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Demographic heterogeneity impacts density-dependent population dynamics

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Abstract Among-individual variation in vital parameters such as birth and death rates that is unrelated to age, stage, sex, or environmental fluctuations is referred to as demographic heterogeneity. This kind of heterogeneity is prevalent in ecological populations, but is almost always left out of models. Demographic heterogeneity has been shown to affect demographic stochasticity in small populations and to increase growth rates for density-independent populations. The latter is due to "cohort selection," where the most frail individuals die out first, lowering the cohort's average mortality as it ages. The importance of cohort selection to population dynamics has only recently been recognized. We use a continuous time model with density dependence, based on the logistic equation, to study the effects of demographic heterogeneity in mortality and reproduction. Reproductive heterogeneity is introduced in three ways: parent fertility, offspring viability, and parent-offspring correlation. We find that both the low-density growth rate and the equilibrium population size increase as the magnitude of mortality heterogeneity increases or as parent-offspring phenotypic correlation increases. Population dynamics are affected by complex interactions among the different types of heterogeneity, and trade-off scenarios are examined which can sometimes reverse the effect of increased heterogeneity. We show that there are a number of different homogeneous approximations to heterogeneous models, but all fail to capture important parts of the dynamics of the full model.

Keywords Demographic heterogeneity · Individual variation · Frailty · Cohort selection · Logistic model

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Introduction

Demographic heterogeneity, among individual variation in vital parameters such as survival and reproduction, is ubiquitous, resulting from 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. 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). Demographic heterogeneity is distinct from (but often confused with) demographic stochasticity. Demographic heterogeneity refers to variation in the underlying vital parameters, while demographic stochasticity refers to the variability in the fates of individuals under specified values of vital parameters.

Demographic heterogeneity has only now begun to receive considerable theoretical attention. Recent studies

have shown that—under many but not all circumstances—survival heterogeneity can have dramatic effects on the population growth rate (Kendall et al. 2011), demographic stochasticity (Fox and Kendall 2002; Kendall and Fox 2002, 2003; Vindenes et al. 2008), and extinction risk of both density-independent populations (Conner and White 1999; Fox 2005; Lloyd-Smith et al. 2005) and density-dependent populations (Robert et al. 2003). Survival heterogeneity has yet to be studied in a deterministic, continuous-time, density-dependent model. There is, however, ample reason to suspect that the effects of heterogeneity may be substantial.

Heterogeneity in reproduction is far more poorly understood than that in survival. In part, this is for a good reason: survival heterogeneity leads to selection within cohorts (Manton et al. 1981; Vaupel and Yashin 1983, 1985; Carey et al. 1992; Vaupel and Carey 1993; Kendall et al. 2011), but reproductive heterogeneity does not (Kendall et al. 2011). However, reproductive heterogeneity may interact with survival heterogeneity and affect population dynamics in ways not obvious from the action of either type of heterogeneity acting alone. Additionally, individuals may vary in reproductive output in several different ways—including their total fertility or its timing, quality of offspring, or their propensity to pass along their traits to their offspring—and these may also affect population dynamics in different ways. Melbourne and Hastings (2008) showed that extinction risk was increased in a stochastic Ricker model by the presence of demographic heterogeneity. An early individual-based model by Uchmański (2000) found that individual weight variability (which results in resource partitioning and reproductive variability) led to slower extinctions (as long as the variation was not too large or too small). Imperfect heritability of initial weights in the population resulted in longer persistence times than that for a population with perfect heritability, which in turn persisted longer than a population with only a single weight class (Uchmański 2000).

Here we study the effects of demographic heterogeneity in survival, reproduction, or both, on explicitly density-dependent populations. We develop models that incorporate three types of reproductive heterogeneity: parent fertility, parent–offspring phenotypic correlation, and initial offspring viability. A central question is how the different types of heterogeneity interact with one another. One interesting result is that if only one of these types of reproductive heterogeneity is operating, it has no effect on deterministic population dynamics unless survival is also heterogeneous.

Are there homogeneous models that capture the important dynamics of heterogeneous models, at least approximately? Such simplifications would clearly be useful. To address this issue requires identifying the appropriate definition of an "average individual." Since survival heterogeneity can lead to changes over time in the distribution of phenotypes within a population (Vaupel and Yashin 1985; Kendall et al. 2011), the average individual within the population at one time is not generally the same as the average individual at another time. We show that appropriately parameterized homogeneous models can describe the asymptotic dynamics (Kendall et al. 2011), but cannot capture the transient behavior of the heterogeneous model.

The model

We start with a version of the single species logistic model in which density dependence only affects reproduction: $\frac{\mathrm{d}}{\mathrm{d}t}N = \beta N\left(1-\frac{N}{K}\right) - \delta N$, where β is the low-density birth rate, δ is the death rate, and K is the maximum possible abundance. We rescale the population by K without changing the notation to arrive at $\frac{\mathrm{d}}{\mathrm{d}t}N = \beta N(1-N) - \delta N$. The equilibrium density is

$$N^* = 1 - \frac{\delta}{\beta}. \tag{1}$$

We introduce a heterogeneous version of this model by including two phenotypes with different birth and death rates. The differential equations 2a–b govern the dynamics of the heterogeneous model.

$$\frac{\mathrm{d}}{\mathrm{d}t}n_1 = (\beta_{11}n_1 + \beta_{21}n_2)(1-n_1-n_2) - \delta_1 n_1 \tag{2a}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}n_2 = (\beta_{12}n_1 + \beta_{22}n_2)(1-n_1-n_2) - \delta_2 n_2 \tag{2b}$$

Mortality is given by the death rates, δ_i, for $i = 1, 2$. Reproduction is determined by the birth rates, β_{ij}, which are the rates at which phenotype i parents reproduce phenotype j offspring. We will refer to n_i as phenotype i's density and $w_i = \frac{n_i}{n_1+n_2}$ as its relative frequency.

The phase space of the heterogeneous model lies in the triangular region $0 \le n_1 + n_2 \le 1$ with $n_i \ge 0$ for $i = 1, 2$. There are two fixed points of concern: extinction at $(0, 0)$ and equilibrium at (n_1^*, n_2^*). These two fixed points are connected by an invariant manifold. In addition, for many parameter values, a population with any initial conditions rapidly converges to the manifold, so we discuss the dynamics along this manifold in detail.

