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Selection in one-sex stage-structured populations

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Hal Caswell

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Abstract

Demographic processes and ecological interactions are central to understanding evolution, and vice versa. We present a novel framework that combines basic Mendelian genetics with the powerful demographic approach of matrix population models. The demographic component of the model may be stage-classified or age-classified, linear or nonlinear, time-invariant or time varying, deterministic or stochastic, and may include dependence on environmental resources or interactions among species. Genotypes may affect, in fully pleiotropic fashion, any mixture of demographic traits (viability, fertility, development) at any points in the life cycle. The dynamics of the stage×genotype structure of the population are given by a nonlinear population projection matrix. We show how to construct this matrix and use it to derive sufficient conditions for a protected genetic polymorphism for the case of linear, time-independent demography. These conditions demonstrate that genotype-specific population growth rates ($\lambda$) do not determine the outcome of selection. Except in restrictive special cases, heterozygote superiority in $\lambda$ is neither necessary nor sufficient for a genetic polymorphism. As a consequence, population growth rate does not always increase and populations can be driven to extinction due to evolutionary suicide. We demonstrate the construction and analysis of the model using data on a color polymorphism in the common buzzard, *Buteo buteo*. The model exhibits a stable genetic polymorphism and declining growth rate, consistent with field data and previous models.
2.1 Introduction

Evolutionary change and population dynamics are both driven by birth and death processes, and these processes provide a fundamental link between the two fields. Evolutionary change in the distribution of genotypes and phenotypes within a population is a consequence of birth and deaths within that population. That is, evolutionary change is a consequence of demographic processes. It has been repeatedly argued that demography is therefore central to understanding evolution (Fussmann et al. 2007; Metcalf and Pavard 2007; Pelletier et al. 2009). Coulson et al. (2006) identified the steps involved in such an eco-evolutionary analysis: a map from genotype to phenotype, from phenotype to demography and from demography to fitness (cf. Lewontin 1974 for an earlier version of these ideas). Our goal here is to provide a model framework that includes ecological and genetic processes operating simultaneously.

Examples of ecological and evolutionary processes operating on similar time scales include the rapid evolution of resistance to antibiotics and pesticides, and rapid life-history responses to environmental changes in urban environments (Schilthuizen 2018). The phenomena of evolutionary rescue, in which genotype dynamics change population growth from negative to positive, and evolutionary suicide, in which the opposite happens, are invoked as general examples of eco-evolutionary outcomes (Jones et al. 2009; Ferriere and Legendre 2013).

To put our results in context, it is useful to recall the various approaches to evolutionary dynamics: population genetics, quantitative genetics, and adaptive dynamics. Each treats the genetic and evolutionary components in its own way, and each has been coupled to ecology and demography in its own way.

Population genetics describes traits determined by a small numbers of genes with potentially large phenotypic effects, in terms of the dynamics of gene and genotype frequencies. The early analyses of Fisher (1930) and Wright (1931) treated population size as fixed. Roughgarden combined population genetics with nonlinear ecological models by writing genotype fitnesses as functions of intra- and inter-specific densities (Roughgarden 1971), but neglected population structure. Charlesworth developed a theory for age-classified population genetics (Charlesworth 1970; Charlesworth and Giesel 1972a; Charlesworth 1972; Charlesworth and Giesel 1972b). Orive (1995) extended Charlesworth’s framework to stage-structured population genetics models to study the effect of clonal reproduction on the evolution of senescence. Tuljapurkar (1982) extended

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1In our terminology, ecological processes are those involved in population growth and interaction. Demographic models are those that analyze ecological processes in terms of internal population structure, distinguishing individuals on the basis of age, developmental stage, size, etc. Demographic models, in our terminology, are a subset of ecological models.
Charlesworth’s results to the case of variable environments by deriving invasion conditions for a new allele into a homozygous, age-structured population with no demographic differences between the sexes.

Quantitative genetics focuses on the dynamics of phenotypic traits determined by large numbers of genes, each with small effects \(\text{[Falconer 1960]}\). These models focus on the components of phenotypic variance (genetic, environmental, genetic×environmental, etc.; \(\text{[Kempthorne 1957]}\)). Selection is described in terms of selection differentials or gradients and the changing patterns of genetic variance and covariance. Quantitative genetic models for changes in phenotype means have been adapted to age-classified \(\text{[Lande 1982b]}\) and stage-classified \(\text{[Barfield et al. 2011]}\) demographic models. A more general structured approach based on an integral projection model for trait distributions has been presented by \(\text{Coulson and Tuljapurkar 2008}\). Childs et al. \(\text{2016}\) extend this framework to include both sexes and develop an extension of the age-structured Price equation for two-sex populations.

Adaptive dynamics \(\text{[Metz et al. 1992; Diekmann 2004]}\) avoids genetics altogether, describing evolution as a series of phenotype substitutions, with one phenotype replacing another until a phenotype is found that can resist invasion by all others. It is used to explore a wide range of complicated ecological scenarios, including nonlinear dynamics, population structure, resource-consumer interactions, and interspecific interactions \(\text{[Dieckmann and Law 1996; Geritz et al. 1998; Dercole and Rinaldi 2008]}\). Adaptive dynamics generally assumes clonal reproduction and infrequent mutations, such that a separation is possible between a slower evolutionary time scale and a faster ecological time scale. Adaptive dynamics has been combined with the framework of integral projection models to study the evolution of function-valued traits \(\text{[Metcalf et al. 2008; Rees and Ellner 2016]}\).

Our results here are squarely in the population genetics tradition, and thus complement recent advances in quantitative genetics using integral projection models \(\text{[Coulson and Tuljapurkar 2008; Coulson et al. 2010; Childs et al. 2016]}\). We present a general connection between population genetics and stage-structured demography. The ecological components of the model may be stage-classified or age-classified, and may be linear or nonlinear, time-invariant or time varying, deterministic or stochastic, and may (although we will not explore that here) include dependence on environmental resources or interactions among species. We allow genotypes to affect \textit{any} of the vital rates (survival, fertility, growth, development, movement, etc.) and to do so in a stage-specific way.

These considerations lead us to a multidimensional matrix population model in which individuals are jointly classified by stage and genotype. Although we
formulate the model for discrete stages, there is no reason to doubt that it could be extended to the Integral Projection Model (IPM) context for the case of continuous i-states. [Coulson et al. (2011)] present an IPM with a genetic component to describe a coat color polymorphism in a population of wolves, although the details of the genetic components are not explicitly laid out. Our results provide a general formulation, applicable to any population, and amenable to analytical manipulations.

Our major results are the methodology for constructing such a model from genotype-specific demographic measurements and a set of analytical conditions that determine whether alleles will coexist in a genetic polymorphism, or if one or another allele will go to fixation.

2.2 Model construction

As in most ecological and demographic studies, we model only females, and suppose that the amount of offspring production is determined by female genotype. The genetic composition of those offspring is determined by the mating process, and thus the genetic composition of potential male mates. This means that we are focusing on traits that affect both female and male survival and transitions, but do not affect male mating success. This would include such traits as, inter alia, predator defense, disease or drug resistance, resource uptake, or migration, that could affect both males and females. It excludes traits such as male courtship displays, ornamental plumage, etc. A two-sex version of the model, which includes males explicitly and relaxes these restrictions, will be presented elsewhere (de Vries and Caswell in prep).

We will assume random mating with respect to stage and genotype, but subject to constraints on which stages take part in reproduction. Assortative mating can be incorporated, but we do not consider that here.

Individuals are jointly classified by stage $(1, \ldots, \omega)$, and genotype $(1, \ldots, g)$. Each genotype is characterized by a matrix of transition probabilities (including survival) and a matrix of reproductive output. These matrices can include time variation or nonlinearities reflecting the environment or density dependence, although we will address those complications elsewhere. Each stage contributes offspring to genotypes at the next time step according to matrices that are determined by the mating system and the population structure.

The matrices, vectors and mathematical operations used in this paper are listed in Table 2.1.
2. Selection in one-sex stage-structured populations

Table 2.1: Mathematical notation used in this paper. Dimensions of vectors and matrices are given where relevant.

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Definition</th>
<th>Dimension</th>
</tr>
</thead>
<tbody>
<tr>
<td>$a$</td>
<td>Number of alleles (2)</td>
<td></td>
</tr>
<tr>
<td>$g$</td>
<td>Number of genotypes (3)</td>
<td></td>
</tr>
<tr>
<td>$\omega$</td>
<td>Number of stages</td>
<td></td>
</tr>
<tr>
<td>$N$</td>
<td>Total population size</td>
<td></td>
</tr>
<tr>
<td>$N_b$</td>
<td>Breeding population size</td>
<td></td>
</tr>
<tr>
<td>$c_i$</td>
<td>Indicator vector for breeding stages in genotype $i$</td>
<td>$\omega \times 1$</td>
</tr>
<tr>
<td>$\mathbf{n}$</td>
<td>Joint stage-genotype vector</td>
<td>$\omega g \times 1$</td>
</tr>
<tr>
<td>$\mathbf{p}$</td>
<td>Joint stage-genotype frequency vector</td>
<td>$\omega g \times 1$</td>
</tr>
<tr>
<td>$p_i$</td>
<td>Genotype frequency vector in genotype $i$</td>
<td>$g \times 1$</td>
</tr>
<tr>
<td>$p'_i$</td>
<td>Genotype frequency vector of the offspring of genotype $i$</td>
<td>$g \times 1$</td>
</tr>
<tr>
<td>$p_b$</td>
<td>Genotype frequency vector in breeding population</td>
<td>$g \times 1$</td>
</tr>
<tr>
<td>$q$</td>
<td>Gene frequency vector in population</td>
<td>$a \times 1$</td>
</tr>
<tr>
<td>$q_i$</td>
<td>Gene frequency vector in genotype $i$</td>
<td>$a \times 1$</td>
</tr>
<tr>
<td>$q_b$</td>
<td>Gene frequency vector in breeding population</td>
<td>$a \times 1$</td>
</tr>
<tr>
<td>$r_i$</td>
<td>Gene frequency in gametes from genotype $i$</td>
<td>$a \times 1$</td>
</tr>
<tr>
<td>$r_b$</td>
<td>Gene frequency in gametes from breeding population</td>
<td>$a \times 1$</td>
</tr>
<tr>
<td>$I_\omega$</td>
<td>Identity matrix</td>
<td>$\omega \times \omega$</td>
</tr>
<tr>
<td>$1_g$</td>
<td>Vector of ones</td>
<td>$g \times 1$</td>
</tr>
<tr>
<td>$e_i$</td>
<td>The $i$th unit vector, with a 1 in the $i$th entry and zeros elsewhere.</td>
<td>various</td>
</tr>
<tr>
<td>$E_{ij}$</td>
<td>A matrix with a 1 in the $(i,j)$ position, and zeros elsewhere.</td>
<td>various</td>
</tr>
<tr>
<td>$\otimes$</td>
<td>Kronecker product</td>
<td></td>
</tr>
<tr>
<td>vec$\mathbf{X}$</td>
<td>The vec operator, which stacks the columns of an $m \times n$ matrix $\mathbf{X}$ into a $mn \times 1$ vector.</td>
<td></td>
</tr>
<tr>
<td>$\mathbf{U}_i$</td>
<td>Demographic transitions for genotype $i$</td>
<td>$\omega \times \omega$</td>
</tr>
<tr>
<td>$\mathbf{F}_i$</td>
<td>Fertility matrix for genotype $i$</td>
<td>$\omega \times \omega$</td>
</tr>
<tr>
<td>$\mathbf{D}_i$</td>
<td>Genotype transitions for stage $i$</td>
<td>$g \times g$</td>
</tr>
<tr>
<td>$\mathbf{H}_i(\mathbf{n})$</td>
<td>Parent-offspring genotype map for stage $i$</td>
<td>$g \times g$</td>
</tr>
</tbody>
</table>

