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Stage-Structured Evolutionary Demography: Linking Life Histories, Population Genetics, and Ecological Dynamics

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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 ecological components of the model may be stage classified or age classified, linear or nonlinear, time invariant or time varying, and deterministic or stochastic. 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 $\times$ 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 (λ) do not determine the outcome of selection. Except in restrictive special cases, heterozygote superiority in λ is neither necessary nor sufficient for a genetic polymorphism. As a consequence, the 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.

Keywords: evolutionary demography, population genetics, eco-evolutionary dynamics, genotype coexistence, heterozygote superiority, protected polymorphisms.

Introduction

Both evolutionary change and population dynamics are 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 births and deaths within that population. That is, evolutionary change is a consequence of demographic processes.¹ 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 (for an earlier version of these ideas, see Lewontin 1974). 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 timescales include the rapid evolution of resistance to antibiotics and pesticides as well as the 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 number 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 (1971) combined

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1. In our terminology, “ecological” processes are those involved in population growth and interaction, and “demographic” models are those that analyze ecological processes in terms of internal population structure, distinguishing individuals on the basis of age, developmental stage, size, and so on. Demographic models, in our terminology, are a subset of ecological models.

population genetics with nonlinear ecological models by writing genotype fitnesses as functions of intra- and interspecific densities but neglected population structure. Charlesworth developed a theory for age-classified population genetics (Charlesworth 1970, 1972; Charlesworth and Giesel 1972*a*, 1972*b*). 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 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 (Falconer 1960). These models focus on the components of phenotypic variance (genetic, environmental, genetic $\times$ environmental, etc.; 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 (Lande 1982*b*) and stage-classified (Barfield et al. 2011) demographic models. A more general structured approach based on an integral projection model for trait distributions has been presented by Coulson and Tuljapurkar (2008). Childs et al. (2016) extend this framework to include both sexes and develop an extension of the age-structured Price equation for two-sex populations.

Adaptive dynamics (Metz et al. 1992; Dieckmann 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 (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 timescale and a faster ecological timescale. Adaptive dynamics has been combined with the framework of integral projection models to study the evolution of function-valued traits (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 (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, linear or nonlinear, time invariant or time varying, and deterministic or stochastic. The ecological components may also include dependence on environmental resources or interactions among species, although we do not include that

here. We allow genotypes to affect 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 that is 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 whether one or another allele will go to fixation.

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, and migration, all of which could affect both males and females. It excludes traits such as male courtship displays, ornamental plumage, and so on. A two-sex version of the model, which includes males explicitly and relaxes these restrictions, will be presented elsewhere (C. de Vries and H. Caswell, unpublished manuscript).

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 ($1, \dots, \omega$) and genotype ($1, \dots, g$). 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 article are listed in table 1.

Table 1: Mathematical notation used in this article

Symbol	Definition	Dimension
a	Number of alleles (2)	
g	Number of genotypes (3)	
ω	Number of stages	
N	Total population size	
N_b	Breeding population size	
$\mathbf{c}_i$	Indicator vector for breeding stages in genotype i	$\omega \times 1$
$\tilde{\mathbf{n}}$	Joint stage-genotype vector	$\omega g \times 1$
$\tilde{\mathbf{p}}$	Joint stage-genotype frequency vector	$\omega g \times 1$
$\mathbf{p}_i$	Genotype frequency vector in genotype i	$g \times 1$
$\mathbf{p}'_i$	Genotype frequency vector of the offspring of genotype i	$g \times 1$
$\mathbf{p}_b$	Genotype frequency vector in breeding population	$g \times 1$
$\mathbf{q}$	Gene frequency vector in population	$a \times 1$
$\mathbf{q}_i$	Gene frequency vector in genotype i	$a \times 1$
$\mathbf{q}_b$	Gene frequency vector in breeding population	$a \times 1$
$\mathbf{r}_i$	Gene frequency in gametes from genotype i	$a \times 1$
$\mathbf{r}_b$	Gene frequency in gametes from breeding population	$a \times 1$
$\mathbf{I}_\omega$	Identity matrix	$\omega \times \omega$
$\mathbf{1}_g$	Vector of ones	$g \times 1$
$\mathbf{e}_i$	The i th unit vector, with a 1 in the i th entry and zeros elsewhere	Various
$\mathbf{E}_{ij}$	A matrix with a 1 in the (i, j) position and zeros elsewhere	Various
$\otimes$	Kronecker product	
$\text{vec}\mathbf{X}$	The vec operator, which stacks the columns of an $m \times n$ matrix $\mathbf{X}$ into an $mn \times 1$ vector	
$\mathbf{U}_i$	Demographic transitions for genotype i	$\omega \times \omega$
$\mathbf{F}_i$	Fertility matrix for genotype i	$\omega \times \omega$
$\mathbf{D}_i$	Genotype transitions for stage i	$g \times g$
$\mathbf{H}_i(\tilde{\mathbf{n}})$	Parent-offspring genotype map for stage i	$g \times g$

