JOURNAL OF Evolutionary Biology

doi: 10.1111/j.1420-9101.2012.02472.x

The stationary distribution of a continuously varying strategy in a class-structured population under mutation–selection–drift balance

L. LEHMANN

Department of Ecology and Evolution, UNIL Sorge, Le Biophore, Lausanne, Switzerland

Keywords:

adaptive dynamics;
adaptive landscape;
convergence stability;
evolutionary stability;
game theory;
inclusive fitness;
kin selection;
mutation–selection–drift balance;
quantitative phenotypes;
stochastic stability.

Abstract

Many traits and/or strategies expressed by organisms are quantitative phenotypes. Because populations are of finite size and genomes are subject to mutations, these continuously varying phenotypes are under the joint pressure of mutation, natural selection and random genetic drift. This article derives the stationary distribution for such a phenotype under a mutation–selection–drift balance in a class-structured population allowing for demographically varying class sizes and/or changing environmental conditions. The salient feature of the stationary distribution is that it can be entirely characterized in terms of the average size of the gene pool and Hamilton's inclusive fitness effect. The exploration of the phenotypic space varies exponentially with the cumulative inclusive fitness effect over state space, which determines an adaptive landscape. The peaks of the landscapes are those phenotypes that are candidate evolutionary stable strategies and can be determined by standard phenotypic selection gradient methods (e.g. evolutionary game theory, kin selection theory, adaptive dynamics). The curvature of the stationary distribution provides a measure of the stability by convergence of candidate evolutionary stable strategies, and it is evaluated explicitly for two biological scenarios: first, a coordination game, which illustrates that, for a multipeaked adaptive landscape, stochastically stable strategies can be singled out by letting the size of the gene pool grow large; second, a sex-allocation game for diploids and haplo-diploids, which suggests that the equilibrium sex ratio follows a Beta distribution with parameters depending on the features of the genetic system.

Introduction

Many phenotypes are quantitative and can be measured on a continuous scale. For instance, allocations of resources to growth, survival, defence, male and female function or offspring production are continuously varying strategies. Body shape and size, rates of transcription, enzymatic fluxes, intensities of desires, dates of first flowering or maximum flight speed are all phenotypes belonging to a continuum. Because of such a prevalence of continuous phenotypes in natural populations, it is relevant to try to understand their evolutionary dynamics and stationary distributions under the joint pressure of mutation, natural selection and random genetic drift. Nevertheless, few studies have analytically addressed the evolution of quantitative phenotypes under the action of these three evolutionary forces, and they often focus on situations of frequency-independent selection, where the recipient of the expression of the phenotype is the actor alone (e.g. Lande, 1976; Bürger *et al.*, 1989; Bürger & Lande, 1994).

Because resources come in finite supply, many phenotypic traits are actually subject to frequency-dependent selection at the intraspecific level, where the behaviour of one individual affects the fitness of others. These include resource competition efforts, mating and foraging tactics, sex ratio, optimal dispersal, parent–offspring conflict, anisogamy, storage effects, levels of social learning or waiting times in attrition fighting. The evolution of continuous phenotypes with frequency-dependent selection is more complicated to analyse than without,

Correspondence: Laurent Lehmann, Department of Ecology and Evolution, UNIL Sorge, Le Biophore, CH-1015 Lausanne, Switzerland.
Tel.: +41 21 692 4183; fax: +41 21 692 4265;
e-mail: laurent.lehmann@unil.ch

and simplifying assumptions are necessary in order to make the analysis tractable. Key assumptions include removing from the analysis one or several evolutionary forces, generally mutation and/or genetic drift, and focusing on a two-allele system coding for mutant and resident phenotypes, where the mutant deviates phenotypically only by small magnitude from the resident. Under these general assumptions, and at the risk of oversimplifying the presentation, one can identify three interrelated approaches for studying the evolution of continuous phenotypes.

The first could be labelled classical evolutionary game or kin selection theory for continuous phenotypes (e.g. Maynard Smith, 1982; Eshel, 1983; Taylor, 1989; Parker & Maynard Smith, 1990; Bulmer, 1994; Taylor & Frank, 1996; Frank, 1998; Pen, 2000; Ohtsuki & Iwasa, 2004; Vincent & Brown, 2005; Lion & Gandon, 2009). Here, the population is assumed to be of total infinite size. Genetic drift at the global scale is thus removed from the model and mutations are not explicitly considered in the formalization, as one is essentially interested in characterizing the end points of the evolutionary dynamics. These are the candidate evolutionary stable strategies (ESS). In practice, they are obtained from phenotypic selection gradients often through the form of the optimization of an individual fitness function (Maynard Smith, 1982; Parker & Maynard Smith, 1990; Vincent & Brown, 2005).

Because stable strategies are identified by comparing the fitness of pairs of strategies, namely, by focusing on the mutant-resident system, implicit in classical evolutionary game theory is an evolutionary dynamic that is assumed decomposable into two time scales (Eshel, 1996; Hammerstein, 1996; Eshel *et al.*, 1998): first, a fast time scale of short-term evolution. This is the time scale during which a novel mutation appears in a population monomorphic for a resident phenotype, and is either eliminated or selected to fixation before any other new mutation appears. The superposition of several of these trait-substitution events yields the second, slower time scale of steady long-term evolution of the phenotype. Evolution is thus regarded as a step-by-step transformation of the phenotype caused by the successive invasion of rare mutant alleles. The orbit of the phenotype in state space eventually converges towards a singular point, a cycle, or is altered forever in a strange attractor (Eshel, 1996; Hammerstein, 1996; Eshel *et al.*, 1998).

The second approach to the evolution of continuous phenotypes is adaptive dynamics. This broadens the first by focusing not only on phenotypic selection gradients but also on the time course of evolution (e.g. Dieckmann & Law, 1996; Geritz *et al.*, 1998; Ferrière *et al.*, 2002; Waxman & Gavrilets, 2005; Champagnat *et al.*, 2006; Dercole & Rinaldi, 2008; Leimar, 2009; Zu *et al.*, 2010). Here, evolution is also assumed to be decomposable into a two-time scale dynamics, but long-term evolution is made more explicit by the incorporation into the formalization of mutation rates and the evaluation of the time dynamics of the phenotype itself. In addition to characterizing candidate ESS and other singular points (phenotypic values at which the local selection gradient vanishes), the adaptive dynamics approach also allows one to explicitly track the changes in phenotype along the orbits in phenotype space towards singular points or through other attractors (Dercole & Rinaldi, 2008). But as under classical game theory, the stochastic effects introduced by genetic drift are often ignored in practice and candidate ESS are obtained from phenotypic selection gradients by way of the optimization of an individual fitness function (Geritz *et al.*, 1998; Dercole & Rinaldi, 2008).

The third approach to the evolution of continuous phenotypes under frequency-dependent selection may be called kin selection (or inclusive fitness) theory for finite populations (e.g. Rousset & Billiard, 2000; Leturque & Rousset, 2002; Roze & Rousset, 2003; Rousset, 2004; Rousset & Ronce, 2004; Taylor *et al.*, 2007a, b). Here, as under the two other approaches, a two-time scale evolutionary dynamic is assumed. As under classical evolutionary game theory, mutations to all possible phenotypes are not explicitly taken into account in the formalization. But, in contrast to the two other approaches, short-term phenotypic evolution is explicitly determined from changes (perturbations) of the fixation probability of a mutant allele introduced into a monomorphic population of residents. The fixation probability captures the effect of both natural selection and random genetic drift on the evolutionary dynamics, from the appearance of a mutant until its loss from or fixation in the population. Importantly, the fixation probability perturbations turn out to be proportional to phenotypic selection gradients for weak selection intensities, so that in practice candidate ESS are obtained from the optimization of an individual fitness function, as under the two other approaches (Leturque & Rousset, 2002; Rousset, 2004).

The identification of singular points of the evolutionary dynamics for continuous phenotype is thus obtained by broadly similar methods throughout evolutionary biology, and whether evolution occurs in finite populations (stochastic systems) or infinite populations (deterministic systems). But in the presence of several singular points, which may occur when the adaptive landscape is multipeaked, the long-term behaviour of a stochastic system may differ markedly from that of a deterministic system. Constant dynamic shocks introduced by the flow of mutations and the sampling effects occurring in finite population may accumulate and tip the balance from one singular point to the other. For a multipeaked fitness landscape, a higher peak may then eventually be singled out by the evolutionary dynamics even if the population can remain locked in a suboptimal peak for a very long time. This state space exploration process due to the interaction between mutation, selection and drift is ingrained in population genetics

(Wright, 1931; Barton *et al.*, 2007; Hartl & Clark, 2007) and used as an equilibrium selection device in game theory (Foster & Young, 1990; Binmore *et al.*, 1995), but it has not been much explored in the context of the evolution of continuous phenotypes.

In this article, the substitution rate approach to the separation between short- and long-term evolution of population genetics (Gillespie, 1983, 1991; Orr, 1998; Sella & Hirsh, 2005) is used in order to derive a diffusion equation for the evolution of a continuous phenotype. Mutation, natural selection and random genetic drift are allowed to jointly affect the evolutionary dynamic when it takes place in a class-structured population with demographically varying class sizes and/or changes in environmental conditions. The approach highlights strong links between the adaptive dynamics framework and the direct fitness (or neighbour-modulated) method of kin selection theory. The article is organized as follows: Model introduces the biological assumptions of the model and specifies the separation of time scales hypothesis. Analysis derives a phenotypic substitution rate for class-structured populations (fast time scale) and a diffusion equation for long-term phenotypic evolution (slow time scale). Stationary Distribution in Terms of Phenotypic Selection Gradient connects the stationary distribution of the slow process to standard phenotypic selection gradients. Applications presents two applications of the stationary distribution, and Discussion discusses the results.

Model

Biological assumptions

Consider a population where individuals express a quantitative phenotype, which may affect the vital rates of the actor (e.g. fecundity, survival, mating) and possibly those of other individuals in the population, the recipients of the actor's phenotype. The quantitative phenotype is assumed to be determined by a one-locus genetic basis with a continuum of possible allelic effects (Kimura, 1965; Bürger, 2000).

The individuals in this population are assumed to be structured into a finite number of classes. This class structure could result from the presence of males and females, of age-classes, of group of individuals located at different positions in the habitat, or from any life-history feature causing different individuals to be in different developmental, physiological or environmental states (Taylor, 1990; Frank, 1998; Caswell, 2000; Rousset, 2004). The number of individuals in class i is written N_i and the vector $\mathbf{s} \equiv (N_0, N_1, N_2 \ldots)$ denotes a state of the population, which gives the number of individuals in each class i at a census point. Individuals in different classes, like males and females, may have different ploidies, and g_i denotes the ploidy of an individual of class i.

When this population is monomorphic for phenotypic value z (no genetic variation) and conditional on its nonextinction, the change in the class structure is assumed to be determined by a transition probability $\Pr(\mathbf{s}' \mid \mathbf{s}, z)$ from state $\mathbf{s}$ in a parental generation to state $\mathbf{s}'$ in the offspring generation (a list of functionals is given in Tables 1 and 2). This defines a homogeneous Markov chain (Karlin & Taylor, 1975; Grimmett & Stirzaker, 2001), which may be driven by both endogenous (demographic) and exogenous (environmental) factors. This Markov chain is assumed irreducible and may then eventually enter the stationary probability $\Pr(\mathbf{s}|z)$ of being in state $\mathbf{s}$ when the population is monomorphic for z (Karlin & Taylor, 1975; Grimmett & Stirzaker, 2001). Under this process, and conditional on producing a class-i individual, an individual of class j with phenotypic value z is assumed to transmit to this descendant a mutant gene that codes for phenotype $z + \delta$ with probability $\mu_{ij}(\delta, z)$, where the mutation distribution is assumed to be symmetric around z, so that it has zero mean. For diploids, the phenotype $z + \delta$ obtains if the class-i offspring is made homozygous for the mutant allele. The model thus allows for dominance (e.g. Roze & Rousset, 2003), and I will refer to a gene coding for phenotype $z + \delta$ as a δ mutant.

Separation of time scales

With a high mutation rate in one or several classes of individuals, the population is very unlikely to ever be

Table 1 List of functionals.

