Evolutionary Ecology Research, 2009, **11**: 1141–1168

A genotype-distinguishing model of senescence

Harry D. Saunders

Decision Processes Incorporated, Danville, California, USA

ABSTRACT

Questions: How does one include genotypic variation in density-independent matrix models and what are the implications of such variation in the context of standard theories of senescence?

Mathematical methods: An extension of standard matrix methods and the application of dynamic systems theory.

Key assumptions: Populations consist of distinct genotypes that express different trade-offs between intrinsic lifespan and fecundity. Reproduction is asexual; alternatively, accounting considers only females. Genotypes do not always breed true: breeding fidelity and accuracy are both imperfect; each genotype can generate offspring of differing genotype according to a normal probability distribution.

Conclusions: Allowing genotypic variation in lifespan/fecundity trade-offs generates predictions conforming to standard theory, including population fecundity trends with age, population mortality trends with age, Williams' Hypothesis, the evolution of semelparity and iteroparity, and differential survival for individuals removed from the influence of an extrinsic death rate. The Euler-Lotka equation and expressions deriving from it generalize to the genotype-distinguishing case. In a departure from conventional thinking, the analysis shows that, even in the presence of genotypes expressing an early-age fecundity advantage, populations can evolve that favour genotypes with lower fecundity whose intrinsic lifespan is longer. The analysis also hints that genotype structure is a determinant of equilibrium population size. A new metric that is laboratory-measurable – mean intrinsic lifespan – follows naturally from the methodology. This turns out to be also a metric of semelparity/iteroparity.

Keywords: dynamic systems, Euler-Lotka equation, fecundity trend, genotype variation, intrinsic lifespan, iteroparity, matrix models, mortality trend, semelparity, senescence, survival curve, Williams' Hypothesis.

INTRODUCTION

The classical evolutionary theory of senescence holds that senescence is a virtually inevitable result of the fact that genes that affect survival or fecundity only early in life have a greater selective impact than genes whose effects express only late in life (Charlesworth, 2000). From this theory have arisen several well-established predictions of expected age-related mortality rates (increasing with age) and fecundity rates (decreasing with age) in

Correspondence: Harry D. Saunders, Managing Director, Decision Processes Incorporated, 2308 Saddleback Drive, Danville, CA 94506-3118, USA. e-mail: hsaunders@decisionprocessesinc.com

populations, the influence of extrinsic death rates (Williams, 1957), the role of antagonistic pleiotropic trade-offs (Kirkwood, 1977), and the evolution of semelparity/iteroparity (Cole, 1954).

This article introduces a methodology for incorporating genetic variation in a population and examines the resulting implications for the theory of senescence. While the methodology is general enough to comprehend any genetic variation that affects fecundity and/or mortality rates at any age, the specific implementation reported here assumes that a population consists of distinct genotypes that express different trade-offs between intrinsic lifespan and fecundity (where here 'genotype' includes any heritable epigenetic traits).

The analysis shows such an assumption leads to predictions that conform to classical senescence theory, but that numerous stable genotype structures are possible. It further shows that the presumed fitness advantage associated with early-age fecundity augmentation does not necessarily lead to a population structure that automatically favours genotypes that express this early-age fecundity benefit.

The motivation for this particular description of genetic variation is the observation that individuals in natural populations often appear to have different intrinsic lifespans, as shown in field/laboratory studies reporting different survival rates for individuals removed from the source of an extrinsic death rate (for instance, Reznick *et al.*, 2004). The presumption of a trade-off between lifespan and fecundity draws its motivation from the antagonistic pleiotropy theory of senescence – detrimental effects shortening lifespan may be the price paid for a fecundity advantage.

The key assumption is that individuals in a population express different lifespan/fecundity traits and that at least some of these may be heritable and not wholly phenotypic. There exist, in other words, distinct genotypes. And if antagonistic pleiotropy is at work, one would expect to distinguish among these genotypes particular pairings of intrinsic lifespan and intrinsic fecundity rate reflecting a trade-off between the two. A common presumption is that such a trade-off is attributable to a metabolic budget constraint that limits an organism's expenditure on the combination of fecundity and somatic maintenance and repair [see Charlesworth (1994), who credits Williams (1966) and Gadgil and Bossert (1970) with the initial idea].

The methodology introduced here relies on an extension of standard matrix methods (originating with Leslie, 1945; formalized by Caswell, 2001). The approach of this article is apparently unique in extending matrix methods to include genotypic sub-cohorts. The methodology is expandable to any number of age classes and genotypes. The use of dynamic systems theory allows a compact and readily computable means to accomplish this easily, simply by enlarging the system transition matrix (an authoritative reference for dynamic systems theory is Luenberger, 1979). This approach has the further benefit of allowing extraction of system eigenvectors and eigenvalues that provide a clear picture of genotype structure, and enables compact extensions of the Euler-Lotka equation and its derivatives. The time dynamic of genotype-specific fitness is also readily observable.

Although reproduction is not explicitly sexual, genotypes of offspring can differ from that of the parent according to a probability distribution that comprehends robustly variable assumptions about fidelity and accuracy of genotypic breeding. This permits the exploration of population dynamics consequences across the full domains of all input variables, including extrinsic death rate, overall fecundity rate, pleiotropic decline rate, and fidelity and accuracy of breeding.

With this framework as a starting point, it is easy to formulate a simple matrix model whose main distinguishing feature is the inclusion of alternative genotypic sub-cohorts

within each age cohort. To depict its generality, the mathematical description first elucidates the general case. Then, a specific implementation (for five age cohorts and five genotypic sub-cohorts within each age cohort) illuminates numerically how the model behaves and what it reveals about senescence dynamics.

It is extremely straightforward to implement the approach in a simple spreadsheet. Two examples, one for five age classes and five genotypes and one for ten age classes and ten genotypes, are posted online (evolutionary-ecology.com/data/2500/).

The resulting structure allows us to conceive of a new metric not available within the usual framework, the mean genotype index $\overline{\Gamma}$, which is a summary measure of the genotype structure indicating the mean intrinsic lifespan of the population. This metric is in principle laboratory-measurable. Specifically, if one were to observe the lifespans of individual organisms in captivity (i.e. in the absence of an extrinsic death rate), one could discern the different intrinsic lifespan-specific genotypes and thereby measure $\overline{\Gamma}$, the mean intrinsic lifespan. Combined with observation of genotype-specific fecundity rates, the intrinsic lifetime/fecundity pairing of each genotype could reveal that genotype's metabolic budget choice and, by then applying this methodology, deepen our understanding of the organism's senescence characteristics. One can envision researchers using this metric to characterize the intrinsic lifetime of a particular population of organisms or to compare different organisms' senescence characteristics.

The conclusion is that specifically tracking alternative lifespan/fecundity-specific genotypes may be a fruitful approach for analysing and exploring a number of senescence-related phenomena of interest to biologists.

The structure of the article is as follows: The next section specifies model assumptions. The subsequent section describes the matrix structure of the dynamic systems model. Then, a mathematical summary shows how the resulting equations are natural generalizations of the Euler-Lotka equation and its derivatives. Following that, numerical solutions show how the model generates results conforming to standard theory. Augmenting sensitivity analyses then demonstrate the key role of alternative genotype structures. The final section summarizes the conclusions of the analysis.

MODEL ASSUMPTIONS

The primary assumption is that within a population there exist alternative genotypes that are characterized by a particular combination of fecundity rate and intrinsic lifespan, and that the longer the intrinsic lifespan the lower will be the fecundity rate permitted by that genotype's metabolic budget. Reproduction is asexual; an alternative assumption is that only females are accounted for.

The following specific assumptions define the framework:

1. *Maximum lifespan*: This population of organisms lives for only A reproductive cycles at maximum ($a = 1, \ldots, A$, where a is age, i.e. number of reproductive cycles).
2. *Genotype lifespan*: The population contains G different genotypes ('genotype index' $g = 1, \ldots, G$). Genotype g is characterized by genetic or heritable epigenetic traits that cause the organism to die from intrinsic causes after g reproductive cycles.
3. *Extrinsic death rate*: An extrinsic death rate d limits survival of individuals of all genotypes to the fraction $1 - d$ each reproductive cycle (except genotype 1 individuals, who all die after one reproductive cycle).

4. *Antagonistic pleiotropy*: Fecundity rates differ among the genotypes. To allow for pleiotropic effects, fecundity declines with genotype index – a pleiotropic fecundity decline parameter π, if not set to zero, depicts a trade-off between longer intrinsic lifespan and fecundity according to the following equation:

$$b = b_0 e^{-\pi(g-1)} \tag{1}$$

where b_0 is a base fecundity level. This ensures that an individual with a lower genotype index (a shorter intrinsic lifespan) will have a higher fecundity rate – each genotype selects a different lifetime maintenance investment program.

5. *Breeding fidelity and accuracy*: The genotypes do not always breed true. Instead, each genotype can generate offspring of differing genotype according to the following one-sided normal probability distribution:

$$\begin{aligned} p(g_P, g_O) &= Pr\ \{\textit{parent of genotype } g_P \textit{ produces offspring of genotype } g_O\} \\ &= \phi k_{g_P}, \quad \textit{if } g_O = g_P \\ &= (1-\phi)\, k_{g_P} e^{-\frac{(g_P - g_O)^2}{\sigma_\alpha^2}}, \quad \textit{if } g_O < g_P \end{aligned} \tag{2}$$

where ϕ is a factor reflecting inherent 'fidelity' of breeding – the degree to which a genotype breeds true to its type – and σ_α is a parameter reflecting breeding dispersion across other genotypes, or its 'accuracy' of breeding. The factor k_{g_P} is chosen so that

$$\sum_{g=1}^{g_P} p(g_P, g_O) = 1.$$

This depiction of inheritance enables consideration of heritable mutations generating maintenance investment program choices in offspring that differ from those of the parent.