When the nonzero equilibrium exists, it is a stable node, the population persists, and $(0, 0)$ is a saddle. Otherwise $(0, 0)$ is a stable node and the population goes extinct.

If at least one phenotype is self-sustaining ($\beta_{ii} > \delta_i$), then the population persists. When neither phenotype is self-sustaining, persistence occurs only if

$$\beta_{12}\beta_{21} > (\beta_{11} - \delta_1)(\beta_{22} - \delta_2). \tag{3}$$

If phenotype i is self-sustaining and dominates phenotype j $\left(\frac{\beta_{ii}}{\delta_i} > \frac{\beta_{jj}}{\delta_j}\right)$, then phenotype j persists ($n_j^* > 0$) only if $\beta_{ij} > 0$.

We analyze this model in several steps. In the "Mortality Heterogeneity" section we analyze the model for the case where mortality alone is heterogeneous; in doing so we generalize results in Kendall et al. (2011). In the "Homogeneous comparisons" section, we examine a number of the possible homogeneous analogs to the heterogeneous mortality model, and ask how well they approximate the heterogeneous model. Finally, in the "Including heterogeneous birth rates" section, we include reproductive heterogeneity and examine the full dynamics of the model.

Mortality heterogeneity

We start by examining death rate heterogeneity with homogeneous birth rates: $\beta_{ij} = \frac{\beta}{2}$ for each i and j. We set the death rates to $\delta_1 = \bar{\delta} + \sigma_\delta$, and $\delta_2 = \bar{\delta} - \sigma_\delta$, where $0 \leq \sigma_\delta < 1$ (without loss of generality, phenotype one is assumed to be on average shorter lived than phenotype two). Time is rescaled so that, $\bar{\delta} = 1$ (the life expectancy of an individual with the mean death rate, $\bar{\delta}$, defines the model timescale).

It is useful to restate the model in terms of the total population abundance, $n = n_1 + n_2$, and the difference in the abundances of the phenotypes, $q = n_2 - n_1$. Equations 2a and 2b become

$$\frac{\mathrm{d}}{\mathrm{d}t}n = \beta n(1-n) - n + \sigma_\delta q \tag{4a}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}q = -q + \sigma_\delta n. \tag{4b}$$

The difference in relative frequencies, $u = w_2 - w_1 = \frac{q}{n}$, is herein referred to as the *population structure*. The relative frequencies sum to one, thus knowing their difference allows one to determine the complete structure of the population. Using the single variable u to denote the structure of the population simplifies some formulae. The population size n and the structure u together give a complete description of the population: $n_1 = n\left(\frac{1-u}{2}\right)$ and $n_2 = n\left(\frac{1+u}{2}\right)$. We use Eqs. 2a–b to describe solutions in phase space and Eqs. 4a–b to describe the dynamics of the population size and structure.

Low-density dynamics

Without density dependence, the population increases unboundedly, approaching an asymptotic growth rate and a stable phenotype distribution. The asymptotic growth rate, r^o, is given by the dominant eigenvalue for $(0,0)$, and the stable phenotype distribution is $u^o = w_2^o - w_1^o$ where $\mathbf{w}^o = (w_1^o, w_2^o)$ is the normalized (to sum to one) eigenvector associated with r^o:

$$u^o = \frac{1}{\sigma_\delta}\left(-\frac{\beta}{2} + \sqrt{\left(\frac{\beta}{2}\right)^2 + \sigma_\delta^2}\right) \tag{5}$$

$$r^o = \frac{\beta}{2} - 1 + \sqrt{\left(\frac{\beta}{2}\right)^2 + \sigma_\delta^2} \tag{6}$$

Increasing the death rate variance, σ_δ^2, while holding the birth rate constant increases the dominance of the longer-lived phenotype within the growing population. Increasing β while holding the death rate variance constant does the opposite (Fig. 1a). The growth rate increases with σ_δ^2 with a negative second derivative, but is nearly linear when β is large (Fig. 1b). This formula is nearly identical to that found by Kendall et al. (2011) where heterogeneity in survival was introduced into a discrete-time density-independent model.

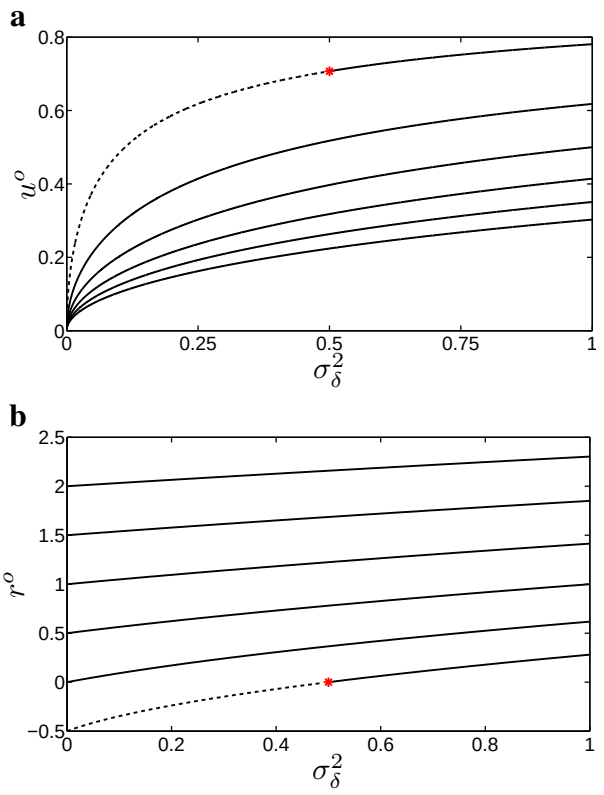

Fig. 1 **a** The difference in relative frequencies on the eigenvector of the zero equilibrium is shown as a function of the death rate variance for β ranging from 0.5 (*upper curve*) to 3 (*lower curve*) in increments of 0.5. **b** The asymptotic growth rate for the density-independent model is shown as a function of the death rate variance for β ranging from 0.5 (*lower curve*) to 3 (*upper curve*) in increments of 0.5. The point $\sigma_\delta^2 = 0.5$ is marked on the $\beta = 0.5$ curve in both panels since this is the critical minimum variance in death rates needed for persistence (the *dashed portion* of the line indicates a subcriticality or equivalently negative growth rate).