Note: Dimensions of vectors and matrices are given where relevant.

The Component Matrices

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

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

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:

$$\tilde{\mathbf{n}}(t) = \text{vec}\mathcal{N}(t). \quad (2)$$

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:

$$\tilde{\mathbf{n}}(t) = \begin{bmatrix} \mathbf{n}_{AA}(t) \\ \mathbf{n}_{Aa}(t) \\ \mathbf{n}_{aa}(t) \end{bmatrix}. \quad (3)$$

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

$$\tilde{\mathbf{n}}(t + 1) = \tilde{\mathbf{A}}[\tilde{\mathbf{n}}]\tilde{\mathbf{n}}(t). \quad (4)$$

The matrix $\tilde{\mathbf{A}}$ depends on $\tilde{\mathbf{n}}$ 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:

$$\begin{aligned} \mathbf{U}_i & \text{demographic transitions for genotype } i \\ & i = 1, \dots, g \quad \omega \times \omega, \\ \mathbf{F}_i & \text{fertility matrix for genotype } i \\ & i = 1, \dots, g \quad \omega \times \omega, \\ \mathbf{D}_i & \text{genotype transitions for stage } i \\ & i = 1, \dots, \omega \quad g \times g, \\ \mathbf{H}_i(\tilde{\mathbf{n}}) & \text{parent-offspring genotype map for stage } i \\ & i = 1, \dots, \omega \quad g \times g. \end{aligned}$$

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 $\mathbf{H}_i(\tilde{\mathbf{n}})$ 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 article, we assume that mating is random with respect to stage and hence that the parent-offspring map is the same for all stages, that is, $\mathbf{H}_i(\tilde{\mathbf{n}}) = \mathbf{H}(\tilde{\mathbf{n}})$. The matrix $\mathbf{H}(\tilde{\mathbf{n}})$ 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 nonreproductive (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 $j = 1, \dots, g$ that show which stages of genotype j take part in mating. That is, the i th entry of $\mathbf{c}_j$ 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:

- $\mathbf{p}$ = genotype frequencies in the overall population,
- $\mathbf{p}_b$ = genotype frequencies in the breeding population,
- $\mathbf{p}_i$ = genotype frequencies in genotype i ($= \mathbf{e}_i$),
- $\mathbf{p}'_i$ = genotype frequencies in the offspring of genotype i .

The size of the breeding population is

$$N_b = \sum_{j=1}^g (\mathbf{e}_j^\top \otimes \mathbf{c}_j^\top) \tilde{\mathbf{n}}, \quad (5)$$

where $\mathbf{e}_j$ 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, $\mathbf{c}_j = \mathbf{c}$ for all genotypes j and

$$N_b = (\mathbf{1}_g^\top \otimes \mathbf{c}^\top) \tilde{\mathbf{n}}, \quad (6)$$

where $\mathbf{1}_g^\top$ is a vector of ones of dimensions $1 \times g$.

The genotype frequency vector in the breeding population is

$$\mathbf{p}_b = \frac{\mathbf{X} \tilde{\mathbf{n}}}{N_b}, \quad (7)$$

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

$$\mathbf{X} = \sum_{i=1}^g (\mathbf{E}_{ii} \otimes \mathbf{c}_i^\top), \quad (8)$$

where $\mathbf{E}_{ii}$ 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, $\mathbf{c}_i = \mathbf{c}$, then

$$\mathbf{X} = (\mathbf{I}_g \otimes \mathbf{c}^\top). \quad (9)$$

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

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

$$\mathbf{q}_i = \mathbf{W} \mathbf{p}_i, \quad (10)$$

$$\mathbf{q}_b = \mathbf{W} \mathbf{p}_b, \quad (11)$$

where²

$$\mathbf{W} = \begin{bmatrix} 1 & 0.5 & 0 \\ 0 & 0.5 & 1 \end{bmatrix}. \quad (12)$$

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

$$\mathbf{L} = \begin{bmatrix} 1-u & v \\ u & 1-v \end{bmatrix}, \quad (13)$$

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:

$$\mathbf{r}_i = \mathbf{L} \mathbf{q}_i. \quad (14)$$

We do not investigate mutations in this article.