Note that genotypes can only generate offspring that have a genotype index less than or equal to their own. This prevents individuals from generating offspring whose intrinsic lifetime is longer than their own. The presumption is that breeding infidelity arises from deleterious heritable genetic mutations that are more common than favourable ones so that mutations leading to longer lifespan are unlikely to occur. However, since it is possible to argue that a mutation leading to a longer lifespan accompanied by a reduction in fecundity could be considered deleterious, a later sensitivity analysis shows the consequences of relaxing this assumption.

MATRIX FORMULATION

It is possible to compactly formulate the above framework as a linear dynamic system. Let x_{ag} be the population of individuals of age a and genotype index g at time t. Then assemble these into vectors categorized by age, so that for age a individuals the vector is:

$$\mathbf{x}_a = \begin{bmatrix} x_{a1} \\ x_{a2} \\ \vdots \\ x_{aG} \end{bmatrix} \tag{3}$$

Now stack these into a state vector partitioned by age cohort:

$$\mathbf{x} = \begin{bmatrix} \mathbf{x}_1 \\ \mathbf{x}_2 \\ \vdots \\ \mathbf{x}_A \end{bmatrix} \tag{4}$$

Then, there is a linear dynamic system that satisfies the following equation:

$$\mathbf{x}_{t+1} = \mathbf{A}\mathbf{x}_t \tag{5}$$

where $\mathbf{A}$ is a Leslie-type transition matrix partitioned by age cohort:

$$\mathbf{A} = \begin{bmatrix} \mathbf{B}_1 & \mathbf{B}_2 & \mathbf{B}_3 & \cdots & \mathbf{B}_A \\ \mathbf{S}_{21} & \mathbf{0} & \mathbf{0} & \cdots & \mathbf{0} \\ \mathbf{0} & \mathbf{S}_{32} & \mathbf{0} & \vdots & \mathbf{0} \\ \vdots & \mathbf{0} & \ddots & \mathbf{0} & \vdots \\ \mathbf{0} & \cdots & \mathbf{0} & \mathbf{S}_{A,A-1} & \mathbf{0} \end{bmatrix} \tag{6}$$

This transition matrix $\mathbf{A}$ contains sub-matrices $\mathbf{B}_a$ (associated with reproduction) and sub-matrices $\mathbf{S}_{a,a-1}$ (associated with survival). Consider each of these in turn.

The *reproduction sub-matrices* deliver offspring from older age cohorts into the first age cohort. Each reproduction sub-matrix accounts for offspring according to genotype. The row and column numbers of the reproduction sub-matrix correspond to genotype indices. For example, the reproduction sub-matrix $\mathbf{B}_1$ is:

$$\mathbf{B}_1 = \begin{bmatrix} b_0 & b_0 e^{-\pi}(1-\phi)k_2 & b_0 e^{-2\pi}(1-\phi)k_3 e^{-\frac{4}{\sigma_a^2}} & \cdots & b_0 e^{-(G-1)\pi}(1-\phi)k_G e^{-\frac{(G-1)^2}{\sigma_a^2}} \\ 0 & b_0 e^{-\pi}\phi k_2 & b_0 e^{-2\pi}(1-\phi)k_3 e^{-\frac{1}{\sigma_a^2}} & \cdots & b_0 e^{-(G-1)\pi}(1-\phi)k_G e^{-\frac{(G-2)^2}{\sigma_a^2}} \\ 0 & 0 & b_0 e^{-2\pi}\phi k_3 & \cdots & b_0 e^{-(G-1)\pi}(1-\phi)k_G e^{-\frac{(G-3)^2}{\sigma_a^2}} \\ \vdots & \vdots & 0 & \ddots & \vdots \\ 0 & 0 & \cdots & 0 & b_0 e^{-(G-1)\pi}\phi k_G \end{bmatrix} \tag{7}$$

And the reproduction sub-matrix $\mathbf{B}_A$ is:

$$\mathbf{B}_A = \begin{bmatrix} 0 & 0 & \cdots & 0 & b_0 e^{-(G-1)\pi}(1-\phi)k_G e^{-\frac{(G-1)^2}{\sigma_a^2}} \\ 0 & 0 & \cdots & 0 & b_0 e^{-(G-1)\pi}(1-\phi)k_G e^{-\frac{(G-2)^2}{\sigma_a^2}} \\ 0 & 0 & \cdots & 0 & b_0 e^{-(G-1)\pi}(1-\phi)k_G e^{-\frac{(G-3)^2}{\sigma_a^2}} \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ 0 & 0 & \cdots & 0 & b_0 e^{-(G-1)\pi}\phi k_G \end{bmatrix} \tag{8}$$

In general, reproduction sub-matrix $\mathbf{B}_a$ has $a-1$ leading columns of zeroes. The first row of each reproduction sub-matrix delivers offspring to genotype 1, the second to genotype 2,

and so forth. So, for instance, the first element of the first row of the sub-matrix in (7) delivers to genotype 1 $b_0 x_{11}$ offspring from age 1 individuals of genotype 1, that is, delivers offspring to genotype 1 at the rate b_0. But this genotype also receives offspring from other genotypes. For example, from age 1 individuals of genotype 2, it receives a fraction $(1 - \phi)k_2$ of their total births; the factor $e^{-\pi}$ reduces total births of this genotype from the rate b_0 to reflect a pleiotropic reduction in fecundity associated with genotype 2.

Similar definitions apply to the other elements of the reproduction sub-matrices $\mathbf{B}_a$, each likewise reflecting the reproductive rates obtained from equations (1) and (2).

Each *survival sub-matrix* contains $A - a$ elements along the lower extreme of its diagonal. For example, the survival sub-matrix $\mathbf{S}_{32}$ is:

$$\mathbf{S}_{32} = \begin{bmatrix} 0 & 0 & 0 & \dots & 0 \\ 0 & 0 & 0 & \dots & 0 \\ 0 & 0 & 1-d & 0 & \vdots \\ \vdots & \vdots & 0 & \ddots & 0 \\ 0 & 0 & \dots & 0 & 1-d \end{bmatrix} \tag{9}$$

In this case, the sub-matrix depicts survival of age 2 individuals to age 3. The two leading columns are zeroes to reflect the fact that no age 2 individuals of genotype 1 or 2 advance to age 3 (their genotype causes them to die from lethal intrinsic causes either at age 1 or age 2). Individuals above genotype 2 have the probability $1 - d$ of surviving to age 3. Survival sub-matrix $\mathbf{S}_{54}$ has four leading columns that are zeros. In general, survival sub-matrix $\mathbf{S}_{a,a-1}$ has $a - 1$ leading columns of zeroes.

Then, given values for b_0, π, ϕ, σ_a, and d, and an initial system state $\mathbf{x}_0$, one can run the system (5) for as many iterations as desired to deliver the system state $\mathbf{x}_t$. This system state will converge to its principal (or dominant) eigenstate $\mathbf{e}$ with population growth rate r (where $1 + r$ is the principal eigenvalue). This eigenstate is independent of the initial system state, so long as this state contains any projection onto the principal eigenvector. By choosing d such that $r = 0$, the system will deliver a stable eigenstate in zero-growth equilibrium.

RELATION TO SINGLE GENOTYPE FORMULATIONS

Classical life-history theory applied to single genotype populations relies heavily on the Euler-Lotka equation and expressions derived from it (see Charlesworth, 1994; Partridge and Mangel, 1999). Table 1 summarizes how these generalize to the genotype-distinguishing case, assuming for the single genotype case a Leslie matrix whose first row is the vector $\mathbf{b}^T$ containing fecundities b_a, $a = 1, \dots, A$ and where s_a, $a = 1, \dots, A$ are survival rates on the sub-diagonal. Appendix A contains a further discussion of these expressions. As discussed there, a key result is that in the genotype-distinguishing case, each genotype grows at the intrinsic growth rate of the population in steady state, so that $\lambda_g = \lambda$ for all g.

As also discussed there, the $\mathbf{b}^T\mathbf{e} = \lambda$ and $\mathbf{B}_g^T\mathbf{e}_g = \lambda$ forms of the Euler-Lotka equation provide intuitive descriptions of the dynamic – the vectors $\mathbf{b}^T$ and $\mathbf{B}_g^T$ contain the age- and genotype-specific fecundities, while the vectors $\mathbf{e}$ and $\mathbf{e}_g$ contain the steady-state age- and genotype-specific survival dynamics, since these eigenvectors depict the steady-state population fractions by age and genotype. Even though in the genotype-distinguishing

Table 1. Comparison of classical single-genotype expressions to genotype-distinguishing-equivalent expressions

