DEMOGRAPHIC RESEARCH
A peer-reviewed, open-access journal of population sciences

DEMOGRAPHIC RESEARCH

VOLUME 38, ARTICLE 8, PAGES 197–226
PUBLISHED 16 JANUARY 2018
http://www.demographic-research.org/Volumes/Vol38/8/
DOI: 10.4054/DemRes.2018.38.8

Research Article

Evolution of fixed demographic heterogeneity from a game of stable coexistence

Stefano Giaimo

Xiang-Yi Li

Arne Traulsen

Annette Baudisch

Contents

Demographic Research: Volume 38, Article 8
Research Article

Evolution of fixed demographic heterogeneity from a game of stable coexistence

Stefano Giaimo[1]

Xiang-Yi Li[2]

Arne Traulsen[3]

Annette Baudisch[4]

Abstract

BACKGROUND
Demographic heterogeneity refers to the observation that – within the same population – trajectories of survival and reproduction differ substantially between individuals. These differences have been found in both natural and captive populations. Models in ecology and evolution that incorporate demographic heterogeneity can improve both our understanding of the evolution of mortality curves and our population management abilities. Current explanations of the origin of demographic heterogeneity mostly revolve around interindividual differences that are either present at birth (fixed heterogeneity) or the result of stochasticity in life history realization (dynamic heterogeneity). Largely neglected remains the possibility that a form of fixed heterogeneity may evolve from interactions between behaviorally distinct individuals through their lifespan.

OBJECTIVE
We suggest one possible way in which heterogeneity in vital rates may evolve. Our approach assumes game theoretic interactions in the population.

METHODS
We combine population matrix models and game theory. We study a stable coexistence game between two types that are initially demographically homogeneous and analyze the effect of mutations that influence the trajectories of survival and reproduction.

[1] Department of Evolutionary Theory, Max Planck Institute for Evolutionary Biology, Plön, Germany. Email: giaimo@evolbio.mpg.de.
[2] Department of Evolutionary Biology and Environmental Studies, University of Zurich, Switzerland.
[3] Department of Evolutionary Theory, Max Planck Institute for Evolutionary Biology, Plön, Germany.
[4] Biodemography Unit, Department of Biology and Department of Public Health, University of Southern Denmark (SDU), Odense, Denmark.

RESULTS
The rise and fixation of mutations can make the population demographically heterogeneous, while the game can preserve the coexistence of different types in the population.

CONCLUSIONS
Frequency-dependent selection can help to explain the evolution of demographic heterogeneity.

CONTRIBUTION
Frequency-dependent selection can maintain already existing demographic heterogeneity in a population without overlapping generations. Here, we show that this form of selection can also be involved in the origin of a form of fixed heterogeneity.

1. Introduction

Evolutionary game theory is an approach to modeling evolutionary change with a focus on fitness as a function of both an individual's traits and the traits of others in the population (Maynard Smith 1982; Hofbauer and Sigmund 1998; Nowak 2006). Evolutionary demographic theory is an approach to modeling evolutionary change with a focus on fitness as the outcome of successive events of survival and reproduction along the lifespan (Stearns 1992; Charlesworth 1994; Caswell 2001). So far, only a few attempts have been made to combine these two approaches (Kokko 1997; Cressman 2003; Chambon-Dubreuil et al. 2006). Yet, it seems instructive to study their interplay, as either approach alone may neglect an important factor driving evolutionary change. Recently, a model was proposed to integrate demography into a standard model of evolutionary game theory (Li et al. 2015). Here, we integrate game theory into a standard model of evolutionary demography. Our objective is to unveil the potential importance of evolutionary games to considerations of life histories. In particular, we show how games may contribute to explain a possible mechanism behind the evolution of a form of fixed heterogeneity in vital rates. Natural populations are usually heterogeneous, i.e., they host a variety of different types. Such variation is the fuel of evolution (Maynard Smith 1995, 1986; Maynard Smith and Szathmáry 1995; Lewontin 1970). Types that have certain traits (e.g., the ability to find food or run, associated with higher survival and reproductive abilities, i.e., higher Darwinian fitness) tend to progressively increase their representation in the population, eventually leading to the evolution of this in terms of a change in the types' relative abundance (Maynard Smith 1995, 1986; Maynard Smith and Szathmáry 1995; Lewontin 1970). The process requires that evolutionarily successful traits are transmitted (e.g., genetically) to descendants (Maynard Smith 1995, 1986; Maynard Smith and Szathmáry 1995; Lewontin 1970).

A form of variation upon which selection may operate is demographic variation, which refers to interindividual diversity in survival and reproductive performances along the lifespan. Such demographic heterogeneity can be of two main kinds. The first kind is fixed demographic heterogeneity, which refers to the presence of individuals within the same population who are expected to follow different trajectories of survival and fertility through their lives. This kind of heterogeneity is called 'fixed' because it derives from individual differences that are present at birth, e.g., genetically coded and remaining unmodified thereafter. Such differences arise as a result of different individual traits with an effect on survival and/or reproduction and, therefore, selection can act upon such forms of heterogeneity. However, while traits underlying interindividual diversity can, in principle, be measured, sometimes they may not be observed and need to be postulated (Vaupel, Manton, and Stallard 1979). Fixed heterogeneity contrasts with dynamic heterogeneity (Tuljapurkar, Steiner, and Orzack 2009). Individuals may transition through a number of stages (reproductive level, spatial location) along their lifespan. Each stage can be associated with stage-specific fertility and survival levels. When the transitioning through stages is governed by a stochastic process, each individual follows a random trajectory. The probabilistic nature of such transitions generates dynamic demographic heterogeneity (Tuljapurkar, Steiner, and Orzack 2009), which need not be based on interindividual intrinsic differences. Individuals with the same propensities to move from one stage to the other may in fact spend different amounts of time in different stages because of random factors. As a consequence, they will realize different survival and reproductive performances. Importantly, dynamic heterogeneity gives rise to a form of variation that cannot be acted upon by natural selection because it is not based on heritable differences (Steiner and Tuljapurkar 2012).

Heterogeneity in vital rates is widespread in wild populations (Vaupel et al. 1998), including humans (Yashin, Iachine, and Harris 1999). Yet it is unclear how much of it is accounted for by either fixed heterogeneity or dynamic heterogeneity (Steiner and Tuljapurkar 2012) and how exactly they can be separated when it comes to analyzing data (Plard et al. 2012; Cam, Aubry, and Authier 2016). Also, more articulated kinds of heterogeneity can be distinguished. For example, genetically identical individuals may sense differently a changing environment and have a different phenotypic response or they may stochastically switch to different phenotypes, some of which may be the best response to the current environment (Kussel and Leibler 2005). For a recent review of the different concepts and models of heterogeneity in demographic rates, see Wilson and Nussey (2010). A better understanding of demographic heterogeneity, however, may be instructive for population management. The growth rate and extinction risk of a population are influenced by the population's level of demographic heterogeneity in a way that homogeneous population models cannot account for (Kendall et al. 2011). Demographic heterogeneity may also help to explain some evolutionarily puzzling observations; for example, results from cohort studies on controlled, captive populations show decelera-

tion of mortality at very late ages (Carey et al. 1992; Curtsinger et al. 1992). This is at odds with the prediction from classical evolutionary theory of senescence, which predicts increasing mortality with age (Hamilton 1966; Charlesworth and Partridge 1997). However, the presence of subcohorts all experiencing a steady increase in mortality with age but at different rates can explain this effect (Curtsinger et al. 1992; Vaupel and Carey 1993; Chen, Zajitschek, and Maklakov 2013), as the aggregate cohort may not display a monotonically increasing mortality with age in that case (Vaupel and Yashin 1985). A similar explanation can be provided based on dynamic heterogeneity (Horvitz and Tuljapurkar 2008), as well as based on some form of heterogeneity that is present at birth yet still potentially modifiable thereafter (Le Bras 1976; Yashin, Vaupel, and Iachine 1994).

A question that has not received much attention is how fixed demographic heterogeneity can evolve, in the first place, and then persist. Certainly, mutation can always inject new variants into the population. The large majority of such mutations are detrimental and are purged by selection, eventually leading to a mutation-selection balance, which leaves room for variation. (For a general model for the equilibrium between mutation and selection in age-structured populations, see Steinsaltz, Evans, and Wachter 2005.) However, the amount of this variation should depend on the mutation rate and the force of selection. When the former is low and the latter strong, persistent variation may be limited. In general, substantial fixed heterogeneity is not trivial to explain because it requires mechanisms that can generate it, while at the same time keeping fitness equal between demographically different types. In the present work, we suggest one possible answer to this question using evolutionary game theory, an approach that lends itself to understanding how natural selection may maintain diversity in a population (Maynard Smith 1982; Nowak 2006; Huang et al. 2012). We propose a model of an age-structured population with two types of individuals involved in a coexistence game. This game allows either subpopulation type to incorporate independent genetic variation that makes the population demographically heterogeneous, while the types maintain equal relative fitness. We start by describing the basic principle behind our model in a model without age structure.

