The Evolution of Phenotypic Polymorphism: Randomized Strategies versus Evolutionary Branching

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ABSTRACT: A population is polymorphic when its members fall into two or more categories, referred to as alternative phenotypes. There are many kinds of phenotypic polymorphisms, with specialization in reproduction, feeding, dispersal, or protection from predators. An individual's phenotype might be randomly assigned during development, genetically determined, or set by environmental cues. These three possibilities correspond to a mixed strategy of development, a genetic polymorphism, and a conditional strategy. Using the perspective of adaptive dynamics, I develop a unifying evolutionary theory of systems of determination of alternative phenotypes, focusing on the relative possibilities for random versus genetic determination. The approach is an extension of the analysis of evolutionary branching in adaptive dynamics. It compares the possibility that there will be evolutionary branching, leading to genetic polymorphism, with the possibility that a mixed strategy evolves. The comparison is based on the strength of selection for the different outcomes. An interpretation of the resulting criterion is that genetic polymorphism is favored over random determination of the phenotype if an individual's heritable genotype is an adaptively advantageous cue for development. I argue that it can be helpful to regard genetic polymorphism as a special case of phenotypic plasticity.

Keywords: genetic polymorphism, bet hedging, evolutionary branching, convergence stability, phenotypic plasticity.

Many populations are polymorphic, in the sense that their members fall into two or more relatively distinct categories with respect to some of their traits. These categories are referred to as morphs or alternative phenotypes. A wide range of phenotypic polymorphisms are found in nature, corresponding to specialization in, for instance, reproduction, feeding, dispersal, or protection from predators (West-Eberhard 2003). For adaptive polymorphisms, where natural selection plays an important role in the origin or maintenance of alternative phenotypes, some advantage from specialization as one phenotype or another is perhaps the most important factor to take into account (Moran 1992; Roff 1996). Frequency dependence, fluctuating environments, and trait-mediated competition for resources are among the general circumstances that may favor polymorphic specialization. Another basic issue is the system of determination of alternative phenotypes. An individual's phenotype could in principle be randomly assigned at some point in development, or it might be genetically determined. The phenotype could also be set by environmental cues acting during development, and there could be combinations of the different mechanisms. My aim here is to develop a theory of the evolution of systems of phenotype determination using the perspective of adaptive dynamics (Metz et al. 1996; Geritz et al. 1998), involving concepts of evolutionary stability and evolutionary branching. My treatment will focus on the relative possibilities for two of the systems, random versus genetic determination, and can be seen as a complement to and a further development of the analysis of evolutionary branching in adaptive dynamics.

In evolutionary thinking about phenotypic polymorphism, contrasting views on the importance of different systems of phenotype determination have been expressed. The traditional view was that discontinuous variation in phenotypes is nearly always genetic (Ford 1971), but more recently there has been a shift toward emphasizing the role of environmental cues (Schlichting and Pigliucci 1998; Nijhout 1999; West-Eberhard 2003). Issues of genetic and random determination of alternative phenotypes have been dealt with in a rather large literature following from early influential articles on multiple-niche polymorphism (Levene 1953) and fluctuating environments (Dempster 1955; Cohen 1966). This field was synthesized and reviewed by Seger and Brockmann (1987), with the overall conclusion that spatial variation tends to promote genetic

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polymorphism, whereas temporal variation may give rise to random determination of alternative phenotypes (bet hedging). An additional idea that competition between relatives could lead to random determination of the phenotype was put forward by Moran (1992). Both sex allocation theory and evolutionary game theory have dealt with the question of whether a mixed evolutionarily stable strategy (ESS) or a genetic polymorphism of pure strategies is more likely as the evolutionary outcome (Kolman 1960; Maynard Smith 1982, 1988; Thomas 1984; Vickery 1988; Frank 1990; Bergstrom and Godfrey-Smith 1998), including the possibility that interactions between relatives could favor a mixed ESS (Grafen 1979; Hines and Haigh 1985).

Although the idea that disruptive selection may play an important role in the evolution of polymorphism has a relatively long history (e.g., Mather 1955; Maynard Smith 1962; Christiansen 1991), more recent developments in adaptive dynamics have been crucial in bringing disruptive selection to the forefront of evolutionary analysis (Metz et al. 1996; Geritz et al. 1998). The process of evolutionary branching is one of the central theoretical constructs of adaptive dynamics, and it leads to the appearance of genetically determined phenotypic alternatives. In my analysis, I will compare the possibility of such a process of evolutionary branching, resulting in genetic polymorphism, with the alternative outcome that a mixed strategy evolves, that is, a genetically monomorphic but phenotypically polymorphic, randomized strategy. There is some similarity to the idea that sexual dimorphism may evolve as an alternative to branching (Bolnick and Doebeli 2003; Van Dooren et al. 2004), in the sense that sex can be regarded as randomly determined. In making this kind of comparison, it is natural to view the evolution of random determination of the phenotype from the perspective of lack of stability of a phenotypically monomorphic strategy in an extended trait space, containing randomized strategies. By investigating how these possibilities depend on the form of the fitness function, one arrives at a general theory of the evolution of random versus genetic determination of phenotypic alternatives. In certain cases, only one of the outcomes would be selectively favored, but if both are possible, the strength of selection, together with the availability of suitable genetic variation, may determine their relative likelihood.