Symbol	Definition
$k(\delta, z)$	Substitution rate of a z population by a δ mutant.
$N_i(\mathbf{s}, z)$	Number of class i individuals in a state $\mathbf{s}$ population fixed for z.
$\bar{N}(z)$	Average number of gene copies in a population fixed for z.
$\Pr(\mathbf{s} \mid z)$	Stationary probability of state $\mathbf{s}$ in a population fixed for z.
$\Pr(\mathbf{s}' \mid \mathbf{s}, z)$	Forward transition probability in a z population from state $\mathbf{s}$ in a parental generation to state $\mathbf{s}'$ in the offspring generation.
$\Pr(\mathbf{s} \mid \mathbf{s}', z)$	Backward transition probability in a z population. This is the probability that a population in state $\mathbf{s}'$ in an offspring generation descends from a population in state in the parental generation (a prime generally refers to an offspring generation)
$w_{ij}(\mathbf{s}', \mathbf{s}, z)$	Expected number of class-i individuals in a population in state $\mathbf{s}'$ descending from a single class j individual in a population fixed for z and in state $\mathbf{s}$.
g_i	Ploidy of a class i individual.
t_{ij}	Probability that a gene in a class i individual is a copy of a gene from a class j individual.
$f_{ij}(\mathbf{s}', \mathbf{s}, z)$	Probability that a gene sampled in a class i individual when the population is in state $\mathbf{s}'$ is a copy of a gene of a class j individual when the population was in state $\mathbf{s}$ in the parental generation.
$\alpha_i(\mathbf{s}, z)$	Probability that a gene randomly sampled from a z population descends from a class i individual in the distant past, conditional on the population being in state $\mathbf{s}$ in the distant past. This is the reproductive value of class i conditional on the population being in state $\mathbf{s}$.

Table 2 List of functionals.

Symbol	Definition
$\mu_{ij}(\delta, z)$	Probability that when a class j individual with phenotype z produces a class i individual, the descendant is a δ mutant.
$\mu(z)$	Probability that a mutation arises in a z population.
$u(\delta, z)$	Probability that, conditional on a mutation arising in a z population, the mutation codes for a δ mutant.
$M_i(\mathbf{s}, \delta, z)$	Number of δ mutants of class i in a z population in state $\mathbf{s}$.
$M(\delta, z)$	Average number of δ mutant produced in a z population.
$p(z(t) = z)$	Probability density function that phenotypic value z obtains at time t. The forward transition probability density from phenotypic state z at time t to $z + \delta$ at time Δt for the process is $p(z(t + \Delta t) = z + \delta \mid z(t) = z)$.
$\pi_i(\mathbf{s}, \delta, z)$	Fixation probability in a z population in state $\mathbf{s}$ of a single δ mutant residing in a class i individual.
$\bar{\pi}_\mu(\delta, z)$	Average fixation probability in a z population of a single δ mutant.
$\bar{\pi}(\delta, z)$	Average fixation probability in a z population of a single δ mutant when the mutation rate is the same across classes.
$\bar{\pi}^\circ_\mu(z)$	Average fixation probability in a z population of a neutral mutant.
$\dot{\bar{\pi}}_\mu(z)$	First-order perturbation of $\bar{\pi}_\mu(\delta, z)$.
$S(z)$	Localized selection gradient: first-order perturbation of $\bar{\pi}(\delta, z)$.

strictly monomorphic for a given trait value and several alleles may simultaneously segregate in the population. This results in a joint demographic and genetic stochastic process describing both the number of individuals in each class in the population and the alleles they carry. A state of this demo-genetic process can be characterized by the number of each allele in each class of individuals, thus yielding a multidimensional Markov chain on an uncountable state space (Meyn & Tweedie, 2009). Owing to the fact that this process has no absorbing states, it may in the long run reach a stationary distribution, which provides the phenotypic distribution in the population under a mutation–selection–drift balance. Nevertheless, the analysis of such a demo-genetic stochastic process is very involved mathematically even in the simple case of a haploid iteroparous panmictic population without class structure (Champagnat & Lambert, 2007),

It is thus relevant to make simplifying assumptions in order to reduce the state space of the process and to allow for more general life-cycle features or life-history modes. This can be achieved by assuming a separation of time between short- and long-term evolution, as it allows reducing the multidimensional Markov chain to a one-dimensional process, whose state space is the range of values z can take. The separation of time into fast and slow process is now fully endorsed and $z(t)$ will denote the value of the evolving phenotype at time t, which refers to the slow time scale. The phenotypic value $z(t)$ is a random variable, and the shorthand notation $p(z,t) \equiv p(z(t) = z \mid z(0) = z_0)$ is used to denote the probability density function that a uniform randomly sampled member of the population at time t expresses phenotypic value z, conditional on the initial phenotypic value in the population being z_0 at $t = 0$.

Call $k(\delta,z)$ the number of δ mutants, which are produced over one iteration of the life cycle in a monomorphic population for z at a demographic equilibrium, and that will fix in the population. If the process was run for a long time, then $k(\delta,z)$ would give the substitution rate per life-cycle iteration of a z population by a δ mutant, but a certain amount of time may occur for this substitution to take place (e.g. Kimura, 1971; Gillespie, 1991). The separation of time-scale assumption for $z(t)$ is introduced by assuming that the substitution by a δ mutant is instantaneous and occurs over an infinitesimally small time step of the $z(t)$ process:

$$\lim_{\Delta t \to 0} \frac{p(z(t + \Delta t) = z + \delta | z(t) = z)}{\Delta t} = k(\delta, z) \qquad (1)$$

for $\delta \neq 0$ where $p(z(t + \Delta t) = z + \delta \mid z(t) = z)$ is the probability density that the population is monomorphic for $z + \delta$ at time $t + \Delta t$ given that it was monomorphic for z at t. Hence, $k(\delta,z)$ gives the instantaneous substitution rate from phenotypic state z to $z + \delta$ at the level of the population.

Analysis

Fast dynamics: phenotypic substitution

Substitution rate

Although the model allows for a fluctuating demography and/or environments in a class-structured population, the substitution rate of a δ mutant can be expressed in a compact form as

$$k(\delta, z) = \bar{N}(z)\mu(z)u(\delta, z)\bar{\pi}_\mu(\delta, z), \qquad (2)$$

where $\bar{N}(z)$ is the average number of gene copies in a population monomorphic for z, $\mu(z)$ is the probability that a randomly sampled gene from this population mutates, $u(\delta,z)$ is the probability that the mutant is a δ mutant, and $\bar{\pi}_\mu(\delta, z)$ is the average fixation probability in a population monomorphic for z of a single δ mutant (see Appendix A, eqns A-1–A-11, for a derivation and Table 3 for explicit expressions of these functionals). The subscript μ in $\bar{\pi}_\mu(\delta, z)$ emphasizes that this quantity may

Table 3 Substitution rate quantities.

Full expressions

$\bar{N}(z) = \sum_{\mathbf{s}}\sum_i g_i N_i(\mathbf{s}, z)\Pr(\mathbf{s} \mid z)$

$\mu(z) = \sum_{\mathbf{s}}\sum_i \left[\int M_i(\mathbf{s}, \delta, z)\mathrm{d}\delta\right]\Pr(\mathbf{s} \mid z)/\bar{N}(z)$

$u(\delta, z) = M(\delta, z)/[\bar{N}(z)\mu(z)]$

$\bar{\pi}_\mu(\delta, z) = \sum_{\mathbf{s}}\sum_i \pi_i(\mathbf{s}, \delta, z)M_i(\mathbf{s}, \delta, z)\Pr(\mathbf{s} \mid z)/M(\delta, z)$

$M_i(\mathbf{s}', \delta, z) = g_i N_i(\mathbf{s}, z)\sum_{\mathbf{s}}\sum_j \Pr(\mathbf{s} \mid \mathbf{s}', z)f_{ij}(\mathbf{s}', \mathbf{s}, z)\mu_{ij}(\delta, z)$

$M(\delta, z) = \sum_{\mathbf{s}}\sum_i M_i(\mathbf{s}, \delta, z)\Pr(\mathbf{s} \mid z)$

Same mutation rate across classes and demographic states

$[\mu_{ij}(\delta, z) = \mu(\delta, z)]$

$\bar{N}(z) = \sum_{\mathbf{s}}\sum_i g_i N_i(\mathbf{s}, z)\Pr(\mathbf{s} \mid z)$

$\mu(z) = \int \mu(\delta, z)\mathrm{d}\delta$

$u(\delta, z) = \mu(\delta, z)/\mu(z)$

$\bar{\pi}(\delta, z) = \sum_{\mathbf{s}}\sum_i \pi_i(\mathbf{s}, \delta, z)g_i N_i(\mathbf{s}, z)\Pr(\mathbf{s} \mid z)/\bar{N}(z)$

depend on the class-specific mutation probability distribution, $\mu_{ij}(\delta,z)$. If individuals in different classes have different mutation rates, the probability that a δ mutant arises in a given class depends on class-specific mutation rates, and this will affect the average fixation probability of the δ mutant.

The expression for the substitution rate given in eqn. 2 connects with at least three previous formalizations. First, it is formally similar to the standard expression for the substitution rate in the field of molecular evolution (Kimura, 1971, eqn. 4.2), which does not consider demographic structures, but stipulates that the substitution rate of a particular mutant depends on the number $\bar{N}(z)\mu(z)u(\delta,z)$ of such mutants produced over one iteration of the life cycle and the fraction $\bar{\pi}_\mu(\delta,z)$ that eventually reaches fixation. Second, for neutral genes, eqn. 2 reduces to the substitution rate for age-structured populations (Charlesworth, 1980; Pollak, 1982). Third and foremost, eqn. 2 reduces to the jump rate derived by Champagnat & Lambert (2007, eqn. 7), who considered the evolution of a continuous phenotype under selection in an iteroparous panmictic population without class structure subject to a birth and death demographic process (e.g. Karlin & Taylor, 1975; Grimmett & Stirzaker, 2001). This result is proved in Appendix A, eqns A-17–A-23, and it points to a first connection with the adaptive dynamics approach.

Substitution rate for small phenotypic deviations

Besides some special very cases, like the Moran process (Ewens, 2004), the fixation probability $\bar{\pi}_\mu(\delta,z)$ cannot be calculated exactly, but it is conveniently approximated by assuming mutants with only small phenotypic deviations relative to the phenotype of residents (Rousset, 2004). This assumption is used by way of a Taylor expansion of the average fixation probability around $\delta = 0$, which gives

$$\bar{\pi}_\mu(\delta,z) = \bar{\pi}^\circ_\mu(z) + \delta\dot{\bar{\pi}}(z) + O(\delta^2), \tag{3}$$

where $\bar{\pi}^\circ_\mu(z) \equiv \bar{\pi}_\mu(0,z)$ is the fixation probability of a neutral mutant, calculated from an evolutionary process where there is no selection ($\delta = 0$), and $\dot{\bar{\pi}}_\mu(z) \equiv \mathrm{d}\bar{\pi}_\mu(\delta,z)/\mathrm{d}\delta$ is the first-order derivative of $\bar{\pi}_\mu(\delta,z)$ with respect to δ evaluated at $\delta = 0$. This Taylor expansion of the fixation probability will be used in the substitution rate (eqn. 2) in order to derive an expression for the probability density function $p(z,t)$.

Slow dynamics: long-term phenotypic evolution

Diffusion equation

Because the instantaneous change in phenotypic value is given by $k(\delta, z)$ (eqn. 1), the $z(t)$ process is entirely determined by $k(\delta, z)$ and the time dynamic of $p(z, t)$ follows a so-called master equation with jump rate given by the substitution rate (Gardiner, 2009, and see Appendix B, eqns B-1 and B-2). But owing to the assumption of small phenotypic deviations (eqn. 3), the function $p(z, t)$ that phenotype z obtains at t satisfies the simpler equation

$$\frac{\partial p(z,t)}{\partial t} = -\frac{\partial}{\partial z}[a(z)p(z,t)] + \frac{1}{2}\frac{\partial^2}{\partial z^2}[b(z)p(z,t)], \tag{4}$$

where

$$\begin{aligned} a(z) &= \bar{N}(z)\mu(z)\sigma^2(z)\dot{\bar{\pi}}_\mu(z) \\ b(z) &= \bar{N}(z)\mu(z)\sigma^2(z)\bar{\pi}^\circ_\mu(z), \end{aligned} \tag{5}$$

are, respectively, the infinitesimal mean and variance of the change in phenotype, which depend on the variance $\sigma^2(z)$ of the mutant step size distribution: $\sigma^2(z) = \int\delta^2 u(\delta, z)\mathrm{d}\delta$ (see Appendix B, eqns. B-4–B-10, for a derivation). The mean change in phenotype, $a(z)$, determines the general direction of evolution of the phenotype, whose sign is given by $\dot{\bar{\pi}}_\mu(z)$. Fluctuations around the mean path due to mutations and genetic drift are described by $b(z)$.