The component matrices

The population state at time $t$ can be described by a stage×genotype distribution

$$\mathbf{N} = \begin{pmatrix}
  n_{11} & \cdots & n_{1g} \\
  \vdots & & \vdots \\
  n_{\omega 1} & \cdots & n_{\omega g}
\end{pmatrix}, \quad (2.1)$$

where $n_{ij}$ represents the number of individuals of stage $i$ and genotype $j$. This two-dimensional array is transformed into a population state vector using the vec operator, which stacks the columns on top of each other:

$$\tilde{n}(t) = \text{vec}\mathbf{N}(t). \quad (2.2)$$

For a single locus with two alleles, say $A$ and $a$, we will identify genotypes 1, 2, and 3 as $AA$, $Aa$, and $aa$, respectively. The population state vector consists of
three genotype-specific population vectors:

\[
\mathbf{n}(t) = \begin{pmatrix}
    n_{AA}(t) \\
    n_{Aa}(t) \\
    n_{aa}(t)
\end{pmatrix}.
\]

(2.3)

The population vector \( \mathbf{n} \) is projected from \( t \) to \( t + 1 \) by a matrix \( \mathbf{\hat{A}}[\mathbf{n}] \), so that

\[
\mathbf{n}(t + 1) = \mathbf{\hat{A}}[\mathbf{n}] \mathbf{n}(t).
\]

(2.4)

The matrix \( \mathbf{\hat{A}} \) depends on \( \mathbf{n} \) because the genotypes of offspring depend on gene frequencies of parents.

The projection matrix is constructed from four sets of matrices representing the demographic and genetic processes:

- \( \mathbf{U}_i \) demographic transitions for genotype \( i \), \( i = 1, \ldots, g \), \( \omega \times \omega \)
- \( \mathbf{F}_i \) fertility matrix for genotype \( i \), \( i = 1, \ldots, g \), \( \omega \times \omega \)
- \( \mathbf{D}_i \) genotype transitions for stage \( i \), \( i = 1, \ldots, \omega \), \( g \times g \)
- \( \mathbf{H}_i(\mathbf{n}) \) parent-offspring genotype map for stage \( i \), \( i = 1, \ldots, \omega \), \( g \times g \)

The matrix \( \mathbf{U}_i \) contains transition and survival probabilities for genotype \( i \). The matrix \( \mathbf{F}_i \) contains stage-specific fertility rates for genotype \( i \). In the absence of genetic structure, these would be the familiar transition and fertility matrices making up a population projection matrix. Because the \( \mathbf{U}_i \) and the \( \mathbf{F}_i \) can differ among genotypes in any way, the model admits any kind of pleiotropy in demographic traits.

The matrix \( \mathbf{D}_i \) contains genotype transition probabilities for individuals in stage \( i \), but because genotypes are fixed within an individual, we set \( \mathbf{D}_i = \mathbf{D} = \mathbf{I}_g \) for all \( i \), where \( \mathbf{I}_g \) is the identity matrix of size \( g \times g \). The matrix \( \mathbf{H}_i(\mathbf{n}) \) is a parent-offspring map, from the genotype of a mother in stage \( i \) to the genotypes of her offspring. The \((k,l)\) entry of \( \mathbf{H}_i \) is the probability that an offspring of a genotype \( l \) mother, of stage \( i \), has genotype \( k \). For the purpose of this paper, we assume that mating is random with respect to stage and hence that the parent-offspring map is the same for all stages, i.e. \( \mathbf{H}_i(\mathbf{n}) = \mathbf{H}(\mathbf{n}) \). The matrix \( \mathbf{H}(\mathbf{n}) \) contains the genetic processes and will be derived in the next section.

**Mating: from genotypes of parents to genotypes of offspring**

To model the Mendelian genetics of offspring production, a description of the genotype and allele structure of the mating population is required. Not every life cycle stage will reproduce, and non-reproductive (e.g., immature) stages play no role in determining the genotype frequencies among offspring. We define the breeding population by a set of indicator vectors \( \mathbf{c}_j \) for \( j = 1, \ldots, g \) that show
which stages of genotype $j$ take part in mating. That is, the $i$th entry of $c_j$ is 1 if stage $i$ of genotype $j$ reproduces, and 0 otherwise.

To describe genotype frequencies in the mating process, we will distinguish four vectors of genotype frequencies:

\[
\begin{align*}
\mathbf{p} & = \text{ genotype frequencies in the overall population} \\
\mathbf{p}_b & = \text{ genotype frequencies in the breeding population} \\
\mathbf{p}_i & = \text{ genotype frequencies in genotype } i = (e_i) \\
\mathbf{p}_i' & = \text{ genotype frequencies in the offspring of genotype } i
\end{align*}
\]

The size of the breeding population is

\[
N_b = \sum_{j=1}^{g} (e_j^T \otimes c_j^T) \hat{\mathbf{n}},
\]

where $e_j$ is a vector $(g \times 1)$ with a 1 in position $j$ and zeros elsewhere, and $\otimes$ indicates the Kronecker product. Breeding stages are allowed to differ among genotypes, in order to study the fate of traits that change reproductive schedules. In the special case where the genotypes do not differ in their reproductive stages, $c_j = c$ for all genotypes $j$ and

\[
N_b = (1_g^T \otimes c^T) \hat{\mathbf{n}},
\]

where $1_g$ is a vector of ones of dimensions $1 \times g$.

The genotype frequency vector in the breeding population is

\[
\mathbf{p}_b = \frac{\mathbf{X} \hat{\mathbf{n}}}{N_b},
\]

where $\mathbf{X}$ is a matrix that combines abundances of breeding stages. If the breeding vectors $c_i$ differ among genotypes, then

\[
\mathbf{X} = \sum_{i=1}^{g} (E_{ii} \otimes c_i^T)
\]

where $E_{ii}$ is a matrix of dimension $g \times g$ with a 1 in the $(i, i)$ location and zeros elsewhere. If the breeding vectors are the same for all genotypes, $c_i = c$, then

\[
\mathbf{X} = (I_g \otimes c^T).
\]

The genotype frequency vector for genotype $i$ is (trivially) $\mathbf{p}_i = e_i$.

The gene frequencies are a function of the genotype frequencies, so that

\[
\mathbf{q}_i = \mathbf{Wp}_i,
\]

\[
\mathbf{q}_b = \mathbf{Wp}_b,
\]
where

\[
\mathbf{W} = \begin{pmatrix}
1 & 0.5 & 0 \\
0 & 0.5 & 1
\end{pmatrix}.
\]

(2.12)

At this point, mutation can be introduced in the production of gametes from
the gene frequency vector. Define a mutation matrix \( \mathbf{L} \) for the two allele case as

\[
\mathbf{L} = \begin{pmatrix}
1 - u & v \\
u & 1 - v
\end{pmatrix}
\]

(2.13)

where \( u \) is the probability of mutation from allele \( A \) to allele \( a \) and \( v \) is the
probability of mutation in the other direction. The allele frequencies in the gametes
produced by a female of genotype \( i \) are the gene frequencies of the mother, modified
by mutation,

\[
\mathbf{r}_i = \mathbf{Lq}_i.
\]

(2.14)

We do not investigate mutations in this paper.

We assume that genotypes affect the survival and transitions of males and fe-
males equally, and that the ratio of males to females in newborns is one. Thus
males and females have the same genotype \( \times \) stage distribution (cf. Charlesworth
1994). Because the genotypes do not affect male mating success, the gene fre-
quencies in the male gamete pool are proportional to the gene frequencies in the
breeding part of the female population, \( \mathbf{q}_b \), modified by mutation,

\[
\mathbf{r}_b = \mathbf{Lq}_b.
\]

(2.15)

Mating and offspring. Now consider a random mother of genotype \( i \) and let
\( \mathbf{p}_i \) be the genotype distribution of her offspring. These offspring are formed by the
random combination of the gametes produced by this female with those produced
by a random member of the breeding population, so that

\[
\mathbf{p}_i = \mathbf{Z (r}_i \otimes \mathbf{r}_b),
\]

(2.16)

This matrix is specific to the two-allele case; with more than one allele, the
exact structure of \( \mathbf{W} \) will depend on how the various diploid genotypes are num-
bered. For example, if the genotypes formed by three alleles are listed in the order
\( (1, 1 1, 1 2, 1 3, 2 1, 2 2, 2 3, 3 1, 3 2, 3 3) \), then the matrix \( \mathbf{W} \), mapping from the
9 genotypes to the 3 alleles, would be

\[
\mathbf{W} = \begin{pmatrix}
1 & .5 & .5 & .5 & 0 & 0 & .5 & 0 & 0 \\
0 & .5 & 0 & .5 & 1 & .5 & 0 & .5 & 0 \\
0 & 0 & .5 & 0 & 0 & .5 & .5 & .5 & 1
\end{pmatrix}.
\]
The matrix $Z$ converts ordered genotypes ($Aa$ separately from $aA$) into unordered genotypes; for the case of one locus and two alleles,

$$Z = \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix}.$$ (2.17)

Writing down the $Z$ matrix for more than two alleles is straightforward after an order of the genotypes in the population vector has been chosen.

Substituting equations (2.7)–(2.10) into equation (2.16) and using the fact that $AC \otimes BD = (A \otimes B)(C \otimes D)$, we find that the distribution of offspring of a mother of genotype $i$ is

$$p'_i = Z (r_i \otimes r_b)$$

$$= \frac{Q (e_i \otimes \tilde{n})}{\sum_{j=1}^{g} (e_j^T \otimes c_j^T)} \tilde{n},$$ (2.19)

where

$$Q = Z (M \otimes M) (W \otimes W) (I_g \otimes X).$$ (2.20)

For a specified number of genotypes and set of breeding stages the matrix $Q$ is a constant.

The matrix $H$

The matrix $H(\tilde{n})$ maps the genotype of the parent to the genotype of the offspring. The $i$th column of the matrix $H$ contains the distribution of offspring of a mother of genotype $i$, $p'_i$, as given in equation (2.19); thus

$$H = \begin{pmatrix} p'_1 & \cdots & p'_g \end{pmatrix},$$ (2.21)

which can be written as

$$H = \sum_{i=1}^{g} p'_i \otimes e_i^T.$$ (2.22)

Combining equation (2.19) and equation (2.22), yields the following equation for the parent-offspring matrix

$$H(\tilde{n}) = \frac{Q \sum_{i=1}^{g} (e_i \otimes \tilde{n} \otimes e_i^T)}{\sum_{j=1}^{g} (e_j^T \otimes c_j^T) \tilde{n}}.$$ (2.23)
The population projection matrix
To project the stage × genotype dynamics, the component matrices, $U_i$, $F_i$, and $H(\tilde{n})$ are incorporated into a population projection matrix. We create a set of block-diagonal matrices $U$, $D$, $F$, and $H$, by putting the corresponding matrices on the diagonal. These block diagonal matrices can be written as

\begin{align*}
U & = \sum_{i=1}^{g} E_{ii} \otimes U_i, \quad (2.24) \\
F & = \sum_{i=1}^{g} E_{ii} \otimes F_i, \quad (2.25) \\
D & = I_\omega \otimes I_g, \quad (2.26) \\
H & = I_\omega \otimes H(\tilde{n}), \quad (2.27)
\end{align*}

where $E_{ii}$ is of dimensions $g \times g$.