We assume that genotypes affect the survival and transitions of males and females equally and that the ratio of males to females in newborns is one. Thus males and females have the same genotype $\times$ stage distribution (see Charlesworth 1994). Because the genotypes do not affect male mating success, the gene frequencies 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:

$$\mathbf{r}_b = \mathbf{L} \mathbf{q}_b, \quad (15)$$

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 combina-

2. 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 numbered. For example, if the genotypes formed by three alleles are listed in the order (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 nine genotypes to the three alleles, would be

$$\mathbf{W} = \begin{bmatrix} 1 & .5 & .5 & .5 & 0 & 0 & .5 & 0 & 0 \\ 0 & .5 & 0 & .5 & 1 & .5 & 0 & .5 & 0 \\ 0 & 0 & .5 & 0 & 0 & .5 & .5 & .5 & 1 \end{bmatrix}.$$

tion of the gametes produced by this female with those produced by a random member of the breeding population, so that

$$\mathbf{p}'_i = \mathbf{Z}(\mathbf{r}_i \otimes \mathbf{r}_b). \quad (16)$$

The matrix $\mathbf{Z}$ converts ordered genotypes (Aa separately from aA) into unordered genotypes; for the case of one locus and two alleles,

$$\mathbf{Z} = \begin{bmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix}. \quad (17)$$

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

Substituting equations (7)–(10) into equation (16) and using the fact that $\mathbf{AC} \otimes \mathbf{BD} = (\mathbf{A} \otimes \mathbf{B})(\mathbf{C} \otimes \mathbf{D})$, we find that the distribution of offspring of a mother of genotype i is

$$\mathbf{p}'_i = \mathbf{Z}(\mathbf{r}_i \otimes \mathbf{r}_b) \quad (18)$$

$$= \frac{\mathbf{Q}(\mathbf{e}_i \otimes \tilde{\mathbf{n}})}{\sum_{j=1}^g (\mathbf{e}_j^T \otimes \mathbf{c}_j^T) \tilde{\mathbf{n}}}, \quad (19)$$

where

$$\mathbf{Q} = \mathbf{Z}(\mathbf{M} \otimes \mathbf{M})(\mathbf{W} \otimes \mathbf{W})(\mathbf{I}_g \otimes \mathbf{X}). \quad (20)$$

For a specified number of genotypes and set of breeding stages, the matrix $\mathbf{Q}$ is a constant.

The Matrix $\mathbf{H}$

The matrix $\mathbf{H}(\tilde{\mathbf{n}})$ maps the genotype of the parent to the genotype of the offspring. The i th column of the matrix $\mathbf{H}$ contains the distribution of offspring of a mother of genotype i , $\mathbf{p}'_i$, as given in equation (19); thus,

$$\mathbf{H} = (\mathbf{p}'_1 \cdots \mathbf{p}'_g), \quad (21)$$

which can be written as

$$\mathbf{H} = \sum_{i=1}^g \mathbf{p}'_i \otimes \mathbf{e}_i^T. \quad (22)$$

Combining equation (19) and equation (22) yields the following equation for the parent-offspring matrix

$$\mathbf{H}(\tilde{\mathbf{n}}) = \frac{\mathbf{Q} \sum_{i=1}^g (\mathbf{e}_i \otimes \tilde{\mathbf{n}} \otimes \mathbf{e}_i^T)}{\sum_{j=1}^g (\mathbf{e}_j^T \otimes \mathbf{c}_j^T) \tilde{\mathbf{n}}}. \quad (23)$$