Equation 4 is a diffusion equation for the change in phenotype (e.g. Kimura, 1964; Karlin & Taylor, 1981; Gillespie, 1991; Ewens, 2004; Gardiner, 2009), where higher-than-second-order moments of phenotypic deviations have been neglected as large δ deviations are assumed unlikely to occur. This diffusion equation is also the solution of the stochastic differential equation for the random variable $z(t)$ (Karlin & Taylor, 1981, p. 376), which has been called the canonical diffusion of adaptive dynamics when it was derived as a limiting result in a haploid iteroparous panmictic population without class structure (Champagnat & Lambert, 2007).

Equation 4 thus points to a second link with the adaptive dynamics approach, and it can be thought of as an extension of the diffusion equation to diploid and/or haplo-diploid systems with class structure and broader life-history modes, thus including spatially structured populations as a special case. The derivation of eqn. 4 is heuristic because the separation of time scales was imposed by way of eqn. 1. The diffusion was not derived as a limiting process when the mutation rate vanishes in a demo-genetic model with multiple alleles as in Champagnat & Lambert (2007). By imposing the separation of time-scale assumption, however, one markedly gains in generality and the connection to previous work is more direct, a point that is illustrated below.

Stationary distribution

The diffusion equation for the phenotype (eqn. 4) describes the evolution of z under the joint action of mutation, natural selection and random genetic drift. But because mutations are constantly introduced into the population, there is no absorbing state with only one allele fixed in the population forever. The stochastic process may then eventually settle into a stationary distribution function $p(z) \equiv \lim_{t\to\infty} p(z, t)$, with the dynamics of z being subject to mutation, selection and

drift but in a balance. This stationary distribution is given by

$$p(z) = \frac{K}{\mu(z)\sigma^2(z)\bar{N}(z)\bar{\pi}^{\circ}_{\mu}(z)} \exp\left[2\int_l^z \frac{\dot{\bar{\pi}}_{\mu}(y)}{\bar{\pi}^{\circ}_{\mu}(y)}\mathrm{d}y\right], \quad (6)$$

where l is the left boundary of the state space and K denotes the normalizing constant, which will be used as such throughout the paper (see Appendix B, eqns B-11 and B-12).

Stationary distribution in terms of phenotypic selection gradient

Adaptive landscape

The stationary distribution $p(z)$ (eqn. 6) remains a complicated expression if $\sigma^2(z)$ depends explicitly on the evolving trait and if different classes of individuals are subject to different mutation rates. But it is reasonable to assume that the mutation machinery, $\sigma^2(z)$ and $\mu(z)$, is independent of the particular value z takes. Further, the mutation rate may be the same in each class, that is, $\mu_{ij}(\delta, z) = \mu(\delta, z)$, which is likely to be the case for sex, stage or geographically structured populations. When this is the case, the neutral fixation probability is simply given by the inverse of the average number of gene copies in the population, $\bar{\pi}^{\circ}_{\mu}(z) = 1/\bar{N}(z)$ (Appendix A, eqns A-12 and A-13), and the stationary distribution can be written as

$$p(z) = K\exp\left[2\int_l^z \bar{N}(y)S(y)\mathrm{d}y\right]. \quad (7)$$

Here, $S(y)$ is the perturbation of the average fixation probability of a δ mutant when the mutation rate is the same across classes, and which does no longer depend on mutation features [$S(y) \equiv \mathrm{d}\bar{\pi}(\delta, y)/\mathrm{d}\delta|_{\delta=0}$, see Table 3 for the expression of $\bar{\pi}(\delta, z)$]. The function $S(y)$ gives the slope of the fixation probability due to the introduction of a δ mutant into the population and can be interpreted of as an invasion condition for the δ mutant (Demetrius & Ziehe, 2007).

The extrema of $p(z)$, which are the most and least likely phenotypic outcomes of evolution, are determined by the extrema of the integral $\int_l^z \bar{N}(y)S(y)\mathrm{d}y$, which can be thought of as an adaptive landscape (Wright, 1931; Barton *et al.*, 2007). The distribution $p(z)$ shows that the exploration of the phenotypic space at an evolutionary steady state varies exponentially with this adaptive landscape. The internal extrema of $p(z)$ satisfy $\mathrm{d}p(z)/\mathrm{d}z = 0$, and by the fundamental theorem of calculus, they satisfy $p(z)\bar{N}(z)S(z) = 0$. As $p(z)$ is positive for all phenotypic values and the Markov chain describing the resident's demography underlying the mutant's gradient $S(z)$ is conditioned on population nonextinction ($\bar{N}(z) > 0$), the internal singular points are the solutions of

$$S(z) = 0. \quad (8)$$

If a given extremum of $p(z)$ is a local or global maximum, then a population of residents that is in small neighbourhood from this singular point is likely to be replaced by a population of mutants that expresses a phenotypic value closer to the singular point. Conversely, if a population is located at a minimum of $p(z)$, then a mutant that expresses a phenotypic value away from that of the resident is likely to invade. The local curvature of $p(z)$ at an extremum thus describes whether a population will converge or diverge from the singular point and should be indicative of the stability by convergence of that point. One can then say that an internal singular point z is stable by convergence (Eshel, 1983; Taylor, 1989; Christiansen, 1991; Rousset, 2004) if $S(z) = 0$ and $\mathrm{d}^2p(z)/\mathrm{d}z^2 < 0$. From eqn. 7 and as $p(z) > 0$ and $N(z) > 0$, this inequality can be written as

$$\frac{\mathrm{d}S(z)}{\mathrm{d}z} < 0. \quad (9)$$

Intuitively, the singular points of $S(z)$ and the strategies stable by convergence should correspond to those points obtained by previous methods (e.g. evolutionary games theory, kin selection theory, adaptive dynamics) when the gene pool is made large. But what exactly is the functional form of $S(z)$?

Localized selection gradient

The interesting feature about $S(z)$ is that it is essentially nothing else but a standard phenotypic selection gradient. In particular, for a haploid semelparous panmictic population of constant size without any further division into classes and family interactions, one has

$$S(z) = \left.\frac{\partial w(z_\bullet, z_0)}{\partial z_\bullet}\right|_{z_\bullet = z_0 = z}, \quad (10)$$

where $w(z_\bullet, z_0)$ is the fitness of a focal individual expressing phenotype $z_\bullet$ when the remaining individuals in the population express phenotype z_0, and the derivative is evaluated at the phenotypic values of the resident (Rousset & Billiard, 2000; Rousset, 2004).

The function $w(z_\bullet, z_0)$ is an individual fitness function as usually used in evolutionary game theory, kin selection (or inclusive fitness) theory or adaptive dynamics (e.g. Maynard Smith, 1982; Eshel, 1983; Taylor, 1989; Parker & Maynard Smith, 1990; Bulmer, 1994; Dieckmann & Law, 1996; Taylor & Frank, 1996; Frank, 1998; Geritz *et al.*, 1998; Vincent & Brown, 2005; Waxman & Gavrilets, 2005; Dercole & Rinaldi, 2008; Leimar, 2009), although fitness means here the expected total number of individuals descending from a focal individual after one full iteration of the life cycle of the organism [thus including itself through survival and its offspring in order to have a full count of gene frequencies over one

life-cycle iteration such that $w(z,z) = 1$]. This shows that the extrema of $p(z)$ correspond to the singular points obtained by evolutionary game theory, kin selection theory or adaptive dynamics. Further, the condition of stability by convergence of these singular points (eqn. 8 and ineq. 9) corresponds to expressions obtained previously (Eshel, 1983; Taylor, 1989; Geritz *et al.*, 1998; Rousset, 2004).

More generally, there may be different classes of individuals in the population, like males and females, or different individuals subject to different numbers of competitors, or living in different regions of the habitat like in families or patches. Then, for the class-structured demographic model introduced above, whose crucial assumption is that the transition probability $\Pr(\mathbf{s}' \mid \mathbf{s},z)$ between demographic states follows a regular homogeneous Markov chain on a countable state space, the function $S(z)$ can be expressed as a 'localized' inclusive fitness effect (Rousset & Billiard, 2000; Leturque & Rousset, 2002; Rousset & Ronce, 2004; Rousset, 2006). This is a demographically explicit version for finite populations of Hamilton's (1964) inclusive fitness effect. The inclusive fitness effect is a relatedness weighted effect on the expected number of each class of offspring over all demographic states of all mutant carriers, which results from the expression of all mutant alleles in the population over all demographic states (see Rousset, 2004 for details and Appendix A, eqns A-12–A-14). Here again, the condition of stability by convergence (eqn. 8 and in eqn. 9) corresponds to the condition defined previously from inclusive fitness effects on fixation probabilities (e.g. Rousset & Ronce, 2004, p. 129).

'Localized' inclusive fitness effect refers to the fact that relatedness is not measured relative to gene identity between pairs of individuals taken at the global scale, but measured relative to the identity between pairs of genes taken at the local scale, the deme of a focal actor, when the population is geographically structured (Lehmann & Rousset, 2010, section 5.b, see also Appendix A right after eqn. A-14). But otherwise, $S(z)$ is similar in baseline structure to the selection gradients obtained by the application of the direct fitness method of kin selection theory (Taylor & Frank, 1996; Frank, 1998; Rousset, 2004; Taylor *et al.*, 2007b). Hence, $S(z)$ and its ramifications connect smoothly to standard phenotypic selection gradients routinely used by evolutionary biologists (Wenseleers *et al.*, 2010), and the stationary distribution $p(z)$ can be calculated from them provided the fitness function(s) and the relatedness coefficients are evaluated for finite population size (e.g. Taylor *et al.*, 2007b).

When the mutation distribution, $\mu_{ij}(\delta,z)$, is not the same across classes, the functional form of $\dot{\bar{\pi}}_\mu(z)$ has not yet been given a clear outline in the literature, and the neutral fixation probability $\bar{\pi}^\circ_\mu(z)$ will no longer be equal to the inverse of the average size of the gene pool. The previous discussion, nevertheless, suggests that it may still be expressed in terms of inclusive fitness effect on fixation probabilities, but one where the average over classes may depend on the class-specific mutation rate, and this may affect the outcome of evolution. More work is needed in order to establish what are the simplest way(s) to evaluate $\dot{\bar{\pi}}_\mu(z)$ in the presence of both class-structured populations and different mutation rates in different classes. This case may be relevant for understanding frequency-dependent selection in age-structured populations where the germ line is separated from the soma. Here, only newborns usually introduce mutations into the gene pool that may ultimately fix in the population as mutations in the soma cannot out-propagate alternatives.

Applications

Multipeaked fitness landscape and coordination

Pairwise interactions

An application of the results is now provided by evaluating the stationary distribution $p(z)$ under a situation of pairwise interactions between individuals in a population of constant size without class structure. The trait z is assumed to vary between zero and one and to describe investment into cooperation. The fecundity of a player with trait value z_1 when meeting a player expressing trait value z_2 is assumed to be given by

$$f(z_1,z_2) = 1 + z_1[z_2R + (1-z_2)S] + (1-z_1)[z_2T + (1-z_2)P], \tag{11}$$

where the interaction setting can be understood by calling R the reward for mutual cooperation, S the sucker's payoff, T the temptation to defect and P the punishment for mutual defection (Hofbauer & Sigmund, 1998).

Depending on the parameter values, three different categories of games can described by the fecundity function $f(z_1,z_2)$: (i) a game with a dominant strategy, like the standard prisoner's dilemma game, which can be obtained by setting $R = B - C$, $S = -C$, $T = B$ and $P = 0$; (ii) a game with an internal equilibrium that is stable in a deterministic model, like the Hawk–Dove game, which can be obtained by setting $R = (B - C)/2$, $S = 0$, $T = B$ and $P = B/2$; and (iii) a game with an internal equilibrium that is unstable in a deterministic model, like a coordination game, which can be obtained by setting $S = T = 0$ and $R > P > 0$. This defines a double-peaked payoff landscape as two individuals playing this game have a higher payoff by coordinating (on whatever action) than those playing opposite actions.

The stationary distribution $p(z)$ will be evaluated for the following life cycle: (i) each of the N haploid adults in the population interacts with another player sampled at random from the population and then produces a large number of juveniles according to the payoff it receives from the interaction and (ii) each adult dies and juveniles compete for vacant breeding spots. Exactly, N individuals reach adulthood and form the next generation.