As in other multistate matrix models, the projection matrix $\tilde{A}(\tilde{n})$ is constructed from the four block matrices,

\begin{equation}
\tilde{A}(\tilde{n}) = K^T D K U + K^T H(\tilde{n}) K F,
\end{equation}

where $K = K_{\omega,g}$ is the vec-permutation matrix (Henderson and Searle 1981). The vec-permutation matrix rearranges the population vector so that genotypes are ordered within stages, that is,

\begin{equation}
\text{vec}N^T = K \text{vec}N.
\end{equation}

For more extensive discussion of the vec-permutation construction, see Hunter and Caswell (2005), Caswell (2012), and Caswell et al. (2018).

The first term, labelled $U$, in $\tilde{A}$ applies the block-diagonal matrix $U$ to generate transitions and survival of extant individuals within genotypes, permutes the resulting vector with $K$, then applies the block-diagonal matrix $D$ to account for changes in genotype among extant individuals (since extant individuals do not generally change their genotype $D$ will be an identity matrix), and finally permutes the vector back to its original form with $K^T$.

The second term, labelled $F$ in $\tilde{A}$, describes reproduction and genotype assignment. First, the block-diagonal matrix $F$ produces offspring, possibly of different birth stages (e.g., seedlings of different sizes) as a function of the genotype of the parent. When they appear, offspring are associated with the genotype of the parent. The vec-permutation matrix $K$ rearranges the vector, and then the block-diagonal matrix $H(\tilde{n})$ allocates the offspring to their genotypes, based on the genotype of their parent and the genotype distribution of the rest of the population. Finally, $K^T$ returns the vector to its original orientation.
Nonlinearity and homogeneity

Even when the demographic components of the model are linear, the system (2.28) is nonlinear, because of the genetic component of the model. The parent-to-offspring genotype transition matrix, and thus the projection matrix \( \tilde{A}(n) \) are nonlinear, but homogeneous of degree zero. That is, for any non-zero scalar \( c \), it is true that \( \tilde{A}(cn) = \tilde{A}(n) \). This implies that dynamics depend on the relative, not the absolute, abundances of the stages. This mathematical fact permits us to switch at will between considering the model as a function of the population vector \( \tilde{n} \) or the frequency vector \( \tilde{p} \), since the two differ only by a proportionality factor.

Homogeneous nonlinear models are familiar in two-sex demographic models (e.g., Hadeler et al. 1988; Iannelli et al. 2005; Caswell 2001). A nonlinear version of Perron-Frobenius theorem guarantees convergence to a constant population structure and exponential population growth under certain conditions in these homogeneous nonlinear models (Nussbaum 1986, 1989). Thus we expect the population to convergence to a stable stage \( \times \) genotype structure, with exponential growth in population size.

In the literature on two-sex models, homogeneous models are often referred to as “frequency-dependent” to distinguish them from density-dependent nonlinear models (Shyu and Caswell 2016b). We do not follow that practice here because it causes confusion with the genetic concept of frequency-dependent fitness. All of our models are homogeneous; none of them are frequency-dependent in the genetic sense.

The block form of the projection matrix

The population projection matrix for the two allele case can be written in terms of 3×3 block matrices which project the three genotype-specific population vectors, \( n_{AA}, n_{Aa}, n_{aa} \), which were introduced in equation (2.3). Taking advantage of the structure of the component matrices, we find that \( A, U, \) and \( \tilde{F} \) can be simplified (Appendix 2.A). The survival and transition matrix \( \tilde{U} \) is block diagonal, because individuals do not change genotype

\[
\tilde{U} = \begin{pmatrix}
U_{AA} & 0 & 0 \\
0 & U_{Aa} & 0 \\
0 & 0 & U_{aa}
\end{pmatrix}.
\] (2.30)

The fertility matrix \( \tilde{F} \) has the following form,

\[
\tilde{F}(\tilde{p}) = \begin{pmatrix}
q_A^b F_{AA} & \frac{1}{2}q_A^b F_{Aa} & 0 \\
q_a^b F_{AA} & \frac{1}{2}F_{Aa} & q_A^b F_{aa} \\
0 & \frac{1}{2}q_a^b F_{Aa} & q_a^b F_{aa}
\end{pmatrix},
\] (2.31)
where \( q_b^A \) and \( q_b^a \) are the frequencies of allele \( A \) and \( a \), respectively, in the breeding part of the population, i.e. they are the elements of the vector \( q_b \) defined by equation (2.11), such that \( q_b^T = (q_b^A, q_b^a)^T \). If the \( F_i \) matrices are linear, the matrix \( \tilde{F} \) is homogeneous and can be written as a function of \( \tilde{p} \) rather than \( \tilde{n} \).

The population projection matrix is the sum of \( \tilde{U} \) and \( \tilde{F} \):

\[
\tilde{A} (\tilde{p}) = \begin{pmatrix}
U_{AA} + q_b^A F_{AA} & \frac{1}{2} q_b^A F_{Aa} & 0 \\
q_b^A F_{AA} & U_{Aa} + \frac{1}{2} F_{Aa} & q_b^A F_{aa} \\
0 & \frac{1}{2} q_b^A F_{Aa} & U_{aa} + q_b^A F_{aa}
\end{pmatrix}.
\]  

(2.32)

The matrix \( \tilde{A} \) is used in equation (2.4) to project trajectories in stage \( \times \) genotype space.

**Stage \( \times \) genotype dynamics**

We consider two examples of projection of the stage structure and genotype composition of the population. In addition to showing how the model can be used, these will provide material for the investigation of analytical conditions for genetic polymorphism.

**A two-stage model.** As a simple example, consider a two stage (juvenile and adult) model, with

\[
U = \begin{pmatrix}
s_J (1 - \gamma) & 0 \\
s_J \gamma & s_A
\end{pmatrix}, \quad F = \begin{pmatrix}
0 & f \\
0 & 0
\end{pmatrix}
\]

(2.33)

where \( s_J \) and \( s_A \) are juvenile and adult survival probabilities, \( \gamma \) is the maturation rate, and \( f \) the adult fertility. Any or all of these parameters may differ among genotypes, so that selection can operate on stage-specific viability, development, and/or fertility; this would lead to genotype-specific versions of each of the matrices (\( U_{AA}, \) etc.).

Figure 2.1 shows two examples of stage \( \times \) genotype dynamics produced by this simple model (model parameters and MATLAB code are given in the Online Supplementary Materials). The \( A \) allele is introduced by adding a small fraction of heterozygote juveniles into a homozygote population. In Figure 2.1b, genotype frequencies converge to a stable polymorphism, even though the population is driven to evolutionary suicide (Figure 2.1a). In Figure 2.1d, the \( A \) allele sweeps to fixation; in so doing it changes population growth from negative to positive (evolutionary rescue; Figure 2.1c). We will return to this example below to examine the criteria for polymorphism, in relation to measures of genotypic growth rates \( \lambda_i \) which will be defined in Section 2.3.
2. Selection in one-sex stage-structured populations

Figure 2.1: Two examples of population dynamics of a two stage (juvenile-adult) Mendelian matrix population model. 2.1a, 2.1b: Introduction of the A allele leads to evolutionary suicide and a genetic polymorphism. 2.1c, 2.1d: Introduction of the A allele leads to evolutionary rescue and fixation of the AA genotype.

A color polymorphism in the Common Buzzard. As an example of how empirical demographic data might be incorporated into the model, we consider a study of a color polymorphism in the common buzzard, Buteo buteo, a bird of prey native to most of Europe and parts of Asia. The buzzard has three color morphs: dark (DD), light (LL) and intermediate (DL). The polymorphism is believed to be a consequence of a one locus system with two alleles, dark and light, where intermediates are heterozygotes [Krüger and Lindström 2001], although recent studies have suggested that the genetics might be more complex [Kappers et al. 2018]. There is evidence of assortative mating in Buteo buteo [Krüger et al. 2001]. However, in the absence of data to quantify the level of assortative mating, we assume random mating for this example.
Table 2.2: The transition matrix and the fertility matrix for *Buteo buteo* females of the intermediate color morph.

\[
U_{DL} = \begin{pmatrix}
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0.8 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0.75 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0.636 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0.714 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0.667 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0.8 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0.625 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0.4 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0.5 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1
\end{pmatrix}
\]

(2.34)

\[
F_{DL} = \begin{pmatrix}
0.05 & 0.41 & 0.42 & 0.45 & 0.56 & 0.48 & 0.61 & 0.27 & 0.40 & 0.54 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix}
\]

(2.35)

Kru¨uger and Lindstr¨om (2001) constructed a Leslie matrix for each color morph using data collected from 1989 through 1999 in eastern Westphalia, Germany. As examples the survival and fertility matrices for females of the intermediate morph are shown in Table 2.2. The other genotype-specific survival and transition matrices are available in the Online Supplementary Materials.

The projection matrix \( \tilde{A} \) is constructed from the genotype specific matrices \( U_i \) and \( F_i \) using equation (2.32). Starting with a population of dark individuals and one heterozygote juvenile, the asymptotic population dynamics are obtained by projecting the population forward using equation (2.4). The abundances in each age class of all three genotypes are shown in Figure 2.2(b). The population growth rate converges to \( \lambda = 0.91 \). Field observations confirm that this *Buteo buteo* population was in decline during the time period used for fitting the model. The marginal distribution over genotypes, integrating over ages in \( \tilde{p} \) is shown in
2. Selection in one-sex stage-structured populations

Figure 2.2: (a) Projected *Buteo buteo* genotype frequencies when the DL heterozygote is introduced at low frequency (0.001) in a homozygous population of DD at its stable age distribution. (b) Projected genotype by age class distribution for the calculations in part (a).

Figure 2.2(a). The light allele increases in frequency until a stable polymorphism is reached.
2.3 Coexistence and genetic polymorphism

The conditions that lead to either fixation of one allele or coexistence of both alleles at a genetic polymorphism are critical to population genetics. In the simplest classical models, the genotypes AA, Aa, and aa are assigned fitnesses \( w_{AA} \), \( w_{Aa} \), and \( w_{aa} \) (e.g. Crow and Kimura (1970)). The two alleles coexist if and only if the heterozygote has the highest fitness, i.e. \( w_{Aa} > w_{AA} \) and \( w_{Aa} > w_{aa} \). The fitnesses \( w_i \) capture, in a single scalar, all the information of genotype effects on survival and reproduction, from one generation to the next, ignoring population structure. To extend this result to a fully stage-structured model is a challenge because the differences among genotypes are not captured by simple scalars, but rather by the matrices \( U_i \) and \( F_i \) and the parameters that determine them.