If the heterogeneous population is initially close to zero and β is small, then solution curves quickly approach this

eigenvector and thus reach the stable structure and growth rate. When the birth rates are large, convergence to the stable structure is slow.

Dynamics near equilibrium

Returning to the density-dependent model, the equilibrium difference in abundances is $q^* = \sigma_\delta n^*$, where the equilibrium population size is

$$n^* = 1 - \frac{1-\sigma_\delta^2}{\beta}. \tag{7}$$

The equilibrium population structure is thus $u^* = \frac{q^*}{n^*} = \sigma_\delta$. The equilibrium population size increases linearly in σ_δ^2, and comparing Eq. 7 to the equilibrium of the logistic model (Eq. 1) suggests that $1-\sigma_\delta^2$ plays the role of an "average death rate" at equilibrium. This intuition is correct: it is the weighted mean death rate of individuals at the equilibrium distribution ($w_1^*\delta_1 + w_2^*\delta_2$). This fact is used later to construct a homogeneous model for comparison.

When β is small and there is little heterogeneity, the manifold is nearly linear, and the dynamics are similar to that of a homogeneous population (Fig. 2a). As the level of heterogeneity is increased, the manifold curves upward, reflecting the bias towards the longer-lived phenotype at equilibrium. When the population is initially small, trajectories in phase space rapidly converge to the manifold for most parameter values (Figs. 2a–c).

However, when the birth rate β is large, the population grows to near equilibrium size rapidly, then growth slows as the population structure slowly shifts towards domination by the longer-lived phenotype (Fig 2d).

As the population grows along the manifold, abundance grows sigmoidally (but not quite logistically) over time (Fig. 3a). The population structure does not start shifting until the abundance is well above zero. The shift in structure produces a decrease in the mean population death rate, which somewhat counteracts the density-dependent reduction in the birth rate. When β is large, the population grows to near equilibrium size rapidly but reaches the equilibrium structure much more slowly (Fig. 3b).

Density dependence increases the bias towards the longer-lived phenotype ($u^* > u^o$). This is caused by the reduction in population growth rate. A growing population has a greater abundance of young individuals whereas older individuals appear in greater numbers in an equilibrium population, and older individuals are more likely to have lower intrinsic mortality.

Cohort dynamics

One can gain insight on the effect of mortality heterogeneity by examining cohort dynamics. With homogeneous repro-

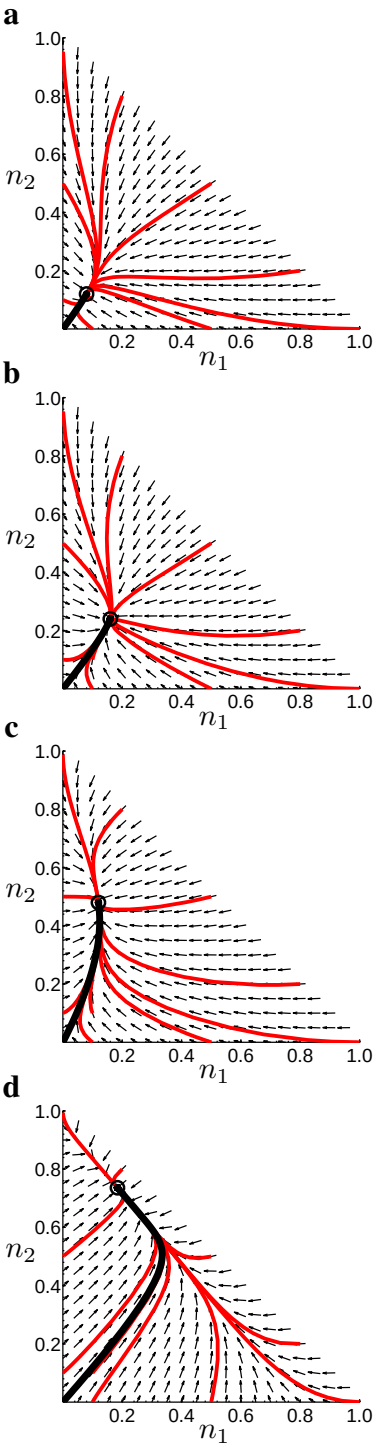

Fig. 2 The direction field in the (n_1, n_2) phase space is shown, for four different parameter combinations. *Arrows* show local direction of motion; *red curves* show sample trajectories for various initial conditions. The manifold connecting $n = 0$ to n^* (marked with $\circ$) is shown as a *thicker black curve* in each graph. When β and σ_δ are both small, **a** $\beta = 1.2, \sigma_\delta = 0.2$ and **b** $\beta = 1.6, \sigma_\delta = 0.2$, the manifold is nearly straight. As heterogeneity is increased, **c** $\beta = 1.6, \sigma_\delta = 0.6$, the longer-lived phenotype increases its dominance in the population. For large β, **d** $\beta = 8, \sigma_\delta = 0.6$, the population grows rapidly to near equilibrium size with little change in structure, but then growth slows as the structure slowly shifts.

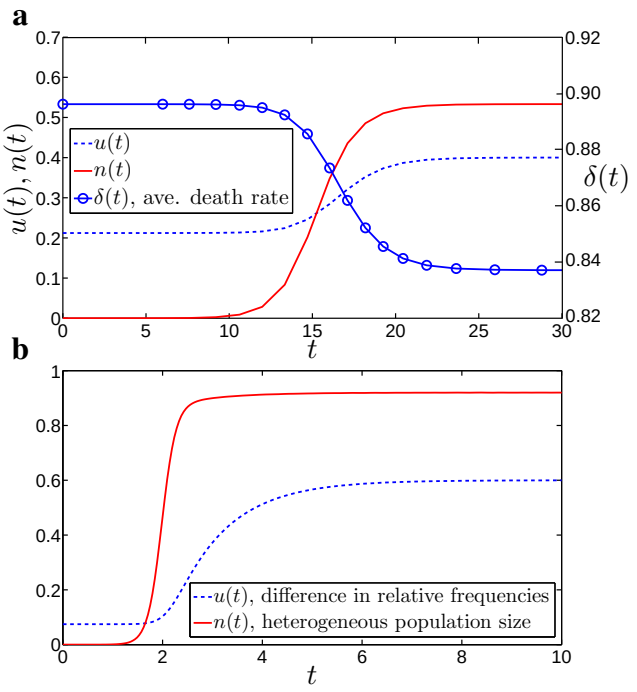

Fig. 3 **a** The difference in relative frequencies of the two phenotypes, total population density, and average death rate along the manifold are shown over time for $\beta = 1.8$ and $\sigma_\delta = 0.4$. **b** The population size and the difference in relative frequencies of the two phenotypes along the manifold over time is shown for $\beta = 8$ and $\sigma_\delta = 0.6$. Phenotype 2 dominates always, but less so initially. The population grows rapidly until just past $t = 2$, but the phenotypic distribution changes little. When the exponential growth phase has ended, the structure begins to shift more gradually towards a greater dominance of the longer-lived phenotype. See Fig. 2d for the phase portrait along the manifold.