The Population Projection Matrix

To project the stage $\times$ genotype dynamics, the component matrices $\mathbf{U}_i$, $\mathbf{F}_i$, and $\mathbf{H}(\tilde{\mathbf{n}})$ are incorporated into a population projection matrix. We create a set of block diagonal

matrices $\mathbf{U}$, $\mathbf{D}$, $\mathbf{F}$, and $\mathbf{H}$ by putting the corresponding matrices on the diagonal. These block diagonal matrices can be written as

$$\mathbf{U} = \sum_{i=1}^g \mathbf{E}_{ii} \otimes \mathbf{U}_i, \quad (24)$$

$$\mathbf{F} = \sum_{i=1}^g \mathbf{E}_{ii} \otimes \mathbf{F}_i, \quad (25)$$

$$\mathbf{D} = \mathbf{I}_\omega \otimes \mathbf{I}_g, \quad (26)$$

$$\mathbf{H} = \mathbf{I}_\omega \otimes \mathbf{H}(\tilde{\mathbf{n}}), \quad (27)$$

where $\mathbf{E}_{ii}$ is of dimensions $g \times g$.

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

$$\tilde{\mathbf{A}}(\tilde{\mathbf{n}}) = \underbrace{\mathbf{K}^T \mathbf{D} \mathbf{K} \mathbf{U}}_{\tilde{\mathbf{U}}} + \underbrace{\mathbf{K}^T \mathbf{H}(\tilde{\mathbf{n}}) \mathbf{K} \mathbf{F}}_{\tilde{\mathbf{F}}}, \quad (28)$$

where $\mathbf{K} = \mathbf{K}_{\omega g}$ 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,

$$\text{vec} \mathcal{N}^T = \mathbf{K} \text{vec} \mathcal{N}. \quad (29)$$

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

The first term in equation (28), labeled $\tilde{\mathbf{U}}$, applies the block diagonal matrix $\mathbf{U}$ to generate transitions and survival of extant individuals within genotypes, permutes the resulting vector with $\mathbf{K}$, applies the block diagonal matrix $\mathbf{D}$ to account for changes in genotype among extant individuals (since extant individuals do not generally change their genotype, $\mathbf{D}$ will be an identity matrix), and finally permutes the vector back to its original form with $\mathbf{K}^T$.

The second term in equation (28), labeled $\tilde{\mathbf{F}}$, describes reproduction and genotype assignment. First, the block diagonal matrix $\mathbf{F}$ 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 $\mathbf{K}$ rearranges the vector, and then the block diagonal matrix $\mathbf{H}(\tilde{\mathbf{n}})$ allocates the offspring to their genotypes, on the basis of the genotype of their parent and the genotype distribution of the rest of the population. Finally, $\mathbf{K}^T$ returns the vector to its original orientation.

Nonlinearity and Homogeneity

Even when the demographic components of the model are linear, the system (28) is nonlinear, because of the genetic component of the model. The parent-to-offspring geno-

type transition matrix, and thus the projection matrix $\tilde{\mathbf{A}}(\tilde{\mathbf{n}})$, are nonlinear but homogeneous of degree zero. That is, for any nonzero scalar c it is true that $\tilde{\mathbf{A}}(c\tilde{\mathbf{n}}) = \tilde{\mathbf{A}}(\tilde{\mathbf{n}})$. 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 $\tilde{\mathbf{n}}$ or the frequency vector $\tilde{\mathbf{p}}$, since the two differ only by a proportionality factor.

Homogeneous nonlinear models are familiar in two-sex demographic models (e.g., Hader et al. 1988; Caswell 2001; Iannelli et al. 2005). A nonlinear version of the 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 converge 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 2016). 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 that project the three genotype-specific population vectors, $\mathbf{n}_{AA}$, $\mathbf{n}_{Aa}$, and $\mathbf{n}_{aa}$, that were introduced in equation (3). Taking advantage of the structure of the component matrices, we find that $\tilde{\mathbf{A}}$, $\mathbf{U}$, and $\tilde{\mathbf{F}}$ can be simplified (app. A; apps. A, B are available online). The survival and transition matrix $\tilde{\mathbf{U}}$ is block diagonal because individuals do not change genotype:

$$\tilde{\mathbf{U}} = \begin{bmatrix} \mathbf{U}_{AA} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{U}_{Aa} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{U}_{aa} \end{bmatrix}. \quad (30)$$