In this article, I first motivate my approach using an example of phenotypic polymorphism in spatially varying environments. Next, I summarize some basic theory of evolutionary stability in multidimensional trait spaces, which is then applied to the evolution of systems of phenotype determination. An important issue is whether genetic polymorphism can be interpreted as an adaptation, and I discuss this matter in the light of the classical treatment of polymorphism in ecological genetics (e.g., Fisher 1958; Ford 1965). To illustrate the theoretical development, I use examples of multiple-niche polymorphism, bet hedging in temporally fluctuating environments, and competitive interactions between relatives. The examples are also studied using individual-based simulations, in order to go beyond the idealized assumptions of the adaptive dynamics analysis. The possible role of the mutational process in generating the kind of genetic variation needed for the evolution of either random or genetic determination of phenotypes is also explored in the simulations.

Spatial Environmental Variation and Polymorphism

Consider a one-dimensional "primary" trait z, which could, for instance, be the level of investment in defense against predation. If the risk of predation is low in some local environments and high in others, it might benefit an organism to have a lower investment, z_1, in some environments, and a higher investment, z_2, in others. When both primary trait values are present in the same population, there is phenotypic polymorphism. One way of studying the evolution of such polymorphism is to start with a phenotypically monomorphic population, say with phenotype z_0, and to investigate if the situation is evolutionarily stable. To allow for mixed strategies, for instance, for an organism to develop primary trait z_1 with probability q_1 and primary trait z_2 with probability q_2, we can use a space of extended traits $x = (z_1, z_2; q_1, q_2)$. Since $q_1 + q_2 = 1$ must hold, this space is effectively three-dimensional. For the special case of $z_1 = z_2 = z$, an extended trait is indistinguishable from the pure strategy to always develop the primary trait z.

In the example, there are two patches or habitat types, characterized by different optimal values $\theta_1 = z_0 - \delta$ and $\theta_2 = z_0 + \delta$ of the primary trait. The survival of an individual with primary trait z, from the time it attempts to settle in a patch up to density regulation, is described by a Gaussian function of the deviation from the optimum in patch number i, with width parameter σ:

$$\alpha_i(z) = a_i \exp\left[-\frac{(z-\theta_i)^2}{2\sigma^2}\right]. \quad (1)$$

For a mixed developmental strategy $x = (z_1, z_2; q_1, q_2)$, the corresponding probability of survival is

$$\beta_i(x) = q_1\alpha_i(z_1) + q_2\alpha_i(z_2). \quad (2)$$

Following the selective phase, there is density regulation to a given carrying capacity, the same for each patch, corresponding to soft selection. Each remaining adult has a large number of offspring and dies. With probability

$1 - m$, an offspring attempts to settle in the patch it was born in, and with probability m, it attempts to settle in the other patch, so that m is a probability of migration. The example is similar to several previously analyzed models with dispersal between two or more different kinds of patches (e.g., Geritz et al. 1998; Kisdi and Geritz 1999; Kisdi 2002).

For gradual evolutionary change of a population that is phenotypically and genetically monomorphic (except that a resident primary trait may be replaced by a nearby mutant primary trait), it turns out that the primary trait z_0 is convergence stable, in the sense that gradual evolution in the space of primary traits will approach this point. For certain parameter values, for instance, for random dispersal over patches ($m = 0.5$) and small separation between patch optima ($\delta^2 < \sigma^2$), the primary trait z_0 will be the endpoint of evolutionary change. However, for certain other parameter values, including more separated patch optima, the point z_0 is a local fitness minimum in the space of primary traits. A process of evolutionary branching may then occur, in which two genetically determined primary trait values appear in the population and diverge from z_0, ending up near the two patch optima. Another possibility is that z_0, regarded as a point $(z_0, z_0; q_1, q_2)$ in the space of extended, randomized strategies, lacks convergence stability in that space, in the sense that gradual evolution starting near the point can lead away from it, ending up at a randomized strategy with the components z_1 and z_2 near the patch optima. The probability q_1 would then also evolve, from some initial value toward $q_1 = 0.5$. An intuitive assessment, which turns out to be correct, might be that a low probability of migration, with local adaptation as a limiting case, favors genetic determination of the phenotype. On the other hand, for the (biologically unrealistic) case of m being close to 1, a line of descent would experience an alteration between patch types, which might favor a mixed strategy.