duction, a cohort is initially split evenly between the phenotypes. The survival to age x within the cohort is given by

$$l(x) = \frac{1}{2}(e^{-(1+\sigma_\delta)x} + e^{-(1-\sigma_\delta)x}). \tag{8}$$

Jensen's inequality can be used to show that this is greater than survivorship in a homogeneous population with death rate one (e^{-x}). The discrepancy increases with both age and the amount of heterogeneity:

$$l(x) \approx e^{-x}\left(1 + \frac{x^2\sigma_\delta^2}{2}\right). \tag{9}$$

The Lotka-Euler equation gives the relationship between the asymptotic exponential growth rate r, $l(x)$, and the birth rate $b(x)$ at age x:

$$1 = \int_0^\infty e^{-rx} l(x) b(x) dx. \tag{10}$$

We use this to examine two cases: exponential growth along the manifold at low density; and population structure at the equilibrium, when $r = 0$.

When the population is growing, it is evident from Eq. 10 that increasing $l(x)$ for some x while holding $b(x)$ constant and not allowing $l(x)$ to decrease for any other x must lead to an increase in r.

At equilibrium, since $r = 0$ and $b(x) \equiv b$ is age independent, Eq. 10 reduces to

$$1 = b\int_0^\infty l(x)dx = \frac{b}{2}\left(\frac{1}{1+\sigma_\delta} + \frac{1}{1-\sigma_\delta}\right) = \frac{b}{1-\sigma_\delta^2}.$$

Since b depends on density ($b(n) = \beta(1-n)$), this sets the density at which growth is zero: n^* satisfies $b(n^*) = 1-\sigma_\delta^2$. Another way of understanding this is to recall that within a stationary population, the crude mortality rate is the inverse of the life expectancy (Cohen 1986). In the heterogeneous model, since a newborn is equally likely to be either phenotype, the average life expectancy of a newborn within the equilibrium population is

$$E\left(\frac{1}{\delta}\right) = \frac{1}{2}\left(\frac{1}{1+\sigma_\delta} + \frac{1}{1-\sigma_\delta}\right) = \frac{1}{1-\sigma_\delta^2}. \tag{11}$$

Therefore, the crude mortality rate within our equilibrium population is

$$\hat{\delta} = \frac{1}{E\left(\frac{1}{\delta}\right)} = 1 - \sigma_\delta^2. \tag{12}$$

This shows that the average death rate in the equilibrium population is the harmonic mean of the death rates, given the distribution of phenotypes at birth.

Homogeneous comparisons

There are several different ways one might calculate average demographic rates to construct a homogeneous model to approximate the heterogeneous population. Accordingly, we parameterize models using the phenotypic distribution at birth, the distribution along the eigenvector near zero ($\mathbf{w}^o$), and the distribution at equilibrium. Finally, we build a synthetic model by approximating the per-capita growth rate curve.

The phenotypic distribution at birth is uniform, giving average death rate $\bar{\delta} = 1$; we call this the *arithmetic mean model*. In the equilibrium population, however, the average death rate is $\hat{\delta} = 1 - \sigma_\delta^2$ and is called the *harmonic mean model*. At low density along the manifold, the average death rate is $\delta^o = \delta_1\left(\frac{1-u^o}{2}\right) + \delta_2\left(\frac{1+u^o}{2}\right)$, and we call this the *$u^o$-mean model*. These homogeneous approximations use the same birth rate parameter β, differing only by the death rate. These three models are summarized in Table 1.

The arithmetic mean model underestimates the low-density growth rate ($\tilde{r}^o = \beta - 1 < r^o$), and the harmonic mean model overestimates it ($\hat{r}^o = \beta - (1-\sigma_\delta^2) > r^o$);

by design, the u^o-mean model matches it exactly. The arithmetic and u^o-mean models have smaller equilibrium population sizes than the harmonic mean model, which (again, by design) exactly matches the heterogeneous model (Fig. 4a).

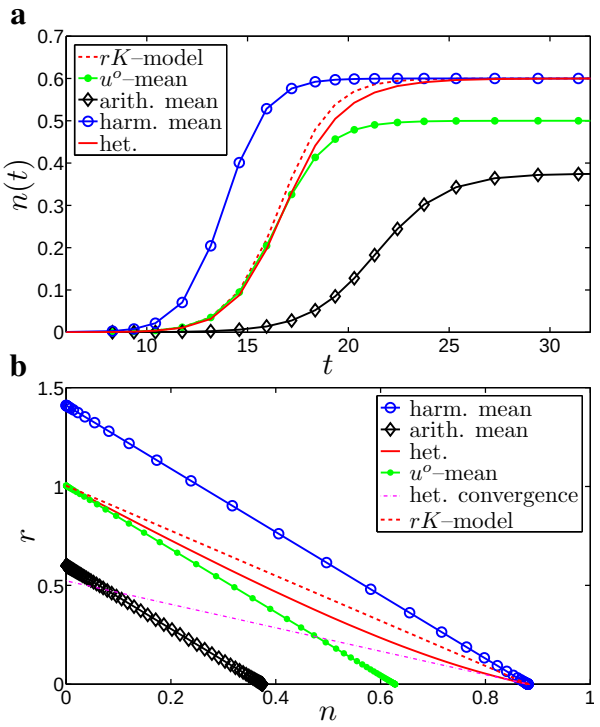

Fig. 4 **a** The population size over time and **b** the per-capita growth rate as a function of population size are shown for the heterogeneous model and all homogeneous comparisons considered for $\beta = 1.6$ and $\sigma_\delta = 0.6$.