2. A population without age structure

We assume a population of two types of individuals, A and B, which is large enough to be considered infinite for our purposes. It is not subject to density dependence, and its dynamics depend on relative frequencies of types; i.e., the relative abundances of types and not the total absolute population abundance has an effect on individual fitness. Generations are nonoverlapping. Individuals are involved in two-player games with the following payoff matrix:

$$\mathbf{M} = \begin{array}{c} \\ A \\ B \end{array} \begin{array}{c} \begin{array}{cc} A & B \end{array} \\ \begin{pmatrix} p_{AA} & p_{AB} \\ p_{BA} & p_{BB} \end{pmatrix} \end{array}, \tag{1}$$

where p_{AA} is the payoff to an individual of type A playing against another A; p_{AB} is the payoff to an individual of type A playing against a B individual; p_{BA} is the payoff to an individual of type B playing against an A individual; p_{BB} is the payoff to an individual of type B playing against another B. We assume that individuals are paired at random to interact in the game. Let N_A and N_B be the number of A and the number of B individuals, respectively, in the population and N the total population size. With a fraction $x = \frac{N_A}{N}$ of individuals of type A, the expected payoffs of an A individual and a B individual are

$$\pi_A(x) = xp_{AA} + (1-x)p_{AB}, \tag{2a}$$
$$\pi_B(x) = xp_{BA} + (1-x)p_{BB}. \tag{2b}$$

The expected fitness of an individual is the sum of a baseline fitness w^0 and expected payoff π (Nowak et al. 2004). The expected fitness values of A and B are then

$$\bar{w}_A(x) = w_A^0 + \pi_A(x), \tag{3a}$$
$$\bar{w}_B(x) = w_B^0 + \pi_B(x). \tag{3b}$$

As we focus on the interplay between these two fitness terms, there is no need to introduce an intensity of selection that would control the relative contribution of the expected payoff to fitness (Nowak et al. 2004).

Evolutionary dynamics is modeled according to the replicator dynamics in discrete time (Hofbauer and Sigmund 1998), which implies that x in generation $t+1$ is given by

$$x(t+1) = x(t)\frac{\bar{w}_A(x(t))}{x\bar{w}_A(x(t)) + (1-x(t))\bar{w}_B(x(t))}. \tag{4}$$

We assume that baseline fitness is initially equal between types, $w_A^0 = w_B^0$, and that A and B play a stable coexistence game, such that each type can invade a homogenous population of the other type. In particular, we assume that payoff values satisfy

$$0 < p_{AA} < p_{BA} < p_{BB} < p_{AB}, \tag{5}$$

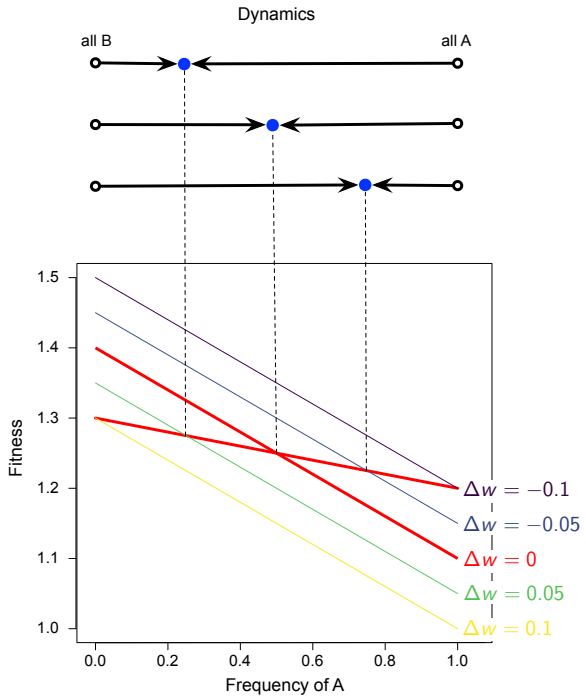
which implies that a homogeneous population of A is less fit than a homogeneous population of B, but both can be invaded by mutants of the other type. The game has a unique interior equilibrium at

$$x^* = \frac{p_{BB} - p_{AB}}{p_{AA} - p_{AB} - p_{BA} + p_{BB}}, \tag{6}$$

which is a stable fixed point of the replicator dynamics.

At the fixed point, the population grows exponentially. Suppose that then, in one type, a mutation arises that leads to a perturbation of the baseline fitness of a small additive amount. The mutation can go to fixation in the subpopulation of that type by either selection or by drift. Typically, selection accounts for cases in which the mutation increases the baseline fitness of the subpopulation, while drift accounts for cases in which the mutation decreases fitness. Is coexistence then still possible? Without the game, payoffs are zero, and there is consequently a fitness difference between the two types. The type with the higher fitness should take over the entire population. With the game, there is the possibility that lower baseline fitness is compensated for by higher payoff in the coexistence game due to frequency-dependent selection.

Figure 1: Change in the stable fixed point with change in baseline fitness



Note: Initially, $w_A^0 = w_B^0 = 1$. Then perturbations to the baseline fitness of A, i.e., $\Delta w \neq 0$, lead to a change in the equilibrium level $\hat{x}$, i.e., intersection, at which stable coexistence is attained. Payoffs are p_{AA} = 0.1, p_{AB} = 0.4, p_{BA} = 0.2 and p_{BB} = 0.3.

Figure 1 shows that the coexistence game is qualitatively robust against such perturbations of the background fitness. For sufficiently small $\Delta w = w_B^0 - w_A^0$, there is a different stable fixed point that still maintains the coexistence between the two subpopulations. Setting $\bar{w}_A = \bar{w}_B$ and solving for x, the stable fixed point is

$$\hat{x} = \frac{\Delta w + p_{BB} - p_{AB}}{p_{AA} - p_{AB} - p_{BA} + p_{BB}}. \tag{7}$$

At this new equilibrium, a difference in expected payoffs between types compensates for $\Delta w \neq 0$. Intuitively, the difference in background fitness appears only in the numerator, as it cancels in the denominator where only the fitness differences of a type appear. In the following, we use $\hat{x}$ to indicate the fixed point when the baseline fitness of the types may be different.

3. A population with age structure

We now consider the age-structured model in which both the relative abundance of types and individual age can affect the dynamics. As before, the population is very large, is not subject to density dependence, and its dynamics depend on relative frequencies of types. Individuals can survive from one step in time to the next, and, therefore, generations overlap. We take A as the focal type to describe the dynamics. After the game, an A individual of age j produces a number

$$\bar{F}_{j,A} = F_{j,A} + \pi_A(x) \tag{8}$$

of offspring. Fertility is the sum of an age- and type-dependent component $F_{j,A} > 0$ and the expected payoff $\pi_A(x)$, which changes with the relative abundance x of A. As payoffs are strictly positive, we have $\bar{F}_{j,A} > 0$. Let $n_{j,A}(t)$ be the number of A individuals in age class j at t and $n_{j,B}(t)$ be the number of B individuals in age class j at t, with the total subpopulations having size $N_A = \sum_j n_{j,A}$ and $N_B = \sum_j n_{j,B}$, and the total population being $N = N_A + N_B$. Then, the A fraction of the population is

$$x(t) = \frac{\sum_j n_{j,A}(t)}{\sum_j n_{j,A}(t) + \sum_j n_{j,B}(t)} = \frac{N_A(t)}{N_A(t) + N_B(t)} = \frac{N_A(t)}{N(t)}. \tag{9}$$

Offspring enter the population in the next time step, to which the parent can survive with a survival probability $S_{j,A}$, which again depends on age and type (but not on the payoff). The dynamics of the A subpopulation is then determined by

$$\underbrace{\begin{bmatrix} n_{1,A}(t+1) \\ n_{2,A}(t+1) \\ \vdots \\ \vdots \\ n_{\omega,A}(t+1) \end{bmatrix}}_{\mathbf{n}_A(t+1)} = \underbrace{\begin{bmatrix} \bar{F}_{1,A}(x) & \bar{F}_{2,A}(x) & \dots & \bar{F}_{\omega-1,A}(x) & \bar{F}_{\omega,A}(x) \\ S_{1,A} & & & & \\ & S_{2,A} & & & \\ & & \ddots & & \\ & & & S_{\omega-1,A} & \end{bmatrix}}_{\bar{\mathbf{L}}_A(x)} \underbrace{\begin{bmatrix} n_{1,A}(t) \\ n_{2,A}(t) \\ \vdots \\ \vdots \\ n_{\omega,A}(t) \end{bmatrix}}_{\mathbf{n}_A(t)} \tag{10}$$

Here, ω is the maximum age class that A individuals can reach. The vector $\mathbf{n}_A(t)$ represents the subpopulation state at t. The matrix $\bar{\mathbf{L}}_A(t)$ is a population projection matrix (PPM) that includes the subpopulation vital rates (i.e., a collective name for age-specific survival and fertilities). It depends directly, via the fertilities, on x and via this also on time t. The PPM and dynamics of B are similarly described.