The purpose of my analysis is to evaluate the relative possibility of these kinds of evolutionary outcomes for situations with general fitness functions. This requires a theory of convergence stability in multidimensional trait spaces, because the space of extended traits $x = (z_1, z_2; q_1, q_2)$ is multidimensional. A characteristic feature of multidimensional evolutionary stability is that genetic correlations between trait components, like the alternative phenotypes z_1 and z_2, can influence stability. The reason is that genetic correlations may cause selection on one trait component to "pull along" another component, which influences the evolutionary dynamics.

For a particular model, like the multiple-niche example here, one could look directly for an evolutionarily stable genetic polymorphism or a stable mixed strategy, instead of using the approach of examining how a monomorphic strategy z_0 might be destabilized. Nevertheless, the latter approach can have considerable value in unifying diverse features of many models.

Evolution in Multidimensional Trait Spaces

In multidimensional trait spaces, genetic variation plays an important role in determining the direction of evolutionary change. With genetic correlations between traits deriving from genes acting pleiotropically or from linkage disequilibrium, the response to selection on one trait will be distributed among several traits. This has been taken into account in theoretical investigations of evolutionary change and evolutionary stability (e.g., Lande 1979; Iwasa et al. 1991; Abrams et al. 1993; Price et al. 1993) and should also apply to adaptive dynamics. In mutation-limited adaptive dynamics for large populations (Metz et al. 1996; Geritz et al. 1998), one considers a succession of mutations that either fail to invade or go to fixation, or possibly produce genetic polymorphism. A basic tool for the analysis is the concept of invasion fitness, which plays the role of determining with what probability a mutant can invade. Invasion fitness corresponds to the mean rate of change of the logarithm of the size of the mutant gene subpopulation, during a phase when the mutant proportion is small (Metz et al. 1992; Rand et al. 1994; Ferriere and Gatto 1995; Dieckmann and Law 1996). For a trait vector x of a genetically monomorphic, resident population, the invasion fitness of a mutant trait vector x' will be denoted $F(x', x)$. The mutant has a positive probability of invading when $F(x', x) > 0$ and no chance of invading when $F(x', x) < 0$. To study gradual evolutionary change, we can introduce the selection gradient, $S(x)$, which has components

$$S_i(x) = \left.\frac{\partial F(x', x)}{\partial x_i'}\right|_{x'=x}, \tag{3}$$

where x_i' is the ith component of the trait vector x'. Provided that mutational increments in the trait values are small, the change over time of x follows the so-called canonical equation

$$\frac{d}{dt}x_i = m(x)\sum_j \mathbf{C}_{ij}(x)S_j(x), \tag{4}$$

where the $\mathbf{C}_{ij}$ are the elements of the covariance matrix of the mutational increments and $m(x)$ is positive and is related to the rate of production of mutations (Dieckmann and Law 1996). The canonical equation provides an approximation of evolutionary change, valid in the limit of large population size, low rate of mutation, and small mu-

tational increments. It has the advantage of taking into account both genetic correlations and natural selection in a relatively simple manner.

Multidimensional Convergence Stability

An $\hat{x}$ where the selection gradient is 0 is called a singular or equilibrium point in the trait space. To investigate whether gradual evolutionary change will converge to such a point, the canonical equation is a useful starting point. Near an equilibrium point $\hat{x}$ in trait space, we can approximate the selection gradient with its first-order Taylor expansion, using the so-called Jacobian matrix of the selection gradient, consisting of first-order derivatives of the components of the selection gradient,

$$\mathbf{J}_{jk} = \frac{\partial S_j(x)}{\partial x_k}\bigg|_{x=\hat{x}} = \frac{\partial^2 F(x', x)}{\partial x_j' \partial x_k'}\bigg|_{x'=x=\hat{x}} + \frac{\partial^2 F(x', x)}{\partial x_j' \partial x_k}\bigg|_{x'=x=\hat{x}}, \tag{5}$$

so that $S_j(x) \approx \sum_k \mathbf{J}_{jk}(x_k - \hat{x}_k)$ for x near $\hat{x}$. Introducing this into the canonical equation, we can analyze the convergence stability of $\hat{x}$ in terms of properties of the Jacobian matrix (the details are given in the appendix in the online edition of the *American Naturalist*). Let us call the Jacobian matrix negative definite when its symmetric part (with elements $[\mathbf{J}_{jk} + \mathbf{J}_{kj}]/2$) is negative definite and similarly for indefiniteness and positive definiteness.