Even along the manifold, heterogeneous mortality renders the population's dynamics fundamentally different from those of the logistic model. This is illustrated by the nonlinear dependence of the per-capita growth rate on density (Fig. 4b).The fact that the average slope is shallower than $-\beta$ is a consequence of the shifting phenotypic structure as the population grows. For small heterogeneous populations on the manifold, the per-capita growth rate curve is tangent to that of the u^o-mean model (with slope $-\beta$), but as the population approaches equilibrium, the asymptotic slope is $\frac{\lambda}{n^*}$ (denoted *heterogeneous convergence* in Fig. 4b). The eigenvalue λ (Eq. 13) gives the rate ($|\lambda|$) at which solutions approach n^* along the associated eigenvector.

$$\lambda = -\frac{\beta}{2} - \sigma_\delta^2 + \sqrt{\frac{\beta^2}{4} - (\beta - 1)(1 - \sigma_\delta^2) + \sigma_\delta^4}. \tag{13}$$

The heterogeneous population converges to equilibrium more slowly than any of the homogeneous models when β is sufficiently large. When β is small, the arithmetic mean model converges to equilibrium slowest. When $\beta < 1.5$, increasing heterogeneity increases $|\lambda|$, otherwise increasing heterogeneity decreases $|\lambda|$.

A linear approximation to the per-capita growth rate curve yields another homogeneous model, with parameters $\beta^{rK} = \frac{r^o}{n^*}$ and $\delta^{rK} = \frac{r^o}{n^*}(1 - n^*)$, and it is called the rK model. The parameters of the rK model depend on the heterogeneity in a more complicated way, but can be rewritten as $\beta^{rK} = \beta\left(\frac{r^o}{\beta - \hat{\delta}}\right)$ and $\delta^{rK} = \hat{\delta}\left(\frac{r^o}{\beta - \hat{\delta}}\right)$. Increasing σ_δ decreases both the birth and death rates, although it increases their difference since $\beta^{rK} - \delta^{rK} = r^o$. Note that the "birth" and "death" rates in this synthetic model do not match the actual demography for any n.

There is an alternative way to formulate the rK model that better matches observable demography. Write $\frac{\mathrm{d}}{\mathrm{d}t}n = \beta n(1-n) - \delta(n)n$, where $\delta(n) = 1 - \sigma_\delta u(n)$ is the average death rate in a population with phenotypic structure $u(n)$; the latter is the structure exhibited by a population growing through size n along the manifold. A linear approximation to $u(n)$ yields

$$\delta(n) = 1 - \sigma_\delta\left(\frac{u^* - u^o}{n^*}n + u^o\right). \tag{14}$$

The resulting homogeneous logistic model has density dependence in both birth and death rates, but it is identical to the rK model. This formulation may be more ecologically intuitive, and it captures the fact that the average death rate declines as the population grows (opposite to the usual competitive effect of density on mortality). However, when reproductive heterogeneity is introduced, this formulation fails, because it introduces a third order term into the differential equation, and is a worse approximation than that for the rK model in Table 1.

Although these homogeneous models match different aspects of the heterogeneous dynamics along the manifold to varying degrees, all of them fail to capture dynamics away from the manifold, which would be important in the presence of environmental perturbations.

Table 1 Homogeneous model parameters

Birth rate	Death rate	Model name
β	1	Arithmetic mean model
β	$1 - \sigma_\delta^2$	Harmonic mean model
β	$1 - \sigma_\delta u^o$	u^o-mean model
$\frac{r^o}{n^*}$	$\frac{r^o}{n^*}(1 - n^*)$	rK model

Including heterogeneous birth rates

We now relax the assumptions that the β_{ij} are all equal (the total birth rate for phenotype i is denoted $\beta_i = \beta_{i1} + \beta_{i2}$). At equilibrium, the probability that a birth results in an

offspring with life expectancy δ_i^{-1} depends on the population structure. The probability that the parent is phenotype i is w_i^* (the proportion of the equilibrium population that is phenotype i). The expected birth rate of a randomly selected parent from the equilibrium population is $\hat{\beta} = \beta_1 w_1^* + \beta_2 w_2^*$, and the rate at which a randomly selected parent gives birth to phenotype i is $\hat{\alpha}_i = \beta_{1i} w_1^* + \beta_{2i} w_2^*$. Thus, at equilibrium, the probability of being born as phenotype i is $\hat{\alpha}_i \hat{\beta}^{-1}$. It still holds that the average death rate at equilibrium is the harmonic mean of the death rates at birth:

$$\hat{\delta} = \frac{1}{E^{u^*}\left(\frac{1}{\delta}\right)} = \left(\frac{1}{\delta_1} \cdot \frac{\hat{\alpha}_1}{\hat{\beta}} + \frac{1}{\delta_2} \cdot \frac{\hat{\alpha}_2}{\hat{\beta}}\right)^{-1} \tag{15}$$

where $E^{u^*}(\cdot)$ is the expectation with respect to the distribution of phenotypes at birth within the equilibrium population. An algebraic exercise verifies that $\hat{\delta} = \delta_1 w_1^* + \delta_2 w_2^*$. At equilibrium, $r = 0$, thus the Lotka-Euler equation becomes:

$$1 = \int_0^\infty \left(\frac{\hat{\alpha}_1}{\hat{\beta}} e^{-(1+\sigma_\delta)x} + \frac{\hat{\alpha}_2}{\hat{\beta}} e^{-(1-\sigma_\delta)x}\right) \hat{\beta}(1-n^*) dx,$$

which can be rearranged to give

$$n^* = 1 - \frac{1}{\hat{\beta} E^{u^*}\left(\frac{1}{\delta}\right)}. \tag{16}$$

Phenotypes can vary with respect to reproduction in three distinct ways. First, individual parents can vary in their fertility, which we parameterize as f_i. Second, the resulting offspring can vary in their initial viability (or survival to adulthood). We parameterize offspring viability as v_{ij}, allowing the viability to depend on both parent phenotype and offspring phenotype. An extensive literature examines consequences of trade-offs between the number of offspring (fertility) and their size (assumed to be closely related to offspring viability) (e.g., Smith and Fretwell 1974; McGinley et al. 1987; Venable 1992; Fox and Czesak 2000; Marshall and Keough 2007; Brown and Shine 2009). Our approach allows for a trade-off between fertility and offspring viability, but does not require it. It is certainly possible for the two traits to be positively correlated—for example, if variation in both traits is determined by site or territory quality, one would expect parents with high fertility to produce offspring with high viability. Finally, parents may vary in their propensities to have offspring of one type or another. This may be caused by genetic heritability (Lynch and Walsh 1998), or by parent–offspring correlation in environmental conditions. More specifically, let h_{ij} be the probability that an individual offspring (prior to viability selection) from a parent of phenotype i has phenotype j.