For technical purposes, we define the matrix

$$\mathbf{K}_A = \begin{bmatrix} 1 & 1 & \dots & 1 & 1 \\ 0 & 0 & \dots & 0 & 0 \\ 0 & 0 & \dots & 0 & 0 \\ \vdots & \vdots & \ddots & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \tag{11}$$

which has the same dimensions as $\mathbf{L}_A$. A similar matrix is formed for B. We can then write the dynamics of the abundances of the two subpopulations as

$$\mathbf{n}_A(t+1) = \bar{\mathbf{L}}_A(x)\mathbf{n}_A(t) = (\mathbf{L}_A + \pi_A(x)\mathbf{K}_A)\mathbf{n}_A(t) \tag{12a}$$
$$\mathbf{n}_B(t+1) = \bar{\mathbf{L}}_B(x)\mathbf{n}_B(t) = (\mathbf{L}_B + \pi_B(x)\mathbf{K}_B)\mathbf{n}_B(t), \tag{12b}$$

in which the matrices $\bar{\mathbf{L}}_A$ and $\bar{\mathbf{L}}_B$ are split up into a frequency-independent part, $\mathbf{L}_A$ and $\mathbf{L}_B$, and a frequency-dependent part, $\pi_A(x)\mathbf{K}_A$ and $\pi_B(x)\mathbf{K}_B$. The matrices $\mathbf{L}_A$ and $\mathbf{L}_B$ are non-negative, as their entries are equal to or greater than zero, and can be shown irreducible (Caswell 2001). Therefore, the Perron-Froebenius theorem applies and we can let λ_A be the Perron root of $\mathbf{L}_A$ and λ_B be the Perron root of $\mathbf{L}_B$. As the effect of expected payoffs is just to increase the value of some entries of these matrices, $\bar{\mathbf{L}}_A(x)$ and $\bar{\mathbf{L}}_B(x)$ are also nonnegative and irreducible and have Perron roots $\bar{\lambda}_A(x)$ and $\bar{\lambda}_B(x)$, respectively.

Without the game, the population dynamics are simply

$$\mathbf{n}_A(t+1) = \mathbf{L}_A\mathbf{n}_A(t) \tag{13a}$$
$$\mathbf{n}_B(t+1) = \mathbf{L}_B\mathbf{n}_B(t). \tag{13b}$$

This linear case is within the scope of the ergodic theorem in demography (Cohen 1979). When a nonzero population state vector is repeatedly multiplied by a constant, non-negative and irreducible PPM, as in our case, the population state vector asymptotically becomes proportional to the leading right eigenvector of that PPM, and the population grows at a rate equal to the Perron root of the PPM. Thus, asymptotically, the population sizes in the system in Equation (13) change as

$$N_A(t+1) = \lambda_A N_A(t) \tag{14a}$$
$$N_B(t+1) = \lambda_B N_B(t), \tag{14b}$$

assuming that the two population state vectors are proportional to the right leading eigenvectors of the matrices $\mathbf{L}$.

We assume that, initially, A and B have the same baseline vital rates, i.e., $\mathbf{L}_A = \mathbf{L}_B$, and that the population is initiated at $x = x^*$. With equal expected payoffs, we obtain $\bar{\mathbf{L}}_A(x^*) = \bar{\mathbf{L}}_B(x^*)$ with corresponding Perron roots at equilibrium $\bar{\lambda}_A(x^*) = \bar{\lambda}_B(x^*)$. Provided the two population state vectors are proportional to the right leading eigenvectors of the respective matrices evaluated at $x = x^*$, the subpopulations of A and B stably grow over time at the same rate. Thus, at the fixed point, we recover linear population dynamics

$$N_A(t+1) = \bar{\lambda}_A(x^*)N_A(t) \tag{15a}$$
$$N_B(t+1) = \bar{\lambda}_B(x^*)N_B(t). \tag{15b}$$

We now introduce a small perturbation to A. In the presence of age structure, mutations can perturb one or more baseline vital rates of this type, but they do not change the number of age classes. As in the demographically unstructured model, we assume that mutations arise and go to fixation in the relevant subpopulation. We do not model the transient phase of population dynamics that starts when the mutation frequency becomes non-negligible and, therefore, leads to a deviation from the game equilibrium and the demographic equilibrium, up to when the mutation becomes fixed, which requires the establishment of new game and demographic equilibria. Such modeling would involve the use of tools for the analysis of the transients in matrix population models (see, e.g., Fox and Gurevitch 2000; Yearsley 2004; Caswell 2007; Stott, Townley, and Hodgson 2011), which would make our model exceedingly complex. Instead we rely on mutant deviations that are sufficiently small so that a smooth passage from one equilibrium to another is guaranteed by stability of coexistence (see below and Appendix). We write A' to indicate that the type has mutated and $\mathbf{L}_{A'}$ to refer to the mutated baseline PPM. The mutation is not neutral, $\lambda_B - \lambda_{A'} \neq 0$. In the absence of the game, the type whose baseline projection matrix has the highest leading eigenvalue takes over. To understand whether the game can preserve coexistence we study the new system

$$\mathbf{n}_{A'}(t+1) = \bar{\mathbf{L}}_{A'}(x)\mathbf{n}_{A'}(t) = (\mathbf{L}_{A'} + \pi_{A'}(x)\mathbf{K}_{A'})\mathbf{n}_{A'}(t) \tag{16a}$$
$$\mathbf{n}_B(t+1) = \bar{\mathbf{L}}_B(x)\mathbf{n}_B(t) = (\mathbf{L}_B + \pi_B(x)\mathbf{K}_B)\mathbf{n}_B(t). \tag{16b}$$

Note, however, that $\pi_{A'}(x) = \pi_A(x)$, as mutations affect only baseline vital rates. We look for values $\hat{x}$ of x in $(0, 1)$ such that the matrices $\bar{\mathbf{L}}_{A'}(\hat{x})$ and $\bar{\mathbf{L}}_B(\hat{x})$ have Perron roots $\bar{\lambda}_{A'}(\hat{x}) = \bar{\lambda}_B(\hat{x}) = \bar{\lambda}(\hat{x})$. In that case, A' and B can coexist at $\hat{x}$ by having equal population growth. This requires demographic stability, i.e., $\mathbf{n}_{A'}$ and $\mathbf{n}_B$ are proportional to the leading right eigenvectors of the relevant matrices.

Finding whether such fixed points exist and are stable is of relevance for the evolution of demographic heterogeneity. Suppose that at the fixed point of the coexistence game a mutant appears in one subpopulation and the mutation reaches fixation within this subpopulation. Assume that a nearby alternative interior fixed point exists and is stable. Then this fixed point is reached. At this new fixed point, the population is demographically heterogeneous. The two subpopulations have the same growth rate, yet their equilibrium PPMs differ for two reasons. First, the mutation has made some baseline vital rates different between types. Second, the expected payoffs between A' and B must be different in order to counteract the nonzero difference between $\lambda_{A'}$ and λ_B.

4. Analysis

4.1 Interior fixed points

The interior fixed points of the dynamics in Equations (16) correspond to coexistences between A' and B. We assume such points are isolated. To retrieve them, we borrow from robust control theory applied to PPMs (Hodgson and Townley 2004; Hodgson, Townley, and McCarthy 2006). In this approach, one considers a PPM (say, $\mathbf{Y}$) that is assumed non-negative and irreducible and, therefore, has a Perron root. A scalar amount $\delta > 0$ is added to some entries of $\mathbf{Y}$. A new matrix is obtained that can be represented as

$$\tilde{\mathbf{Y}} = \mathbf{Y} + \delta \mathbf{p}\mathbf{q}^T, \tag{17}$$

where $\mathbf{p}$ is a column vector and $\mathbf{q}^T$ (here T indicates vector transposition) is a row vector such that the product $\mathbf{p}\mathbf{q}^T$ is a sparse matrix of 0s and 1s of the same dimensions as $\mathbf{Y}$, where nonzero entries of $\mathbf{p}\mathbf{q}^T$ correspond to entries of $\mathbf{Y}$ to which δ is added. As $\tilde{\mathbf{Y}}$ is also non-negative and irreducible, let z be its Perron root. Following Hodgson and Townley (2004), we write the usual eigenvector problem for this matrix as

$$\tilde{\mathbf{Y}}\mathbf{c} = (\mathbf{Y} + \delta \mathbf{p}\mathbf{q}^T)\mathbf{c} = z\mathbf{c} \tag{18}$$

for some nonzero eigenvector $\mathbf{c}$, which is guaranteed positive by the Perron-Froebenius theorem. Subtracting $\mathbf{Y}\mathbf{c}$ on both sides, this equation becomes $\delta \mathbf{p}\mathbf{q}^T\mathbf{c} = (z\mathbf{I} - \mathbf{Y})\mathbf{c}$. If z is not an eigenvalue of $\mathbf{Y}$, the inverse of $(z\mathbf{I} - \mathbf{Y})$ exists. Multiplying on the left, first, by $(z\mathbf{I} - \mathbf{Y})^{-1}$ and, then, by $\mathbf{q}^T$ both sides of the equation, we obtain $\delta \mathbf{q}^T(z\mathbf{I} - \mathbf{Y})^{-1}\mathbf{p}\mathbf{q}^T\mathbf{c} = \mathbf{q}^T\mathbf{c}$. Note that $\mathbf{q}^T\mathbf{c}$ is a nonzero scalar. Dividing the equation by $\mathbf{q}^T\mathbf{c}$, we get $\delta \mathbf{q}^T(z\mathbf{I} - \mathbf{Y})^{-1}\mathbf{p} = 1$. Let $G(z) = \mathbf{q}^T(z\mathbf{I} - \mathbf{Y})^{-1}\mathbf{p}$, then we obtain the relationship

$$\delta \mathbf{q}^T(z\mathbf{I} - \mathbf{Y})^{-1}\mathbf{p} = \delta G(z) = 1. \tag{19}$$

The function $G(z)$ is called the transfer function and is a rational function in z implicitly relating δ with z (Hodgson and Townley 2004; Hodgson, Townley, and McCarthy 2006).