Since the \( w_i \) represent a kind of growth rate, it has seemed natural, at least since Fisher (1930), to search for ways to use a measure of the rate of growth implied by the demography of each genotype as a measure of “fitness.” The approach was extended by Hamilton (1966) to include the sensitivity analysis of growth rate, considered as a measure of selection pressure, and has formed the basis of several decades of evolutionary life-history research (e.g., Emlen 1970, Stearns 1992, Roff 1992, Metz et al. 1992, Caswell 2001), and eventually became, in the form of invasion fitness, the basis for adaptive dynamics (Dercole and Rinaldi 2008, Geritz et al. 1998). Lande incorporated population growth rate into quantitative genetic models in an age structured version of the breeder’s equation (Lande 1982a, b).

These attempts have in common the exclusion of explicit genotype dynamics. Charlesworth, however, developed an extensive theory for genetics in age-structured models (Charlesworth 1970, 1972, 1994). We will return to a comparison of our approach with that of Charlesworth in the Discussion.

Genotype-specific growth rates. To examine the relation of the full stage×genotype dynamics to the concept of fitness as measured by a growth rate, we define here the genotype-specific growth rates

\[
\begin{align*}
\lambda_{AA} &= \rho(U_{AA} + F_{AA}) \\
\lambda_{Aa} &= \rho(U_{Aa} + F_{Aa}) \\
\lambda_{aa} &= \rho(U_{aa} + F_{aa})
\end{align*}
\]  

where \( \rho(·) \) denotes the largest eigenvalue of a matrix, also referred to as the spectral radius. The problem, of course, is that in a genetically mixed population each genotype contributes offspring to, and receives offspring from, other genotypes. The growth rates defined by (2.36)-(2.38) are thus hypothetical, because in those rates, each genotype is credited with all of its offspring.
If the genotype-specific population growth rates functioned as fitnesses, then
the genotypes would coexist when the heterozygote has the highest fitness; i.e.,
when
\[ \lambda_{Aa} > \lambda_{AA} \]  \hspace{1cm} (2.39)
\[ \lambda_{Aa} > \lambda_{aa}. \]  \hspace{1cm} (2.40)

As we will see, this is true only in special cases.

**Protected genetic polymorphism**

The dynamics of \( \tilde{n} \) take place in a \( \omega g \)-dimensional space defined by combinations of \( \omega \) stages and \( g \) genotypes (with \( g = 3 \) in the present context). The \( \omega \)-dimensional subspaces defined by the homozygous genotypes \( AA \) and \( aa \) are referred to as boundaries. In the absence of mutation, dynamics on the boundaries remain on the boundaries\(^3\) and are given by the projection matrices for the homozygous genotypes.

Coexistence of the two alleles is guaranteed if the boundary subspaces are both unstable; that is, if allele \( A \) can invade a population of \( aa \) individuals and allele \( a \) can invade a population of \( AA \) individuals, then the population can never reach a homozygote state again once both alleles are present in the population (since both alleles grow when rare). Mutual invasibility therefore leads to a protected genetic polymorphism. This approach was introduced in the study of spatially structured populations (Levene 1953; Prout 1968); for a complete summary, see Nagylaki (1992, Chap. 6).

The model reduces to a linear matrix model on the boundary, and hence will either grow or shrink exponentially while converging to a stable population structure. To define the boundary equilibria, the model is transformed from the population vector \( \tilde{n} \), which grows exponentially, to the frequency vector \( \tilde{p} \), which converges to an equilibrium. This transformation is possible because of the homogeneity of \( \tilde{A} \). The dynamics of \( \tilde{p} \) are given by

\[ \tilde{p}(t + 1) = \frac{\tilde{A}[\tilde{p}(t)]\tilde{p}(t)}{\|\tilde{A}[\tilde{p}(t)]\tilde{p}(t)\|}, \]  \hspace{1cm} (2.41)

where \( \|a\| \) indicates the 1-norm of the vector \( a \), defined as the sum of the absolute values of the entries of the vector \( a \). Equilibria of (2.41), denoted by \( \hat{p} \), satisfy

\[ \hat{p} = \frac{\tilde{A}[\hat{p}]\hat{p}}{\|\tilde{A}[\hat{p}]\hat{p}\|}. \]  \hspace{1cm} (2.42)

\(^3\)Technically, the boundaries are invariant under the dynamics specified by \( \tilde{A}[\tilde{n}] \).
The stability of a boundary equilibrium to invasions by the other allele is determined by the dominant eigenvalue of the Jacobian matrix of the linearization of (2.41) at the boundary equilibrium. We denote the dominant eigenvalues by ζ_AA and ζ_aa for the AA and aa boundaries respectively. The strong ergodic theorem ensures that the boundary equilibria are stable to perturbations inside the boundary. Therefore if the dominant eigenvalue of the Jacobian is larger in magnitude than 1, the associated eigenvector must be pointing into the interior which implies the invading allele increases when rare.

The Jacobian matrix
\[ M = \frac{d\tilde{p}(t+1)}{d\tilde{p}(t)} \bigg|_{\hat{p}}, \]  

is obtained by differentiating equation (2.41) and evaluating the resulting derivative at the boundary equilibrium. The derivation is a lengthy exercise in matrix calculus (Verdy and Caswell 2008; Caswell 2008, 2018), presented in detail in Appendix 2.B. The result shows that \( M \) is a block upper triangular matrix given by equation (2.B57) in Appendix 2.B. The diagonal blocks, corresponding to growth of the AA, Aa, and aa genotypes, determine the stability of the boundary equilibrium. We use this matrix to determine the coexistence for protected polymorphism in three cases: the general model, a model restricted to a single reproducing stage, and a model that eliminates population structure, corresponding to the classical population genetics model. We compare each of these results to the naive expectation based on the genotype-specific population growth rate.

**The general case**

The following set of theorems and corollaries are derived in Appendix 2.B. For a model of the form given by equations (2.24), the eigenvalues of the Jacobian matrix associated with growth in the Aa direction are,

\[ \zeta_{AA} = \frac{1}{\lambda_{AA}} \rho \left( U_{Aa} + \frac{1}{2} F_{Aa} + \frac{1}{2p_b} (F_{AA}\hat{p}_{AA}) \otimes c_{Aa}^T \right) \]  

(2.44)

\[ \zeta_{aa} = \frac{1}{\lambda_{aa}} \rho \left( U_{Aa} + \frac{1}{2} F_{Aa} + \frac{1}{2p_b} (F_{aa}\hat{p}_{aa}) \otimes c_{Aa}^T \right) \]  

(2.45)

where \( \lambda_{AA} \) and \( \lambda_{aa} \) are given by equations (2.36) and (2.38), respectively, and \( p_b \) is the fraction of the stable stage distribution that is in a breeding stage for the boundary homozygote.

**Theorem 1** A protected polymorphism occurs when both boundary equilibria are unstable, i.e.

\[ \zeta_{AA} > 1 \]  

(2.46)

\[ \zeta_{aa} > 1 \]  

(2.47)
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or

\[ \rho \left( U_{Aa} + \frac{1}{2} F_{Aa} + \frac{1}{2p_b} (F_{AA} \hat{p}_{AA} \otimes c_{Aa}^T) \right) > \lambda_{AA}, \]  
\[ \rho \left( U_{Aa} + \frac{1}{2} F_{Aa} + \frac{1}{2p_b} (F_{aa} \hat{p}_{aa} \otimes c_{Aa}^T) \right) > \lambda_{aa}. \]  

(2.48)

(2.49)

It is worth noting that the data required to evaluate (2.48) and (2.49) are all obtained from linear life-history calculations: the matrices \( U_{Aa} \) and \( F_{Aa} \) describing the heterozygote, the fertility matrices \( F_{AA} \) and \( F_{aa} \) describing the fertility of the homozygotes, the stable stage distributions \( \hat{p}_{AA} \) and \( \hat{p}_{aa} \) of the homozygotes, and the vector \( c_{Aa} \) that defines the breeding stages for the heterozygote.

The difference between the conditions (2.48) and (2.49) on the one hand, and the simple (but erroneous) comparison of genotype-specific growth rates,

\[ \rho (U_{Aa} + F_{Aa}) > \lambda_{AA}, \]  
\[ \rho (U_{Aa} + F_{Aa}) > \lambda_{aa}. \]  

(2.50)

(2.51)

is the result of both genetic and demographic complexity. When the \( a \) allele invades the \( AA \) boundary equilibrium, it produces almost exclusively heterozygotes [hence the appearance of \( U_{Aa}, F_{Aa}, \) and \( c_{Aa} \) in (2.48)]. But the gametes of these heterozygotes combine with gametes of the much more abundant \( AA \) homozygotes [hence the appearance of \( F_{AA} \)]. In addition, fertility and zygote formation depend on the demographic stage structure of the \( AA \) heterozygotes [hence the appearance of \( \hat{p}_{AA} \) in (2.48)]. None of these complexities are captured by the oversimplified heterozygote growth rates in (2.51). The situation for the \( A \) allele invading the \( aa \) boundary, in equation (2.49), is the same.

**Special case 1: a single reproducing stage**

Theorem 1 provides conditions for polymorphism that apply regardless of the stage structure or the ways in which genotype affects the life cycle. It is instructive to consider some special, simplified cases, in which the relation between the full conditions and the genotype-specific population growth rates is clear.

First, we consider a restriction to a single reproducing stage. When there is only one reproducing stage, the eigenvalues of the Jacobian matrix associated with perturbations in the \( Aa \) direction on the \( AA \) and \( aa \) boundaries, equations (2.44) and (2.45), reduce to,

\[ \zeta_{AA} = \frac{1}{\lambda_{AA}} \rho \left( U_{Aa} + \frac{1}{2} (F_{Aa} + F_{AA}) \right) \]  
\[ \zeta_{aa} = \frac{1}{\lambda_{aa}} \rho \left( U_{Aa} + \frac{1}{2} (F_{Aa} + F_{aa}) \right) \]  

(2.52)

(2.53)
see Appendix 2.B for a derivation of both equations.

**Corollary 1** For a model of the form given by equations (2.24)-(2.28) with only one reproducing stage, a protected polymorphism occurs when both boundary equilibria are unstable, i.e.

\[
\rho \left( U_{Aa} + \frac{1}{2} (F_{Aa} + F_{AA}) \right) > \rho (U_{AA} + F_{AA}) \quad (2.54)
\]

\[
\rho \left( U_{Aa} + \frac{1}{2} (F_{Aa} + F_{aa}) \right) > \rho (U_{aa} + F_{aa}) \quad (2.55)
\]

If the genotypes differ only in survival and transitions (the stage-structured analogue of viability selection), so that \( F_{AA} = F_{Aa} = F_{aa} \), then the coexistence condition reduces to heterozygote superiority in \( \lambda \). If fertility is also affected by genotype, it does not.

Once again, the criteria for polymorphism reflect the two processes by which new \( Aa \) individuals are created: an \( Aa \) female randomly mating with a resident allele from the gamete pool and a resident homozygote female randomly mating with the invading allele from the gamete pool (all other matings are second-order processes).