Letting b represent the mean fertility of the two phenotypes, we define $f_1 = b(1-f)$ and $f_2 = b(1+f)$ with $-1 < f < 1$. Thus phenotype one is assumed to have the lower fertility when f is positive; when f is negative and $\sigma_\delta > 0$ there is a trade-off between fertility and mortality. Assuming that the probability an offspring shares its parent's phenotype does not depend on the parent's phenotype, we set $h_{ij} = \frac{1}{2}(1-h)$ when $i \neq j$, and $h_{ij} = \frac{1}{2}(1+h)$ when $i = j$ for $-1 < h < 1$. The parameter h is related to the level of phenotypic correlation between parent and offspring. We consider here only the simplest case of heterogeneity in offspring viability: that viability depends only on offspring phenotype rather than on parent phenotype as well. Thus we let $v_{i1} = \frac{1}{2}(1-v)$ and $v_{i2} = \frac{1}{2}(1+v)$ where $-1 < v < 1$. We let $b = 2\beta$ and arrive at the birth rate parametrization shown in Table 2.

Plugging the parametrization from Table 2 into Eqs. 2a–b and using the heterogeneous death rates as in the "Mortality heterogeneity" section gives:

$$\begin{aligned}\frac{\mathrm{d}}{\mathrm{d}t} n =& \beta(1+vhf)n(1-n) \\ &+ \beta(vh+f)q(1-n) - n + \sigma_\delta q\end{aligned} \tag{17a}$$

$$\begin{aligned}\frac{\mathrm{d}}{\mathrm{d}t} q =& \beta(v+hf)n(1-n) \\ &+ \beta(vf+h)q(1-n) - q + \sigma_\delta n.\end{aligned} \tag{17b}$$

An important feature of these equations is that v, h, and f appear in all possible combinations. This means that they can interact, so that particular combinations of these parameters may 'override' the effect one would expect from considering these parameters singly.

Our analysis proceeds as follows. First, we examine reproductive heterogeneity when mortality is homogeneous, and then we examine how mortality and reproductive heterogeneity interact with one another. Because these interactions become quite complex, we primarily focus on the cases where parent–offspring phenotypic correlation is zero.

Table 2 The birth rate parametrization is shown

Rate Parameter	Formula
β_{11}	$\frac{\beta}{2}(1-f)(1+h)(1-v)$
β_{12}	$\frac{\beta}{2}(1-f)(1-h)(1+v)$
β_{21}	$\frac{\beta}{2}(1+f)(1-h)(1-v)$
β_{22}	$\frac{\beta}{2}(1+f)(1+h)(1+v)$

The magnitude of heterogeneity in fertility is given by f, that in offspring viability by v, and parent–offspring phenotypic correlation is given by h. Each of the parameters v, h, and f are constrained to the interval $(-1, 1)$. Phenotype two has higher fertility when $f > 0$ and higher offspring viability when $v > 0$. Offspring are more likely to be the same phenotype as their parent when $h > 0$.

Reproductive heterogeneity only

If reproduction is the sole source of heterogeneity ($\sigma_\delta = 0$), the equilibrium population size is

$$n^* = 1 - \frac{1}{\beta(1+vhf)+\beta(vh+f)u^*}. \tag{18}$$

The formula for the equilibrium structure u^* depends on the sign of $vh + f$: when $vh + f > 0$:

$$u^* = -\frac{(1-h)(1-vf)}{2(vh+f)} + \sqrt{\left(\frac{(1-h)(1-vf)}{2(vh+f)}\right)^2 + \frac{v+hf}{vh+f}} \tag{19}$$

in which case $\beta_1 < \beta_2$; when $vh + f < 0$ (which gives $\beta_1 > \beta_2$):

$$u^* = -\frac{(1-h)(1-vf)}{2(vh+f)} - \sqrt{\left(\frac{(1-h)(1-vf)}{2(vh+f)}\right)^2 + \frac{v+hf}{vh+f}}; \tag{20}$$

and if $vh + f = 0$ ($\beta_1 = \beta_2$):

$$u^* = -\frac{v+hf}{(1-h)(1-vf)}. \tag{21}$$

The manifold connecting zero and n^* is linear, so the population structure does not change along it: the average birth and death rates are constant. An initially small population (with arbitrary structure) reaches the stable structure quickly, then grows logistically thereafter. The population on the manifold is modeled exactly by the harmonic mean model, u^o-mean model, and rK model; all of which are identical when mortality is homogeneous.

If only a single form of birth rate heterogeneity is included (that is if only one of v, h, or f is nonzero), then the average birth rate parameter along the manifold is β. Thus, heterogeneity has no effect on deterministic population dynamics.

On the manifold, phenotype two dominates ($u^* > 0$) if $v + hf > 0$. Defining $\alpha_i = \beta_{1i} + \beta_{2i}$ gives a quantification for the appearance rate of phenotype i (distinguished from phenotype i's reproductive rate, β_i). When $v + hf > 0$, $\alpha_2 > \alpha_1$ means that a cohort (in the equilibrium population) is initially dominated by longer-lived individuals. Because there is no cohort selection, the initial cohort structure translates directly to the equilibrium phenotypic structure within the population.