We can adopt this approach to capture exactly the effect of expected payoffs on the Perron roots of $\mathbf{L}_{A'}$ and $\mathbf{L}_B$. The structure of the change induced by expected payoffs for A' is represented by $\mathbf{K}_{A'}$, which we can express by setting $\mathbf{K}_{A'} = \mathbf{p}_{A'}\mathbf{q}_{A'}^T = [1,0,0,..,0]^T[1,1,1,..,1]$. The magnitude of the change is represented by the expected payoffs so that $\delta = \pi_{A'}$. We adopt the same procedure for B. We then write the transfer functions for A' as $\pi_{A'}\mathbf{q}_{A'}^T(z\mathbf{I}_{A'} - \mathbf{L}_{A'})^{-1}\mathbf{p}_{A'} = \pi_{A'}G_{A'}(z) = 1$ and for B as $\pi_B\mathbf{q}_B^T(z\mathbf{I}_B - \mathbf{L}_B)^{-1}\mathbf{p}_B = \pi_B G_B(z) = 1$. Using the definition of expected payoffs in Equations (2) and solving for x, we can write for the two types

$$x = \frac{(G_{A'}(z))^{-1} - p_{AB}}{p_{AA} - p_{AB}} \tag{20a}$$

$$x = \frac{(G_B(z))^{-1} - p_{BB}}{p_{BA} - p_{BB}}. \tag{20b}$$

Equating these two expressions, we obtain

$$(p_{BA} - p_{BB})[(G_{A'}(z))^{-1} - p_{AB}] = (p_{AA} - p_{AB})[(G_B(z))^{-1} - p_{BB}]. \tag{21}$$

This can be written as

$$p_{AA}p_{BB} - p_{AB}p_{BA} = \frac{p_{AA} - p_{AB}}{G_B(z)} - \frac{p_{BA} - p_{BB}}{G_{A'}(z)}. \tag{22}$$

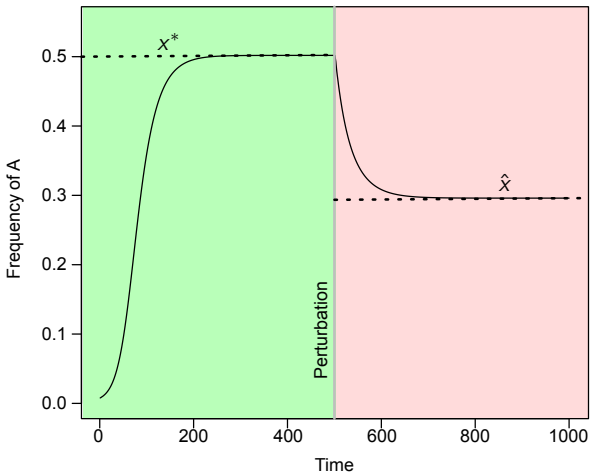
The left side of this expression is the determinant of the payoff matrix $\det(\mathbf{M})$. Dividing through Equation (22) by this determinant, we get

$$1 = \frac{1}{\det(\mathbf{M})}\left(\frac{p_{AA} - p_{AB}}{G_B(z)} - \frac{p_{BA} - p_{BB}}{G_{A'}(z)}\right). \tag{23}$$

The right side of this expression is a rational function in z. Any real root $\hat{z}$ of Equation (23) corresponds to the stable growth rate shared by A' and B at fixed points $\hat{x}$ of the dynamics in Equation (16). Relevant roots $\hat{z}$ must be strictly within $\bar{\lambda}_B(0)$ and $\bar{\lambda}_B(1)$ so that $\hat{x}$ is constrained to the $(0,1)$ interval. The corresponding value of $\hat{x}$ for some root $\hat{z}$ can be retrieved from either expression in Equation (20) by setting $z = \hat{z}$. Properties of such fixed points (e.g., linear stability, existence, and uniqueness)

are derived in the Appendix. These properties guarantee that when the system is at an initial stable fixed point and A mutates to A', then a new nearby interior fixed point (if it exists) should be reached. A numerical example is given in Figure 2.

Figure 2: Dynamics of the frequency of A and the stable fixed point under a perturbation



$$\mathbf{L}_A = \mathbf{L}_B = \begin{bmatrix} 0.20 & 0.3 & 0.8 & 1.0 & 0.9 & 0.4 \\ 0.45 & & & & & \\ & 0.6 & & & & \\ & & 0.9 & & & \\ & & & 0.8 & & \\ & & & & 0.5 & \end{bmatrix}$$

$$\mathbf{L}_{A'} = \begin{bmatrix} 0.2 & 0.25 & 1.20 & 1.00 & 0.8 & 0.2 \\ 0.4 & & & & & \\ & 0.57 & & & & \\ & & 0.95 & & & \\ & & & 0.75 & & \\ & & & & 0.3 & \end{bmatrix}$$

Note: A population in which A and B have equal vital rates (see inset PPM) is initiated and the dynamics of x are followed for 1,000 time steps. Initial state vectors are $[1,1,1,1,1,1]^T$ for A and $\frac{994}{6}[1,1,1,1,1,1]^T$ for B. Initial total population size is $1,000$ and $x(0) = \frac{6}{1000}$. Neither subpopulation is at the stable age distribution implied by the common PPM. At $t = 500$, A is mutated to A' (see inset PPM). Payoffs are $p_{AA} = 0.1$, $p_{AB} = 0.4$, $p_{BA} = 0.2$ and $p_{BB} = 0.3$.

4.2 Fixed-point sensitivity

The model in Equation (23) is challenging to analyze with respect to a specific perturbation that changes $\mathbf{L}_A$ into $\mathbf{L}_{A'}$. However, it is possible to formulate a simpler, approximate model similar to Equation (7), provided successive mutations that lead from A to A' affect only a single vital rate and are of small effect, and expected payoffs are also small.

Consider the initial scenario in which A and B have identical vital rates. Define the matrix $\mathbf{L}$ with nonzero entries $S_j = S_{j,A} = S_{j,B}$ and $F_j = F_{j,A} = F_{j,B}$ and Perron root $\lambda = \lambda_A = \lambda_B$ with corresponding right (column) $\mathbf{u}$ and left (row) $\mathbf{v}^T$ eigenvectors normalized so that $\sum_j u_j = 1$ and $\mathbf{v}^T\mathbf{u} = 1$. The vector $\mathbf{u}$ is proportional to the stable age distribution for the matrix population model $\mathbf{L}$, while $\mathbf{v}$ is usually called the reproductive

value vector, as its j component is proportional to the relative contribution made to the future population by individuals of age j (Caswell 2001). Define the matrix $\mathbf{K} = \mathbf{K}_A = \mathbf{K}_B$. Suppose that some entries of $\mathbf{L}$ depend on a parameter θ so that we can write $\mathbf{L}(\theta)$. Differentiating the eigenvector equation $\lambda = \mathbf{v}^T\mathbf{L}(\theta)\mathbf{u}$ with respect to θ and evaluating at $\theta = 0$ while eigenvectors are assumed constant leads to

$$\left.\frac{\partial\lambda}{\partial\theta}\right|_{\theta=0} = \mathbf{v}^T\left.\frac{\partial\mathbf{L}}{\partial\theta}\right|_{\theta=0}\mathbf{u}, \tag{24}$$

which corresponds to a classic result (Caswell 2001). We can use Equation (24) for two purposes. First, we can capture the linear change in λ due to expected payoffs by setting $\theta = \pi$. Then,

$$\mathbf{L}(\pi) = \begin{bmatrix} F_1 + \pi & F_2 + \pi & \dots & F_\omega + \pi \\ S_1 & & & \\ & \ddots & & \\ & & S_{\omega-1} & \end{bmatrix} \tag{25}$$

and the sensitivity of λ to expected payoffs is

$$\left.\frac{\partial\lambda}{\partial\pi}\right|_{\pi=0} = \mathbf{v}^T\left.\frac{\partial\mathbf{L}}{\partial\pi}\right|_{\pi=0}\mathbf{u} = \mathbf{v}^T\mathbf{K}\mathbf{u} = v_1. \tag{26}$$

Second, we can use Equations (24) and (26) to get an expression for the sensitivity of the initial fixed point to mutations in one type. Using Equation (26), we have that, to a linear approximation, the Perron root of $\bar{\mathbf{L}}_A(x) = \mathbf{L}_A + \pi_A(x)\mathbf{K}$ is $\bar{\lambda}_A(x) \approx \lambda_A + v_1\pi_A(x)$ and the Perron root of $\bar{\mathbf{L}}_B(x) = \mathbf{L}_B + \pi_B(x)\mathbf{K}$ is $\bar{\lambda}_B(x) \approx \lambda_B + v_1\pi_B(x)$. Therefore, at a fixed point of the dynamics in Equation (15), we have

$$\lambda_A + v_1\pi_A(\hat{x}) \approx \lambda_B + v_1\pi_B(\hat{x}) \tag{27}$$

solving this for $\hat{x}$ by using Equation (2),

$$\hat{x} \approx \frac{\lambda_B - \lambda_A + v_1(p_{BB} - p_{AB})}{v_1(p_{AA} - p_{AB} - p_{BA} + p_{BB})}. \tag{28}$$