Selection gradient

Because by construction the gradients $\dot{\pi}_\mu(z)$ or $S(z)$ depend only on first-order effects of selection (eqn. 7), effects of order δ^2 on payoffs are not needed. To the first order in δ, the average fecundity of a focal individual with phenotype $z_\bullet$ (average of eqn. 11 over $N-1$ possible partners) can be simply written as $f(z_\bullet,z_0)$, where z_0 is the average phenotype of an individual randomly sampled from the population and excluding the focal individual. Hence, to the first order in δ, the function of an average can be taken in place of the average of a function (Rousset, 2004, p. 95). With this, the direct fitness function of a focal individual can be written as

$$w(z_\bullet,z_0)=\frac{f(z_\bullet,z_0)}{f(z_0^{\mathrm{R}},z_0^{\mathrm{R}})}, \tag{12}$$

which is the ratio of the average fecundity $f(z_\bullet,z_0)$ of a focal individual to the average fecundity $f(z_0^{\mathrm{R}},z_0^{\mathrm{R}})$ in the population, where $z_0^{\mathrm{R}}=z_\bullet/N+(N-1)z_0/N$ is the average phenotype in the population including the focal individual.

Substituting eqn. 12 into eqn. 10, one obtains

$$S(z)=\frac{(N-2)(P+R-S-T)z-(N-2)P+(N-1)S-T}{Nf(z,z)}. \tag{13}$$

Depending on the parameter values, $S(z)$ may be positive or negative for all z so that $z^*=1$ and $z^*=0$ are candidate singular points, as well as the internal point

$$z^*=\frac{(N-2)P-(N-1)S+T}{(N-2)(P+R-S-T)}, \tag{14}$$

which was derived previously by other methods for discrete strategies (Schaffer, 1988; Wild & Taylor, 2004).

As eqn. 14 may be either a stable or unstable internal equilibrium in a deterministic model, which corresponds to, respectively, a maxima or a minima of the adaptive landscape, the stationary distribution may be multipeaked. When all parameter values are nonzero, this stationary distribution takes a somewhat complicated expression, which is presented in Appendix C (eqn. C-2). The two cases of Hawk–Dove and coordination game will now be considered separately as these are the most relevant here.

Hawk–Dove game

In order to obtain the Hawk–Dove game (Maynard Smith, 1982), the parameterization $R=(B-C)/2$, $S=0$, $T=B$ and $P=B/2$ is used. With this, the internal singular point becomes $z^*=BN/\{C(N-2)\}$ and is convergence stable. The stationary distribution is

$$p(z)=K\left(1-\frac{Cz^2}{B+2}\right)^{N-2}\exp\left(-\frac{2BN\tan^{-1}\left(\frac{Cz}{\sqrt{-(B+2)C}}\right)}{\sqrt{-(B+2)C}}\right), \tag{15}$$

which is plotted in Fig. 1 and illustrates that the variance of the distribution is reduced as population size increases. When population size becomes very large, stochastic effects due to mutation and genetic drift become very small, and all the weight of the distribution tends to be put on the singular point $z^*=BN/\{C(N-2)\}\simeq B/C$. The distribution thus becomes strongly peaked at this point, and as $N\to\infty$, this becomes the only phenotypic value observed at a steady state, thereby recovering the results found from deterministic models with otherwise exactly similar assumptions (Rousset, 2004, p. 89).

Coordination game

In order to obtain a coordination game, the parameterization $S=T=0$ and $R>P>0$ is used. For this case, there are two singular points, $z^*=0$ and $z^*=1$, which are convergence stable, and an internal equilibrium is given by $z^*=P/(P+R)$, which is not convergence stable. In a deterministic model, the internal equilibrium is unstable and evolution will lead to equilibrium $z^*=0$ if $z_0<P/(P+R)$, and $z^*=1$ if $z_0>P/(P+R)$, where z_0 is the initial condition of the system. Hence, the outcome of evolution is path dependent as it depends on initial conditions.

In a finite population, the stationary distribution for the coordination game is obtained from eqn. 2 as

$$p(z)=K\left(\frac{1+z^2R+(1-z)^2P}{1+P}\right)^{N-2}, \tag{16}$$

which is convex with maxima at $z=0$ and $z=1$ (Fig. 2). When $z=0$, we have $p(z)=K$, whereas when $z=1$, we have $p(z)=K[(1+R)/(1+P)]^{N-2}$. If $R>P$, this function grows very large as N becomes large. Because the

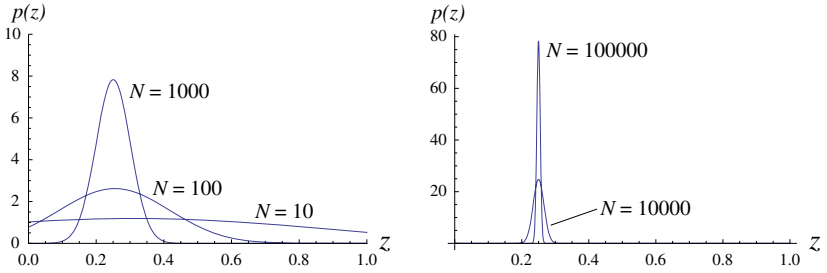

Fig. 1 Stationary distribution $p(z)$ for the Hawk–Dove game (eqn. 15). The different curves in the two panels correspond to different population sizes (N), whereas the other parameters values are hold constant and given by $B=0.1$ and $C=0.4$, which gives $z^*=0.25$ as the singular point if the population size is made very large.

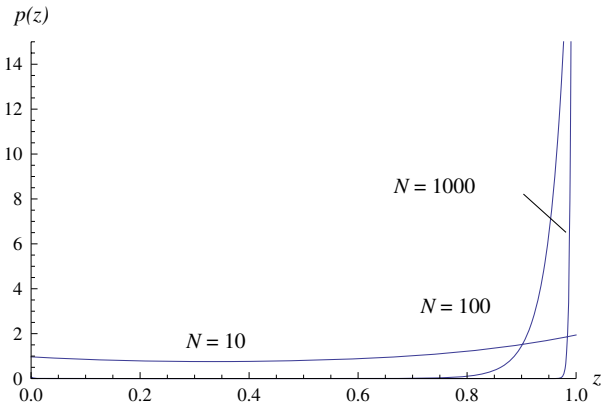

Fig. 2 Stationary distribution $p(z)$ for the coordination game (eqn. 16). The different curves correspond to different population sizes (N), whereas the other parameters values are hold constant and given by $R = 0.2$ and $P = 0.1$. As population size grows large, more probability mass accumulates on $z = 1$, and this becomes the only equilibrium point when $N \to \infty$. This is the stochastically stable state of the system.

probability distribution is normalized, all probability mass tends to accumulates around $z = 1$ as $N \to \infty$, which determines the stochastically stable state of the system (Foster & Young, 1990).

That the system is very likely to reside on phenotypic value $z = 1$ when population size grows large is an instance of an equilibrium selection among alternative equilibria (Foster & Young, 1990; Binmore *et al.*, 1995). This illustrates that when the stochastic shocks in the system are made very small, here by blowing up the size of the gene pool, the analysis of the stationary distribution allows to remove the path dependence of the evolution of z that occurs in the deterministic process.

The equilibrium $z = 1$ can be called payoff dominant as coordinating on this strategy leads to a higher payoff than coordinating on equilibrium $z = 0$ ($R > P$). More generally, for the coordination game $S = 0$, $R > T > 0$, $R > P > 0$ and $P + T > R$, the equilibrium point $z = 1$ is still payoff dominant but the equilibrium $z = 0$ is called risk dominant (Kandori *et al.*, 1993; Binmore *et al.*, 1995). This stems from the fact that if players are unsure of the strategy of their partner and assign a probability 1/2 to each of their partner's action, the expected payoff of playing the risk dominant strategy exceeds that of playing the payoff dominant strategy (Kandori *et al.*, 1993; Binmore *et al.*, 1995).

In a deterministic model, the basin of attraction of the risk dominant strategy is larger than that of the payoff dominant strategy. One then expects that in a model with stochastic shocks, the population spends more time fixed on the risk dominant strategy (Kandori *et al.*, 1993; Binmore *et al.*, 1995). Although previous work has shown that the payoff dominant strategy may be selected for under certain conditions in finite populations with discrete strategies (Binmore *et al.*, 1995), a numerical analysis of the stationary distribution for the game with continuous strategies (eqns 2–5) suggests that the limiting stationary distribution tends to be concentrated on the risk dominant strategy. A detailed analysis of this problem is beyond the scope of this paper.

Multiploidy and sex allocation

Assumptions and fitness functions

The model in the last section assumed the presence of only a single class of individuals that were haploid. We now turn to an application with males and females. The evolving trait z is assumed to determine the primary sex ratio produced by a female and can be thought of as the fraction of resources allocated to producing females (or to their survival to the reproductive stage), whereas $1 - z$ represents the fraction of resources allocated to males (Taylor, 1988; Taylor & Frank, 1996; Frank, 1998). The population is assumed to be of constant size, and for simplicity, an equal number N of males and females are assumed to reach the stage where they can reproduce.

The sex ratio is further assumed to be entirely under maternal control and the evolution of z will be analysed for a diploid and a haplo-diploid genetic system, where males are haploid. The life cycle is as follows: (i) Each of the N mated females in the population produces a large number of male and female juveniles, where the ratio of the number of females to males that reach the stage of density-dependent competition is determined by the evolving trait. Individuals of the parental generation die. (ii) Density-dependent competition occurs and exactly N males and females are sampled to form the next generation of adults. (iii) Each female mates a large number of times randomly with the available males.

As the trait is under maternal control, any male, regardless of its genotype, will have the same fitness contribution to the next generation as any other male. In order to evaluate selection on a mutant allele, one then needs to consider only the fitness of females because females with different genotypes will make different contributions to the gene pool in the next generation (Taylor & Frank, 1996; Frank, 1998). We can then focus on a random female and write expressions for her fitness contribution through females, w_{ff}, and males, w_{mf}, where w_{ij} is the expected number of class-i individuals descending from a single class j individual (Table 1). Under the life cycle described above, fitness through sex i of a focal female depends on the number of sex i offspring produced by that female relative to the average number of sex i offspring produced in the population. This gives $w_{\mathrm{ff}}(z_\bullet, z_{\mathrm{f}}) = z_\bullet / z_{\mathrm{f}}^{\mathrm{R}}$ and $w_{\mathrm{fm}}(z_\bullet, z_{\mathrm{f}}) = (1 - z_\bullet)/(1 - z_{\mathrm{f}}^{\mathrm{R}})$, where $z_\bullet$ is the average phenotype of the focal female and $z_{\mathrm{f}}^{\mathrm{R}} = z_\bullet/N + (N - 1)z_{\mathrm{f}}/N$ is the average phenotype of females in the population including the focal female, whereas z_{f} is the average phenotype excluding the focal female.

Selection gradient and stationary distribution

For the sex-allocation model, the localized inclusive fitness effect can be written as

$$S(z) = \left(\alpha_f t_{ff} \frac{\partial w_{ff}(z_\bullet, z_f)}{\partial z_\bullet} + \alpha_m t_{mf} \frac{\partial w_{mf}(z_\bullet, z_f)}{\partial z_\bullet} \right) L, \quad (17)$$

where α_i is the reproductive value of class i and t_{ij} is the probability that a gene in a class i individual descends from a class j individual (Appendix C, eqns C-6 and C-7). For a diploid system, we have $t_{ij} = 1/2$ for all i and j, which implies $\alpha_m = 1/2$ and $\alpha_f = 1/2$, whereas for a haplo-diploid system where males are haploid, we have $t_{ff} = 1/2$, $t_{fm} = 1/2$, $t_{mf} = 1$ and $t_{mm} = 0$, which implies $\alpha_m = 1/3$ and $\alpha_f = 2/3$ (Taylor, 1988; Taylor & Frank, 1996; Frank, 1998). In both cases, $\alpha_f t_{ff} = \alpha_m t_{mf}$.

The selection gradient also depends on the factor of proportionality L, which accounts for evolution occurring in a finite population and is specific to the mating system (see Rousset, 2004 for details). It quantifies the extent to which two genes taken in a focal female are more likely to be identical than two genes taken at random from two different females (Appendix C, eqns C-7 and C-8). The proportionality factor L was equal to '1' under the haploid assumptions leading to eqn. 10, and it is here equal to 1/2 for diploids (as in deterministic models, Frank, 1998) and $(9N - 4)/(18N - 6)$ for haplo-diploids, which reduces to 1/2 when population size becomes large (Appendix C, eqn. C-10).