The fertility matrix $\tilde{\mathbf{F}}$ has the following form:

$$\tilde{\mathbf{F}}(\tilde{\mathbf{p}}) = \begin{bmatrix} q_A^b \mathbf{F}_{AA} & \frac{1}{2} q_A^b \mathbf{F}_{Aa} & \mathbf{0} \\ q_a^b \mathbf{F}_{AA} & \frac{1}{2} \mathbf{F}_{Aa} & q_A^b \mathbf{F}_{aa} \\ \mathbf{0} & \frac{1}{2} q_a^b \mathbf{F}_{Aa} & q_a^b \mathbf{F}_{aa} \end{bmatrix}, \quad (31)$$

where q_A^b and q_a^b are the frequencies of allele A and a , respectively, in the breeding part of the population; that is, they are the elements of the vector $\mathbf{q}_b$ defined by equation (11), such that $\mathbf{q}_b^T = (q_A^b, q_a^b)^T$. If the $\mathbf{F}_i$ matrices are linear, the matrix $\tilde{\mathbf{F}}$ is homogeneous and can be written as a function of $\tilde{\mathbf{p}}$ rather than $\tilde{\mathbf{n}}$.

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

$$\tilde{\mathbf{A}}(\tilde{\mathbf{p}}) = \begin{bmatrix} \mathbf{U}_{AA} + q_A^b \mathbf{F}_{AA} & \frac{1}{2} q_A^b \mathbf{F}_{Aa} & \mathbf{0} \\ q_a^b \mathbf{F}_{AA} & \mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} & q_A^b \mathbf{F}_{aa} \\ \mathbf{0} & \frac{1}{2} q_a^b \mathbf{F}_{Aa} & \mathbf{U}_{aa} + q_a^b \mathbf{F}_{aa} \end{bmatrix}. \quad (32)$$

The matrix $\tilde{\mathbf{A}}$ is used in equation (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

$$\mathbf{U} = \begin{bmatrix} s_j(1 - \gamma) & 0 \\ s_j\gamma & s_A \end{bmatrix}, \quad (33)$$

$$\mathbf{F} = \begin{bmatrix} 0 & f \\ 0 & 0 \end{bmatrix},$$

where s_j and s_A are juvenile and adult survival probabilities, γ is the maturation rate, and f is 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 ($\mathbf{U}_{AA}$ and so on).

Figure 1 shows two examples of stage $\times$ genotype dynamics produced by this simple model (model parameters and Matlab code are provided in a zip file, available online).³ The A allele is introduced by adding a small fraction of heterozygote juveniles into a homozygote population. In figure 1b, genotype frequencies converge to a stable polymorphism, even though the population is driven to evolutionary suicide (fig. 1a). In figure 1d, the A allele sweeps to fixation; in so doing, it changes population growth from negative to positive (evolutionary rescue; fig. 1c). We will return to this example below to examine the criteria for polymorphism in relation to measures of genotypic growth rates λ_i , which will be defined in the section “Coexistence and Genetic Polymorphism.”

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

3. Code that appears in *The American Naturalist* is provided as a convenience to readers. It has not necessarily been tested as part of peer review.

Figure 1: Two examples of population dynamics of a two-stage (juvenile-adult) Mendelian matrix population model. *a, b*, Introduction of the *A* allele leads to evolutionary suicide and a genetic polymorphism. *c, d*, Introduction of the *A* allele leads to evolutionary rescue and fixation of the *AA* genotype. A color version of this figure is available online.

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 *B. 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.

Krüger and Lindström (2001) constructed a Leslie matrix for each color morph using data collected from 1989 through 1999 in eastern Westphalia, Germany. As examples, the sur-

vival and fertility matrices for females of the intermediate morph are shown in box 1. The other genotype-specific survival and transition matrices are available in the zip file.

The projection matrix $\hat{A}$ is constructed from the genotype-specific matrices U_i and F_i using equation (32). Starting with a population of dark individuals and one heterozygote juvenile, the population dynamics are obtained by projecting the population forward using equation (4). The abundances in each age class of all three genotypes are shown in figure 2*b*. The population growth rate converges to $\lambda = 0.91$. Field observations confirm that this *B. buteo* population was in decline during the time period used for fitting the model. The marginal distribution over genotypes, integrating over ages in $\tilde{\mathbf{p}}$, is shown in figure 2*a*.

Box 1: The transition matrix and the fertility matrix for *Buteo buteo* females of the intermediate color morph

$$\mathbf{U}_{\text{DL}} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0.8 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0.75 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0.636 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0.714 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0.667 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0.8 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0.625 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0.4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0.5 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \end{bmatrix} .$$

$$\mathbf{F}_{\text{DL}} = \begin{bmatrix} 0.05 & 0.41 & 0.42 & 0.45 & 0.56 & 0.48 & 0.61 & 0.27 & 0.40 & 0 & 0.54 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

The light allele increases in frequency until a stable polymorphism is reached.