**Special case 2: an unstructured population**

An even simpler special case results from completely eliminating demographic structure and reducing the model to an unstructured one. In this case, the matrices \( U \) and \( F \) reduce to scalars: the survival probability \( s_i \) and the fertility \( f_i \). In this case, Theorem 1 reduces to the following corollary. For an unstructured version of the model given by equations (2.24)-(2.28), the eigenvalues of the Jacobian matrix, associated with the \( Aa \) direction, at the \( AA \) and \( aa \) boundaries are

\[
\zeta_{AA} = \frac{1}{\lambda_{AA}} \left( s_{Aa} + \frac{1}{2} (f_{Aa} + f_{AA}) \right) \quad (2.56)
\]

\[
\zeta_{aa} = \frac{1}{\lambda_{aa}} \left( s_{Aa} + \frac{1}{2} (f_{Aa} + f_{AA}) \right) \quad (2.57)
\]

**Corollary 2** A protected polymorphism occurs when both boundary equilibria are unstable, i.e.

\[
s_{Aa} + \frac{1}{2} (f_{Aa} + f_{aa}) > s_{aa} + f_{aa} \quad (2.58)
\]

\[
s_{Aa} + \frac{1}{2} (f_{Aa} + f_{AA}) > s_{AA} + f_{AA} \quad (2.59)
\]
or
\[
\begin{align*}
    s_{Aa} + \frac{1}{2} f_{Aa} & > s_{aa} + \frac{1}{2} f_{aa} \\
    s_{Aa} + \frac{1}{2} f_{Aa} & > s_{AA} + \frac{1}{2} f_{AA}
\end{align*}
\] (2.60) (2.61)

Under viability selection alone (i.e., \( f_{Aa} = f_{aa} = f_{AA} \)) or fertility selection alone (i.e., \( s_{Aa} = s_{aa} = s_{AA} \)), the conditions for protected polymorphism reduce to heterozygote advantage in \( \lambda \). This is the result familiar from classical population genetics. However, when genotypes differ in both fertility and viability, heterozygote advantage is neither necessary nor sufficient for polymorphism, a result that agrees with a large literature on fertility selection in unstructured populations [Hadeler and Liberman 1975; Pollak 1978; Feldman et al. 1983].

2.4 Examples revisited

Armed with the results of Theorem 1, we return to the two examples considered in Figure 2.1 (the two-stage model) and Figure 2.2 (the common buzzard color polymorphism).

**Two-stage population.** In the two-stage example of evolutionary suicide (Figure 2.1a,b), the heterozygote is inferior as measured by its growth rate \( \lambda_{Aa} \), but both boundary equilibria are unstable, so that a protected polymorphism results:

\[
\begin{align*}
    \lambda_{AA} &= 0.989 \\
    \lambda_{Aa} &= 0.966 \\
    \lambda_{aa} &= 1.003 \\
    \zeta_{AA} &= 1.012 \\
    \zeta_{aa} &= 1.007
\end{align*}
\] (2.62)

In the case leading to evolutionary rescue (Figure 2.1c,d), the heterozygote is superior in growth rate \( \lambda_{Aa} \), but the \( AA \) boundary equilibrium is stable and the \( aa \) boundary equilibrium is unstable, so that the population converges to fixation of the \( A \) allele:

\[
\begin{align*}
    \lambda_{AA} &= 1.018 \\
    \lambda_{Aa} &= 1.029 \\
    \lambda_{aa} &= 0.990 \\
    \zeta_{AA} &= 0.983 \\
    \zeta_{aa} &= 1.004
\end{align*}
\] (2.63)

In these cases, the outcome of selection is not determined by the relationships among genotype-specific growth rates.

**Common buzzard.** In the population of Figure 2.2, the heterozygote genotype DL has the highest population growth rate, and both boundary equilibria are
unstable, resulting in a protected polymorphism:

\[
\begin{align*}
\lambda_{DD} &= 0.48 \\
\lambda_{DL} &= 1.04 \\
\lambda_{LL} &= 0.68 \\
\zeta_{DD} &= 1.891 \\
\zeta_{LL} &= 1.493
\end{align*}
\] (2.64)

In Figure 2.2(b), the DL genotype is introduced at low frequency in a population of DD individuals at its stable age distribution. Thus at the beginning, we see the smooth exponential decay of the DD genotype (blue, dotted lines) and dramatic fluctuations in the age and genotype composition of the DL and LL components (green/yellow solid lines and orange/red dashed lines). Eventually (at about 20 years) the entire joint age-genotype population structure stabilizes, and the population continues to decline. The rate of decline differs from that of the initial DD population, reflecting the new, polymorphic, population structure. The time scale for convergence of population dynamics is the same as the time scale over which the genotype distribution converges (Figure 2.2(a)). Thus ecological and evolutionary processes are clearly operating on comparable time scales in this case.

The genotype frequencies reach stable proportions of 18% light, 68% intermediate, and 14% dark individuals. Observed population frequencies for the period 1989–1999 reported by Krüger and Lindström (2001) are 29% light, 65% intermediate, and 6.0% dark. Whether this agreement is close or not is less interesting than the fact that the values indicate how much difference the neglected details of the buzzard life cycle (particularly assortative mating) can make.
2.5 Discussion

Gene frequency dynamics and population dynamics are both driven by the demographic processes of birth and death. Combining Mendelian genetics and demographic models makes it possible to analyze genotype×stage dynamics of species with arbitrarily complex life cycles. Such a demographic genetic model lays bare the choices about how genotypes map to phenotypes (see Coulson et al. [2006]) by specifying the $U_i$ and $F_i$ matrices for each genotype. The model allows an arbitrary degree of pleiotropy in genotype effects on the demographic phenotype. The result is the nonlinear matrix model in equations (2.24)–(2.28), that projects stage×genotype population structures (Figures 2.1 and 2.2). The model leads to analytical conditions for the maintenance of a genetic polymorphism (Theorem 1).

The data required to parametrize this Mendelian matrix model is the same as that required to parametrize any demographic model: survival, transition, and fertility rates of individuals in each $i$-state. These typically appear in entries of matrices $U$ and $F$. Now those rates must be measured for each genotype, appearing in the entries of the matrices $U_i$ and $F_i$. From such genotype-specific demographic data, one can obtain all the usual demographic output, in addition to the stage×genotype structure dynamics. For a comprehensive review of demographic models containing multiple classifications, see Caswell et al. (2018).

Our framework extends a well-developed body of work on the genetics of age-structured populations (Charlesworth 1994) by allowing any kind of demographic structure and by connecting the results to the mathematics of matrix population models. Other studies have explored this connection, but were limited to species-specific models (e.g. Coulson et al. [2011]’s study of a wolf population) or to equilibrium conditions (Diekmann et al. 2003).

Despite empirical evidence for the importance of fertility selection (Anderson and Watanabe 1974; Travis 1988; Pincheira-Donoso and Hunt 2017), general coexistence conditions for genotypes in the presence of fertility and viability selection have posed a challenge as it remains unclear how the fertility of a mating is determined. The consequences of different assumptions about the fertility of a mating were worked out for unstructured models in a series of papers between the 1950s and the 1980s (Penrose 1947; Owen 1953; Bodmer 1965; Pollak 1978; Hadeler and Liberman 1975; Feldman et al. 1983; Pollak 1978; and Clark and Feldman 1986) found that including differences between genotypes in fertility as well as survival results in the mean fitness in the population not always increasing. Our results extend this conclusion to a structured population genetic model, and to cases where selection may operate on other rates besides viability and fertility (e.g. growth or development).
2.5 Discussion

Our model is a one-sex model, so we implicitly restrict attention to traits with the same effect in male and female survival and development. This corresponds to standard practice in population ecology, where demographic analyses typically focus on females. Relaxing this assumption by including both sexes makes it possible to analyze, e.g., traits affecting male mating success, or female energy allocation. Sexual antagonism (i.e., intralocus sexual conflict, in which an allele has positive effects on one sex and negative effects on the other) are analyzed in a two-sex version of our model (de Vries and Caswell 2018b). Sexual antagonism has been shown to lead, in some cases, to evolutionary suicide, both theoretically (Kokko and Brooks 2003), and experimentally (Doherty et al. 2003; Martins et al. 2018).

In addition to a fully two-sex version of the model, the framework presented here can be extended in other ways. For example, more complicated ecological interactions can be included, such as non-linear demography, time-dependent demographic rates, interactions among species, or dependence on environmental resources. The model can also be extended to include more genetic details, including non-random mating, more than two alleles, or mutations. A time-dependent version of the model will make it applicable to species with both sexual and clonal reproduction (Orive 2001; Orive et al. 2017), such as rotifers (Zweerus et al. 2017).

It is well known that genotype-specific population growth rates ($\lambda_i$) are not reliable proxies for fitness when modeling sexual reproduction, because genotypes do not only produce copies of themselves. Nevertheless, there is a huge informal life-history theory literature that assumes selection can be described in terms of some version of $\lambda$ (or the related generational measure $R_0$).

Our Theorem 1 provides a general solution to the question of genotype coexistence by protected polymorphism, and we have shown that $\lambda$ can be a proxy for fitness, given certain simplifying assumptions (no fertility differences between genotypes and only one reproducing stage). Charlesworth’s extensive analysis of age-classified selection found several conditions under which heterozygote superiority in the genotype-specific intrinsic rate of increase $r = \log \lambda$ leads to unstable boundary equilibria and therefore to a protected polymorphism. One condition assumes weak selection. Another relaxes the assumption of weak selection but assumes no demographic differences between the sexes. Both cases involve issues relating to the relative rates of convergence of age structure and gene frequencies (e.g., Charlesworth 1994, p. 150).

We need not invoke weak selection, and by considering a gene that affects female fertility but does not affect male reproductive success, we also deviate from Charlesworth’s assumption of no demographic differences between the sexes.
The search for restrictive conditions under which heterozygote superiority in λ or r determines polymorphism becomes less compelling given the results from Theorem 1 based on ζ_{AA} and ζ_{aa}.

The framework presented in this paper makes genotype frequencies just one more type of demographic structure, differing from age, size, or stage structure only in the details of the reproduction process and the nonlinearity this creates. This approach to incorporating population genetics into matrix models has two advantages. First, our mathematical formulation makes it possible to obtain analytical results, such as the conditions for a protected polymorphism derived in this paper. Second, the detailed derivation of the model lays bare the assumptions required and therefore simplifies the task of extending the model to relax those assumptions, for example, by including more than two alleles, incorporating mutations, or adding males.

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Appendix 2.A  The projection matrix $\tilde{\mathbf{A}}$

In this Appendix, we will derive equation (2.32) from the main text, repeated here for convenience,

$$
\tilde{\mathbf{A}}(\tilde{\mathbf{p}}) = \begin{pmatrix}
U_{AA} + q_A^b F_{AA} & \frac{1}{2} q_A^b F_{Aa} & 0 \\
q_A^b F_{AA} & U_{Aa} + \frac{1}{2} F_{Aa} & q_A^b F_{aa} \\
0 & \frac{1}{2} q_A^b F_{Aa} & U_{aa} + q_a^b F_{aa}
\end{pmatrix}, \quad (2.A1)
$$

$$
= \tilde{\mathbf{U}} + \tilde{\mathbf{F}}. \quad (2.A2)
$$

We start with the survival and transition matrix, $\tilde{\mathbf{U}}$. Substituting equation (2.24),

$$
\mathbf{U} = \sum_{i=1}^{g} E_{ii} \otimes \mathbf{U}_i, \quad (2.A3)
$$

into the equation for $\tilde{\mathbf{U}}$ yields,

$$
\tilde{\mathbf{U}} = \sum_{i=1}^{g} K^T \mathbb{D} \mathbf{K} (E_{ii} \otimes \mathbf{U}_i). \quad (2.A4)
$$

Because individuals do not change their genotype once they are born,

$$
\tilde{\mathbf{U}} = \begin{pmatrix}
U_{AA} & 0 & 0 \\
0 & U_{Aa} & 0 \\
0 & 0 & U_{aa}
\end{pmatrix}, \quad (2.A5)
$$

where we have set $g = 3$.