Setting $h = 0$ simplifies the model allowing us to examine the interactions between heterogeneity in fertility and viability:

$$\frac{\mathrm{d}}{\mathrm{d}t}n = \beta n(1-n) + \beta f q(1-n) - n \tag{22a}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}q = \beta v q(1-n) - q. \tag{22b}$$

The equilibrium population size is

$$n^* = 1 - \frac{1}{\beta(1+fv)}, \tag{23}$$

and the asymptotic density-independent growth rate is

$$r^o = \beta(1+fv) - 1. \tag{24}$$

The population structure along the manifold is $u^o = u^* = v$ (the manifold is linear since mortality is homogeneous). The equilibrium structure is determined by the amount of viability heterogeneity. Viability is effectively a juvenile survival. A cohort of newly recruited adults has structure v, and absent mortality heterogeneity, this structure does not change as the cohort ages.

The equilibrium population size and low-density growth rate both increase with increasing fv. When $fv > 0$, one phenotype has both lower viability and lower fertility than the other, and is thus weaker in all respects. If $fv < 0$, then there is a trade-off (one phenotype has a higher fertility while the other has higher viability). In this trade-off situation, increasing the magnitude of either type of heterogeneity lowers n^* and r^o.

The average density-independent birth rate along the manifold (regardless of population size) is $\beta(1 + fv)$ since the population structure is v. In fact, along the manifold the population dynamics are logistic with effective birth rate parameter $\tilde{\beta} = \beta(1 + fv)$: $\frac{\mathrm{d}}{\mathrm{d}t}n = \tilde{\beta}n(1-n) - n$.

Note that swapping the values of f and v has no effect on n^* or r^o, but it alters the equilibrium population structure.

Heterogeneity in both mortality and reproduction

We now examine the full model with demographic heterogeneity in both mortality and reproduction. Using Eqs. 17a–b, the equilibrium population size is given by

$$n^* = 1 - \frac{1-\sigma_\delta u^*}{\beta(1+vhf)+\beta(vh+f)u^*}, \tag{25}$$

and the low-density growth rate is

$$r^o = \beta(1+vhf) + \beta(f+vh)u^o - 1 + \sigma_\delta u^o. \tag{26}$$

We can get analytical formulae for u^* and u^o (see Appendix: Population Structure), but they are very large and unwieldy; plugging them into the above equations provides little insight. Numerical analysis reveals that the dependence of u^* and u^o on each type of heterogeneity is complex, but

one clear pattern is that increasing h always increases n^* and r^o, and that both are symmetric across the line $f + v = 0$ (symmetric with respect to swapping the values of β_{12} and β_{21}).

The former effect seems intuitive, because it allows the more successful phenotype to produce a greater number of successful offspring. However, the relationship cannot be easily seen analytically.

When there is no correlation between parent and offspring phenotypes ($h = 0$) the analysis is tractable. Initially, we also set $v = 0$ to examine how heterogeneity in fertility and mortality interact to affect population dynamics. The differential equations are now:

$$\frac{\mathrm{d}}{\mathrm{d}t}n = \beta n(1-n) + \beta f q(1-n) - n + \sigma_\delta q \tag{27a}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}q = -q + \sigma_\delta n. \tag{27b}$$

The equilibrium population size is

$$n^* = 1 - \frac{1-\sigma_\delta^2}{\beta(1+f\sigma_\delta)}, \tag{28}$$

the equilibrium structure is $u^* = \sigma_\delta$, and the low-density growth rate is

$$r^o = \frac{\beta}{2} - 1 + \sqrt{\frac{\beta^2}{4} + (\beta f + \sigma_\delta)\sigma_\delta}. \tag{29}$$

Both n^* and r^o are increasing functions of f. Cohort dynamics, and the resulting population structure, depend on the amount of mortality heterogeneity.

When mortality and fertility are positively correlated ($f > 0$), increasing the magnitude of heterogeneity always increases r^o and n^*. However, when $f < 0$, the population has a trade-off: the phenotype with higher fertility has higher mortality. Increasing the magnitude of fertility heterogeneity (making it more negative) decreases r^o and n^*. If $f = -\sigma_\delta$, then the equilibrium population size is $n^* = 1 - \frac{1}{\beta}$ which is the same as that for a population with the arithmetic mean demographic rates, but the dynamics away from n^* are different.

To understand how r^o and n^* change when heterogeneity in mortality is manipulated, we take derivatives:

$$\frac{\mathrm{d}}{\mathrm{d}\sigma_\delta}r^o = \frac{1}{2}\frac{\beta f + 2\sigma_\delta}{\sqrt{\frac{\beta^2}{4} + \sigma_\delta(\beta f + \sigma_\delta)}} \tag{30}$$

$$\frac{\mathrm{d}}{\mathrm{d}\sigma_\delta}n^* = \frac{f(1+\sigma_\delta^2) + 2\sigma_\delta}{\beta^2(1+f\sigma_\delta^2)}. \tag{31}$$

Increasing mortality heterogeneity can decrease both n^* and r^o but only if a sufficiently strong trade-off is present ($f < -\frac{2\sigma_\delta}{\beta}$ for r^o; $f < -\frac{2\sigma_\delta}{1+\sigma_\delta^2}$ for n^*).

When viability is heterogeneous ($v \neq 0$), the equilibrium population size becomes

$$n^* = 1 - \frac{1-\sigma_\delta u^*}{\beta(1+fu^*)}, \tag{32}$$

where the equilibrium population structure is

$$u^* = \frac{v+\sigma_\delta}{1+v\sigma_\delta}. \tag{33}$$

Viability heterogeneity acts on the equilibrium population size by shifting the equilibrium population structure u^* away from σ_δ; increasing viability heterogeneity v increases u^*. Increasing v only decreases n^* if $f < -\sigma_\delta$ (because u^* is increased, and $\frac{\mathrm{d}}{\mathrm{d}u^*}n^* < 0$). This is the condition for phenotype two to have a lower expected fitness, and increasing v increases the rate at which phenotype two enters the population effectively decreasing the mean fitness of the population.

Discussion

This is one of the few published treatments of dynamics of heterogeneous density-dependent populations and is the first published study to isolate the effects of several types of demographic heterogeneity (in the absence of stochasticity) for a continuous-time density-dependent model. As a result, both the model and its analysis lead to important insights about the nature of demographic heterogeneity, the potential importance of interactions between different components of demographic heterogeneity, the effects of heterogeneity on population dynamics, and the extent to which homogeneous models are useful approximations.