	Algebraic expressions	**Dynamic systems formulation**
Single-genotype Leslie model	Euler-Lotka Equation: $\sum_{a=1}^{A} l_a m_a \lambda^{-a} = 1$	Euler-Lotka equation: $\mathbf{b}^T\mathbf{e} = \lambda$ (where $\mathbf{e}$ is the principal eigenvector)
	$\frac{\partial r}{\partial s_a} = \frac{\sum_{x=a}^{A} l_x m_x \lambda^{-x}}{s_a \sum_{a=1}^{A} a l_a m_a \lambda^{-a}}$	$\frac{\partial r}{\partial s_a} = \frac{f_a e_{a-1}}{\mathbf{f}^T\mathbf{e}}$
	$\frac{\partial r}{\partial m_a} = \frac{l_a \lambda^{-a}}{\sum_{a=1}^{A} a l_a m_a \lambda^{-a}}$ (where $\lambda = e^r$ is the principal eigenvalue, and $l_a = \prod_{i=2}^{a} s_i$ and $l_1 = 1$)	$\frac{\partial r}{\partial m_a} = \frac{f_1 e_a}{\mathbf{f}^T\mathbf{e}}$ (where $\mathbf{f}^T$ is the left eigenvector corresponding to $\mathbf{e}$)
Genotype-distinguishing model	No analytically concise equivalent	Euler-Lotka-equivalent equations: $\mathbf{B}_g^T\mathbf{e}_g = \lambda_g = \lambda,\ g = 1, \ldots, G$ (where $\mathbf{B}_g^T$ is the gth row of $\mathbf{A}$ and $\mathbf{e}_g$ is $\mathbf{e}$ normalized such that e_{1g} is unity)
		$\frac{\partial r}{\partial s_{ag}} = \frac{f_{ag} e_{a-1,g}}{\mathbf{f}^T\mathbf{e}}$
		$\frac{\partial r}{\partial m_{ag_P g_O}} = \frac{f_{1g_P g_O} e_{a\ g_P g_O}}{\mathbf{f}^T\mathbf{e}}$
		$\frac{\partial r}{\partial m_{g_P g_O}} = \frac{f_{1g_P g_O} \sum_{a=1}^{A} e_{a\ g_P g_O}}{\mathbf{f}^T\mathbf{e}}$ (where g_P and g_O are the genotype numbers of parent and offspring, respectively)

case fecundity rates affect survival dynamics, fecundity and survival dynamics become analytically separable. Positive changes in either augment the principal eigenvalue, $\lambda = 1 + r$, and thus the steady-state growth rate. (Note that some authors use a continuous-time version of λ, where $\lambda = e^r$.)

Accordingly, to the extent r is considered a measure of fitness, positive changes to fecundity and survival increase fitness. The derivatives indicate the change to the intrinsic growth rate associated with changes to the individual elements of the respective transition matrices. While parameter-explicit analytic forms of these derivatives are difficult to obtain for large $G = A = N$ (since analytic forms for the eigenvectors are difficult to obtain), quantifying them is easy, as shown in the spreadsheets (evolutionary-ecology.com/data/2500/) for $G = A = 5$ and $G = A = 10$.

MEAN INTRINSIC LIFESPAN

The object of interest is senescence. A new measure that emerges by including genotypes is *mean intrinsic lifespan* (mean genotype index):

$$\overline{\Gamma} = \frac{\sum_{g=1}^{G} g \sum_{a=1}^{A} e_{ag}}{\sum_{a=1}^{A} \sum_{g=1}^{G} e_{ag}} \tag{10}$$

where g is the genotype index and e_{ag} is the element (a,g) of the system eigenvector $\mathbf{e}$. The function $\overline{\Gamma}$ is the steady-state mean genotype index (weighted by genotype population size) in equilibrium. This function measures the population's mean age to death from intrinsic causes that would occur in the absence of an extrinsic death rate. It is, however, a function of in-the-wild extrinsic death rate as well as base fecundity, fecundity decline, and breeding fidelity and accuracy, since all these input parameters determine the eigenstate and thus the distribution's shape.

GENOTYPE FITNESS

If one adopts the view that intrinsic growth rate r is an appropriate measure of fitness, it is straightforward to develop equations that measure this when the population is not in steady state.

Let $N_g(t)$ be the number of individuals of genotype g. Then, the fitness of genotype g in reproductive period t is:

$$\lambda_g(t) = \frac{N_g(t+1)}{N_g(t)} \tag{11}$$

(Of course, if the population has achieved steady state, $\lambda_g(t) = \lambda_g$ for all t.) However, the contribution to the fitness of the whole population from genotype g will depend on its population size relative to the whole population. Accordingly, one can define genotype g's contribution to total population fitness as:

$$\hat{\lambda}_g(t) = \frac{N_g(t+1)}{N_g(t)} \left(\sum_{a=1}^{A} x_{ag}(t) \Big/ \sum_{a=1}^{A} \sum_{g=1}^{G} x_{ag}(t) \right) \tag{12}$$

where $x_{ag}(t)$ is the population of age a individuals of genotype g at time t and the expression in brackets is the fraction of individuals of genotype g in the whole population.

The relationship shown in Table 1 allows us to express this differently, but equivalently. Noting that $\mathbf{B}_g^T\mathbf{e}_g = \lambda_g = \lambda$, $g = 1, \ldots, G$ in steady state, we can write the fitness contribution of genotype g in reproductive period t as:

$$\hat{\lambda}_g(t) = \mathbf{B}_g^T\mathbf{x}_g \left(\sum_{a=1}^{A} x_{ag}(t) \Big/ \sum_{a=1}^{A}\sum_{g=1}^{G} x_{ag}(t) \right) \tag{13}$$

where, as with $\mathbf{e}_g$, $\mathbf{x}_g$ is normalized such that $x_{1g} = 1$.

RESULTS – BASE CASE

The results of this section and the next one, which numerically illustrate the behaviour of the system (5), rely on the parameter values shown in Table 2, together with the assumption that $A = G = 5$. Note that to provide a direct comparison among cases, the extrinsic death rate d must be adjusted in each case to result in zero-growth equilibrium (any growth rate could have been chosen). One can then consider each case as representing a separate population with its own parameter values, each in equilibrium. Each generates a different eigenvector. Summing elements of the resulting eigenvector by either age or genotype allows one to predict the equilibrium population for each age cohort or genotype.

Fecundity rate trends

As shown in Fig. 1, the Base Case input values generate a declining trend of per capita fecundity rate with age, for the entire population. This result is robust to a wide range of parameter values. (Note that a base fecundity rate of 65% means that, on average, each female individual of genotype 1 produces 0.65 female offspring per reproductive cycle.)

Declining reproduction with age is not necessarily what one would automatically assume *a priori* with a structure like this. Each age class contains a mixture of genotypes, each with different fecundity rates. Categorized by genotype, one would expect to see declining

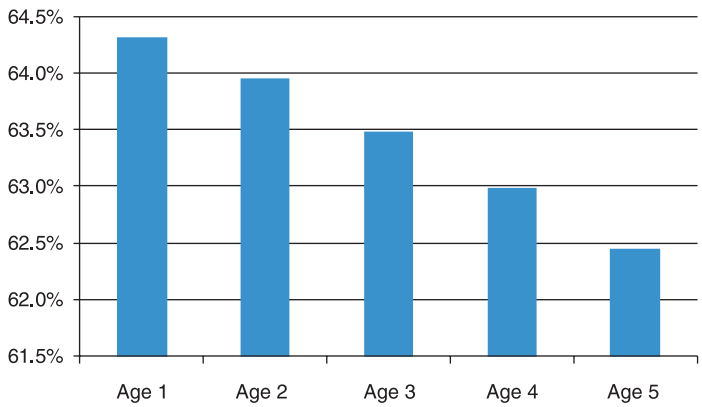

Fig. 1. Fecundity rate trend with age (one-period fecundity).

fecundity owing to the parameter π, which measures the strength of a pleiotropic decline in fecundity as the genotype index increases. But the fecundity trend with age is less obvious. Nonetheless, categorized by age class, this result still holds. This is simply because genotypes of larger genotype index, whose fecundity is lower, will be a larger fraction of older age cohorts' populations than of younger ones.

Incremental fecundity rates (the first derivative of fecundity rate with age) are therefore negative, as expected. However, the change in incremental fecundity (the second derivative of fecundity rate with age) can in certain circumstances be positive (discussed later).

Mortality rate trends

Mortality also follows the classical trend of increasing mortality with age, as shown in Fig. 2. At each age, individuals experience a probability d of extrinsic death, which cumulates over time, so mortality rate increases with age. Incremental mortality by age is positive, in accordance with classical theory. Again, however, there are circumstances where the second derivative of mortality rate changes sign.

Fidelity and accuracy of breeding

The Base Case parameters result in the distribution by genotype in the eigenstate shown in Fig. 3 (note that the distribution of genotypes will not, in general, be observed).

Genotype 2 is favoured in terms of its contribution to population size for this set of parameters, but all genotypes play a role in this stable population. Of course, different parameters generate much different distributions. Mean intrinsic lifespan in this case is $\overline{\Gamma} = 2.332$, indicating that the average survival time in this steady-state population is 2.332 reproductive cycles in the absence of an extrinsic death rate. Recall from the relation $\mathbf{B}_g^T \mathbf{e}_g = \lambda_g = \lambda$, $g = 1, \ldots, G$ that all genotypes grow at the same rate (in this case at zero).

Fidelity and accuracy of breeding play a central role in determining this distribution. With ϕ and σ_α set as in Table 1 (Base Case) – that is, with offspring genotypes being identical to parent genotypes 70% of the time and dispersion of remaining offspring being fairly tightly confined to nearby genotypes – there is nonetheless a relatively large spread of

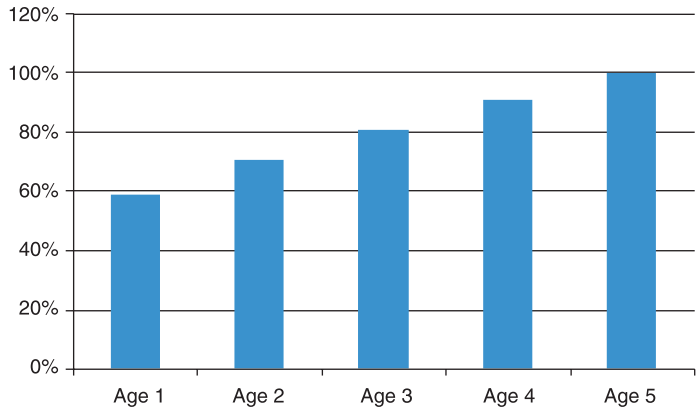

Fig. 2. Mortality rate trend with age.