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, that is, $w_{Aa} > w_{AA}$ and $w_{Aa} > w_{aa}$. The fitnesses w_i capture, in a single scalar, all of the information on 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 $\mathbf{U}_i$ and $\mathbf{F}_i$ 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 pres-

sure, and has formed the basis of several decades of evolutionary life-history research (e.g., Emlen 1970; Metz et al. 1992; Roff 1992; Stearns 1992; Caswell 2001) and eventually became, in the form of invasion fitness, the basis for adaptive dynamics (Geritz et al. 1998; Dercole and Rinaldi 2008). Lande incorporated population growth rate into quantitative genetic models in an age-structured version of the breeder's equation (Lande 1982a, 1982b).

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 "Discussion."

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

$$\lambda_{AA} = \rho(\mathbf{U}_{AA} + \mathbf{F}_{AA}), \quad (34)$$

$$\lambda_{Aa} = \rho(\mathbf{U}_{Aa} + \mathbf{F}_{Aa}), \quad (35)$$

$$\lambda_{aa} = \rho(\mathbf{U}_{aa} + \mathbf{F}_{aa}), \quad (36)$$

Figure 2: *a*, Projected *Buteo buteo* genotype frequencies when the intermediate (DL) heterozygote is introduced at low frequency (0.001) in a homozygous dark (DD) population at its stable age distribution. *b*, Projected genotype by age class distribution for the calculations in *a*.

where $\rho(\cdot)$ 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 equations (34)–(36) 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, that is, when

$$\lambda_{Aa} > \lambda_{AA}, \quad (37)$$

$$\lambda_{Aa} > \lambda_{aa}. \quad (38)$$

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

Protected Genetic Polymorphism

The dynamics of $\tilde{\mathbf{n}}$ take place in a ωg -dimensional space defined by combinations of ω stages and g genotypes (with $g = 3$ in the present context). The ω -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 and are given by the projection matrices for the homozygous genotypes.⁴

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 popula-

tion 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 $\tilde{\mathbf{n}}$, which grows exponentially, to the frequency vector $\tilde{\mathbf{p}}$, which converges to an equilibrium. This transformation is possible because of the homogeneity of $\tilde{\mathbf{A}}$. The dynamics of $\tilde{\mathbf{p}}$ are given by

$$\tilde{\mathbf{p}}(t+1) = \frac{\tilde{\mathbf{A}}[\tilde{\mathbf{p}}(t)]\tilde{\mathbf{p}}(t)}{\|\tilde{\mathbf{A}}[\tilde{\mathbf{p}}(t)]\tilde{\mathbf{p}}(t)\|}, \quad (39)$$

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 $\mathbf{a}$. Equilibria of equation (39), denoted by $\hat{\mathbf{p}}$, satisfy

$$\hat{\mathbf{p}} = \frac{\tilde{\mathbf{A}}[\hat{\mathbf{p}}]\hat{\mathbf{p}}}{\|\tilde{\mathbf{A}}[\hat{\mathbf{p}}]\hat{\mathbf{p}}\|}. \quad (40)$$

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 equation (39) 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. There-

4. Technically, the boundaries are invariant under the dynamics specified by $\tilde{\mathbf{A}}[\tilde{\mathbf{n}}]$.

fore, if the dominant eigenvalue of the Jacobian is larger in magnitude than 1, the associated eigenvector must be pointing into the interior, which implies that the invading allele increases when rare.

The Jacobian matrix

$$\mathbf{M} = \frac{d\tilde{\mathbf{p}}(t+1)}{d\tilde{\mathbf{p}}(t)} \bigg|_{\tilde{\mathbf{p}}}, \quad (41)$$

is obtained by differentiating equation (39) and evaluating the resulting derivative at the boundary equilibrium. The derivation is a lengthy exercise in matrix calculus (Verdy and Caswell 2008; Caswell 2008, 2019), presented in detail in appendix B. The result shows that $\mathbf{M}$ is a block upper triangular matrix given by equation (B57). 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 B. For a model of the form given by equations (24)–(28), the eigenvalues of the Jacobian matrix associated with growth in the *Aa* direction are