At the initial fixed point (i.e., prior to perturbations on either type), $\lambda_B - \lambda_A = 0$ and Equation (28) is exact, as it gives the fixed point $\hat{x} = x^*$. To understand the effect that a perturbation θ on some nonzero entry of $\mathbf{L}_A$ has on the initial fixed point, we differentiate

Equation (28) with respect to θ. We do so by treating v_1 as constant. We also keep in mind that the perturbation involves only A and, therefore, λ_B is not a function of θ. We then obtain an expression for the fixed-point sensitivity to the perturbation

$$\left.\frac{\partial x}{\partial \theta}\right|_{\theta=0} \approx -\frac{1}{v_1(p_{AA}-p_{AB}-p_{BA}+p_{BB})}\left.\frac{\partial \lambda}{\partial \theta}\right|_{\theta=0}. \tag{29}$$

Note that the denominator in this expression is negative and, therefore,

$$\left.\frac{\partial x}{\partial \theta}\right|_{\theta=0} \propto \left.\frac{\partial \lambda}{\partial \theta}\right|_{\theta=0}. \tag{30}$$

We can thus linearly approximate the new fixed point that pertains to the dynamics in Equation (16) after the perturbation from the initial equilibrium x^* where A and B have identical vital rates as

$$\hat{x} \approx x^* - \frac{\theta}{v_1(p_{AA}-p_{AB}-p_{BA}+p_{BB})}\left.\frac{\partial \lambda}{\partial \theta}\right|_{\theta=0}. \tag{31}$$

We further assume that the mutation perturbs fertilities in an additive fashion, while it perturbs survival in a multiplicative fashion, as usual in population genetic models with age structure (Charlesworth 1994), and that the perturbation is limited to a single vital rate. Then, using Equation (24),

$$\left.\frac{\partial \lambda}{\partial \theta}\right|_{\theta=0} = \begin{cases} v_1 u_j, & \text{if perturbation is on } F_j \\ v_{j+1} u_j S_j, & \text{if perturbation is on } S_j, \end{cases} \tag{32}$$

as shown in (Caswell 1978). A classical result in evolutionary demography about the relative magnitudes of these derivatives in populations that are not decreasing in size is

$$v_1 u_j \geq v_1 u_{j+1} \tag{33}$$

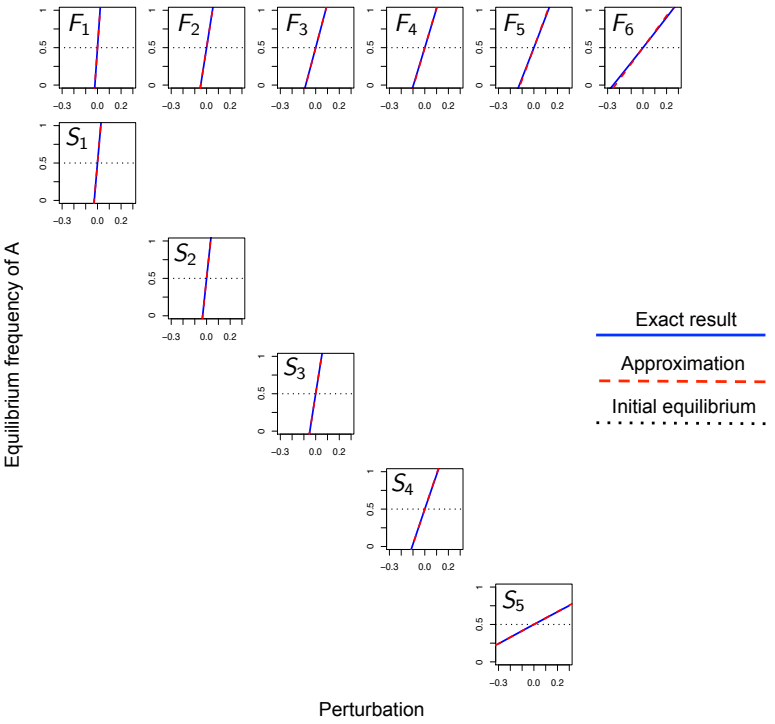
$$v_{j+1} u_j S_j \geq v_{j+2} u_{j+1} S_{j+1} \tag{34}$$

(Hamilton 1966; Charlesworth 1994; Caswell 2001).

Intuitively, this means that perturbations at older ages affect the population dynamics less. Population growth is more sensitive to an additive change in fertility, or a multiplicative change in survival, at an earlier age rather than at later ages. In our case, this means that coexistence is more likely to be preserved when a late life vital rate is perturbed compared to when the perturbation hits an early life vital rate. In Figure 3, we apply Equation

(31) to each nonzero entry of a projection matrix $\mathbf{L}_A$ while keeping the initially identical matrix $\mathbf{L}_B$ constant. Figure 3 shows that the fixed point is less sensitive to late life, as opposed to early life perturbations in vital rates.

Figure 3: Fixed-point sensitivity analysis



Note: The fixed point $\hat{x}$ is plotted as a function of the perturbation in a single vital rate of type A, separately for each vital rate, while B is kept unperturbed. Entries of the PPM of A and B are those in the shared PPM in Figure 2. Payoffs are $p_{AA} = 0.01$, $p_{AB} = 0.04$, $p_{BA} = 0.02$ and $p_{BB} = 0.03$.

5. Numerical exploration

5.1 Pleiotropy

Mutations can perturb more than one vital rate, at more than one age and in different directions (e.g., increasing one vital rate while decreasing another). We refer to this as

pleiotropy. In addition, mutations can hit either type A or B, and the magnitude may not always be small, as it is required to be by the model in Equation (31). Vital rate perturbations of this kind appear to be of particular interest for the study of demographic heterogeneity, as they should lead to a higher demographic diversity between the two types compared to perturbations limited to a single vital rate. Instead of using the approximate model of the previous section, here we numerically explore the exact model in Equation (23) to understand more precisely how much demographic heterogeneity the stable coexistence game can tolerate without one type dominating the other when both subpopulations separately incorporate pleiotropic mutations.

To this aim, we parametrize the set of baseline vital rates that are initially shared between A and B. Survival follows a Gompertz mortality function, which is commonly used to model mammalian mortality (Gage 1998, 2001). In continuous time, Gompertz mortality at age t is $\mu(t) = ae^{bt}$ where $a > 0$ gives baseline mortality and b is the rate of aging. In discrete time, this translates to

$$S_j = \exp\left(-\int_j^{j+1} \mu(y)\mathrm{d}y\right) = \exp\left(-\int_j^{j+1} ae^{by}\mathrm{d}y\right) = \exp\left[-\frac{a}{b}\left(e^{b(j+1)} - e^{bj}\right)\right]. \tag{35}$$

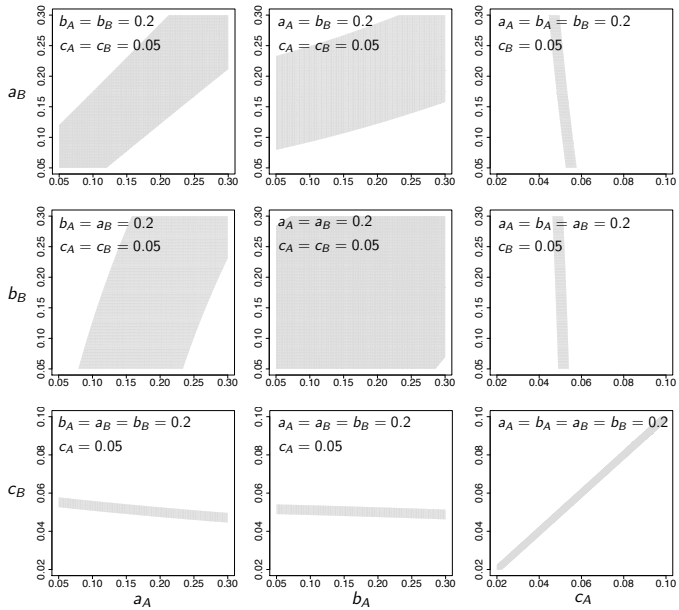
Survival can either decline ($b > 0$), improve ($b < 0$), or stay constant ($b = 0$) with age. As for fertility, we set

$$F_j = cj(\omega + 1 - j)^2, \tag{36}$$

where ω is the last age class. Thus, fertility is a third-degree polynomial in j, similar to that commonly used to model primate fertility (Gage 1998). With fixed $c > 0$, F_j is positive at all ages, increases from age 1 to a peak at some intermediate age, and then declines to the last age class.

We can now perturb a, b, and c as a proxy to the effect of the fixation of pleiotropic mutations.

Figure 4: Regions of stable coexistence between two types under pleiotropic mutations



Note: The shaded area indicates regions of stable coexistence between A and B for the given parameter combinations. Payoffs are $p_{AA} = 0.1$, $p_{AB} = 0.4$, $p_{BA} = 0.2$, and $p_{BB} = 0.3$. Here there are always $\omega_A = \omega_B = 6$ age classes.

We look at whether stable coexistence is possible when each type is perturbed for only one of the three above parameters. Figure 4 explores all possible pairwise combinations of pleiotropic perturbations and reports for each pair of perturbation values whether there is an equilibrium frequency $\hat{x}$ at which the two types have equal fitness. Figure 4 shows that stable coexistence is preserved for quite ample regions of the parameter space when perturbations involve survival parameters. This supports the robustness of the coexistence game against small, nonneutral pleiotropic mutations that arise and fixate within each subpopulation. However, we also see that the amount by which fertility can be perturbed while keeping coexistence is much narrower. This is explained by the fact that, in our model, a change in fertility has a very strong effect on fitness.