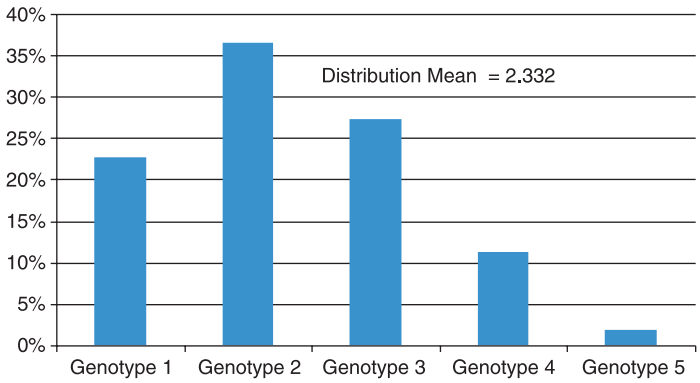

Fig. 3. Distribution of population across genotypes.

genotypes realized in the eigenstate of Fig. 3. Later sensitivity analysis (in the Results – Sensitivities section) shows how these parameters influence the distribution.

Importantly, even though genotype 1 has a fecundity advantage over genotype 2, genotype 2 is favoured in terms of population size. This suggests that selection pressures favouring early fecundity-enhancing genes can be offset by selection pressures acting against their survival (genotype 1 survives for only one reproductive cycle).

This much is consistent with standard theory. However, since $\overline{\Gamma}$ can take larger values than shown here even while the intrinsic lifespan of each genotype is fixed, this cautions against a broad conclusion that selection pressures must always favour early-acting genes that enhance fecundity. In fact, as shown later, the larger the early fecundity advantage π, the larger is $\overline{\Gamma}$ for a fixed population growth rate. This points to the conclusion that the subtleties of genotype structure need always to be considered: a new mutation favouring early-age fecundity can readily shift the mean lifespan of a population towards longer-lived, lower-fecundity genotypes.

Survival curves

This approach enables the creation of survival curves. The experiments of Reznick *et al.* (2004) on Trinidadian guppies, *Poecilia reticulata*, rely on observation of survival curves. In their experiments, they took second-generation offspring of wild-caught females subject to different predation regimes and raised them in an environment devoid of an extrinsic death rate. They observed that different populations exhibited different survival rates.

One can readily emulate survival curves by running the model to zero-growth equilibrium, extracting the age 1 individuals from the eigenvector (offspring), and allowing them to survive as long as their genotype permits with $d = 0$. More specifically, for a particular parameter setting, the system (5) is run to zero-growth equilibrium and a new system vector is created that looks like (4) except that the $\mathbf{x}_1$ elements are the corresponding elements of the eigenvector and all other elements are zero. Then, this new vector is run through the system (5) with certain elements of $\mathbf{A}$ changed: all fecundity rates are set to zero (no need to track offspring), and the extrinsic death rate d is set to zero. The total surviving population is calculated at each reproductive cycle by summing all the elements of the system state.

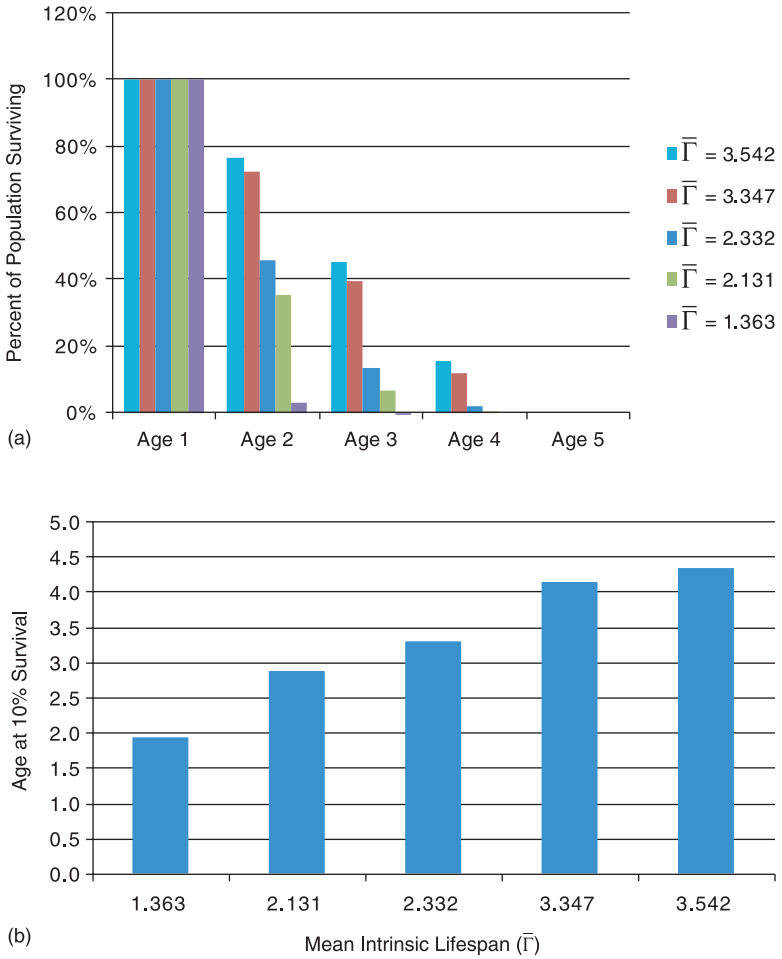

Fig. 4. (a) Survival curves for different values of ($\overline{\Gamma}$). (b) Age at which 10% survival is reached (ages are interpolated).

Figure 4 shows the result for five different input parameter settings leading to five different values of $\overline{\Gamma}$. Not surprisingly, a population with longer mean intrinsic lifespan will exhibit a slower decline in survival. The implication is that populations with different genotype distributions will exhibit different survival characteristics, and they can be calculated.

Methodology-wise, the significance is that this approach allows exploration of how genotype structure influences survival curves.

RESULTS – SENSITIVITIES

This section describes the sensitivities summarized in Table 2.

Table 2. Parameter value settings for Base Case and sensitivity analyses

	Base Case	Sensitivities
b_0	65%	35% to 100%
π	1%	0% to 15%
ϕ	70%	40% to 95%
σ_α	2.00	0.10 to 10.00
d	36% (for zero-growth equilibrium)	adjusted to achieve zero-growth equilibrium

Population-level fecundity rates and $\overline{\Gamma}$

The mean intrinsic lifespan parameter $\overline{\Gamma}$ is a declining linear function of the base fecundity rate b_0. That is, the lower the fecundity rate, the longer the mean intrinsic lifespan. Although this may appear counterintuitive, it becomes comprehensible by recalling that each $\overline{\Gamma}$, b_0 pairing satisfies zero-growth equilibrium conditions. That is, the lower the fecundity rate, the lower will be the extrinsic death rate required to produce zero-growth equilibrium of the population. This result is then explicable as a consequence of Williams' Hypothesis (Williams, 1957), which states that a lower extrinsic death rate is associated with delayed senescence.

While b_0 affects all genotypes uniformly, the pleiotropic fecundity decline π affects them differentially. That is, since the fecundity rate is governed by the equation $b = b_0 e^{-\pi(g-1)}$, the higher the value of π, the lower will be the fecundity rate of any particular genotype g. This reflects a trade-off between fecundity and lifespan, and the higher the value of π, the more onerous the trade-off. A higher value of π represents a more restrictive metabolic budget constraint for any given value of b_0.

While small increases in π from their Base Case level (1%) reduce $\overline{\Gamma}$, Fig. 5 shows that mean intrinsic lifespan increases for high values of the fecundity decline. Again, recalling that in zero-growth equilibrium lower fecundity corresponds to a lower extrinsic death rate, the result is an understandable consequence of Williams' Hypothesis.

This illustrates the point made in connection with Fig. 3 that early-age (i.e. low genotype index) fecundity augmentation does not necessarily translate into a selection advantage for such genotypes.

Figure 1 showed that this genotype-distinguishing approach leads to a declining fecundity rate with age. Figure 6 shows that the per capita fecundity rate decrease with age is larger with lower $\overline{\Gamma}$. As it happens, the actual per capita fecundity rate versus age is higher with lower $\overline{\Gamma}$, but the rate of decline is also higher. This is because a population consisting largely of genotypes of lower index bears a disproportionate pleiotropic burden – genotypes of lower index have a higher fecundity rate as a result of equation (1), but also are removed from the population more quickly, thus rapidly reducing the population's fecundity rate.

A somewhat curious result appears for $\overline{\Gamma} = 1.363$. Incremental fecundity rate becomes more negative for age 2 individuals advancing to age 3, but age 3 individuals advancing to age 4 show a reduction in this decline rate. This is because, for this setting of parameters, population by genotype in the eigenvector shows a strong decline between genotypes 2 and 3 and a smaller decline between genotypes 3 and 4.

Between age class 2 and age class 3, therefore, a large proportion of the population – the more fecund genotype 2 individuals – exits the population. Between age class 3 and 4, the more fecund genotype 3 individuals exiting the population represent a smaller proportion

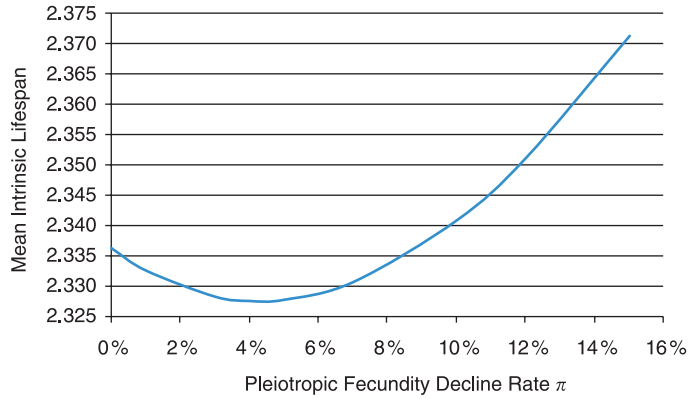

Fig. 5. Effect on mean intrinsic lifespan of increasing the pleiotropic cost.