The fertility matrix, $\tilde{\mathbf{F}}$, is rewritten in a similar way. We substitute equation (2.25),

$$
\mathbf{F} = \sum_{i=1}^{g} E_{ii} \otimes \mathbf{F}_i, \quad (2.A6)
$$

and equation (2.27),

$$
\mathbb{H} = \mathbf{I}_\omega \otimes \mathbf{H} (\mathbf{n}), \quad (2.A7)
$$

into the fertility part of equation (2.A2), yielding

$$
\tilde{\mathbf{F}} = K^T \mathbb{H} (\mathbf{n}) \mathbf{K} \mathbf{F} \quad (2.A8)
$$

$$
= \sum_{i=1}^{g} K^T (\mathbf{I}_\omega \otimes \mathbf{H}) \mathbf{K} (E_{ii} \otimes \mathbf{F}_i), \quad (2.A9)
$$

where the explicit dependence of the matrix $\mathbf{H}$ on $\mathbf{n}$ was dropped to avoid a proliferation of brackets. Note that $K^T (\mathbf{A} \otimes \mathbf{B}) \mathbf{K} = \mathbf{B} \otimes \mathbf{A}$ (Magnus and Neudecker 1979), so

$$
\tilde{\mathbf{F}} = \sum_{i=1}^{g} (\mathbf{H} \otimes \mathbf{I}_\omega) (E_{ii} \otimes \mathbf{F}_i). \quad (2.A10)
$$
Finally use \((A \otimes B)(C \otimes D) = AC \otimes BD\) to write
\[
\tilde{F} = \sum_{i=1}^{g} (HE_{ii}) \otimes F_i.
\] (2.A11)

To continue, the entries of the parent-offspring matrix, \(H\), are needed. The parent-offspring matrix is defined as,
\[
H = \begin{pmatrix} p'_1 & p'_2 & p'_3 \end{pmatrix},
\] (2.A12)
where \(p'_i\) is the genotype frequency vector in the offspring of a parent of genotype \(i\). We ignore mutations, so that the gene frequencies in the gametes are the same as the gene frequencies in the breeding population,
\[
r_i = q_i, \quad r_b = q_b.
\] (2.A13, 2.A14)

The offspring are formed by the random combination of gametes when a parent of genotype \(i\) mates with a member of the breeding population, so that
\[
p'_i = Z(q_i \otimes q_b),
\] (2.A15)
where \(q_i\) and \(q_b\) are respectively the vector of gene frequencies in gametes of genotype \(i\) and the vector of gene frequencies in gametes of the entire breeding population. The matrix \(Z\) is
\[
Z = \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix}.
\] (2.A16)

The three genotypes have the following gene frequency vectors:
\[
q_{AA} = \begin{pmatrix} 1 \\ 0 \end{pmatrix} \quad q_{Aa} = \begin{pmatrix} \frac{1}{2} \\ \frac{1}{2} \end{pmatrix} \quad q_{AA} = \begin{pmatrix} 0 \\ 1 \end{pmatrix}
\] (2.A17)

We denote the entries of \(q_b\), the vector of gene frequencies in gametes of the entire breeding population, as
\[
q_b = \begin{pmatrix} q_A^b \\ q_a^b \end{pmatrix}.
\] (2.A18)

Finally, substituting \(Z\) and \(q_{AA}\) into equation (2.A15) for \(i = 1\) yields,
\[
p'_1 = Z(r_1 \otimes r_b) = \begin{pmatrix} q_A^b \\ q_a^b \\ 0 \end{pmatrix}.
\] (2.A19)
Similarly, $\mathbf{p}_2'$ and $\mathbf{p}_3'$ can be expressed in terms of the parental genotype frequencies, this yields the following matrix $\mathbf{H}$

$$
\mathbf{H} = \begin{pmatrix}
q_A^b & \frac{1}{2}q_A^b & 0 \\
q_a^b & \frac{1}{2} & q_A^b \\
0 & \frac{1}{2}q_a^b & q_a^b
\end{pmatrix}.
$$

(2.A20)

The final step is to substitute the above expression for $\mathbf{H}$ into equation (2.A11), which yields

$$
\tilde{\mathbf{F}}(\tilde{\mathbf{p}}) = \begin{pmatrix}
q_A^b F_{AA} & \frac{1}{2}q_A^b F_{Aa} & 0 \\
q_a^b F_{AA} & \frac{1}{2}F_{Aa} & q_A^b F_{aa} \\
0 & \frac{1}{2}q_a^b F_{Aa} & q_a^b F_{aa}
\end{pmatrix}.
$$

(2.A21)
Appendix 2.B  Coexistence conditions

In this Appendix, we will derive the linearization of the model defined by the transition matrix (2.28) at the boundary equilibria, and prove that the eigenvalues of the Jacobian matrix $M$ are given by Theorem 1.

**Linear demography, any number of reproducing stages**

We consider first the general case, in which demography is described by an age- or stage-structured life cycle, with any number of reproducing stages. We assume that the demographic component of the model is linear (nonlinear demography will be considered elsewhere). The dynamics of the population frequency vector are

$$\hat{\mathbf{p}}(t + 1) = \frac{\hat{\mathbf{A}}(\hat{\mathbf{p}}(t))\hat{\mathbf{p}}(t)}{\|\hat{\mathbf{A}}(\hat{\mathbf{p}}(t))\hat{\mathbf{p}}(t)\|},$$

(2.B1)

where $\|\mathbf{a}\|$ indicates the 1-norm of the vector $\mathbf{a}$, defined as the sum of the absolute values of the entries of the vector $\mathbf{a}$. The matrix $\hat{\mathbf{A}}$ is given by equation (2.32),

$$\hat{\mathbf{A}}(\hat{\mathbf{p}}) = \begin{pmatrix}
U_{AA} + q_A^b F_{AA} & \frac{1}{2} q_A^b F_{Aa} & 0 \\
q_A^b F_{AA} & U_{AA} + \frac{1}{2} F_{AA} & q_A^b F_{aa} \\
0 & \frac{1}{2} q_a^b F_{Aa} & U_{aa} + q_a^b F_{aa}
\end{pmatrix}.$$  

(2.B2)

An equilibrium solution, $\hat{\mathbf{p}}$, must satisfy

$$\hat{\mathbf{p}} = \frac{\hat{\mathbf{A}}(\hat{\mathbf{p}})\hat{\mathbf{p}}}{\mathbf{1}^T g_\omega \hat{\mathbf{A}}(\hat{\mathbf{p}})\hat{\mathbf{p}}},$$

(2.B3)

where the one norm can be replaced by $\mathbf{1}^T g_\omega \hat{\mathbf{A}}(\hat{\mathbf{p}})\hat{\mathbf{p}}$ because $\hat{\mathbf{p}}$ is nonnegative.

The Jacobian matrix at an equilibrium $\hat{\mathbf{p}}$ is

$$M = \left. \frac{d\hat{\mathbf{p}}(t + 1)}{d\hat{\mathbf{p}}^T(t)} \right|_{\hat{\mathbf{p}}}.$$  

(2.B4)

This differentiation is carried out using matrix calculus; for details of the methodology see [Magnus and Neudecker (1985, 1988)]; for ecological presentations see [Caswell (2007b, 2008, 2018)]. Matrix calculus makes extensive use of the vec operator, which stacks the columns of a matrix; e.g.,

$$\text{vec} \begin{pmatrix}
a & b \\
c & d
\end{pmatrix} = \begin{pmatrix}
a \\
c \\
b \\
d
\end{pmatrix}$$

(2.B5)

and of the fact that

$$\text{vec} \mathbf{ABC} = (\mathbf{C}^T \otimes \mathbf{A}) \text{vec} \mathbf{B}$$

(2.B6)
for any conformable matrices $A$, $B$, and $C$, where $\otimes$ is the Kronecker product.

For notational convenience, define a matrix $B$ as

$$B(\tilde{p}) = \frac{\tilde{A}(\tilde{p})}{1 \otimes \omega \tilde{A}(\tilde{p})\tilde{p}},$$  \hfill (2.B7)

so that

$$\tilde{p}(t + 1) = B(\tilde{p}(t))\tilde{p}(t).$$  \hfill (2.B8)

Differentiate equation (2.B8) to obtain

$$d\tilde{p}(t + 1) = Bd\tilde{p}(t) + \left(dB\right)\tilde{p}(t),$$ \hfill (2.B9)

where the explicit dependence of $B$ on $\tilde{p}$ has been omitted to avoid a clutting of brackets. Apply the vec operator to both sides, to obtain

$$d\tilde{p}(t + 1) = Bd\tilde{p}(t) + (\tilde{p}^T(t) \otimes I_{\omega g})d\text{vec}B.$$ \hfill (2.B10)

Thus

$$M = \left. \frac{d\tilde{p}(t + 1)}{d\tilde{p}(t)} \right|_{\tilde{p}} = \left. B(\tilde{p}) + (\tilde{p}^T(t) \otimes I_{\omega g}) \left. \frac{\partial \text{vec}B(\tilde{p})}{\partial \tilde{p}^T} \right|_{\tilde{p}} \right).$$ \hfill (2.B11)

(Verdy and Caswell 2008)

We will express the Jacobian matrix $M$ in terms of the genotype specific matrices, $U_i$ and $F_i$, the genotype-specific growth rates, and the boundary equilibrium population structures. We choose to analyze the Jacobian at the $AA$ boundary; the expression at the $aa$ boundary can be derived afterwards by symmetry.