The selection gradient can be evaluated explicitly as

$$S(z) = \frac{a_g}{2\bar{N}} \frac{1 - 2z}{z(1 - z)}, \quad (18)$$

where the size of the gene pool, $\bar{N}$, is equal to $4N$ for diploids and $3N$ for haplo-diploids, and

$$a_g = \begin{cases} N - 1 & \text{diploids} \\ \frac{(N-1)(9N-4)}{(9N-3)} & \text{haplo-diploids.} \end{cases} \quad (19)$$

For both a diploid and a haplo-diploid system, a_g grows with population size and it becomes approximately equal to N for large population size. It now remains to integrate the expression for $S(z)$ in order to uncover the stationary distribution (eqn. 6). This yields

$$p(z) = \frac{z^{a_g}(1 - z)^{a_g}}{\int_0^1 y^{a_g}(1 - y)^{a_g} \mathrm{d}y}, \quad (20)$$

which is a Beta distribution with parameter $1+a_g$ and it satisfies eqn. 4 at equilibrium. Hence, the mean sex ratio is 1/2, which is the value of z predicted by previous applications of evolutionary game theory for infinite population size but with otherwise similar assumptions (Taylor & Frank, 1996; Frank, 1998), and is independent of the features of the genetic system. By contrast, the variance of the Beta sex-ratio distribution is given by $1/(12 + 8a_g)$. This results in a lower variance in the sex-ratio distribution for diploids in small populations, and the variance vanishes for the diploid and haplo-diploid system as population size becomes very large.

Discussion

The stationary distribution of a one-locus continuous phenotype under a mutation–selection–drift balance in a class-structured population has been derived under the assumptions of weak selection intensities and a separation of time scales between short- and long-term evolution. If mutation rates are the same across classes and the mutation machinery is independent of the evolving phenotype, the stationary distribution can be entirely characterized in terms of the average size of the gene pool and Hamilton's (1964) inclusive fitness effect for demographically structured populations of finite size (Rousset, 2004; Rousset & Ronce, 2004; Taylor *et al.*, 2007b).

The stationary distribution shows that the exploration of the phenotypic space at steady state varies exponentially with the inclusive fitness effect cumulated over state space, which determines an adaptive landscape (eqn. 7, Figs 1 and 2). For a multipeaked fitness landscape, the various peaks of the landscape are those phenotypes that are candidate evolutionary stable strategies. The curvature of the stationary distribution at a candidate evolutionary stable strategy provides a natural measure of its stability by convergence (eqn. 8 and ineq. 9), which is consistent with those obtained in previous analyses (Eshel, 1983; Taylor, 1989; Geritz *et al.*, 1998; Rousset, 2004).

The results of this paper support Gillespie's (1991) enthusiasm that the separation of time between long- and short-term evolution makes tractable an apparently intractable model, which captures realistic aspects of natural populations, such as finite size, frequency-dependent selection, class structure, varying demography and mutation rates. Further, the stationary distribution of the phenotype can be expressed in terms of standard quantities; namely, phenotypic selection gradient obtained as derivatives of individual fitness functions weighted by relatedness coefficients, which are commonly used in evolutionary biology (Wenseleers *et al.*, 2010). These standard approaches thus allows one to obtain an approximate, but calculable estimate of the phenotypic distribution, the drift load or the variance in phenotype maintained in a population at a mutation–selection–drift balance, to which relaxing assumptions can be compared. In addition to identifying candidate evolutionary stable strategies, the stationary distribution also allows one to select among such alternatives and to identify stochastically stable strategies (Foster & Young, 1990, Binmore *et al.*, 1995) by letting the average size of the gene pool grow large.

The results of this paper also point to connections between the adaptive dynamics framework (Dieckmann & Law, 1996; Geritz *et al.*, 1998; Dercole & Rinaldi, 2008) and the direct fitness method of kin selection theory

(Taylor & Frank, 1996; Rousset, 2004; Wenseleers *et al.*, 2010). The model developed here was inspired by these two approaches: in particular, the emphasis of adaptive dynamics on evaluating the time dynamics of evolving phenotypes and the emphasis of kin selection theory for finite populations on stochastic elements affecting the fate of mutant alleles. Here, as has already been suggested for branching points determination (Ajar, 2003), the direct fitness method of kin selection theory can be envisioned as adaptive dynamics at the intraspecific level with mutant–mutant interactions. Such interactions are difficult to avoid in small populations, as two interacting individuals are likely to descend from the same recent common ancestor.

One main limitation of the model from a theoretical point of view is the heuristic assumption of a separation of time scales between short- and long-term evolution (eqn. 1). Conditions on the mutation rate guaranteeing convergence to the separation of time scales would be interesting to document and could be addressed by more mathematically inclined research. Evaluating expressions for the average fixation probability of a mutant in the presence of varying mutation rates across classes should also be interesting, as this is relevant for the evolution in age-structured populations. From a more biological perspective, one main limitation of the model is its one-dimensional phenotypic nature. Addressing the co-evolution of multiple phenotypic traits and/or multispecies interactions opens avenues for future explorations.

Acknowledgments

I thank Émile Ajar and a reviewer for helpful comments on this paper. I am grateful to another reviewer for suggesting that the curvature of the stationary distribution may be indicative of stability.

References

Ajar, E. 2003. Analysis of disruptive selection in subdivided populations. *BMC Evol. Biol.* **3**: 22.

Barton, N.H., Briggs, D., Eisen, J.A. Goldstein, D.B. & Patel, N.H., 2007. *Evolution*. Cold Spring Harbor Laboratory Press, New York.

Binmore, K., Samuelson, L. & Vaughan, R., 1995. Musical chairs: modeling noisy evolution. *Games Econ. Behav.* **11**: 1–35.

Bulmer, M., 1994. *Theoretical Evolutionary Ecology*. Sinauer Associates, MA.

Bürger, R. 2000. *The Mathematical Theory of Selection, Recombination, and Mutation*. John Wiley and Sons, New York.

Bürger, R. & Lande, R. 1994. On the distribution of the mean and variance of a quantitative trait under mutation-selection-drift balance. *Genetics* **138**: 901–912.

Bürger, R., Wagner, G.P. & Stettinger, F., 1989. How much heritable variation can be maintained in finite populations by mutation-selection balance? *Evolution* **43**: 1748–1766.

Caswell, H. 2000. *Matrix Population Models*. Sinauer Associates, MA.

Champagnat, N. & Lambert, A. 2007. Evolution of discrete populations and the canonical diffusion of adaptive dynamics. *Ann. Appl. Probab.* **17**: 102–155.

Champagnat, N., Ferrière, R. & Méléard, S., 2006. Unifying evolutionary dynamics: from individual stochastic processes to macroscopic models. *Theor. Popul. Biol.* **69**: 297–321.

Charlesworth, B. 1980. *Evolution in Age-Structured Populations*. Cambridge University Press, Cambridge.

Christiansen, F.B. 1991. On conditions for evolutionary stability for a continuously varying character. *Am. Nat.* **138**: 37–50.

Demetrius, L. & Ziehe, M. 2007. Darwinian fitness. *Theor. Popul. Biol.* **72**: 323–345.

Dercole, F. & Rinaldi, S. 2008. *Analysis of Evolutionary Processes: The Adaptive Dynamics Approach and Its Applications*. Princeton University Press, Princeton, NJ.

Dieckmann, U. & Law, R. 1996. The dynamical theory of coevolution: A derivation from stochastic ecological processes from stochastic ecological processes. *J. Math. Biol.* **34**: 579–612.

Eshel, I. 1983. Evolutionary and continuous stability. *J. Theor. Biol.* **103**: 99–111.

Eshel, I. 1996. On the changing concept of evolutionary population stability as a reflection of a changing point of view in the quantitative theory of evolution. *J. Math. Biol.* **34**: 485–510.

Eshel, I., Feldman, M. & Bergman, A. 1998. Long-term evolution, short-term evolution, and population genetic theory. *J. Theor. Biol.* **191**: 391–396.

Ewens, W.J. 2004. *Mathematical Population Genetics*. Springer-Verlag, New York.

Ferrière, R., Bronstein, J.L., Rinaldi, S., Law, R. & Gauduchon, M. 2002. Cheating and the evolutionary stability of mutualisms. *Proc. Biol. Sci.* **269**: 773–780.

Foster, D. & Young, H.P. 1990. Stochastic evolutionary game dynamics. *Theor. Popul. Biol.* **38**: 219–232.

Frank, S.A. 1998. *Foundations of Social Evolution*. Princeton University Press, Princeton, NJ.

Gardiner, C.W. 2009. *Stochastic Methods*, 4th edn. Springer-Verlag, Berlin.

Geritz, S.A.H., Kisdi, E., Meszéna, G. & Metz, J.A.J. 1998. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. *Evol. Ecol.* **12**: 35–57.

Gillespie, J.H. 1983. A simple stochastic gene substitution model. *Theor. Popul. Biol.* **23**: 202–215.

Gillespie, J.H. 1991. *The Causes of Molecular Evolution*. Oxford University Press, Oxford.

Gillespie, J.H. 2004. *Population Genetics: A Concise Guide*. Johns Hopkins, Baltimore & London.

Grimmett, G. & Stirzaker, D. 2001. *Probability and Random Processes*. Oxford University Press, Oxford.

Hamilton, W.D. 1964. The genetical evolution of social behaviour, 1. *J. Theor. Biol.* **7**: 1–16.

Hammerstein, P. 1996. Darwinian adaptation, population genetics and the streetcar theory of evolution. *J. Math. Biol.* **34**: 511–532.

Hartl, D. & Clark, A.G. 2007. *Principles of Population Genetics*, 4th edn. Sinauer, MA.

Hofbauer, J. & Sigmund, K. 1998. *Evolutionary Games and Population Dynamics*. Cambridge University Press, Cambridge.

Kandori, M., Mailath, G. & Rob, R. 1993. Learning, mutation, and long run equilibria in games. *Econometrica* **61**: 29–56.

Karlin, S. 1968. Equilibrium behavior of population genetic models with non-random mating: part ii: pedigrees, homozygosity and stochastic models. *J. Appl. Probab.* **5**: 487–566.

Karlin, S. & Taylor, H.M. 1975. *A First Course in Stochastic Processes*. Academic Press, San Diego.
Karlin, S. & Taylor, H.M. 1981. *A Second Course in Stochastic Processes*. Academic Press, San Diego.
Kimura, M. 1964. Diffusion models in population genetics. *J. Appl. Probab.* **1**: 177–232.
Kimura, M. 1965. A stochastic model concerning the maintenance of genetic variability in quantitative character. *Proc. Natl. Acad. Sci. U.S.A.* **54**: 731–736.
Kimura, M. 1971. Theoretical foundation of population genetics at the molecular level. *Theor. Popul. Biol.* **2**: 174–208.
Kimura, M. & Crow, J.F. 1964. The number of alleles that can be maintained in a finite population. *Genetics* **49**: 725–738.
Lande, R. 1976. Natural selection and random genetic drift in phenotypic evolution. *Evolution* **30**: 314–334.
Lehmann, L. and Rousset, F. 2010. How life-history and demography promote or inhibit the evolution of helping behaviors. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* **365**: 2599–2617.
Leimar, O. 2009. Multidimensional convergence stability. *Evol. Ecol. Res.* **11**: 191–208.
Leturque, H. & Rousset, F. 2002. Dispersal, kin competition, and the ideal free distribution in a spatially heterogeneous population. *Theor. Popul. Biol.* **62**: 169–180.
Lion, S. & Gandon, S. 2009. Habitat saturation and the spatial evolutionary ecology of altruism. *J. Evol. Biol.* **22**: 1487–1502.
Malécot, G. 1975. Heterozygosity and relationship in regularly subdivided populations. *Theor. Popul. Biol.* **8**: 212–241.
Maynard Smith, J. 1982. *Evolution and the Theory of Games*. Cambridge University Press, Cambridge.
Meyn, S. and Tweedie, R.L. 2009. *Markov Chains and Stochastic Stability*, 2nd edn. Cambridge University Press, Cambridge.
Ohtsuki, H. & Iwasa, Y. 2004. How should we define goodness? reputation dynamics in indirect reciprocity. *J. Theor. Biol.* **231**: 107–120.
Orr, H.A. 1998. The population genetics of adaptation: the distribution of factors fixed during adaptive. *Evolution* **52**: 935–949.
Parker, G.A. & Maynard Smith, J. 1990. Optimality theory in evolutionary biology. *Science* **349**: 27–33.
Pen, I. 2000. Reproductive effort in viscous populations. *Evolution* **54**: 293–297.
Pollak, E. 1982. The rate of mutant substitution in populations with overlapping generations. *Genet Res.* **40**: 89–94.
Rousset, F. 2002. Inbreeding and relatedness coefficients: what do they measure? *Heredity* **88**: 371–380.
Rousset, F. 2003. A minimal derivation of convergence stability measures. *J. Theor. Biol.* **221**: 665–668.
Rousset, F. 2004. *Genetic Structure and Selection in Subdivided Populations*. Princeton University Press, Princeton, NJ.
Rousset, F. 2006. Separation of time scales, fixation probabilities and convergence to evolutionarily stable states under isolation by distance. *Theor. Popul. Biol.* **69**: 165–179.
Rousset, F. & Billiard, S. 2000. A theoretical basis for measures of kin selection in subdivided populations: finite populations and localized dispersal. *J. Evol. Biol.* **13**: 814–825.
Rousset, F. & Ronce, O. 2004. Inclusive fitness for traits affecting metapopulation demography. *Theor. Popul. Biol.* **65**: 127–141.
Roze, D. & Rousset, F. 2003. Selection and drift in subdivided populations: a straightforward method for deriving diffusion approximations and applications involving dominance, selfing and local extinctions. *Genetics* **165**: 2153–2166.
Schaffer, M.E. 1988. Evolutionarily stable strategies for a finite population and a variable contest size. *J. Theor. Biol.* **132**: 469–478.
Sella, G. & Hirsh, A.E. 2005. The application of statistical physics to evolutionary biology. *Proc. Natl. Acad. Sci. USA.* **102**: 9541–9546.
Taylor, P. 1988. Inclusive fitness models with two sexes. *Theor. Popul. Biol.* **34**: 145–168.
Taylor, P.D. 1989. Evolutionary stability in one-parameter models under weak selection. *Theor. Popul. Biol.* **36**: 125–143.
Taylor, P. 1990. Allele-frequency change in a class-structured population. *Am. Nat.* **135**: 95–106.
Taylor, P.D. & Frank, S.A. 1996. How to make a kin selection model. *J. Theor. Biol.* **180**: 27–37.
Taylor, P.D., Day, T. & Wild, G. 2007a. Evolution of cooperation in a finite homogeneous graph. *Nature* **447**: 469–472.
Taylor, P.D., Day, T. & Wild, G. 2007b. From inclusive fitness to fixation probability in homogeneous structured populations. *J. Theor. Biol.* **249**: 101–110.
Vincent, T.L. & Brown, J.S. 2005. *Evolutionary Game Theory, Natural Selection, and Darwinian Dynamics*. Cambridge University Press, Cambridge.
Waxman, D. & Gavrilets, S. 2005. 20 questions on adaptive dynamics. *J. Evol. Biol.* **18**: 1139–1154.
Wenseleers, T., Gardner, A. & Foster, K.R. 2010. Social evolution theory: a review of methods and approaches. In: *Social Behaviour: Genes, Ecology and Evolution* (T. Szekely, A. Moore & J. Komdeur, eds), pp. 132–158. Cambridge University Press, Cambridge.
Wild, G. & Taylor, P. 2004. Fitness and evolutionary stability in game theoretic models of finite populations. *Proc. Biol. Sci.* **271**: 2345–2349.
Wolfram, S. 2003. *Mathematica*, 5th edn. Cambridge University Press, Cambridge.
Wright, S. 1931. Evolution in Mendelian populations. *Genetics* **16**: 97–159.
Zu, J., Mimura, M. & Wakano, J.Y. 2010. The evolution of phenotypic traits in a predator–prey system subject to allee effect. *J. Theor. Biol.* **262**: 528–543.

