogeneity on the population growth rate. Unfortunately, biologically sensible survival models that incorporate both age-dependent mortality and frailty variance have only been developed for continuous-time models, so incorporating these effects in the current model framework is not trivial.

Three important extensions include investigating demographic heterogeneity within stages (e.g., juveniles and adults) in stage-structured models, examining interactions between heterogeneity and density dependence, and incorporating demographic or environmental stochasticity into the model. Using a somewhat different modeling framework, Stover et al. (2011) introduced demographic heterogeneity into a density-dependent model, finding that increasing survival heterogeneity increases the equilibrium density. The asymptotic phenotype structure shifts as the population grows from low density to equilibrium, so that the dynamics can only be approximated by a homogeneous model if survival is assumed to *increase* with density. Heterogeneity in fertility has no effect on the dynamics, but heterogeneity in both fertility and offspring viability changes the equilibrium abundance (positively or negatively, depending on the correlations between the two parameters). Stover et al. (2011) assumed that the competitive interactions among individuals was not affected by the demographic phenotype; relaxing this assumption is an obvious direction for further work.

We expect stochasticity to introduce two additional effects of demographic heterogeneity. First, heterogeneity may impact the variance due to demographic stochasticity (Fox and Kendall 2002, Kendall and Fox 2003, Vindenes et al. 2008). Second, environmental stochasticity creates the potential for the phenotype distribution to be perturbed away from its stable distribution. The resulting transient dynamics may cause the mean and variance of the stochastic growth rate to

be rather different from the "equivalent" homogeneous model. Developing a framework to look at the additive and interactive effects of heterogeneity on cohort selection, the demographic variance, and the response to environmental perturbations is key to fully understanding the patterns in individual-based models such as those presented by Conner and White (1999); stochastic loop analysis may be helpful here (Claessen 2005). This can also have evolutionary significance as species adapt to rapid change—for example, heritable phenotypic variation leading to higher invasibility (Lavergne and Molofsky 2007), or any species adapting to environmental change such as fragmentation or climate change (Boulding and Hay 2001, Boyce et al. 2006, Willi and Hoffmann 2009). Lande and Shannon (1996) demonstrated that the relationship between genetic variance in a population and population persistence is rather complex: genetic variation can either increase or reduce the extinction risk, depending on the temporal pattern of environmental variation. How that conclusion changes in a population experiencing cohort selection, which we contend is very common, is an important question for further research.

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APPENDIX A

The derivation of Eq. 1 using nonlinear averaging of the survivorship function (*Ecological Archives* E092-169-A1).

APPENDIX B

The demonstration that the age-specific mean annual survival and population growth rate are not simple consequences of nonlinear averaging (*Ecological Archives* E092-169-A2).

APPENDIX C

An analysis of a model with continuously distributed demographic traits (*Ecological Archives* E092-169-A3).

APPENDIX D

A demonstration that mean life span fails to estimate mean survival in the heterogeneous population (*Ecological Archives* E092-169-A4).

APPENDIX E

The loop analysis of the two-type model (*Ecological Archives* E092-169-A5).