5.2 Cohort mortality

The average mortality in a cohort of same-age individuals from a demographically heterogeneous population differs from the mortalities observed in cohorts from composing subpopulations (Curtsinger et al. 1992; Vaupel and Carey 1993; Chen, Zajitschek, and Maklakov 2013; Vaupel and Yashin 1985). Here we show that interesting patterns of average cohort mortality can be retrieved from points inside coexistence regions like those explored in the previous section. Each point in this region represents a population that is characterized by a certain equilibrium frequency $\hat{x}$ of A individuals and by the two projection matrices of A and B evaluated at equilibrium. In both subpopulations, age-specific mortality follows a Gompertz function (defined above), possibly with different values for the parameters a and b between the two subpopulations. To observe the average mortality in a cohort of age 1 individuals that are sampled from this population, we adapt the methods of Vaupel and Yashin (1985). From the equilibrium projection matrices of A and B, we compute the respective equilibrium birth rates m_A and m_B. Here, m is defined as the stable fraction of individuals that, at each time step, enter the first age class at demographic stability. Thus, if at demographic stability u_j is the stable fraction of individuals in age class j and F_j is the number of individuals in age class 1 at $t+1$ per individual in age class j at t,

$$m = \sum_j u_j F_j. \tag{37}$$

The initial fraction of age 1 individuals of type A at demographic stability is

$$\vartheta_1 = \frac{\hat{x} m_A}{\hat{x} m_A + (1-\hat{x}) m_B}, \tag{38}$$

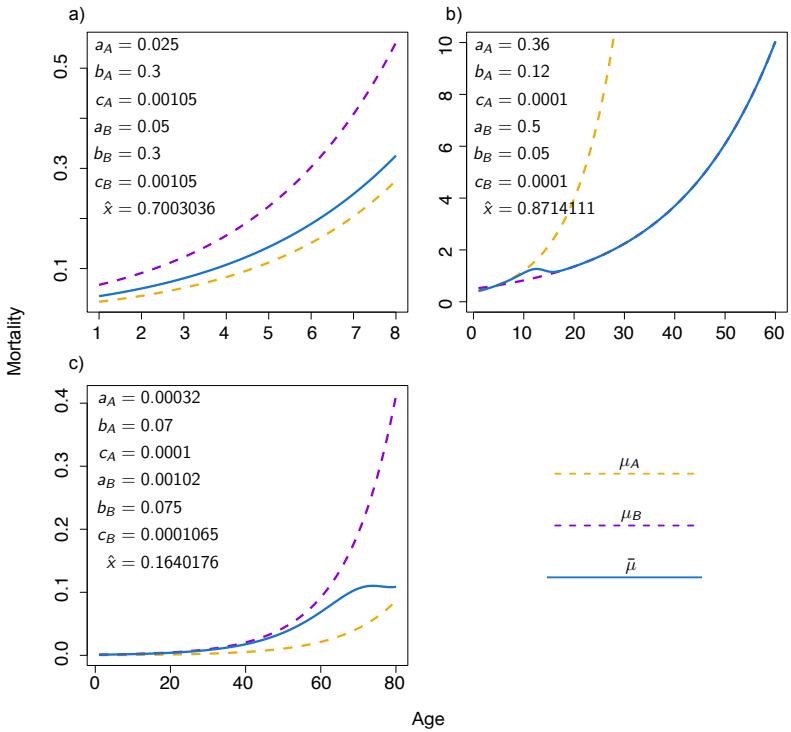
the remaining fraction $1-\vartheta_1$ being of type B. We then look at mortality in the cohort of individuals of age 1 composed of a fraction ϑ_1 of A and a fraction $(1-\vartheta_1)$ of B. The proportion $p_A(y)$ of the A subcohort that survives at least to age y is $\exp\left(-\int_1^y \mu_A(t)\mathrm{d}t\right)$. The corresponding parameter for B, $p_B(y)$, is found analogously. When our cohort is of age y, the fraction of A individuals is

$$\vartheta_y = \frac{\vartheta_1 p_A(y)}{\vartheta_1 p_A(y) + (1-\vartheta_1) p_B(y)}, \tag{39}$$

and average mortality at y in the cohort is

$$\bar{\mu}(y) = \vartheta_y \mu_A(y) + (1-\vartheta_y)\mu_B(y). \tag{40}$$

Figure 5: Average cohort mortality at coexistence



Note: The mortality for A and B (dashed lines) is plotted against age, up to the maximum attainable age, together with the average mortality (continuous line) in a cohort composed of a mixture of A and B. Both types have the same maximum age. The initial cohort composition is a function of the equilibrium fraction $\hat{x}$ of A and the birth rates of A and B at coexistence. Payoffs are $p_{AA} = 0.1$, $p_{AB} = 0.4$, $p_{BA} = 0.2$ and $p_{BB} = 0.3$.

Figure 5 shows the average cohort mortality for three different cases of coexistence along with the underlying mortality in the component subcohorts. Panel a) shows simple exponentially increasing average cohort mortality with age. Approximately this form of mortality characterizes adult life in many mammal species (Promislow 1991). Panel b) displays increasing average cohort mortality with age with a slight 'bump' in mortality at young adult ages. This is a pattern that is typical of human male demography (Carey and Judge 2000), where the passage to adulthood is marked by slightly increased risk of death. Panel c) shows average cohort mortality that levels off at late ages. This form of mortality is documented in flies, nematodes, and humans (Vaupel et al. 1998).

6. Conclusions

The emergence and maintenance of demographic heterogeneity in a population can be explained by some form of frequency-dependent selection (Le Cunff, Baudisch, and Pakdaman 2013). Le Cunff, Baudisch, and Pakdaman (2013) used stochastic agent-based simulations to show that types with discrete generations that differently allocate resources between survival and reproduction along their life may have equal fitness. For example, in their model, individuals that invest in early reproduction have a selective advantage in the first part of life over individuals that invest in early survival; in late life, the situation is reversed. An important numerical result of Le Cunff, Baudisch, and Pakdaman (2013) is that, in a population with an initially random distribution of allocation strategies, frequency-dependent selection eventually produces a quasi-stationary distribution of types. The average mortality in a cohort of individuals from the quasi-stable population qualitatively resembles that observed in cohorts of captive organisms and in humans (Le Cunff, Baudisch, and Pakdaman 2013).

In Le Cunff, Baudisch, and Pakdaman (2013), however, the initial population is assumed heterogeneous, and generations are not overlapping. Some questions are then left unanswered: How can a population become heterogeneous in the first place? Can types with different survival and reproductive strategies also stably coexist when generations overlap? When generations overlap, does the age distribution of the population play a role in the dynamics of demographic heterogeneity? In the present study, we addressed the first two open questions. As for the third, we simply note here that there may be non-intuitive consequences of selection on different fitness components in finite populations, depending on the initial stable age distribution (Li et al. 2016).

As for the first question, we showed that demographic heterogeneity may evolve from populations that initially are homogeneous from a demographic point of view, but that contain two behavioral types. On the background of a stable coexistence game between these two behaviorally distinct types, mutations can perturb vital rates of either type generating heterogeneity, yet coexistence is preserved. Provided that game payoffs are important in determining fitness, the game is qualitatively robust against perturbations, and frequency-dependent selection is key in maintaining diversity. Therefore, a form of fixed demographic heterogeneity may emerge from mutations in homogeneous populations. It should be kept in mind that our fundamental assumption is that the initial population is composed of two behaviorally different types, and such difference has a genetic basis, e.g., overdominance (Hofbauer and Sigmund 1998; Traulsen and Reed 2012). This may not always be a precondition for demographic heterogeneity, however. The presence of heterogeneous vital rates in a population is a prominent explanation of late-life deceleration of mortality observed in some species. Studies observed such deceleration both in cohorts of genetically heterogeneous medflies (Carey et al. 1992) and in highly inbred strains of flies (Curtsinger et al. 1992). This observation shows that

demographic heterogeneity is not necessarily connected with fixed heterogeneity, as we assume, and other forms of heterogeneity may play a role, as reviewed in the introduction section.

With regard to the second question, we showed that with overlapping generations also, frequency-dependent selection can maintain demographic heterogeneity. Differently from Le Cunff, Baudisch, and Pakdaman (2013), in our framework we opted for a deterministic model in which fitness is identified with the population growth rate. As a drawback, the presence of overlapping generations has complicated the analysis of our model. Therefore, we used numerical methods to find regions of coexistence under pleiotropic mutations. Using perturbation theory, we performed fixed-point sensitivity analysis to study how infinitesimal perturbations on single vital rates impact on the stable equilibrium between types. Our analysis predicted a higher level of heterogeneity in late life. This may be somehow expected in the light of the classic result, indicating that selective dynamics in age-structured populations in a constant environment show diminished sensitivity to changes in late-life fitness components (Hamilton 1966; Charlesworth 1994; Caswell 2001). Yet it is relevant to see how a similar result can be derived from a game theoretical setting that represents a different context from the constant fitness scenario assumed in the derivation of this classic result. We have not considered the scenario in which the environment stochastically changes with time so that vital rates fluctuate in response to it. The population then has a stochastic growth rate (Tuljapurkar 1990). The analysis of this scenario is much more complicated, as it requires the sensitivities of the stochastic growth rate, which involve the second derivatives of the leading eigenvalue of the average projection matrix (Caswell 2001). As a consequence, the relationship between fitness in early life and fitness in late life may also be less transparent than in the case of a constant environment (e.g., Orzack and Tuljapurkar 1989).