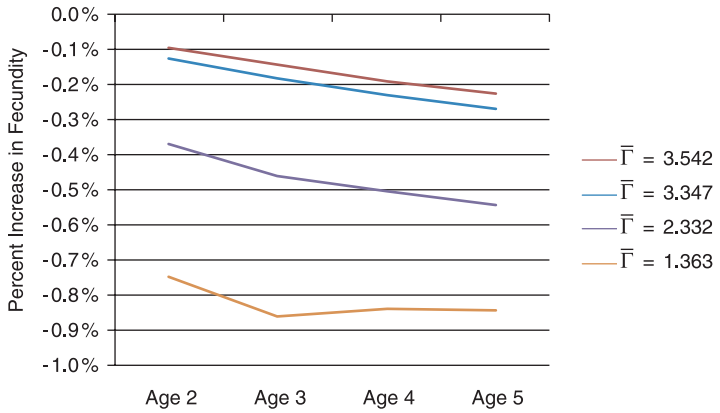

Fig. 6. Incremental fecundity by age as a function of mean intrinsic lifespan.

of the population. This results in a smaller decline in population fecundity between age 3 and age 4.

Such subtleties suggest that accounting for genotypes, not just age classes, carries with it the promise of deeper insights into trends in the fecundity rate of a given population.

Mortality rates as a function of $\overline{\Gamma}$

Incremental mortality trends show similar characteristics, where mortality is defined as $M(t) = \frac{-1}{S(t)} \frac{dS}{dt}$, where $S(t)$ is the fraction of individuals who were alive at time 0 who survive to age t.

In particular, the higher $\overline{\Gamma}$ is, the greater the slope of the mortality curve. In other words, the longer the mean intrinsic lifespan, the more rapidly mortality increases with age. This is because age 1 mortality for a high $\overline{\Gamma}$ population is much lower than for a low $\overline{\Gamma}$ population, but must eventually reach 100%. Said differently, a population characterized largely by inherently longer-lived individuals will be more subject to extrinsic than intrinsic death at

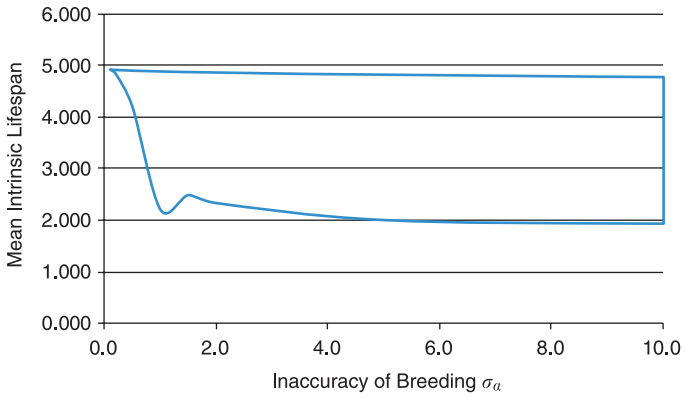

Fig. 7. Mean intrinsic lifespan as a function of breeding accuracy (with $\phi = 0.7$).

early ages but will bear the rapidly increasing cumulative effects of extrinsic death, combined with increasing intrinsic death, as age advances. In contrast, a population characterized largely by inherently shorter-lived individuals begins with a high intrinsic mortality at age 1 and has less 'distance' to go before experiencing 100% mortality from both intrinsic and extrinsic causes.

Similar to fecundity rates, a lower $\overline{\Gamma}$ reveals incremental mortality rates that sometimes shift in the sign of their derivative. Comparable arguments show that the strongly declining population by genotype is the cause.

As with fecundity rates, this suggests that accounting for genotypes, not just age classes, carries with it the promise of deeper insights into population mortality rate trends.

$\overline{\Gamma}$ as a function of breeding fidelity and accuracy

Increasing the breeding fidelity factor ϕ increases mean intrinsic lifespan. Additionally, the higher the breeding accuracy implied by σ_α, the higher the absolute level of $\overline{\Gamma}$. This suggests that the higher the breeding accuracy and fidelity, the higher will be the mean intrinsic lifespan.

Figure 7 shows that the greater the accuracy of breeding (the lower is σ_α), the greater is the mean intrinsic lifespan. Comparable figures show that increased breeding fidelity likewise increases mean intrinsic lifespan.

This result suggests that the lower the mutation rate affecting genotypic choice of maintenance investment program, the greater will be the propensity for an organism to contain long-lived individuals. Together with results shown previously for b_0 and π, this suggests that organisms exhibiting low overall fecundity rates and/or high pleiotropic decline rates, if they are also organisms subject to infrequent mutations affecting their maintenance investment program choices, will be organisms characterized by long intrinsic lifespans.

Total population size

A potentially interesting result follows from using a genotype-distinguishing approach. That is, there appears to be a strong connection between $\overline{\Gamma}$ and total population size

achievable by an organism in zero-growth equilibrium. By way of example, if the Base Case value of b_0 is reduced from 65% to 50%, the value of $\overline{\Gamma}$ increases from 2.332 to 2.945 but the zero-growth equilibrium population size is decreased 74%. Changes to other inputs that likewise increase $\overline{\Gamma}$ (with the notable exception of fecundity decline rate π) all show this phenomenon. No explanation for this result is readily apparent.

Nonetheless, the strong indication of this analysis is that genotype structure is a determinant of zero-growth equilibrium population size.

Semelparity and iteroparity

$\overline{\Gamma}$ also has implications for semelparity and iteroparity. A population characterized by $\overline{\Gamma} = 1$ will contain only individuals of genotype 1, that is, individuals who will die from intrinsic causes at age 1, irrespective of the extrinsic death rate. But a low value of $\overline{\Gamma}$ must be offset by a high base fecundity rate for zero-growth equilibrium to be achieved. Thus, such a population will in equilibrium exhibit high fecundity but will live for only one reproductive cycle – both hallmarks of a semelparous population. Similarly, a population having large $\overline{\Gamma}$ will exhibit a longer intrinsic lifetime with a propensity for repeated reproduction cycles (the actual realization of which will depend on the extrinsic death rate) but with lower fecundity – in other words, will exhibit a propensity towards iteroparity.

The implication is that the determinants of semelparity and iteroparity for populations are the same as the determinants of $\overline{\Gamma}$, namely the base fecundity rate b_0, the pleiotropic decline rate π, and fidelity and accuracy of breeding, ϕ and σ_a.

Specifically, the lower the base fecundity rate, the higher the propensity towards iteroparity; the higher the pleiotropic decline rate (the more onerous the fecundity/lifespan budget constraint), the higher the propensity towards iteroparity; and the higher the fidelity and accuracy of breeding, the higher the propensity towards iteroparity.

A question that naturally arises is when would iteroparous genotypes be able to invade and displace a semelparous population?

If one assumes a semelparous population in equilibrium and retains the assumption that parent genotypes cannot produce offspring with a longer intrinsic lifespan than their own, the latter assumption means iteroparous genotypes can never invade a semelparous population. However, if one relaxes this latter assumption and permits individuals a distribution of offspring applied symmetrically upward as well as downward, a semelparous system state quickly converges to an iteroparous one. This says invasion of iteroparity into a purely semelparous population is entirely dependent on whether genotypes can in reality mutate to generate in offspring a longer lifespan/lower fecundity maintenance investment program as against a shorter lifespan/higher fecundity one.

However, as seen in previously described results, iteroparous populations can exist having only one-sided (downward) genotype offspring distributions. Can more (or less) iteroparous genotypes invade such a population?

Given an iteroparous population, a positive perturbation to any genotype-specific element of the transition matrix (fecundity rate or survival rate) will increase that genotype's intrinsic growth rate relative to genotypes of higher index (it will increase the intrinsic growth rate of lower genotypes since each genotype can generate offspring of lower genotype index – and of higher genotype index if upward symmetry is allowed). In the short run, this causes the growth rate of these genotypes to increase in relative terms, the degree to which depends on the magnitudes of π, ϕ, and σ_a, and also on the genotype index of the

transition matrix element in question. In the long run, intrinsic growth rate for all genotypes will revert to the intrinsic growth rate of the population, since $\mathbf{B}_g^T\mathbf{e}_g = \lambda$, $g = 1, \ldots, G$, but the value of $\overline{\Gamma}$ will be adjusted. Generally speaking, a positive change to a transition matrix element will have a greater likelihood of increasing $\overline{\Gamma}$ to the extent the genotype index of the perturbed element is greater than $\overline{\Gamma}$, but this depends also on the magnitudes of π, ϕ, and σ_a.

Accordingly, even knowing that the determinants of $\overline{\Gamma}$ are those that determine a population's propensity towards semelparity or iteroparity is insufficient to provide useful insight into how these transition matrix-specific determinants will change $\overline{\Gamma}$. Owing to the subtleties of the aforementioned interactions, and notwithstanding the ability to calculate the derivatives as in Table 1, the derivatives $\partial\overline{\Gamma}/\partial\pi$, $\partial\overline{\Gamma}/\partial\phi$, and $\partial\overline{\Gamma}/\partial\sigma_a$ permit no obvious analytic solution, let alone the derivatives $\partial\overline{\Gamma}/\partial a_{ij}$, where a_{ij} is the i,j th element of the transition matrix.