We define the scalar function $f(\tilde{p})$ as

$$f(\tilde{p}) = \frac{1}{\tilde{1}_g \tilde{A}(\tilde{p})\tilde{p}},$$ \hfill (2.B13)

so that

$$B(\tilde{p}) = f(\tilde{p})\tilde{A}(\tilde{p}).$$ \hfill (2.B14)

Where it does not create confusion, we will drop the explicit dependence of $\tilde{A}$, $B$, and $f$ on $\tilde{p}$. Differentiate equation (2.B14) and take the vec of both sides to obtain

$$d\text{vec}B = \text{vec}\tilde{A}(df) + f d\text{vec}\tilde{A},$$ \hfill (2.B15)

or

$$\frac{\partial \text{vec}B}{\partial \tilde{p}^T} = \text{vec}\tilde{A} \frac{\partial f}{\partial \tilde{p}^T} + f(\tilde{p}) \frac{\partial \text{vec}\tilde{A}}{\partial \tilde{p}^T}. \hfill (2.B16)$$
Differentiating \( f \) in equation (2.B13) gives

\[
df = \frac{-1}{\left( \Omega_\omega^T \tilde{A}(\hat{p})\hat{p} \right)^2} \left[ \Omega_\omega^T \left( \frac{d\tilde{A}}{dA} \right) \hat{p} + \Omega_\omega^T \tilde{A} d\hat{p} \right]. \tag{2.B17}\]

At the \( AA \) boundary, \( \tilde{A}(\hat{p})\hat{p} = \lambda_{AA} \hat{p} \), and therefore

\[
\Omega_\omega^T \tilde{A}(\hat{p})\hat{p} = \lambda_{AA}. \tag{2.B18}\]

Evaluate the differential of \( f \) at the boundary and use equation (2.B18) to obtain

\[
df = \frac{-1}{\lambda_{AA}^2} \left[ \Omega_\omega^T \left( \frac{d\tilde{A}}{dA} \right) \hat{p} + \Omega_\omega^T \tilde{A} d\hat{p} \right]. \tag{2.B19}\]

The first term in this sum, \( \Omega_\omega^T \left( \frac{d\tilde{A}}{dA} \right) \hat{p} = 0 \) when evaluated at the boundary. To see this, recall that the population vector at the \( AA \) boundary is

\[
\hat{p} = \begin{pmatrix} \hat{p}_{AA} \\ 0 \\ 0 \end{pmatrix}. \tag{2.B20}\]

The last two block columns of the matrix \( d\tilde{A} \) are therefore multiplied by zero, yielding

\[
\Omega_\omega^T \left( \frac{d\tilde{A}}{dA} \right) \hat{p} = \Omega_\omega^T F_{AA} \hat{p} - \Omega_\omega^T d_{AA} (dq_A + dq_A^b), \tag{2.B21}\]

\[
= 0, \tag{2.B22}\]

because

\[
q_a^b + q_A^b = 1, \tag{2.B23}\]

\[
dq_a^b + dq_A^b = 0. \tag{2.B24}\]

Substituting equation (2.B19) into equation (2.B15) and evaluating at the boundary yields

\[
dvec{B} = \frac{-1}{\lambda_{AA}^2} \text{vec} \tilde{A} \left[ \Omega_\omega^T \tilde{A} d\hat{p} \right] + \frac{1}{\lambda_{AA}} \text{dvec} \tilde{A}, \tag{2.B25}\]

or

\[
\frac{\partial \text{vec} B}{\partial \hat{p}^T} \biggm|_{\hat{p}} = \frac{-1}{\lambda_{AA}^2} \left( \text{vec} \tilde{A} \right) \left( \Omega_\omega^T \tilde{A} \right) + \frac{1}{\lambda_{AA}} \frac{\partial \text{vec} \tilde{A}}{\partial \hat{p}^T} \biggm|_{\hat{p}}. \tag{2.B26}\]
Finally substituting the expression above into equation (2.B12) yields an expression for the Jacobian matrix:

\[
M = B(\hat{p}) + (\hat{p}^T \otimes I_{\omega_g}) \frac{\partial \text{vec} B}{\partial \hat{p}^T}_{\hat{p}},
\]

\[
= B(\hat{p}) - \frac{1}{\lambda_{AA}^2} (\hat{p}^T \otimes I_{\omega_g}) (\text{vec} \hat{A}) \left( I_{\omega_g}^T \hat{A} \right)
\]

\[
+ \frac{1}{\lambda_{AA}} (\hat{p}^T \otimes I_{\omega_g}) \frac{\partial \text{vec} \hat{A}}{\partial \hat{p}^T}_{\hat{p}}
\]

where we have identified the three terms as \(A\), \(B\), and \(C\). We address each of these in turn.

**Derivation of Term \(A\).** Evaluating \(B(\hat{p})\) from equation (2.B14), we find that

\[
B(\hat{p}) = \frac{1}{\lambda_{AA}} \left( \begin{array}{c|c|c}
U_{AA} + F_{AA} & 1/2 F_{Aa} & 0 \\
0 & U_{Aa} + 1/2 F_{Aa} & F_{aa} \\
0 & 0 & U_{aa}
\end{array} \right).
\]

**Derivation of Term \(B\).** This term is given by

\[
\otimes = - \frac{1}{\lambda_{AA}^2} (\hat{p}^T \otimes I_{\omega_g}) (\text{vec} \hat{A}) \left( I_{\omega_g}^T \hat{A} \right).
\]

Using Roth’s theorem, this simplifies to

\[
(\hat{p}^T \otimes I_{\omega_g}) \text{vec} \left( \hat{A} (\hat{p}) \right) = \text{vec} \left( I_{\omega_g} \hat{A}(\hat{p}) \hat{p} \right) = \lambda_{AA} \hat{p},
\]

so that

\[
\otimes = - \frac{1}{\lambda_{AA}} \hat{p} \left( I_{\omega_g}^T \hat{A} (\hat{p}) \right).
\]

Substituting \(\hat{p}\) from equation (2.B20) into equation (2.B32) and writing the result in terms of the block matrices yields

\[
\otimes = - \frac{1}{\lambda_{AA}} \left( \begin{array}{c|c|c}
\hat{p}_{AA} \otimes I_{\omega_g} (U_{AA} + F_{AA}) & \hat{p}_{AA} \otimes I_{\omega_g} (U_{Aa} + F_{Aa}) & \hat{p}_{AA} \otimes I_{\omega_g} (U_{aa} + F_{aa}) \\
0 & 0 & 0 \\
0 & 0 & 0
\end{array} \right).
\]
Derivation of Term \((\mathbb{C})\) Term \((\mathbb{C})\) requires the most elaborate derivation. We first derive a useful expression for vec \(\tilde{A}\) in terms of its component block matrices. Recall that \(\tilde{A}\) can be decomposed into nine \(\omega \times \omega\) block matrices, as in equation (2.32),

\[
\tilde{A}(\tilde{p}) = \begin{pmatrix}
    U_{AA} + F_{AA}q_A^b & \frac{1}{2}F_{Aa}q_A^a & 0 \\
    F_{AA}q_a^b & U_{Aa} + \frac{1}{2}F_{Aa} & F_{aa}q_a^b \\
    0 & \frac{1}{2}F_{Aa}q_a^b & U_{aa} + F_{aa}q_a^b
\end{pmatrix}
\] (2.B34)

Denote the blocks by \(A_{ij}\), so that \(\tilde{A}\) can be written as

\[
\tilde{A} = \sum_{i,j=1}^{3} E_{ij} \otimes A_{ij},
\] (2.B35)

where we have used the definition of the matrix \(E_{ij} = e_i e_j^T\). Using the fact that \(AC \otimes BD = (A \otimes B)(C \otimes D)\), equation (2.B36) can be rewritten as

\[
\tilde{A} = \sum_{i,j=1}^{3} (e_i \otimes A_{ij}) (e_j^T \otimes I_\omega).
\] (2.B37)

Next use the identity \(\sum_i (e_i \otimes I_\omega) A_{ij} = \sum_i e_i \otimes A_{ij}\) to write

\[
\tilde{A} = \sum_{i,j=1}^{3} (e_i \otimes I_\omega) A_{ij} (e_j^T \otimes I_\omega).
\] (2.B38)

and apply the vec operator to obtain

\[
\text{vec} \tilde{A} = \sum_{i,j=1}^{3} (e_j \otimes I_\omega) \otimes (e_i \otimes I_\omega) \text{vec} A_{ij}.
\] (2.B39)

Armed with this expression for vec \(A\), we analyze term \((\mathbb{C})\) in the Jacobian. Replace the derivative of vec \(A\) with equation (2.B39), such that

\[
\frac{1}{\lambda_{AA}} (\hat{p}^T \otimes I_{\omega g}) \frac{\partial \text{vec} A}{\partial \hat{p}^T} = \frac{1}{\lambda_{AA}} \sum_{i,j=1}^{3} (e_j \otimes I_\omega) \otimes (e_i \otimes I_\omega) \frac{\partial \text{vec} A_{ij}}{\partial \hat{p}^T},
\] (2.B40)

where all derivatives are evaluated at \(\hat{p}\). Use \((A \otimes B)(C \otimes D) = AC \otimes BD\) to rewrite

\[
(\hat{p}^T \otimes I_{\omega g}) \left[ (e_j \otimes I_\omega) \otimes (e_i \otimes I_\omega) \right]=\left[ \hat{p}^T (e_j \otimes I_\omega) \right] \otimes \left[ I_{\omega g} (e_i \otimes I_\omega) \right].
\] (2.B41)
Substituting this expression into the right hand side of equation (2.B40) yields
\[
\frac{1}{\lambda_{AA}} (\hat{p}^T \otimes I_{\omega g}) \frac{\partial \text{vec}A}{\partial \hat{p}^T} = \frac{1}{\lambda_{AA}} \sum_{i,j=1}^{3} \left[ \hat{p}_i^T (e_j \otimes I_{\omega}) \right] \otimes \left[ I_{\omega g} (e_i \otimes I_{\omega}) \right] \frac{\partial \text{vec}A_{ij}}{\partial \hat{p}^T}. 
\]
(2.B42)

Substitute \( \hat{p}^T = (\hat{p}_{AA}^T, 0, 0) \) into the right-hand side of equation (2.B42), so that only terms with \( j = 1 \) are nonzero, yielding
\[
\frac{1}{\lambda_{AA}} (\hat{p}^T \otimes I_{\omega g}) \frac{\partial \text{vec}A}{\partial \hat{p}^T} = \frac{1}{\lambda_{AA}} \sum_{i=1}^{3} \left[ \hat{p}_{AA}^T \otimes (e_1 \otimes I_{\omega}) \right] \frac{\partial \text{vec}A_{11}}{\partial \hat{p}^T},
\]
(2.B43)

Write down each term in the sum over \( i \) and take the derivative of \( \text{vec}A_{11} = \text{vec} (U_{AA} + q_{b}^b F_{AA}) \) and \( \text{vec}A_{21} = \text{vec} (q_{a}^b F_{AA}) \) to obtain
\[
\frac{1}{\lambda_{AA}} (\hat{p}^T \otimes I_{\omega g}) \frac{\partial \text{vec}A}{\partial \hat{p}^T} = \frac{1}{\lambda_{AA}} \left[ \hat{p}_{AA}^T \otimes (e_1 \otimes I_{\omega}) \right] \frac{\partial \text{vec}(F_{AA})}{\partial \hat{p}^T}. 
\]
(2.B44)

Finally apply Roths theorem (Roth 1934), \((C^T \otimes A) \text{vec}B = \text{vec}ABC\), to the equation above (with \( C^T = \hat{p}_{AA}^T \), \( A = (e_1 \otimes I_{\omega}) \), and \( \text{vec}B = \text{vec}F_{AA} \)) to write
\[
\frac{1}{\lambda_{AA}} (\hat{p}^T \otimes I_{\omega g}) \frac{\partial \text{vec}A}{\partial \hat{p}^T} = \frac{1}{\lambda_{AA}} \left[ \text{vec} ((e_1 \otimes I_{\omega}) F_{AA} \hat{p}_{AA}) - \text{vec} ((e_2 \otimes I_{\omega}) F_{AA} \hat{p}_{AA}) \right] \frac{\partial q_{b}^b}{\partial \hat{p}^T}. 
\]
(2.B45)

where, throughout, all derivatives are evaluated at \( \hat{p} \). Written in terms of block matrices this expression yields
\[
\frac{1}{\lambda_{AA}} (\hat{p}^T \otimes I_{\omega g}) \frac{\partial \text{vec}A}{\partial \hat{p}^T} = 
\frac{1}{\lambda_{AA}} \begin{pmatrix}
(F_{AA} \hat{p}_{AA}) \otimes \frac{\partial q_{b}^b}{\partial \hat{p}_{AA}^T} & (F_{AA} \hat{p}_{AA}) \otimes \frac{\partial q_{a}^b}{\partial \hat{p}_{AA}^T} & (F_{AA} \hat{p}_{AA}) \otimes \frac{\partial q_{a}^b}{\partial \hat{p}_{AA}^T} \\
-(F_{AA} \hat{p}_{AA}) \otimes \frac{\partial q_{a}^b}{\partial \hat{p}_{AA}^T} & -(F_{AA} \hat{p}_{AA}) \otimes \frac{\partial q_{b}^b}{\partial \hat{p}_{AA}^T} & -(F_{AA} \hat{p}_{AA}) \otimes \frac{\partial q_{a}^b}{\partial \hat{p}_{AA}^T} \\
0 & 0 & 0
\end{pmatrix}. 
\]
(2.B46)