Interestingly, results based on pleiotropic mutations in our model can qualitatively mimic some mortality patterns that have been observed in natural and captive cohorts. This encourages us to further explore the potential of crossing demographic thinking with game theoretic approaches to explain patterns of mortality, and fertility, observed in nature.

7. Acknowledgments

Funding for Stefano Giaimo was provided by the Max Planck International Research Network on Aging (MaxNetAging).

References

Cam, E., Aubry, L.M., and Authier, M. (2016). The conundrum of heterogeneities in life history studies. *Trends in Ecology and Evolution* 31(11): 872–886. doi:10.1016/j.tree.2016.08.002.

Carey, J.R. and Judge, D.S. (2000). Mortality dynamics of aging. *Generations* 24: 19–24.

Carey, J., Liedo, P., Orozco, D., and Vaupel, J. (1992). Slowing of mortality rates at older ages in large medfly cohorts. *Science* 258(5081): 457–461. doi:10.1126/science.1411540.

Caswell, H. (1978). A general formula for the sensitivity of population growth rate to changes in life history parameters. *Theoretical Population Biology* 14(2): 215–230. doi:10.1016/0040-5809(78)90025-4.

Caswell, H. (2001). *Matrix population models*. Sunderland: Sinauer Associates.

Caswell, H. (2007). Sensitivity analysis of transient population dynamics. *Ecology Letters* 10(1): 1–15. doi:10.1111/j.1461-0248.2006.01001.x.

Chambon-Dubreuil, E., Auger, P., Gaillard, J.M., and Khaladi, M. (2006). Effect of aggressive behaviour on age-structured population dynamics. *Ecological Modelling* 193(3–4): 777–786. doi:10.1016/j.ecolmodel.2005.09.005.

Charlesworth, B. (1994). *Evolution in age-structured populations*. Cambridge: Cambridge University Press.

Charlesworth, B. and Partridge, L. (1997). Ageing: Levelling of the grim reaper. *Current Biology* 7(7): R440–R442. doi:10.1016/S0960-9822(06)00213-2.

Chen, H., Zajitschek, F., and Maklakov, A.A. (2013). Why ageing stops: Heterogeneity explains late-life mortality deceleration in nematodes. *Biology Letters* 9(5): 1–4. doi:10.1098/rsbl.2013.0217.

Cohen, J.E. (1979). Ergodic theorems in demography. *Bulletin of the American Mathematical Society (New Series)* 1(2): 275–295.

Cressman, R. (2003). *Evolutionary dynamics and extensive form games*. Cambridge: MIT Press.

Curtsinger, J., Fukui, H., Townsend, D., and Vaupel, J. (1992). Demography of genotypes: Failure of the limited life-span paradigm in drosophila melanogaster. *Science* 258(5081): 461–463. doi:10.1126/science.1411541.

Fox, G.A. and Gurevitch, J. (2000). Population numbers count: Tools for near-term demographic analysis. *The American Naturalist* 156(3): 242–256. doi:10.1086/303387.

Gage, T.B. (1998). The comparative demography of primates: With some comments on the evolution of life histories. *Annual Review of Anthropology* 27: 197–221.

Gage, T.B. (2001). Age-specific fecundity of mammalian populations: A test of three mathematical models. *Zoo Biology* 20(6): 487–499. doi:10.1002/zoo.10029.

Hamilton, W.D. (1966). The moulding of senescence by natural selection. *Journal of Theoretical Biology* 12(1): 12–45.

Hodgson, D.J. and Townley, S. (2004). Methodological insight: Linking management changes to population dynamic responses: The transfer function of a projection matrix perturbation. *Journal of Applied Ecology* 41(6): 1155–1161. doi:10.1111/j.0021-8901.2004.00959.x.

Hodgson, D.J., Townley, S., and McCarthy, D. (2006). Robustness: Predicting the effects of life history perturbations on stage-structured population dynamics. *Theoretical Population Biology* 70(2): 214–224. doi:10.1016/j.tpb.2006.03.004.

Hofbauer, J. and Sigmund, K. (1998). *Evolutionary games and population dynamics*. Cambridge: Cambridge University Press.

Horvitz, C.C. and Tuljapurkar, S. (2008). Stage dynamics, period survival, and mortality plateaus. *The American Naturalist* 172(2): 203–215.

Huang, W., Haubold, B., Hauert, C., and Traulsen, A. (2012). Emergence of stable polymorphism driven by evolutionary games between mutants. *Nature Communications* 3: 919.

Kendall, B.E., Fox, G.A., Fujiwara, M., and Nogeire, T.M. (2011). Demographic heterogeneity, cohort selection, and population growth. *Ecology* 92(10): 1985–1993. doi:10.1890/11-0079.1.

Kokko, H. (1997). Evolutionarily stable strategies of age-dependent sexual advertisement. *Behavioral Ecology and Sociobiology* 41(2): 99–107. doi:10.1007/s002650050369.

Kussel, E. and Leibler, S. (2005). Phenotypic diversity, population growth, and information in fluctuating environments. *Science* 309(5743): 2075–2078.

Le Bras, H. (1976). Lois de mortalité et âge limite [laws of mortality and the life span]. *Population* 31(3): 655–692.

Le Cunff, Y., Baudisch, A., and Pakdaman, K. (2013). How evolving heterogeneity distributions of resource allocation strategies shape mortality patterns. *PLoS Computational Biology* 9(1): e1002825. doi:10.1371/journal.pcbi.1002825.

Lewontin, R. (1970). The units of selection. *Annual Review of Ecology and Systematics*

1: 1–18.

Li, X.Y., Giaimo, S., Baudisch, A., and Traulsen, A. (2015). Modeling evolutionary games in populations with demographic structure. *Journal of Theoretical Biology* 380: 506–515.

Li, X.Y., Kurokawa, S., Giaimo, S., and Traulsen, A. (2016). How life history can sway the fixation probability of mutants. *Genetics* 203: 1297–1313.

Maynard Smith, J. (1982). *Evolution and the theory of games*. Cambridge: Cambridge University Press.

Maynard Smith, J. (1986). *The problems of biology*. Oxford: Oxford University Press.

Maynard Smith, J. (1995). *The theory of evolution*. Cambridge: Cambridge University Press.

Maynard Smith, J. and Szathmáry, E. (1995). *The major transitions in evolution*. Oxford: W.H. Freeman.

Meyer, C.D. (2015). Continuity of the Perron root. *Linear and Multilinear Algebra* 63(7): 1332–1336. doi:10.1080/03081087.2014.934233.

Nowak, M.A., Sasaki, A., Taylor, C., and Fudenberg, D. (2004). Emergence of cooperation and evolutionary stability in finite populations. *Nature* 428: 646–650.

Nowak, M. (2006). *Evolutionary dynamics*. Cambridge: Harvard University Press.

Orzack, S. and Tuljapurkar, S. (1989). Population dynamics in variable environments: VII: The demography and evolution of iteroparity. *American Naturalist* 133: 901–923.

Plard, F., Bonenfant, D., Delorme, D., and Gaillard, J. (2012). Modeling reproductive trajectories of roe deer females: Fixed or dynamic heterogeneity? *Theoretical Population Biology* 82(4): 317–328. doi:10.1016/j.tpb.2012.03.006.

Promislow, D. (1991). Senescence in natural populations of mammals: A comparative study. *Evolution* 45(8): 1869–1887.

Stearns, S. (1992). *The evolution of life histories*. Oxford: Oxford University Press.

Steiner, U.K. and Tuljapurkar, S. (2012). Neutral theory for life histories and individual variability in fitness components. *Proceedings of the National Academy of Sciences* 109(12): 4684–4689. doi:10.1073/pnas.1018096109.

Steinsaltz, D., Evans, S.N., and Wachter, K.W. (2005). A generalized model of mutation–selection balance with applications to aging. *Advances in Applied Mathematics* 35(1): 16–33. doi:10.1016/j.aam.2004.09.003.

Stott, I., Townley, S., and Hodgson, D.J. (2011). A framework for studying transient

dynamics of population projection matrix models. *Ecology Letters* 14(9): 959–970. doi:10.1111/j.1461-0248.2011.01659.x.

Traulsen, A. and Reed, F.A. (2012). From genes to games: Cooperation and cyclic dominance in meiotic drive. *Journal of Theoretical Biology* 299: 120–125.

Tuljapurkar, S. (1990). *Population dynamics in variable environments*. Berlin: Springer.

Tuljapurkar, S., Steiner, U.K., and Orzack, S. (2009). Dynamic heterogeneity in life histories. *Ecology Letters* 12(1): 93–106. doi:10.1111/j.1461-0248.2008.01262.x.

Vaupel, J.W. and Carey, J.R. (1993). Compositional interpretations of medfly mortality. *Science* 260(5114): 1666–1667. doi:10.1126/science.8503016.