Once again, this points to the benefits of exploiting numerical solutions. The spreadsheets (evolutionary-ecology.com/data/2500/) allow the user to alter b_0, π, ϕ, and σ_a, perturb transition matrix elements a_{ij}, and to toggle between one-sided and two-sided symmetry of breeding and observe the effects on $\overline{\Gamma}$, now interpretable as a metric of semelparity/iteroparity.

DISCUSSION AND CONCLUSIONS

It appears the hypothesis that populations consist of different genotypes expressing differing and heritable lifespan/fecundity trade-offs is one capable of readily delivering a number of results consistent with standard evolutionary theories of senescence. While this is not a particularly astonishing finding, the methodology introduced in this article may provide a convenient means for analysing and exploring the genotypic structure of observed populations, if such structure indeed exists.

The particular approach to considering genotypic variation reported here, by extending standard matrix methods and applying dynamic systems theory, leads to compact extensions of the Euler-Lotka equation and its derivatives, facilitating interpretation of numerical solutions in a way familiar to researchers.

But consideration of genotypic variation in populations allows us to conceive of a new metric, mean intrinsic lifespan $\overline{\Gamma}$, not available in the usual framework where only single genotypes are considered, that is in principle laboratory-measurable and could provide a means to compare different organisms, or different populations of a single organism subject to differing determinants of $\overline{\Gamma}$.

The results reported in connection with fecundity rate trends suggest that declining fecundity rate with age may be a consequence of declining fecundity by genotype; declining fecundity by genotype may embody a deeper explanation of this observed phenomenon.

As with fecundity rates, the results relating to mortality rate trends suggest that accounting for genotypes, not just age classes, carries with it the promise of deeper insights into population mortality rate trends and that such genotypes may represent an underlying cause of observed mortality trends.

The results reported in connection with breeding fidelity and accuracy point to the conclusion that the subtleties of genotype structure can lead to counterintuitive results: a new mutation favouring early-age fecundity can readily shift the mean lifespan of a

population towards longer-lived, lower-fecundity genotypes. Said differently, if one compares two identically growing populations, one with an early age fecundity advantage versus the other, one expects to find that the early age fecundity-advantaged population has a longer mean lifespan – that is, consists largely of genotypes that suffer a relative fecundity disadvantage, in contrast to conventional presumptions.

The results further indicate that the determinants of semelparity and iteroparity for populations are the same as the determinants of $\overline{\Gamma}$, namely the base fecundity rate b_0, the pleiotropic decline rate π, and fidelity and accuracy of breeding, ϕ and σ_a. This leads naturally to the supposition that explicitly considering the genotype structure may prove fruitful in understanding the semelparity/iteroparity propensity of a given population.

Differential survival among individuals, observed in natural populations, appears as a natural consequence of considering a genotype structure that distinguishes different fecundity/lifespan trade-offs; this means the approach reported here allows exploration of how genotype structure influences survival curves.

In summary, the analysis suggests that consideration of alternative genotype structures can reveal subtleties not otherwise available relating to mortality and fecundity rate trends, the selection dynamics associated with early-expressing fecundity advantages, the propensity towards semelparity or iteroparity, and differential survival rates within populations. Presuming the existence of alternative genotype structures, and that they are measured (e.g. by observing fecundity rates and survival curves in captivity), applying this methodology may make available new insights regarding the role of extrinsic hazards in shaping genotype structures, regarding an organism's fidelity and accuracy of breeding, and regarding the somatic maintenance and fecundity choices underlying an organism's tendency towards semelparity or iteroparity.

The results also hint that genotype structure is a determinant of equilibrium population size.

ACKNOWLEDGEMENTS

I acknowledge the generous and time-consuming support and encouragement of David Reznick. I also am deeply indebted to Marc Mangel for substantive contributions to both the content and presentation of these ideas and particularly for prodding me to extend the Euler-Lotka equation. I thank Troy Day for taking the time to review various manuscripts and offer insightful advice. Andres Lopez-Sepulcre provided several very key suggestions and Peter Abrams made a number of useful observations on an early version. I further thank Daniel Johnson for early encouragement and Joan Roughgarden for directing me to the right literature.

REFERENCES

Caswell, H. 2001. *Matrix Population Models*. Sunderland, MA: Sinauer Associates, Inc.

Charlesworth, B. 1994. *Evolution in Age-Structured Populations*. Cambridge: Cambridge University Press.

Charlesworth, B. 2000. Fisher, Medawar, Hamilton and the evolution of aging. *Genetics*, **156**: 927–931.

Cole, L.C. 1954. The population consequences of life history phenomena. *Q. Rev. Biol.*, **29**: 103–137.

Demetrius, L. 1969. The sensitivity of population growth rate to perturbations in the life-cycle components. *Math. Biosci.*, **4**: 129–136.

Gadgil, M. and Bossert, W.H. 1970. Life history consequences of natural selection. *Am. Nat.*, **102**: 52–64.
Goodman, L.A. 1971. On the sensitivity of the intrinsic growth rate to changes in the age-specific birth and death rates. *Theor. Popul. Biol.*, **2**: 339–354.
Hamilton, W.D. 1966. The moulding of senescence by natural selection. *J. Theor. Biol.*, **12**: 12–45.
Kirkwood, T.B.L. 1977. Evolution of ageing. *Nature*, **270**: 301–304.
Leslie, P.H. 1945. On the use of matrices in certain population mathematics. *Biometrika*, **33**: 183–212.
Luenberger, D.G. 1979. *Introduction to Dynamic Systems: Theory, Models, and Applications*. New York: John Wiley & Sons, Inc.
Medawar, P.B. 1952. *An Unsolved Problem of Biology*. London: H.K. Lewis and Co. Ltd.
Partridge, L. and Mangel, M. 1999. Messages from mortality: the evolution of death rates in the old. *Trends Ecol. Evol.*, **14**: 438–442.
Reznick, D.N., Bryant, M.J., Roff, D., Ghalambor, C.K. and Ghalambor, D.E. 2004. Effect of extrinsic mortality on the evolution of senescence in guppies. *Nature*, **431**: 1095–1099.
Williams, G.C. 1957. Pleiotropy, natural selection, and the evolution of senescence. *Evolution*, **11**: 398–411.
Williams, G.C. 1966. *Adaptation and Natural Selection*. Princeton, NJ: Princeton University Press.

APPENDIX A: EXTENSION OF THE EULER-LOTKA EQUATION

Unfortunately, the algebraic Euler-Lotka equation does not extend in a mathematically elegant or analytically productive way to a genotype-distinguishing model. However, a dynamic systems formulation provides a compact generalization of its equivalent. (This Appendix uses notation departing from that of the main text to better match traditional usage.)

Application to a simple Leslie matrix

The simplest way to illustrate the generalization is by first comparing the Euler-Lotka equation to its dynamic systems counterpart equation when applied to a standard Leslie formulation. If **A** is a Leslie matrix, the dynamic system is

$$\mathbf{x}_{t+1} = \mathbf{A}\mathbf{x}_t \tag{A1}$$

where

$$\mathbf{A} = \begin{bmatrix} m_1 & m_2 & m_3 & \dots & m_A \\ s_2 & 0 & 0 & \dots & 0 \\ 0 & s_3 & 0 & \dots & 0 \\ 0 & 0 & \ddots & \ddots & \vdots \\ 0 & \dots & 0 & s_N & 0 \end{bmatrix} \tag{A2}$$

When the system state is aligned with an eigenvector, the system satisfies

$$\mathbf{A}\mathbf{e} = \lambda\mathbf{e} \tag{A3}$$

where **e** is an eigenvector and λ is its corresponding eigenvalue. ['Aligned' means this: if one considers the system state as a vector pointing in some direction in $\mathcal{R}^N$ space

from the origin, and the eigenvector likewise an arrow pointing in another direction, the system state arrow will rotate around in this space with each application of the transition matrix (one can think of this matrix as a rotation operator applied at each time t) until it converges to exactly lie on top of the eigenvector arrow (direction-wise; magnitude-wise it can differ).]

If one defines

$$l_1 = 1$$
$$l_a = \prod_{i=2}^{a} s_i \tag{A4}$$

and solves for the characteristic equation from $det(\mathbf{A} - \lambda\mathbf{I}) = 0$, the result is the Euler-Lotka equation

$$\sum_{a=1}^{A} l_a m_a \lambda^{-a} = 1 \tag{A5}$$

The roots of this equation are the eigenvalues. The focus is on the principal eigenvector, whose corresponding eigenvalue has the largest (real) magnitude. The system state will always converge to the principal eigenvector so long as the system state has any projection onto it. In real-world systems, the principal eigenvector almost always takes hold and dominates any other eigenvector, since even a slight system state perturbation at any point in time introduces a small projection onto the principal eigenvector. In steady state, all eigenvector elements – and therefore their sum (total population, in this case) – will grow at the rate determined by the principal eigenvalue.

If r is the growth rate of the population (classically, the 'intrinsic growth rate'), the principal eigenvalue can be expressed either as $\lambda = e^r$ (continuous case) or $\lambda = 1 + r$ (discrete case). In this case, the Euler-Lotka equation takes the form

$$\sum_{a=1}^{A} l_a m_a \frac{1}{(1+r)^a} = 1$$

or (A6)

$$\sum_{a=1}^{A} l_a m_a e^{-ra} = 1$$

Total differentiation of (the continuous version of) this equation with respect to s_a and m_a, and judicious substitution of (A5) into the result (and in the limit that changes are small), yields

$$\frac{\partial r}{\partial s_a} = \frac{\sum_{x=a}^{A} l_x m_x \lambda^{-x}}{s_a \sum_{a=1}^{A} a l_a m_a \lambda^{-a}} \tag{A7}$$

and

$$\frac{\partial r}{\partial m_a} = \frac{l_a \lambda^{-a}}{\sum_{a=1}^{A} a l_a m_a \lambda^{-a}} \tag{A8}$$

These are the standard classical expressions (see, for instance, Partridge and Mangel, 1999).