$$\zeta_{AA} = \frac{1}{\lambda_{AA}} \rho \left(\mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{AA} \hat{\mathbf{p}}_{AA}) \otimes \mathbf{c}_{Aa}^T \right), \quad (42)$$

$$\zeta_{aa} = \frac{1}{\lambda_{aa}} \rho \left(\mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{aa} \hat{\mathbf{p}}_{aa}) \otimes \mathbf{c}_{Aa}^T \right), \quad (43)$$

where λ_{AA} and λ_{aa} are given by equations (34) and (36), 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, that is,

$$\zeta_{AA} > 1, \quad (44)$$

$$\zeta_{aa} > 1, \quad (45)$$

or

$$\rho \left(\mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{AA} \hat{\mathbf{p}}_{AA}) \otimes \mathbf{c}_{Aa}^T \right) > \lambda_{AA}, \quad (46)$$

$$\rho \left(\mathbf{U}_{Aa} + \frac{1}{2} \mathbf{F}_{Aa} + \frac{1}{2p_b} (\mathbf{F}_{aa} \hat{\mathbf{p}}_{aa}) \otimes \mathbf{c}_{Aa}^T \right) > \lambda_{aa}. \quad (47)$$

It is worth noting that the data required to evaluate conditions (46) and (47) are all obtained from linear life-history calculations: the matrices $\mathbf{U}_{Aa}$ and $\mathbf{F}_{Aa}$ describing the heterozygote, the fertility matrices $\mathbf{F}_{AA}$ and $\mathbf{F}_{aa}$ describing the fertility of the homozygotes, the stable stage distributions $\hat{\mathbf{p}}_{AA}$ and $\hat{\mathbf{p}}_{aa}$ of the homozygotes, and the vector $\mathbf{c}_{Aa}$ that defines the breeding stages for the heterozygote.

The difference between conditions (46) and (47), on the one hand, and the simple (but erroneous) comparison of genotype-specific growth rates,

$$\rho(\mathbf{U}_{Aa} + \mathbf{F}_{Aa}) > \lambda_{AA}, \quad (48)$$

$$\rho(\mathbf{U}_{Aa} + \mathbf{F}_{Aa}) > \lambda_{aa}, \quad (49)$$

on the other, 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 $\mathbf{U}_{Aa}$, $\mathbf{F}_{Aa}$, and $\mathbf{c}_{Aa}$ in condition [46]). But the gametes of these heterozygotes combine with the gametes of the much more abundant *AA* homozygotes (hence, the appearance of $\mathbf{F}_{AA}$). In addition, fertility and zygote formation depend on the demographic stage structure of the *AA* homozygotes (hence, the appearance of $\hat{\mathbf{p}}_{AA}$ in condition [46]). None of these complexities are captured by the oversimplified heterozygote growth rates in condition (49). The situation for the *A* allele invading the *aa* boundary in condition (47) 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 (42) and (43), reduce to

$$\zeta_{AA} = \frac{1}{\lambda_{AA}} \rho \left(\mathbf{U}_{Aa} + \frac{1}{2} (\mathbf{F}_{Aa} + \mathbf{F}_{AA}) \right), \quad (50)$$

$$\zeta_{aa} = \frac{1}{\lambda_{aa}} \rho \left(\mathbf{U}_{Aa} + \frac{1}{2} (\mathbf{F}_{Aa} + \mathbf{F}_{aa}) \right). \quad (51)$$

See “Special Case: A Single Reproducing Stage” in appendix B for a derivation of both equations.

COROLLARY 1: For a model of the form given by equations (24)–(28) with only one reproducing stage, a protected polymorphism occurs when both boundary equilibria are unstable, that is,

$$\rho\left(\mathbf{U}_{Aa} + \frac{1}{2}(\mathbf{F}_{Aa} + \mathbf{F}_{AA})\right) > \rho(\mathbf{U}_{AA} + \mathbf{F}_{AA}), \quad (52)$$

$$\rho\left(\mathbf{U}_{Aa} + \frac{1}{2}(\mathbf{F}_{Aa} + \mathbf{F}_{aa})\right) > \rho(\mathbf{U}_{aa} + \mathbf{F}_{aa}). \quad (53)$$