Appendix A: substitution rate

Arbitrary mutation distributions

In this appendix, eqn. 2 of the main text for the substitution rate $k(\delta, z)$ is derived. This gives the expected number of δ mutants produced in a monomorphic population for z and that will fix in the population. This is also the expectation over all demographic states of the expected number $N_{\mathrm{Fix}}(\mathbf{s}, \delta, z)$ of δ mutants produced in a monomorphic population for z in state $\mathbf{s}$ and that will ultimately fix in the population. Namely,

$$k(\delta, z) = \sum_{\mathbf{s}} N_{\mathrm{Fix}}(\mathbf{s}, \delta, z) \Pr(\mathbf{s} \mid z) \tag{A-1}$$

with

$$N_{\mathrm{Fix}}(\mathbf{s}, \delta, z) = \sum_{\mathbf{s}'} \sum_{j} \sum_{i} \pi_i(\mathbf{s}', \delta, z) w_{ij}(\mathbf{s}', \mathbf{s}, z) g_i t_{ij} \times \mu_{ij}(\delta, z) N_j(\mathbf{s}, z) \Pr(\mathbf{s}' \mid \mathbf{s}, z), \tag{A-2}$$

where $\pi_i(\mathbf{s}', \delta, z)$ is the fixation probability of a δ mutant when arising as a single copy in a class i individual when

the population is monomorphic for z and in demographic state $\mathbf{s}'$; w_{ij} ($\mathbf{s}'$, $\mathbf{s}$; z) is the expected number of class-i individuals in a population in state $\mathbf{s}'$ produced by a single class-j individual in a population in state $\mathbf{s}$ and monomorphic for z; g_i is the ploidy of a class i individual; t_{ij} is the probability that a gene randomly sampled in a class-i offspring descends from a class-j individual ($g_i t_{ij}$ is the number of class-i genes descending from a class-j individual); $\mu_{ij}(\delta,z)$ is the probability that this gene codes for phenotypic deviation δ; and $N_j(\mathbf{s},z)$ is the number of individuals in class j in demographic state $\mathbf{s}$ in a population monomorphic for z.

In order to simplify eqns A-1 and A-2, it is useful to use

$$\Pr(\mathbf{s}|\mathbf{s}',z) \equiv \frac{\Pr(\mathbf{s}'\,|\,\mathbf{s},z)\Pr(\mathbf{s}\,|\,z)}{\Pr(\mathbf{s}'\,|\,z)}, \tag{A-3}$$

which is the backward transition probability that a population in state $\mathbf{s}'$ in the offspring generation (a prime always refers to the offspring generation) derives from a population in state $\mathbf{s}$ in the parental generation (e.g. Karlin, 1968; Rousset & Ronce, 2004). We will also use

$$\begin{aligned} f_{ij}(\mathbf{s}',\mathbf{s},z) &\equiv \frac{w_{ij}(\mathbf{s}',\mathbf{s},z)g_i t_{ij} N_j(\mathbf{s},z)}{g_i N_i(\mathbf{s}',z)} \\ &= \frac{w_{ij}(\mathbf{s}',\mathbf{s},z)t_{ij} N_j(\mathbf{s},z)}{N_i(\mathbf{s}',z)}, \end{aligned} \tag{A-4}$$

which is the probability that a gene sampled in a class i individual when the population is in state $\mathbf{s}'$ in the offspring generation is a copy of a gene of a class j individual when the population was in state $\mathbf{s}$ in the parental generation (e.g. Charlesworth, 1980; Rousset & Ronce, 2004), and is obtained as the ratio of the number of genes in class i descending from class j to the total number of genes in class i.

Substituting these expressions into eqns A-1 and A-2 yields

$$k(\delta,z) = \sum_{\mathbf{s}'}\sum_i \pi_i(\mathbf{s}',\delta,z)M_i(\mathbf{s}',\delta,z)\Pr(\mathbf{s}'\,|\,z), \tag{A-5}$$

where

$$M_i(\mathbf{s}',\delta,z) = g_i N_i(\mathbf{s}',z)\sum_{\mathbf{s}}\sum_j \Pr(\mathbf{s}\,|\,\mathbf{s}',z)f_{ij}(\mathbf{s}',\mathbf{s},z)\mu_{ij}(\delta,z) \tag{A-6}$$

is the number of δ mutants in class i when the population is in demographic state $\mathbf{s}'$.

Equation A-6 allows us to evaluate the expected number $M(\delta,z)$ of δ mutants produced in a population monomorphic for z. This is

$$M(\delta,z) = \sum_{\mathbf{s}}\sum_i M_i(\mathbf{s},\delta,z)\Pr(\mathbf{s}\,|\,z). \tag{A-7}$$

and as $\int M(\delta,z)\mathrm{d}\delta$ gives the total expected number of mutants produced in the population, regardless of their type, we have

$$u(\delta,z) \equiv \frac{M(\delta,z)}{\int M(\delta,z)\mathrm{d}\delta} = \frac{M(\delta,z)}{\bar{N}(z)\mu(z)}, \tag{A-8}$$

which is the probability that, among all possible mutants, a δ mutant is produced in a population monomorphic for z, where $\mu(z) = \int M(\delta,z)\mathrm{d}\delta/\bar{N}(z)$ is the probability that a mutant arises in the population and $\bar{N}(z) = \sum_{\mathbf{s}} N(\mathbf{s},z)\Pr(\mathbf{s}\,|\,z)$ is the expected total number of homologous genes in the population, which is the average over all states of the total number $N(\mathbf{s},z) = \sum_i N_i(\mathbf{s},z)g_i(z)$ of genes when the population is in state $\mathbf{s}$.

Equation A-6 also allows us to compute

$$\begin{aligned} q_i(\mathbf{s},\delta,z) &= \frac{M_i(\mathbf{s},\delta,z)}{\sum_i M_i(\mathbf{s},\delta,z)} \\ q(\mathbf{s},\delta,z) &= \frac{\left[\sum_i M_i(\mathbf{s},\delta,z)\right]\Pr(\mathbf{s}\,|\,z)}{M(\delta,z)}, \end{aligned} \tag{A-9}$$

where $q_i(\mathbf{s},\delta,z)$ is the probability that a δ mutant arises in an class-i individual when the population is in state $\mathbf{s}$ and when the parental generation is monomorphic for z and $q(\mathbf{s},\delta,z)$ is the probability that the δ mutant arises when the population is in state $\mathbf{s}$. Averaging $\pi_i(\mathbf{s},\delta,z)$ over these quantities yields

$$\begin{aligned} \bar{\pi}_\mu(\delta,z) &\equiv \sum_{\mathbf{s}}\sum_i \pi_i(\mathbf{s},\delta,z)q_i(\mathbf{s},\delta,z)q(\mathbf{s},\delta,z) \\ &= \sum_{\mathbf{s}}\sum_i \pi_i(\mathbf{s},\delta,z)M_i(\mathbf{s},\delta,z)\Pr(\mathbf{s}\,|\,z)/M(\delta,z). \end{aligned} \tag{A-10}$$

which is the average fixation probability of a single δ mutant in the population and where the subscript μ emphasizes that the fixation probability may depend on the mutation distributions $\mu_{ij}(\delta,z)$.

With the above quantities (eqns A-6–A-10), algebraic rearrangements show that we can write eqn. A-5 as

$$k(\delta,z) = \bar{N}(z)\mu(z)u(\delta,z)\bar{\pi}_\mu(\delta,z). \tag{A-11}$$

Same mutation distributions across classes

The above expressions can be simplified when $\mu_{ij}(\delta,z) = \mu(\delta,z)$. In this case, we have

$$\begin{aligned} M_i(\mathbf{s}',\delta,z) &= g_i N_i(\mathbf{s}',z)\sum_{\mathbf{s}}\sum_j \Pr(\mathbf{s}\,|\,\mathbf{s}',z)f_{ij}(\mathbf{s}',\mathbf{s},z)\mu(\delta,z) \\ &= \mu(\delta,z)g_i N_i(\mathbf{s}',z)\underbrace{\sum_{\mathbf{s}}\Pr(\mathbf{s}\,|\,\mathbf{s}',z)}_{1}\underbrace{\sum_j f_{ij}(\mathbf{s}',\mathbf{s},z)}_{1} \\ &= \mu(\delta,z)g_i N_i(\mathbf{s}',z), \end{aligned} \tag{A-12}$$

whereby $M(\delta,z) = \mu(\delta,z)\bar{N}(z)$. The average fixation probability (eqn. A-10) then no longer depends on the mutation rate and can be written as

$$\bar{\pi}(\delta,z) = \sum_{\mathbf{s}}\sum_i \pi_i(\mathbf{s},\delta,z)\frac{g_i N_i(\mathbf{s},z)\Pr(\mathbf{s}\,|\,z)}{\bar{N}(z)}. \tag{A-13}$$

Under neutrality, $\pi_i(\mathbf{s},0,z) = \alpha_i(\mathbf{s},z)/[g_i N_i(\mathbf{s},z)]$, where $1/[g_i N_i(\mathbf{s},z)]$ is the initial mutant frequency in class i and

$\alpha_i(\mathbf{s},z)$ is the probability that a gene randomly sampled in the population descends in a distant past τ from an individual from class i, conditional on the population being in state $\mathbf{s}$ in the distant past τ and monomorphic for z (Leturque & Rousset, 2002; Rousset & Ronce, 2004). The reproductive value $\alpha_i(\mathbf{s},z)$ also provides the ultimate contribution to the population of a gene lineage presently taken in a class i individual when the population is in state $\mathbf{s}$. As $\sum_i \alpha_i(\mathbf{s}, z) = 1$, the average fixation probability of a neutral mutant reduces to $\bar{\pi}(0, z) = 1/\bar{N}(z)$.