However, there is an alternate approach that both gives deeper insight into the Euler-Lotka equation and sets the stage for generalization to a genotype-distinguishing model.

Observe that the eigenvector of equation (A3), when normalized to make the first element unity (any multiple of the eigenvector is also an eigenvector), is

$$\mathbf{e} = \begin{bmatrix} 1 \\ \frac{s_2}{\lambda} \\ \frac{s_2 s_3}{\lambda} \\ \vdots \\ \frac{\prod_{a=2}^{A} s_a}{\lambda^{A-1}} \end{bmatrix} = \begin{bmatrix} l_1 \\ \frac{l_2}{\lambda} \\ \frac{l_3}{\lambda^2} \\ \vdots \\ \frac{l_A}{\lambda^{A-1}} \end{bmatrix} \tag{A9}$$

Now, designate the first row of $\mathbf{A}$ as the vector $\mathbf{m}^T$ (where the transpose superscript indicates a row vector) and multiply $\mathbf{m}^T$ by $\mathbf{e}/\lambda$:

$$[m_1 \quad m_2 \quad m_3 \quad \ldots \quad m_A] \begin{bmatrix} \frac{l_1}{\lambda} \\ \frac{l_2}{\lambda^2} \\ \frac{l_3}{\lambda^3} \\ \vdots \\ \frac{l_A}{\lambda^A} \end{bmatrix} = \sum_{a=1}^{A} l_a m_a \lambda^{-a} = 1 \tag{A10}$$

It is obvious that the Euler-Lotka equation can then be written as

$$\mathbf{m}^T \frac{\mathbf{e}}{\lambda} = 1$$

or

$$\mathbf{m}^T \mathbf{e} = \lambda \tag{A11}$$

The principal eigenvector **e** depicts the long-term steady-state system state, with each element in a fixed and stable ratio to the others. The principal eigenvector **e** thus depicts the steady-state survival dynamics with respect to age contained in the expression l_a. It also contains the 'discounting' effect that reduces the fecundity contribution to population size of each age cohort with age if the population is growing. Similarly, since **e** depicts the steady-state population distribution versus age, and $\mathbf{m}^T$ depicts fecundity with age, (A11) shows the steady-state per-reproductive-period fecundity of the population assuming age cohort 1 were to contain a single individual. In zero-growth equilibrium, i.e. when $r = 0$ so that $\lambda = 1$, the population exactly replaces itself each reproductive cycle. In general, the greater the fecundities embedded in $\mathbf{m}^T$, the greater the population growth rate.

An alternative interpretation is that $\mathbf{m}^T\mathbf{e}$ is the expected lifetime contribution to the population of a fecund individual of age 1. If $\lambda = 1$, this individual's progeny is expected to exactly replace it over its lifetime. When λ exceeds unity, this individual's contribution to the population will be reduced at each succeeding age because the population is growing; this reduction is indicated in (A9) by larger values in the denominators of the elements of **e**. Likewise, if λ is less than unity, this individual's contribution is augmented at each age.

Not surprisingly, total differentiation of (A11) delivers equations (A7) and (A8), since (A11) is essentially the same equation as (A5).

Note that the eigenvector (A9) contains no elements related to fecundity rates. Fecundity and survival are in this sense separable parts of the system dynamic. This is what allows for the analytically clean functional forms of equations (A5), (A7), and (A8). The elegance of the algebraic Euler-Lotka equation is the serendipitous consequence of this separability.

Equations (A7) and (A8) can be alternately specified, following Charlesworth (1994) [who credits Hamilton (1966), Demetrius (1969), and Goodman (1971) with its development] in a more compact way that is generalizable to the genotype-distinguishing model. Let $\mathbf{f}^T$ be the left eigenvector of the system (A1) and (A2) corresponding to **e**. Then equation (A7) becomes:

$$\frac{\partial r}{\partial s_a} = \frac{f_a e_{a-1}}{\mathbf{f}^T\mathbf{e}} \tag{A12}$$

And equation (A8) becomes:

$$\frac{\partial r}{\partial m_a} = \frac{f_1 e_a}{\mathbf{f}^T\mathbf{e}} \tag{A13}$$

Extension to a genotype-distinguishing model

The elegance of the Euler-Lotka equation is lost when the model is extended to comprehend differing lifespan genotypes. Fortunately, however, the integrity of equation (A11) is fundamentally maintained.

It is easiest to illustrate the difficulties by considering a genotype-distinguishing model with $A = 2$, $G = 2$. Replace the transition matrix **A** in equation (A3) with the following:

$$\mathbf{A} = \left[\begin{array}{cc:cc} m_{11} & m_{12} & 0 & m_{12} \\ 0 & m_{22} & 0 & m_{22} \\ \hdashline 0 & 0 & 0 & 0 \\ 0 & s_2 & 0 & 0 \end{array}\right] \tag{A14}$$

The characteristic equation for this system is

$$m_{11}m_{22}s_2\lambda^{-2} + m_{22}\,(m_{11} - s_2)\,\lambda^{-1} + \lambda - (m_{11} + m_{22}) = 0 \tag{A15}$$

(Note that one of the eigenvalues is zero. This is the consequence of row 3 of **A** having zero entries. For higher-order systems, there will be several such eigenvalues.) It is apparent that cross-terms and additive terms prevent reduction to a simple form paralleling the Euler-Lotka equation. For higher-order models, the difficulty is compounded as the number of cross terms and additive terms expands.

However, something close to equation (A11) is nonetheless preserved. The following system of equations allows extraction of the principal eigenvector:

$$\mathbf{A} - \lambda\mathbf{I} = 0 \tag{A16}$$

This system of equations also delivers the relationship

$$m_{22} = \frac{\lambda^2}{s_2 + \lambda} \tag{A17}$$

The principal eigenvector is of the form

$$\mathbf{e} = \begin{bmatrix} 1 \\ -\dfrac{\lambda(m_{11} - \lambda)}{m_{12}\,(s_2 + \lambda)} \\ 0 \\ -\dfrac{s_2\,(m_{11} - \lambda)}{m_{12}\,(s_2 + \lambda)} \end{bmatrix} \tag{A18}$$

This is a form of the eigenvector normalized such that its first element is unity. Designating this form as $\mathbf{e}_1$, likewise define $\mathbf{e}_2$ as

$$\mathbf{e}_2 = \begin{bmatrix} -\dfrac{m_{12}\,(s_2 + \lambda)}{\lambda(m_{11} - \lambda)} \\ 1 \\ 0 \\ \dfrac{s_2}{\lambda} \end{bmatrix} \tag{A19}$$

Note that these eigenvectors contain terms involving fecundity rates, unlike the simple Leslie matrix case. Fecundity and survival are no longer cleanly separable. The 'survival' dynamic contained in the eigenvector in this case is such that the steady-state population of each genotype is affected by the fidelity and accuracy of breeding, with genotype 1 being

augmented by offspring from genotype 2. With multiple genotypes, the eigenvector contains many such terms.

Nevertheless, designating the first row of **A** as the vector $\mathbf{m}_1^T$ we have

$$\mathbf{m}_1^T\mathbf{e}_1 = \lambda \tag{A20}$$

Similarly, drawing on relationship (A17), we see that

$$\mathbf{m}_2^T\mathbf{e}_2 = \lambda \tag{A21}$$

Although the $\mathbf{e}_g$ contain terms involving fecundity rates, they nonetheless wholly characterize survival and contribution dynamics in the same way seen in equation (A11), and can be interpreted similarly. Thus, equations (A20) and (A21) allow a conceptual, and analytic, separability of steady-state fecundity and survival/contribution dynamics.

It is still possible to obtain expressions comparable to (A7) and (A8) by total differentiation of (A20) and (A21). For example,

$$\frac{\partial r}{\partial s_2} = \frac{m_{22}\lambda}{m_{22}s_2 + \lambda^2} \tag{A22}$$

and

$$\frac{\partial r}{\partial m_{22}} = \frac{s_2 + \lambda}{m_{22}s_2 + \lambda^2} \tag{A23}$$

However, these expressions become increasingly intractable as the number of age classes and genotypes grows, depriving the resulting expressions of any simple interpretation paralleling (A7) and (A8).

Instead, (A12) and (A13) can be generalized to the genotype-distinguishing case by appealing to the dynamic systems approach:

$$\frac{\partial r}{\partial s_{ag}} = \frac{f_{ag}e_{a-1,g}}{\mathbf{f}^T\mathbf{e}} \tag{A24}$$

and

$$\frac{\partial r}{\partial m_{ag_Pg_O}} = \frac{f_{1g_Pg_O}e_{a\,g_Pg_O}}{\mathbf{f}^T\mathbf{e}} \tag{A25}$$

where g_P and g_O are the genotype indices of parent and offspring, respectively. In the implementation reported in this article, fecundity is a function of genotype only, not age. The resulting expression corresponding to (A25) is

$$\frac{\partial r}{\partial m_{g_Pg_O}} = \frac{f_{1g_Pg_O}\sum_{a=1}^{A} e_{a\,g_Pg_O}}{\mathbf{f}^T\mathbf{e}} \tag{A26}$$

Since for $A = G = N$ the transition matrix is of dimension $N^2 \times N^2$ and the eigenvector is of dimension N^2, deriving analytic expressions for the eigenvectors becomes increasingly difficult. Practically, this means one is reduced to measuring these derivatives by means of numerical modelling. However, this is easy. The spreadsheets calculate these derivatives for $A = G = 5$ and $A = G = 10$ (evolutionary-ecology.com/data/2500/).