If the genotypes differ only in survival and transitions (the stage-structured analogue of viability selection), so that $\mathbf{F}_{AA} = \mathbf{F}_{Aa} = \mathbf{F}_{aa}$, then the coexistence condition reduces to heterozygote superiority in λ . 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 $\mathbf{U}$ and $\mathbf{F}$ 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 (24)–(28), the eigenvalues of the Jacobian matrix, associated with the *Aa* direction, at the *AA* and *aa* boundaries are

$$\zeta_{AA} = \frac{1}{\lambda_{AA}} \left(s_{Aa} + \frac{1}{2}(f_{Aa} + f_{AA}) \right), \quad (54)$$

$$\zeta_{aa} = \frac{1}{\lambda_{aa}} \left(s_{Aa} + \frac{1}{2}(f_{Aa} + f_{AA}) \right). \quad (55)$$

COROLLARY 2: A protected polymorphism occurs when both boundary equilibria are unstable, that is,

$$s_{Aa} + \frac{1}{2}(f_{Aa} + f_{aa}) > s_{aa} + f_{aa}, \quad (56)$$

$$s_{Aa} + \frac{1}{2}(f_{Aa} + f_{AA}) > s_{AA} + f_{AA}, \quad (57)$$

or

$$s_{Aa} + \frac{1}{2}f_{Aa} > s_{aa} + \frac{1}{2}f_{aa}, \quad (58)$$

$$s_{Aa} + \frac{1}{2}f_{Aa} > s_{AA} + \frac{1}{2}f_{AA}, \quad (59)$$

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 λ . 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).

Examples Revisited

Armed with the results of theorem 1, we return to the two examples considered in figure 1 (the two-stage model) and figure 2 (the common buzzard color polymorphism).

Two-stage population. In the two-stage example of evolutionary suicide (fig. 1a, 1b), the heterozygote is inferior as measured by its growth rate λ_{Aa} , but both boundary equilibria are unstable, so that a protected polymorphism results:

$$\begin{aligned} \lambda_{AA} &= 0.989, & \zeta_{AA} &= 1.012, \\ \lambda_{Aa} &= 0.966, & \zeta_{aa} &= 1.007. \end{aligned} \quad (60)$$

In the case leading to evolutionary rescue (fig. 1c, 1d), the heterozygote is superior in growth rate λ_{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:

$$\begin{aligned} \lambda_{AA} &= 1.018, & \zeta_{AA} &= 0.983, \\ \lambda_{Aa} &= 1.029, & \zeta_{aa} &= 1.004. \\ \lambda_{aa} &= 0.990, \end{aligned} \quad (61)$$

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, the heterozygote genotype DL has the highest population growth rate and both boundary equilibria are unstable, resulting in a protected polymorphism:

$$\begin{aligned} \lambda_{DD} &= 0.48, & \zeta_{DD} &= 1.891, \\ \lambda_{DL} &= 1.04, & \zeta_{LL} &= 1.493. \\ \lambda_{LL} &= 0.68, \end{aligned} \quad (62)$$

In figure 2b, 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 timescale for convergence of population dynamics is the same as the timescale over which the genotype distribution converges (fig. 2a). Thus, ecological and evolutionary processes are clearly operating on comparable timescales 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 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.

Discussion

Both gene frequency dynamics and population dynamics are driven by the demographic processes of birth and death. Combining Mendelian genetics and demographic models makes it possible to analyze genotype $\times$ 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 (24)–(28) that projects stage $\times$ genotype population structures (figs. 1, 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 of the usual demographic output in addition to the stage $\times$ 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., the study of a wolf population by Coulson et al. [2011]) or to equilibrium conditions (Diekmann et al. 2003).

Despite empirical evidence for the importance of fertility selection (Anderson and Watanabe 1974; Pincheira-Donoso and Hunt 2017; Travis 1988), 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 articles between the late 1940s and the 1980s (Penrose 1947; Owen 1953; Bodmer 1965; Haderler and Liberman 1975; Pollak 1978; 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 genetics model and to cases where selection may operate on other rates besides viability and fertility (e.g., growth or development).

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 (C. de Vries and H. Caswell, unpublished manuscript). 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 nonlinear demography, time-dependent demographic rates, interactions among species, and dependence on environmental resources. The model can also be extended to include more genetic details, including nonrandom mating, more than two alleles, and 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 (λ_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 literature on life-history theory that assumes selection can be described in terms of some version of λ (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 λ 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 males and females. 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 article 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 article. 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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