Death rate heterogeneity increases the low-density growth rate, a phenomenon that is caused by cohort selection (Vaupel and Yashin 1985) and has been observed in discrete-time population models (Kendall et al. 2011). In addition, by lowering the mean death rate in the equilibrium population, cohort selection increases the equilibrium density. As the population grows from low density to the equilibrium, the average mortality rate declines; which is not strong enough to create an Allee effect in the population, but does run counter to the usual effect of density on mortality.

Two-sex models with demographic differences among males and females (e.g., Caswell and Weeks 1986; Jenouvrier et al. 2010) provide an interesting contrast to our work. Differential survival between the sexes—along with other sources of demographic variation, such as differences in maturation times for males and females—is recognized as a primary driver of imbalances in the population sex ratio (Girondot and Pieau 1993; Bessa-Gomes et al. 2004; Veran and Beissinger 2009), through a process that is directly analogous to cohort selection. This shifts the average survival in

the population (as in our model), but in the two-sex models only female survival has a direct influence on the population dynamics. Under most mating systems, however, a strong sex ratio bias can reduce fertility (because of inability to find mates), with the result that survival heterogeneity among genders can reduce asymptotic population growth rates (e.g., Jenouvrier et al. 2010), opposite to the effect we show in our models.

A key feature of our approach is the inclusion of basic reproductive heterogeneity in three different ways that are not mutually exclusive: heterogeneity in parent fertility, in offspring quality, and in parent–offspring correlation. Although heterogeneity in parent–offspring correlation was not treated explicitly in our analysis, numerical analysis suggests that the distribution of offspring phenotypes varies among the different parent types due to the interaction of fertility, offspring quality, and parent–offspring correlation. When only a single type of reproductive heterogeneity is present and mortality is homogeneous, deterministic population dynamics are unaffected; prior results indicating that reproductive heterogeneity increases extinction risk (e.g., Robert et al. 2003; Melbourne and Hastings 2008) require the presence of demographic stochasticity.

An important consequence of this model is that the three types of reproductive heterogeneity occur in all possible combinations in the dynamic equations 17a–b and in the equilibrium expressions for the population size and low-density growth rate (Eqs. 25–26). Previous treatments of demographic heterogeneity have not addressed these interactions.

Trade-offs can appear in several varieties between mortality, fertility, and offspring quality. In many trade-off scenarios, increasing the magnitude of heterogeneity of either source of variation decreases both the growth rate and the equilibrium population size. However other cases exist—such as a weak trade-off between mortality and fertility—where incrementally increasing the magnitude of heterogeneity in either will increase the equilibrium population size and growth rate.

In addition to changing the equilibrium population size, stable phenotypic distribution, and growth rate, heterogeneity can have significant effects on both transient and asymptotic dynamics. Although in many cases the population appears to follow a sigmoidal curve, there are important differences. Perhaps the most important of these is that the shifting population structure results in a nonlinear per-capita growth rate curve and shifting average demographic rates.

There are multiple ways to make homogeneous approximations to the heterogeneous model. However, none of the approaches we used here is a good approximation for all settings. The distribution of phenotypic traits within a population can change as it grows, making a single homogeneous approximation necessarily inaccurate. For a density-dependent population with homogeneous reproduction and heterogeneous mortality, merely taking the average death rate leads to underestimates of the equilibrium population size and low-density growth rate. Parameterizing a model with the average life expectancy leads to an overestimate of the low-density growth rate, but does capture the correct equilibrium.

We assumed an underlying model that is asymptotically stable—the continuous logistic model. It is an open question how heterogeneity might affect the dynamics of models capable of more complex dynamics, such as those involving lags in density dependence. Jones and Ellner (2007) studied the effects of rapid evolution (resulting from genetic variability) on a predator–prey system and showed that the "cost of a good defense" affects whether or not and what type of cycles develop. Similarly, we might expect a more complex set of behaviors in models with heterogeneity in response to density.

Directly testing many of the predictions of our models (such as the increase in equilibrium density caused by survival heterogeneity) in real populations would require comparisons between homogeneous and heterogeneous populations with exactly the same mean demography, an empirically infeasible task even in laboratory populations. However, it should be relatively straightforward to look for the proximate mechanism that underlies the increased equilibrium, namely the steady decline in the average death rate as the population increases towards equilibrium. Experimental populations could be constructed either from mixtures of genotypes known to differ strongly in survival probabilities, or (perhaps more simply) by artificially imposing heterogeneity, much as Dennis et al. (2001) artificially changed birth and death rates. By imposing heterogeneity of varied strength, one could directly test this prediction.

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Appendix: Population Structure

We solve $\frac{\mathrm{d}}{\mathrm{d}t}n(t) = 0$ and $\frac{\mathrm{d}}{\mathrm{d}t}q(t) = 0$ for $u = \frac{q}{n}$ and arrive at two quadratic equations, one for $n(t) = n^*$ and the other for $n(t) = 0$. The equilibrium structure, u^*, is the solution to the equation $au^2 + bu + c = 0$ whose coefficients are

$$a = (vh + f) + \sigma_\delta(vf + h),$$

$$b = (1 - h)\Big((1 - vf) + \sigma_\delta(v - f)\Big),$$

and

$$c = -\Big((v + hf) + \sigma_\delta(1 + vhf)\Big).$$

If $a \neq 0$, then the equation is quadratic, and there are two candidate solutions. When $f > -h\frac{v+\sigma_\delta}{1+\sigma_\delta v}$, u^* is given by the '+' root, and alternatively it is given by the '−' root. When $a = 0$ the equation is linear, and $u^* = -c/b$.

If $v > -\frac{hf+\sigma_\delta}{1+hf\sigma_\delta}$, then phenotype two dominates the equilibrium population ($u^* > 0$), and if $v < -\frac{hf+\sigma_\delta}{1+hf\sigma_\delta}$, then phenotype one dominates the equilibrium population ($u^* > 0$).

The structure along the eigenvector out of the origin, u^o, is the solution to the equation $au^2 + bu + c = 0$ whose coefficients are

$$a = \beta(vh + f) + \sigma_\delta,$$

$$b = \beta(1 - h)(1 - vf),$$

and

$$c = -\beta(v + hf) - \sigma_\delta.$$

Similar to the equilibrium structure, when the coefficient $a > 0$, then we take the '+' root, and when $a < 0$, then we take the '−' root.

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