Vaupel, J.W., Carey, J.R., Christensen, K., Johnson, T.E., Yashin, A.I., Holm, N.V., Iachine, I.A., Kannisto, V., Khazaeli, A.A., Liedo, P., Longo, V.D., Zeng, Y., Manton, K.G., and Curtsinger, J.W. (1998). Biodemographic trajectories of longevity. *Science* 280(5365): 855–860. doi:10.1126/science.280.5365.855.

Vaupel, J.W., Manton, K.G., and Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography* 16(3): 439–454. doi:10.2307/2061224.

Vaupel, J.W. and Yashin, A.I. (1985). Heterogeneity's ruses: Some surprising effects of selection on population dynamics. *The American Statistician* 39(3): 176–185.

Wilson, A.J. and Nussey, D.H. (2010). What is individual quality? An evolutionary perspective. *Trends in Ecology and Evolution* 25(4): 207–214. doi:10.1016/j.tree.2009.10.002.

Yashin, A.I., Iachine, I.A., and Harris, J.R. (1999). Half of the variation in susceptibility to mortality is genetic: Findings from Swedish twin survival data. *Behavior Genetics* 29(1): 11–19. doi:10.1023/A:1021481620934.

Yashin, A.I., Vaupel, J.W., and Iachine, I.A. (1994). A duality in aging: The equivalence of mortality models based on radically different concepts. *Mechanisms of Ageing and Development* 74(1): 1–14. doi:10.1016/0047-6374(94)90094-9.

Yearsley, J.M. (2004). Transient population dynamics and short-term sensitivity analysis of matrix population models. *Ecological Modelling* 177(3): 245–258. doi:10.1016/j.ecolmodel.2003.12.053.

Appendix

Linear stability

Here we establish the linear stability of interior fixed points of the dynamics in Equation (16). Along with the set of inequalities in (5), we also assume

$$p_{AA} < p_{BA} < p_{BB} < p_{AB} < 1, \tag{41}$$

so that payoffs make a limited contribution to fitness. First of all, $u_{j,A'}(t) = \frac{n_{j,A'}}{N_{A'}}(t)$ and $u_{j,B}(t) = \frac{n_{j,B}}{N_B}(t)$, i.e., the current fractions of the A and B subpopulations in age class j. Then, using Equation (10), we write a dynamic equation for x, which is now the A' fraction of the population, and we implicitly define a function $f(x(t))$,

$$\begin{aligned} x(t+1) &= \frac{N_{A'}(t+1)}{N_{A'}(t+1) + N_B(t+1)} \\ &= \frac{\sum_j n_{j,A'}(t)(\bar{F}_{j,A'}(t) + S_{j,A'})}{\sum_j n_{j,A'}(t)(\bar{F}_{j,A'}(t) + S_{j,A'}) + \sum_j n_{j,B}(t)(\bar{F}_{j,B}(t) + S_{j,B})} \\ &= \frac{x(t)N(t)\sum_j u_{j,A'}(t)(\bar{F}_{j,A'}(t) + S_{j,A'})}{x(t)N(t)\sum_j u_{j,A'}(t)(\bar{F}_{j,A'}(t) + S_{j,A'}) + (1-x(t))N(t)\sum_j u_{j,B}(t)(\bar{F}_{j,B}(t) + S_{j,B})} \\ &= \underbrace{\frac{x(t)W_{A'}(t)}{x(t)W_{A'}(t) + (1-x(t))W_B(t)}}_{f(x(t))}, \end{aligned} \tag{42}$$

where it is assumed that $S_{\omega,A} = S_{\omega,B} = 0$ and where $W_{A'}(t) = \sum_j u_{j,A'}(t)(\bar{F}_{j,A'}(t) + S_{j,A'})$ and $W_B(t) = \sum_j u_{j,B}(t)(\bar{F}_{j,B}(t) + S_{j,B})$. To simplify notation, we set

$$M(t) = x(t)W_{A'}(t) + (1-x(t))W_B(t). \tag{43}$$

We differentiate W using Equations (2) and (8),

$$\frac{\partial W_{A'}}{\partial x} = \sum_j u_{j,A'}(p_{AA} - p_{AB}) = (p_{AA} - p_{AB})\sum_j u_{j,A'} = p_{AA} - p_{AB}, \tag{44}$$

and, similarly,

$$\frac{\partial W_B}{\partial x} = \sum_j u_{j,B}(p_{BA} - p_{BB}) = (p_{BA} - p_{BB})\sum_j u_{j,B} = p_{BA} - p_{BB}. \tag{45}$$

Thus,

$$\frac{\partial M}{\partial x} = W_{A'} + x(p_{AA} - p_{AB}) + p_{BA} - p_{BB} - W_B - x(p_{BA} - p_{BB}), \tag{46}$$

and, finally,

$$\frac{\partial f}{\partial x} = \frac{1}{M^2}\left[M\left(W_{A'} + xp_{AA} - xp_{AB}\right) - xW_{A'}\frac{\partial M}{\partial x}\right]. \tag{47}$$

We evaluate this expression at some interior equilibrium $x(t) = \hat{x}$ recalling that, at this equilibrium, we also have demographic stability and, therefore, $N_A(t+1) = \bar{\lambda}(\hat{x})N_A(t)$ and $N_B(t+1) = \bar{\lambda}(\hat{x})N_B(t)$ where $\bar{\lambda}(\hat{x}) = \bar{\lambda}_A(\hat{x}) = \bar{\lambda}_B(\hat{x})$ is the leading eigenvalue shared by $\bar{\mathbf{L}}_{A'}(\hat{x})$ and $\bar{\mathbf{L}}_B(\hat{x})$. Hence, as $W_{A'}(\hat{x}) = W_B(\hat{x}) = \bar{\lambda}(\hat{x})$,

$$\left.\frac{\partial f}{\partial x}\right|_{x=\hat{x}} = 1 + \frac{\hat{x}(1-\hat{x})(p_{AA} - p_{AB} - p_{BA} + p_{BB})}{\bar{\lambda}(\hat{x})}. \tag{48}$$

Because of the inequalities in (5) and (41),

$$-1 < p_{AA} - p_{AB} - p_{BA} + p_{BB} < 0. \tag{49}$$

Note also that in Equation (48) we should have $\bar{\lambda} \geq 1(\hat{x})$, as it makes sense to focus only on populations that are not going extinct, i.e., geometric growth per time unit should not be smaller than unity. Thus,

$$0 < \frac{\hat{x}(1-\hat{x})}{\bar{\lambda}(\hat{x})} < \frac{1}{4}, \tag{50}$$

and

$$1 - \frac{1}{4} < \left.\frac{\partial f}{\partial x}\right|_{x=\hat{x}} < 1. \tag{51}$$

We then conclude that the interior fixed point $x = \hat{x}$ is linearly stable. We have assumed that A' and B may have different vital rates. However, the same result holds for A and B when the two types share the same vital rates. This means that when the system is at an initial stable fixed point and A mutates to A', a new nearby interior fixed point (if it exists) should be reached. A numerical example is given in Figure 2.

Existence and uniqueness

In the previous section, the interior fixed point $x = \hat{x}$ was arbitrary. Hence, linear stability is shown for every interior fixed point. Suppose that f has more than one interior fixed

point. Then there are at least two distinct interior fixed points, say, $\hat{x}_1$ and $\hat{x}_2$ with $\hat{x}_1 < \hat{x}_2$. By our stability result, $1 - \frac{1}{4} < \frac{\partial f}{\partial x}|_{x=\hat{x}_1} < 1$ and $1 - \frac{1}{4} < \frac{\partial f}{\partial x}|_{x=\hat{x}_2} < 1$. Therefore, there is some $0 < h \ll \hat{x}_2 - \hat{x}_1$ such that $f(\hat{x}_1 + h) < \hat{x}_1 + h$ and $f(\hat{x}_2 - h) > \hat{x}_2 - h$. By the intermediate value theorem, f must have at least one other fixed point, say, $\hat{x}_3$ between $\hat{x}_1$ and $\hat{x}_2$ and such that $\frac{\partial f}{\partial x}|_{x=\hat{x}_3} > 1$. But this means that $\hat{x}_3$ is linearly unstable contradicting the fact that every interior fixed point of f is linearly stable. Therefore, if a stable interior fixed point exists, then it is unique.

The Perron root of a matrix is continuous in the matrix entries (Meyer 2015). The Perron roots $\bar{\lambda}_A(x)$ and $\bar{\lambda}_B(x)$ are continuous in their respective entries $\bar{F}_{j,A}(x)$ and $\bar{F}_{j,B}(x)$, which in turn are continuous in x via expected payoffs. Hence, $\bar{\lambda}_A(x)$ and $\bar{\lambda}_B(x)$ are continuous in x and, by the intermediate value theorem, the function $\bar{\lambda}_A(x) - \bar{\lambda}_B(x)$ has at least one root in the $(0, 1)$ interval when the following condition holds

$$\text{sgn}\left(\bar{\lambda}_{A'}(0) - \bar{\lambda}_B(0)\right) \neq \text{sgn}\left(\bar{\lambda}_{A'}(1) - \bar{\lambda}_B(1)\right), \tag{52}$$

where $\text{sgn}(\cdot)$ is the sign function. This condition is sufficient for the existence of a unique interior fixed point in our system in Equation (16), but it is not necessary. If the condition is false, the function $\bar{\lambda}_A(x) - \bar{\lambda}_B(x)$ may still have a root, provided that the x axis is tangential to it at a point. However, we do not consider this possibility here.