When the δ mutant is not neutral, the perturbation $S(z) = d\bar{\pi}(\delta, z)/d\delta|_{\delta=0}$ of the average fixation probability (eqn. A-13) of the δ mutant has been given a distinctive outline by Rousset & Ronce (2004), eqn. 23) for the case without dominance as it can be written under the form of an inclusive fitness effect:

$$S(z) = \lim_{v\to 0} \frac{1}{1-Q_o} \times \left[\sum_{\mathbf{s}} \sum_{\mathbf{s}'} \sum_{i} \sum_{j} \sum_{c\in\mathscr{A}} \alpha_i(\mathbf{s}', z) \frac{\partial}{\partial z_c} \left[f_{ij}(\mathbf{s}', \mathbf{s}, \mathbf{z}) \Pr(\mathbf{s}' \mid \mathbf{s}, \mathbf{z}) \right] Q_{jc}(\mathbf{s}, z) \Pr(\mathbf{s} \mid z) \right], \tag{A-14}$$

where $\partial[f_{ij}(\mathbf{s}', \mathbf{s}, \mathbf{z})\Pr(\mathbf{s}' \mid \mathbf{s},\mathbf{z})]/\partial z_c$ is the change, due to all actors of category c expressing the mutant deviation δ, of the probability that a gene taken in a class i individual in a state $\mathbf{s}'$ population descends from an individual in class j and from a population in state $\mathbf{s}$ (the derivatives are evaluated at $z_c = z$ for all c). The set of actors $\mathscr{A} = \{\bullet, 0, 1, 2, \ldots\}$ includes a representative individual (focal individual) with phenotype denoted $z_\bullet$ and each class of individual because individuals from any class may affect the vital rates of a focal individual belonging to any class. The functions $\Pr(\mathbf{s}' \mid \mathbf{s}, \mathbf{z})$ and

$$f_{ij}(\mathbf{s}', \mathbf{s}, \mathbf{z}) = \frac{w_{ij}(\mathbf{s}', \mathbf{s}, \mathbf{z}) t_{ij} N_j(\mathbf{s}, z)}{N_i(\mathbf{s}', z)}, \tag{A-15}$$

now depend on mutant phenotypes through the vector $\mathbf{z} \equiv (z_\bullet, z_0, z_1, z_2, \ldots)$ of average phenotypes of the individuals in each class (see Rousset, 2004; Rousset & Ronce, 2004 for details).

The phenotypic selection gradient $S(z)$ also depends on the mutation rate v used to evaluate the coefficients Q in a neutral model in a finite population (Malécot, 1975; Gillespie, 2004). For practical applications, the infinite alleles model of mutation is convenient to use (Kimura & Crow, 1964). With this, $Q_{jc}(\mathbf{s},z)$ gives the probability that two homologous genes randomly sampled, one from class j and the other from class c, are identical-by-descent (Rousset, 2004). More specifically, when the two genes are sampled in a focal actor of class j [$Q_{j\bullet}(\mathbf{s},z)$ coefficient], they are sampled with replacement. In the remaining cases, the two genes are assumed to be randomly sampled from two distinct individuals (without replacement), one from class j and the other from class c, which describes an actor–recipient relation. One can also write $S(z)$ in terms of identity coefficients where genes are always sampled with replacement (Rousset & Ronce, 2004, eqn. 23). Either way yields the same results provided the definition of the actors' phenotypes appearing in $f_{ij}(\mathbf{s}', \mathbf{s}, \mathbf{z})\Pr(\mathbf{s}' \mid \mathbf{s}, \mathbf{z})$ matches the definition of the identity coefficients (Rousset, 2004). Finally, we need Q_o, which is defined from

$$1 - Q_o = 2v\bar{N}(z) + O(v^2). \tag{A-16}$$

With this, in a geographically structured population of constant size $\bar{N}(z)$, Q_o is equivalent to the probability of identity between a pair of homologous genes sampled without replacement in the same patch (Leturque & Rousset, 2002; Rousset, 2002, 2003, 2004); hence, the term 'localized' inclusive fitness effect. Equation 2 was obtained by rearranging eqn. 23 of Rousset & Ronce (2004) and using $1/(1 - Q_o)$ in place of their $1/[2v\bar{N}(z)]$.

Birth–death reproductive process

Here, an expression for $k(\delta,z)$ will be given for a haploid panmictic population without class structure and following a birth–death reproductive protocol (Karlin & Taylor, 1975; Grimmett & Stirzaker, 2001). This allows to connect the present formalization with the model of Champagnat & Lambert (2007). For a birth–death reproductive protocol, a demographic state can be taken to be population size n ($\mathbf{s} \equiv n$) and $b(n,z)$ will denote the birth rate of a single individual in a population of size n that is monomorphic for z and $d(n,z)$ denotes the death rate of an individual in a population of size n (Champagnat & Lambert, 2007, p. 2).

For a birth and death process, the transition probability $\Pr(n' \mid n,z)$ from size n in a parental generation to size n' in an offspring generation is nonzero per time interval h only for the following transitions

$$\begin{aligned} \Pr(n+1 \mid n,z) &= b(n,z)nh + o(h) \text{ ('birth' in the population)} \\ \Pr(n-1 \mid n,z) &= d(n,z)nh + o(h) \text{ ('death' in the population)} \\ \Pr(n \mid n,z) &= 1 - [b(n,z) + d(n,z)]nh \\ &\quad + o(h) \text{ ('no transition')}. \end{aligned} \tag{A-17}$$

There is no class structure under this birth–death setting, and the average fixation probability (eqn. A-10) can be written as

$$\bar{\pi}_\mu(\delta,z) = \sum_{n'} \pi(n',z,\delta) M(n',z,\delta) \Pr(n',z)/M(\delta,z), \tag{A-18}$$

where $\pi(n',z,\delta)$ is the fixation probability of a single δ mutant when it arises in a population of size n'. Because eqn. A-17 entails that at most one mutant can enter the population per time step, which occurs when there is a

birth in the population, the number of δ mutants that enter a population of size n' is

$$M(n',z,\delta)=\sum_n f(n',n,z)\mu(n',n,z,\delta)\Pr(n\,|\,n',z), \quad \text{(A-19)}$$

where $f(n',n,z)$ is the probability that a gene taken in a population of size n' in the offspring generation, which was of size n in the parental generation, descends from an individual in the parental generation. We also have $\mu(n',n,z,\delta)$, which is the probability that a δ mutant is produced when the parental generation is of size n and the descendant generation is of size n'. Here, the mutation rate is written as a function of demographic states, and this could also have been done for the model introduced above [eqns A-1–A-11 could have been written in terms of $\mu_{ij}(\mathbf{s}', \mathbf{s}, z, \delta)$], but introducing this dependence will not change the final expressions, and as it seems rather specific to birth–death processes, it was not introduced above for ease of presentation.

Because mutations can occur only when there is a birth, one has $\mu(n+1, n, z, \delta) \geq 0$, zero otherwise, and the shorthand notation $\mu(z,\delta) \equiv \mu(n+1, n, z, \delta)$ will be used. Further, $f(n+1, n) = 1$ and $f(n, n) = 1$, $f(\cdot, n) = 0$ otherwise. With this, we have

$$\begin{aligned} M(n',z,\delta) &= f(n',n'-1,z)\mu(n',n'-1,z,\delta)\Pr(n'-1\,|\,n',z) \\ &= \mu(z,\delta)\Pr(n'-1\,|\,n',z), \end{aligned} \quad \text{(A-20)}$$

where $\Pr(n'-1 \mid n',z)$ is the probability that a population of size n' descends from a population of size $n'-1$ (backward transition probability). As a δ mutant can be produced in a z population only if it is not extinct, we have $M(n', z, \delta) = 0$ for $n' < 2$, otherwise

$$\begin{aligned} M(n',z,\delta)\Pr(n',z) &= \mu(z,\delta)\Pr(n'-1\,|\,n',z)\Pr(n',z) \\ &= \mu(z,\delta)\Pr(n'\,|\,n'-1,z)\Pr(n'-1,z) \\ &= \mu(\delta,z)b(n'-1,z)(n'-1) \\ &\quad \Pr(n'-1,z)h+o(h), \end{aligned} \quad \text{(A-21)}$$

where care must be taken with the notations in this equation as $\Pr(n'-1 \mid n',z)$ has to be read as a backward transition probability, whereas $\Pr(n' \mid n'-1,z)$ as a forward transition probability, which is given by eqn. A-17. From the above

$$\begin{aligned} M(\delta,z) &= \sum_{n'} M(n',z,\delta)\Pr(n',z) \\ &= \sum_{n'\geq 2} \mu(\delta,z)b(n'-1,z)(n'-1)\Pr(n'-1,z)h+o(h) \\ &= \sum_{n\geq 1} \mu(\delta,z)b(n,z)n\Pr(n,z)h+o(h), \end{aligned} \quad \text{(A-22)}$$

and the average fixation probability can be written

$$\bar{\pi}_\mu(\delta,z)=\sum_{n\geq 1}\pi(n+1,z,\delta)\frac{b(n,z)n\Pr(n,z)}{\sum_{n\geq 1}b(n,z)n\Pr(n,z)}+o(h). \quad \text{(A-23)}$$

Substituting eqns A-22 and A-23 along with eqn. A-8 into eqn. A-11 and assuming that the substitution rate is for a continuous time reproductive process evaluated as a rate per unit time h as $h \to 0$; that is, $k(\delta,z) = \lim_{h\to 0}\bar{N}(z)\mu(z)u(\delta,z)\bar{\pi}_\mu(\delta,z)/h$, produces

$$k(\delta,z)=\underbrace{\mu(z,\delta)\left[\sum_{n\geq 1}b(n,z)n\Pr(n,z)\right]}_{\beta(z)M(z,\delta)}\underbrace{\bar{\pi}_\mu(\delta,z)}_{\chi(z,z+\delta)}. \quad \text{(A-24)}$$

This is the jump rate for the continuous time reproductive scheme derived by Champagnat & Lambert (2007, eqn. 7), where their notations are used in the underbraces in order to highlight the connection.

Appendix B: trait-substitution sequence

Master equation

Here, a partial differential equation for $p(z,t)$ expressed in terms of the substitution rate $k(\delta,z)$ will be presented by applying standard results of stochastic processes derived in Gardiner (2009). In particular, the probability density function $p(z,t)$ of a Markov chain with instantaneous transition rate

$$\lim_{\Delta t\to 0}\frac{p(z(t+\Delta t)=z'\,|\,z(t)=z)}{\Delta t}\equiv T(z'\,|\,z) \quad \text{(B-1)}$$

from state z to z' for $z' \neq z$ follows the so-called master equation

$$\frac{\partial p(z,t)}{\partial t}=\int[T(z\,|\,z')p(z',t)-T(z'\,|\,z)p(z,t)]\mathrm{d}z', \quad \text{(B-2)}$$

where the first term describes the fact that $p(z,t)$ is increased by all changes from state z' to z and the second term that t is decreased by all changes from z to z' (Gardiner, 2009, eqn. 3.5.2).

In order to express eqn. B-2 in terms of $k(\delta,z)$, I follow directly along the lines of Gardiner (2009, p. 276) and make a change of variables in eqn. B-2 by substituting $z' = z-\delta$ in the first term, $z' = z+\delta$ in the second term, and then use

$$T(z+\delta\,|\,z)\equiv k(\delta,z), \quad \text{(B-3)}$$

whereby the master equation becomes

$$\frac{\partial p(z,t)}{\partial t}=\int[k(z-\delta,\delta)p(z-\delta,t)-k(\delta,z)p(z,t)]\mathrm{d}\delta \quad \text{(B-4)}$$

(Gardiner, 2009, eqn. 11.2.24).

By way of a Taylor expansion of eqn. B-4 around $\delta = 0$, we obtain

$$\begin{aligned}\frac{\partial p(z,t)}{\partial t} &= \int\left(\sum_{n=1}^{\infty}\frac{(-\delta)^n}{n!}\frac{\partial^n}{\partial z^n}[k(\delta,z)p(z,t)]\right)\mathrm{d}\delta \\ &= \sum_{n=1}^{\infty}\frac{(-1)^n}{n!}\frac{\partial^n}{\partial z^n}[\alpha_n(z)p(z,t)],\end{aligned} \tag{B-5}$$

where

$$\alpha_n(z) = \int \delta^n k(\delta,z)\mathrm{d}\delta \tag{B-6}$$

is the n-th jump moment of the substitution process.