This approach enables the tracking of population dynamics in a way counterpart to the Euler-Lotka equation and associated derivatives, but by individual genotype:

$$\mathbf{m}_g^T \mathbf{e}_g = \lambda,\ g = 1, \ldots, G \tag{A27}$$

Equations (A27) contain all the richness of content described for the simple Leslie case as described in connection with equation (A11), except here they are genotype specific. Similarly, equations (A24) through (A26) deliver the counterpart survival and fecundity derivatives by (age and) genotype.

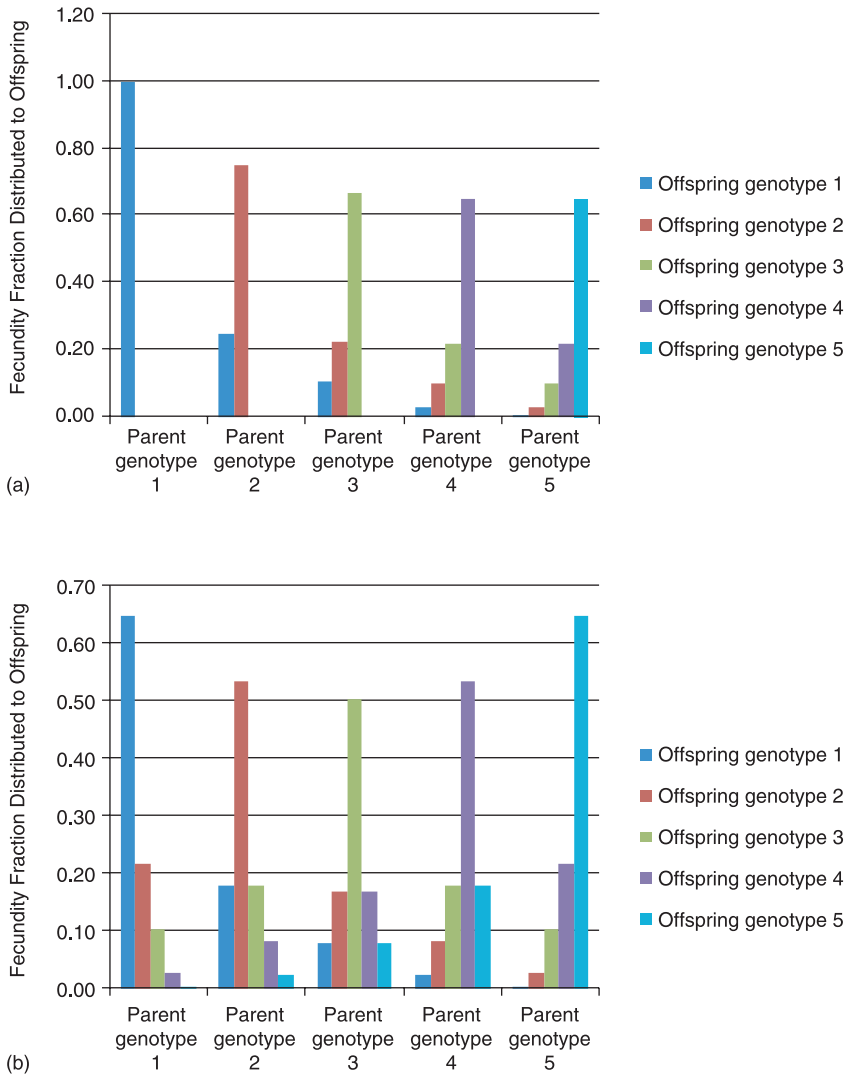

Fig. B1. (a) Base Case breeding distribution across genotypes. (b) Distribution allowing upward symmetry (bottom panel).

APPENDIX B: PARENT GENOTYPES PRODUCE OFFSPRING WITH LONGER INTRINSIC LIFESPAN THAN THEIR OWN

A central assumption embedded in equation (2) is that parent genotypes can only give rise to offspring whose genotype index is less than or equal to their own. Although this seems a sensible assumption, the consequences of relaxing this assumption merit examination.

In this sensitivity case, the breeding fidelity and accuracy parameters are set to the Base Case values reflected in Fig. 1 (i.e. $\phi = 0.7$, $\sigma_\alpha = 2.0$), but the distribution of offspring is allowed to apply symmetrically upward as well as downward, as shown in Fig. B1. Not surprisingly, the result is a rightward shift in the genotype structure and an increase in $\overline{\Gamma}$ (Fig. B2).

If one believes that deleterious mutations could result in offspring with longer intrinsic lifespan but that suffer a corresponding fecundity reduction, the methodology introduced here can accommodate such a framework.

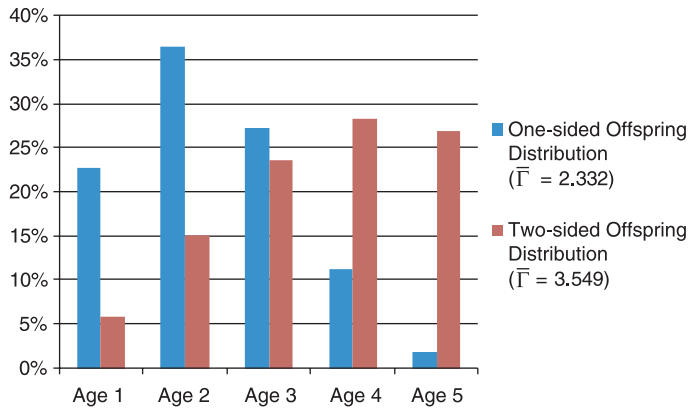

Fig. B2. Genotype distributions with and without longer lifespan offspring.

APPENDIX C: ADDITIONAL NOTES ON MODEL BEHAVIOUR WITH HIGH FECUNDITIES

The methodology reported here permits the exploration of population dynamics consequences across the full domains of all input variables, but there are certain cases that require explanation, particularly in connection with high fecundity rates and zero-growth equilibrium conditions.

Even in a single-genotype traditional Leslie matrix formulation, for a fecundity rate applied uniformly across age cohorts in excess of 100% (i.e. a rate allowing for more than one viable female offspring per female per reproductive cycle), the total population will rise in unbounded fashion and zero-growth equilibirum is not feasible, even for an extrinsic death rate arbitrarily close to 100%. This is perhaps not surprising, since the methodology ignores any density dependence. But it would seem at first blush to impose a serious limitation of the approach for researchers wishing to apply it to organisms that appear to be of relatively fixed size yet exhibit fecundity rates well in excess of 100% per reproductive cycle.

The problem reveals itself by considering a simple 2×2 single-genotype Leslie model:

$$\mathbf{x}_{t+1} = \begin{bmatrix} b & b \\ s & 0 \end{bmatrix} \mathbf{x}_t \tag{C1}$$

The eigenvalues of this system are

$$\lambda = \frac{b \pm \sqrt{b^2 + 4bs}}{2} \tag{C2}$$

If we attempt to require zero-growth equilibrium (i.e. $\lambda = 1$), from (C2) we find that

$$b = \frac{1}{1+s} \tag{C3}$$

It is thus apparent that it is not possible to have zero-growth equilibrium with any $b > 1$ (since $s > 0$).

The genotype-distinguishing case suffers from the same problem, since in general the matrix $\mathbf{B_1} \neq \mathbf{0}$. The numerical solutions shown in the main text have all assumed $b_0 < 1$ (i.e. $b_0 < 100\%$).

Fortunately, this problem disappears with the addition of a single further assumption: that the first age cohort is non-reproductive.

For the singe-genotype Leslie case, the system then becomes

$$\mathbf{x}_{t+1} = \begin{bmatrix} 0 & b \\ s & 0 \end{bmatrix} \mathbf{x}_t \tag{C4}$$

The eigenvalues of this system are

$$\lambda = \pm \sqrt{bs} \tag{C5}$$

and the zero-growth equilibrium condition is $bs = 1$. Thus, in this case, b can take arbitrarily large values and still generate zero-growth equilibrium with s sufficiently small.

As with the single-genotype case, with this assumption of a non-reproductive age 1 cohort ($\mathbf{B_1} = \mathbf{0}$), the genotype-distinguishing case can accommodate arbitrarily large fecundity rates alongside zero-growth equilibrium. One can then consider the extrinsic death rate that delivers this zero-growth equilibrium as arising in part from density-dependent factors that check unlimited growth. While this is imperfect, and the methodology will allow a more sophisticated depiction of density dependence (to be reported in a future article), the current implementation nonetheless allows high fecundity populations in zero-growth equilibrium to be modelled in a sensible fashion given this added assumption.

Numerical solutions for such populations generally require many more reproductive cycles to achieve equilibrium. Note that it is only in equilibrium conditions (whether zero-growth or steady-state growth) that the Euler-Lotka-equivalent conditions, $\mathbf{B}_g^T \mathbf{e}_g = \lambda$, are satisfied. Also, it is frequently the case that such populations exhibit oscillatory behaviour for a time. This is an indication that at least one of the sub-dominant eigenvalues is a complex number. While this may be indicative of a real phenomenon in some populations, such oscillations disappear as the principal eigenvector takes hold and equilibrium is reached.

To address these issues, the example spreadsheets (evolutionary-ecology.com/data/2500/) allow the user to extend indefinitely the number of reproductive cycles as the model seeks zero-growth equilibrium and to toggle off reproduction for age 1 individuals, thus allowing researchers to explore high fecundity populations. These features are explained in the User Guide contained in each spreadsheet.