Equation (2.B46) requires the derivative of the frequency of allele \( A \) in the gamete pool with respect to the population frequency vector:
\[
\frac{\partial q_{b}^b}{\partial \hat{p}^T}. 
\]
(2.B47)

Differentiating equation (2.11) from the main text,
\[
q_{A} = e_{1}^T q_{b} = e_{1}^T W_{b}, 
\]
(2.B48)
yields
\[
\frac{\partial q_{b}^b}{\partial \hat{p}^T} = e_{1}^T W_{b} \frac{\partial p_{b}}{\partial \hat{p}^T}. 
\]
(2.B49)
Combine equations (2.5) and (2.7) from the main text to write
\[ p_b = \frac{\sum_{i=1}^{g} (E_{ii} \otimes c_i^T) \tilde{p}}{\sum_{j=1}^{g} (e_j^T \otimes c_j^T) \tilde{p}}, \]  
(2.B50)
where we can substitute \( \tilde{p} \) for \( \tilde{n} \) because of homogeneity. The denominator gives the fraction of individuals in the total population that are in a breeding stage,
\[ \sum_{j=1}^{g} (e_j^T \otimes c_j^T) \tilde{p} = p_b. \]  
(2.B51)
Taking the derivative of \( p_b \) yields
\[ \frac{\partial p_b}{\partial \tilde{p}^T} \bigg|_{\tilde{p}} = \frac{p_b \sum_{i=1}^{3} (E_{ii} \otimes c_i^T) - \sum_{i=1}^{3} (E_{ii} \otimes c_i^T) \hat{p} \sum_{j=1}^{3} (e_j^T \otimes c_j^T)}{p_b^2}. \]  
(2.B52)
Writing above expression as a matrix yields
\[ \frac{\partial p_b}{\partial \tilde{p}^T} \bigg|_{\tilde{p}} = 1 \frac{p_b}{p_b^2} \begin{pmatrix} 0 & -c_{Aa}^T & -c_{aa}^T \\ 0 & c_{Aa}^T & 0 \\ 0 & 0 & c_{aa}^T \end{pmatrix}. \]  
(2.B53)
Substituting equation (2.B53) into equation (2.B49) leads to
\[ \frac{\partial q_b^A}{\partial \tilde{p}^T} \bigg|_{\tilde{p}} = e_1^T W \frac{\partial p_b}{\partial \tilde{p}^T} \bigg|_{\tilde{p}} \]  
(2.B54)
\[ = \begin{pmatrix} 0 & -\frac{1}{2} c_{Aa}^T - \frac{c_{aa}^T}{p_b} \end{pmatrix}. \]  
(2.B55)
Finally, plugging equation (2.B55) into (2.B46) yields
\[ \frac{1}{\lambda_{AA}} (p^T \otimes I_{\omega g}) \frac{\partial \text{vec} A}{\partial \tilde{p}^T} \bigg|_{\tilde{p}} = \frac{1}{\lambda_{AA}} \begin{pmatrix} 0 & -\frac{1}{2p_b} (F_{AA} \hat{p}_{AA}) \otimes c_{Aa}^T & -\frac{1}{p_b} (F_{AA} \hat{p}_{AA}) \otimes c_{aa}^T \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}. \]  
(2.B56)

The Jacobian matrix. Combining the terms by substituting equations (2.B29), (2.B33), and (2.B56) into equation (2.B28), we obtain the Jacobian:
\[ M = \frac{1}{\lambda_{AA}} \begin{pmatrix} U_{AA} + F_{AA} & \frac{1}{2} F_{Aa} \\ 0 & U_{Aa} + \frac{1}{2} F_{Aa} \\ 0 & 0 & U_{aa} \end{pmatrix} \]
\[ - \frac{1}{\lambda_{AA}} \begin{pmatrix} \hat{p}_{AA} \otimes I_{\omega}^T (U_{AA} + F_{AA}) & \hat{p}_{AA} \otimes I_{\omega}^T (U_{Aa} + F_{Aa}) & \hat{p}_{AA} \otimes I_{\omega}^T (U_{aa} + F_{aa}) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \]
\[ + \frac{1}{\lambda_{AA}} \begin{pmatrix} 0 & -\frac{1}{2p_b} (F_{AA} \hat{p}_{AA}) \otimes c_{Aa}^T & -\frac{1}{p_b} (F_{AA} \hat{p}_{AA}) \otimes c_{aa}^T \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}. \]  
(2.B57)
2.B Coexistence conditions

**Eigenvalues of the Jacobian.** The Jacobian matrix, given by equation (2.B.57), is upper block triangular, so the eigenvalues of $\mathbf{M}$ are the eigenvalues of the diagonal blocks,

\[
\begin{align*}
\mathbf{M}_{11} &= \frac{1}{\lambda_{AA}} \left( \mathbf{U}_{AA} + \mathbf{F}_{AA} - \mathbf{p}_{AA} \otimes \mathbf{1}_s^T (\mathbf{U}_{AA} + \mathbf{F}_{AA}) \right) \\
\mathbf{M}_{22} &= \frac{1}{\lambda_{AA}} \left( \mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{AA} \mathbf{p}_{AA}) \otimes \mathbf{c}_{Aa}^T \right) \\
\mathbf{M}_{33} &= \frac{1}{\lambda_{AA}} \mathbf{U}_{aa}
\end{align*}
\] 

(2.B.58)

The largest absolute eigenvalue of the Jacobian, i.e. the spectral radius $\rho(\mathbf{M})$, determines the stability of the boundary equilibrium. The block $\mathbf{M}_{11}$ projects perturbations within the $AA$ boundary, and because $\mathbf{p}$ is stable to perturbations in that boundary, $\rho(\mathbf{M}_{11}) < 1$. Block $\mathbf{M}_{33}$ projects perturbations in the $aa$ direction. In the neighbourhood of the $AA$ equilibrium, $aa$ homozygotes are negligibly rare, and thus $\mathbf{M}_{33}$ normally does not determine the stability of $\mathbf{M}$. An exception occurs when $\lambda_{AA} < \rho(\mathbf{U}_{aa}) < 1$. That is, if the $AA$ population is declining sufficiently rapidly, the $aa$ homozygote may increase in frequency simply by declining to extinction more slowly. If the homozygous $AA$ population is stable or increasing, so that $\lambda_{AA} \geq 1$, this cannot happen. Similarly, if $\mathbf{U}_{aa}$ is age-classified with a maximum age, $\rho(\mathbf{U}_{aa}) = 0$, and the phenomenon can not happen. We neglect this pathological case in our discussions.

We thus focus our stability analysis on growth in the $Aa$ direction, which means we focus on the middle block,

\[
\mathbf{M}_{22} = \frac{1}{\lambda_{AA}} \left( \mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{AA} \mathbf{p}_{AA}) \otimes \mathbf{c}_{Aa}^T \right).
\] 

(2.B.61)

The largest absolute value of the eigenvalues of this matrix, the dominant eigenvalue, evaluated at the $AA$ boundary, denoted by $\zeta_{AA}$, is

\[
\zeta_{AA} = \frac{1}{\lambda_{AA}} \rho \left( \mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{AA} \mathbf{p}_{AA}) \otimes \mathbf{c}_{Aa}^T \right).
\] 

(2.B.62)

By symmetry, at the $aa$ boundary,

\[
\zeta_{aa} = \frac{1}{\lambda_{aa}} \rho \left( \mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{aa} \mathbf{p}_{aa}) \otimes \mathbf{c}_{Aa}^T \right).
\] 

(2.B.63)

If both boundaries are unstable, then both alleles will coexist. The coexistence conditions are therefore given by

\[
\rho \left( \mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{AA} \mathbf{p}_{AA}) \otimes \mathbf{c}_{Aa}^T \right) > \lambda_{AA},
\] 

(2.B.64)

\[
\rho \left( \mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{aa} \mathbf{p}_{aa}) \otimes \mathbf{c}_{Aa}^T \right) > \lambda_{aa}.
\] 

(2.B.65)

This completes the derivation of Theorem 1.
2. Selection in one-sex stage-structured populations

Special case: a single reproducing stage

Corollary 1 uses the eigenvalues \( \zeta_{AA} \) and \( \zeta_{aa} \) for the special case in which the life cycle contains only a single breeding stage, but with no restrictions on the newborn and juvenile stages.

Our goal is to simplify the final term in the expressions for \( \zeta_{AA} \) and \( \zeta_{aa} \) by showing that

\[
\frac{1}{2p_b}(F_{AA}\hat{p}_{AA}) \otimes e_{\omega}^\top = \frac{1}{2} F_{AA} \quad \text{(2.B66)}
\]

\[
\frac{1}{2p_b}(F_{aa}\hat{p}_{aa}) \otimes e_{\omega}^\top = \frac{1}{2} F_{aa}. \quad \text{(2.B67)}
\]

Without loss of generality, let the reproducing stage be the last stage, stage \( \omega \), for all 3 genotypes. Then \( c_i = c = e\omega, \hat{p}_{AA}(\omega) = p_b, \) and \( \hat{p}_{aa}(\omega) = p_b. \) The matrix \( F_{AA} \) has a single nonzero column, denoted by \( f_{AA} \), giving the production of the various types of offspring produced by the reproducing stage, i.e.

\[
F_{AA} = \left( \begin{array}{cccc}
0 & \cdots & 0 & f_{AA}
\end{array} \right). \quad \text{(2.B68)}
\]

Thus

\[
\frac{1}{2p_b}(F_{AA}\hat{p}_{AA}) \otimes e_{\omega} = \frac{1}{2p_b} p_b f_{AA} \otimes e_{\omega}^\top, \quad \text{(2.B69)}
\]

\[
= \frac{1}{2} F_{AA}. \quad \text{(2.B70)}
\]

Likewise,

\[
\frac{1}{2p_b}(F_{aa}\hat{p}_{aa}) \otimes e_{\omega}^\top = \frac{1}{2p_b} p_b f_{aa} \otimes e_{\omega}^\top, \quad \text{(2.B71)}
\]

\[
= \frac{1}{2} F_{aa}. \quad \text{(2.B72)}
\]

Substituting (2.B70) and (2.B72) into \( \zeta_{AA} \) and \( \zeta_{aa} \) yields Corollary 1.