Diffusion equation

Here, a diffusion equation for $p(z, t)$ will be derived from eqn. B-5 by taking into account only jumps of small magnitude in δ. In order to obtain the limit of small jumps, the mutation distribution $u(\delta, z)$ is assumed to have been rescaled so that it describes a distribution of only small phenotypic deviation around z. For an unscaled mutation distribution, say $u(\epsilon, z)$, which can accommodate phenotypic deviations ϵ of any length, a rescaled distribution allowing only for small deviations can be obtained as $u(\epsilon/\delta, z)/\delta$, which integrates up to one: $\int[u(\epsilon/\delta, z)/\delta]\mathrm{d}\delta = 1$ (Dercole & Rinaldi, 2008).

For ease of presentation, I assume that the distribution $u(\delta,z)$ used in the main text has been rescaled to allow only small jumps and substitute eqn. 3 of the main text into eqn. 2, whereby

$$k(\delta,z) = \bar{N}(z)\mu(z)u(\delta,z)\left[\bar{\pi}_{\mu}^{\circ}(z) + \delta\dot{\bar{\pi}}_{\mu}(z)\right] + O(\delta^2), \tag{B-7}$$

which, on substitution into the jump moments (eqn. B-6), gives

$$\begin{aligned}\alpha_1(z) &= \bar{N}(z)\mu(z)\int\left[\delta\bar{\pi}_{\mu}^{\circ}(z) + \delta^2\dot{\bar{\pi}}_{\mu}(z) + O(\delta^3)\right]u(\delta,z)\mathrm{d}\delta \\ \alpha_2(z) &= \bar{N}(z)\mu(z)\int\left[\delta^2\bar{\pi}_{\mu}^{\circ}(z) + O(\delta^3)\right]u(\delta,z)\mathrm{d}\delta \\ \alpha_n(z) &= \bar{N}(z)\mu(z)\int O(\delta^3)u(\delta,z)\mathrm{d}\delta \text{ if } n > 2.\end{aligned} \tag{B-8}$$

The mutation distribution was assumed symmetric, which implies that the mean deviation is zero: $\int\delta u(\delta, z)\mathrm{d}\delta = 0$. Defining $\sigma^2(z) \equiv \int\delta^2 u(\delta,z)\mathrm{d}\delta$ as the variance of the mutant deviation when the reference phenotype is z and assuming that higher central moments vanish, $\int\delta^n u(\delta, z)\mathrm{d}\delta \to 0$ for $n > 2$, gives

$$\begin{aligned}\alpha_1(z) &= \bar{N}(z)\mu(z)\sigma^2(z)\dot{\bar{\pi}}_{\mu}(z) \\ \alpha_2(z) &= \bar{N}(z)\mu(z)\sigma^2(z)\bar{\pi}_{\mu}^{\circ}(z) \\ \alpha_n(z) &= 0 \text{ if } n > 2.\end{aligned} \tag{B-9}$$

Substituting these jump moments back into eqn. B-5 yields

$$\frac{\partial p(z,t)}{\partial t} = -\frac{\partial}{\partial z}[a(z)p(z,t)] + \frac{1}{2}\frac{\partial^2}{\partial z^2}[b(z)p(z,t)], \tag{B-10}$$

which is a diffusion equation for the change in phenotype, where $a(z) \equiv \alpha_1(z)$ and $b(z) \equiv \alpha_2(z)$ are the infinitesimal mean and variance of the process (e.g. Kimura, 1964; Karlin & Taylor, 1981; Gillespie, 1991; Ewens, 2004; Gardiner, 2009).

Stationary distribution

The long-term phenotypic distribution is given by $p(z) = \lim_{t\to\infty} p(z, t)$, which is characterized by an evolutionary steady-state $\partial p(z)/\partial t = 0$. In order to obtain the probability density function $p(z)$, it is useful to express eqn. 4 as $\partial p(z, t)/\partial t = -\partial J(z, t)/\partial z$, where $J(z, t)$ is the probability flux through z at time t (Kimura, 1964, p. 187; Gillespie, 1991, p. 157; Gardiner, 2009, p. 119). At steady state, the probability flux $J(z) = \lim_{t\to\infty} J(z,t)$ is given by

$$J(z) = a(z)p(z) - \frac{1}{2}\frac{\partial}{\partial z}[b(z)p(z)]. \tag{B-11}$$

The diffusion process is assumed to have reflecting boundaries so that $J(z) = 0$ for all $z \in [l, r]$ (Gardiner, 2009, pp. 119–121). The stationary distribution can then be obtained by substituting $y(z) = \log(p(z))$ into $J(z) = 0$, which gives $a(z) - (1/2)\partial b(z)/\partial z - (1/2)b(z)\partial y(z)/\partial z = 0$ (e.g. Kimura, 1964; Gillespie, 1991; Ewens, 2004; Gardiner, 2009), and can be solved by integration to give

$$p(z) = \frac{K}{b(z)}\exp\left[2\int_l^z\frac{a(y)}{b(y)}\mathrm{d}y\right], \tag{B-12}$$

where K is a normalization constant ensuring that $\int_l^r p(z)\mathrm{d}z = 1$.

Appendix C: application

Pairwise interaction game

Here, we present the explicit expression for $p(z)$ for the pairwise interaction game. From eqns 11 and 20, one has

$$S(z) = \frac{(N-2)(P+R-S-T)z - (N-2)P + (N-1)S - T}{N\left[1 + z(S+T) + z^2(R-S-T) + P(1-z)^2\right]}. \tag{C-1}$$

Although the calculation of the integral $\int_0^z 2NS(y)\mathrm{d}y$ for the stationary distribution (eqn. 7) is difficult, it can readily be achieved by a computational software program such as Mathematica (Wolfram, 2003). The stationary distribution can then be written as

$$p(z) = K\mathrm{e}^{-X(z)N}Y(z)^{N-2}, \tag{C-2}$$

where

$$Y(z) = \frac{1 + P(1-z)^2 + z[S + T + z(R-S-T)]}{1+P}. \tag{C-3}$$

and

$$X(z) = \frac{2(T-S)[\tan^{-1}((S+T-2P(1-z)+2z(R-S-T))/V)+\tan^{-1}((2P-S-T)/V)]}{V} \quad \text{(C-4)}$$

with $V = \sqrt{4R+4P(R+1)-(S+T)(4+S+T)}$ and $\tan^{-1}(x)$ is the inverse function of the tangent.

When $z = 0$, we have from eqns C-2 and C-3 that $p(0) = K$. The strategy $z = 1$ then gets more probability mass if

$$e^{-X(1)N}Y(1)^{N-2} > 1 \quad \text{(C-5)}$$

because it entails $p(1) > K$. For a coordination game with parameterization $R > P > 0$, $R > T > 0$, and $S = 0$, it can be checked numerically that if the condition $P + T > R$ holds, then $e^{-X(1)N}Y(1)^{N-2}$ usually goes to zero as N grows very large. Hence, the stochastic system tends to spend most of its time on the risk dominant strategy ($z = 0$), rather than on the payoff dominant strategy ($z = 1$).

Sex-allocation game

Here, we derive the expression of $S(z)$ for the sex-allocation model by simplifying eqn. A-14 according to the specificity of the assumptions described in the main text. The population is assumed to be of constant size, and there are only two classes of individuals (males and females) with equal number of adults in each class. This entails that the fitness functions do not depend on demographics states, $f_{ij} = w_{ij}$ in eqn. A-14, and the sum over i and j in eqn. A-14 runs over the set f, m of classes of individuals. Further, the reproductive values and the probabilities of identity-by-descent do not depend on the evolving trait. As the sex ratio is assumed to be fully under maternal control, the set of actors can be written as $\mathscr{A} = \{\bullet, \mathrm{f}\}$ and the vector of phenotypes as $\mathbf{z} \equiv (z_\bullet, z_f)$, where $z_\bullet$ is the phenotype of a focal female and z_f is the average phenotype among females in the population but excluding the focal female ($z_\mathrm{f}^\mathrm{R} = z_\bullet/N + (N-1)z_\mathrm{f}/N$ is the average phenotype of females in the population including the focal female). Further, any male regardless of its genotype will have the same fitness through sons and daughters than any other male. So the fitness contributions of males are constants and can be removed from the calculations of the selection gradient (e.g. Taylor & Frank, 1996; Frank, 1998).

From all these considerations, it follows that eqn. A-14 can be simplified to

$$S(z) = \lim_{v\to 0}\frac{1}{1-Q_\mathrm{o}}\left[\alpha_\mathrm{f} t_\mathrm{ff}\left\{\frac{\partial w_\mathrm{ff}(z_\bullet, z_\mathrm{f})}{\partial z_\bullet}Q_\mathrm{f} + \frac{\partial w_\mathrm{ff}(z_\bullet, z_\mathrm{f})}{\partial z_\mathrm{f}}Q_\mathrm{ff}\right\}\right.$$
$$\left.+\alpha_\mathrm{m} t_\mathrm{mf}\left\{\frac{\partial w_\mathrm{mf}(z_\bullet, z_\mathrm{f})}{\partial z_\bullet}Q_\mathrm{f} + \frac{\partial w_\mathrm{mf}(z_\bullet, z_\mathrm{f})}{\partial z_\mathrm{f}}Q_\mathrm{ff}\right\}\right], \quad \text{(C-6)}$$

where Q_ff is the probability of identity between two homologous genes randomly sampled in two different females and $Q_\mathrm{f} \equiv Q_{\mathrm{f}\bullet}$ is the probability of identity between two homologous genes randomly sampled with replacement from the same female (coancestry with self). This is equal to $Q_\mathrm{f} = (1+F)/2$, where F is the probability of identity between homologous genes taken in the same individual (inbreeding coefficient).

Using the zero sum property of the partial derivatives [e.g. $\partial w_\mathrm{ff}(z_\bullet, z_\mathrm{f})/\partial z_\bullet = -\partial w_\mathrm{ff}(z_\bullet, z_\mathrm{f})/\partial z_\mathrm{f}$, Rousset, 2004], we can further reduce the inclusive fitness effect to

$$S(z) = \left(\alpha_\mathrm{f} t_\mathrm{ff}\frac{\partial w_\mathrm{ff}(z_\bullet, z_\mathrm{f})}{\partial z_\bullet} + \alpha_\mathrm{m} t_\mathrm{mf}\frac{\partial w_\mathrm{mf}(z_\bullet, z_\mathrm{f})}{\partial z_\bullet}\right)L, \quad \text{(C-7)}$$

where

$$L \equiv \lim_{v\to 0}\left(\frac{Q_\mathrm{f}-Q_\mathrm{ff}}{1-Q_\mathrm{o}}\right). \quad \text{(C-8)}$$

In order to close the model, we need the expressions for Q_f, Q_ff and Q_o. These can be obtained by applying standard calculations for probabilities of identity-by-descent (Karlin, 1968; Rousset, 2004). For instance, for a haplo-diploid model with haploid males and diploid females, the probabilities of identity-by-descent satisfy the following recursions at equilibrium

$$\begin{aligned}
Q_\mathrm{ff} &= \gamma\left[\frac{1}{4}(P_\mathrm{f}Q_\mathrm{f} + (1-P_\mathrm{f})Q_\mathrm{ff}) + \frac{1}{2}Q_\mathrm{fm}\right.\\
&\quad\left.+\frac{1}{4}(P_\mathrm{m} + (1-P_\mathrm{m})Q_\mathrm{mm})\right]\\
Q_\mathrm{fm} &= \gamma\left[\frac{1}{2}(P_\mathrm{f}Q_\mathrm{f} + (1-P_\mathrm{f})Q_\mathrm{ff}) + \frac{1}{2}Q_\mathrm{fm}\right]\\
Q_\mathrm{mm} &= \gamma[P_\mathrm{f}Q_\mathrm{f} + (1-P_\mathrm{f})Q_\mathrm{ff}]\\
Q_\mathrm{f} &= \frac{1+F}{2}\\
F &= \gamma Q_\mathrm{fm},
\end{aligned} \quad \text{(C-9)}$$

where $\gamma = (1-v)^2$ and $P_\mathrm{f} = P_\mathrm{m} = 1/N$ are the probabilities that two individuals descend from, respectively, the

same female and male (e.g. Taylor, 1988). Solving these equations and using $\bar{N}(z) = 3N$ in eqn. A-16, one can then evaluate eqn. C-8. Similar calculations for a diploid reproductive system (where the right members of the three first lines in eqn. 8 are all equal to the right member of the first line) and using $\bar{N}(z) = 4N$ in eqn. A-16 show that

$$L = \begin{cases} \frac{1}{2} & \text{diploid} \\ \frac{9N-4}{18N-6} & \text{haplo-diploid.} \end{cases} \tag{C-10}$$

Received 19 September 2011; revised 22 November 2011; accepted 9 January 2012