We have the following stability criterion: if the Jacobian matrix of the selection gradient is negative definite at an equilibrium point $\hat{x}$, the point will be asymptotically stable for any positive definite covariance matrix $\mathbf{C}$ in the canonical equation. This is called strong convergence stability and means that the point would be convergence stable for any fixed pattern of genetic correlations appearing near $\hat{x}$. If the Jacobian matrix instead is indefinite, the convergence stability of $\hat{x}$ can depend on the form of the covariance matrix $\mathbf{C}$, whereas if $\mathbf{J}$ is positive definite, $\hat{x}$ is not convergence stable for any positive definite $\mathbf{C}$.

For a one-dimensional trait space, the criterion corresponds to traditional convergence stability (Eshel and Motro 1981; Eshel 1983; Taylor 1989; Christiansen 1991), and it is a summary and generalization of previous work (e.g., Lessard 1990; Abrams et al. 1993; Motro 1994; Marrow et al. 1996; Matessi and Di Pasquale 1996; Metz et al. 1996; Weissing 1996; Eshel et al. 1997; Geritz et al. 1998). As suggested by the name, strong convergence stability is in practice sufficient for convergence stability, but it is worth noting that very special mechanisms of generating mutational increments in a multidimensional trait space could cause gradual adaptive evolution to move away from a strongly convergence stable point (Leimar 2001, 2005).

Stabilizing and Disruptive Selection

For an equilibrium $\hat{x}$, the invasion fitness $F(x', \hat{x})$ may have a local maximum at $x' = \hat{x}$, corresponding to stabilizing selection, so that $\hat{x}$ is locally uninvadable. If the point is also strongly convergence stable, $\hat{x}$ can be called a continuously stable strategy (CSS), following the terminology introduced by Eshel and Motro (1981). A sufficient condition for stabilizing selection is that the matrix of mutant second derivatives of invasion fitness, the so-called Hessian matrix of invasion fitness, with elements

$$\mathbf{H}_{jk} = \frac{\partial^2 F(x', x)}{\partial x_j' \partial x_k'}\bigg|_{x'=x=\hat{x}}, \tag{6}$$

is negative definite. If the Hessian matrix instead is indefinite or positive definite, so that $F(x', \hat{x})$ as a function of x' has a saddle or a local minimum at $x' = \hat{x}$, there will be disruptive selection at the equilibrium point, at least along some direction in trait space. If the point is convergence stable, evolutionary branching at $\hat{x}$ will be possible.

For a one-dimensional trait space, there is at present a good theoretical understanding of gradual evolution near an equilibrium point (Metz et al. 1996; Geritz et al. 1998). For a multidimensional trait space, all possible outcomes have not been fully explored, but some are relevant to the evolution of phenotype determination. For instance, suppose that the Jacobian matrix is indefinite and the Hessian matrix is positive definite at $\hat{x}$. Depending on the nature of genetic correlations, there may then be evolution toward $\hat{x}$ followed by branching, corresponding to the evolution of genetic phenotype determination. Alternatively, because of the indefinite Jacobian matrix, there might be evolution first toward and then away from $\hat{x}$ in another direction, corresponding to the evolution of random phenotype determination.

Genetic versus Random Phenotype Determination

The idea developed here is to use the theory above to classify evolutionary stability and instability of points that correspond to "pure strategies" in an extended trait space X containing stochastic mixtures of primary traits. For simplicity, I only consider mixtures of at most two primary traits, so an extended trait can be written as $x = (z_1, z_2; q_1, q_2)$, with $q_1 + q_2 = 1$, where q_μ is the probability that an individual develops primary trait z_μ. A pure strategy is an extended trait that is phenotypically mono-

morphic, $x = (z, z; q_1, q_2)$, corresponding to the primary trait z, and I will compare the possibility of evolutionary branching at such an x with the alternative that a stochastic mixture of two different primary traits z_1 and z_2 evolves. For this purpose, several first- and second-order derivatives of invasion fitness are needed. To achieve a simpler presentation, the primary trait space Z is taken to be one-dimensional, but this assumption is relaxed in the appendix, where more detailed derivations appear.

Let us start by focusing on the primary trait space Z. With $f(z', z)$, the invasion fitness for mutant primary trait z' in a resident population with primary trait z, we write the selection gradient in the primary trait space as

$$\left.\frac{\partial f(z', z)}{\partial z'}\right|_{z'=z} = s(z). \tag{7}$$

To study evolutionary stability, we also need the following second derivatives:

$$\left.\frac{\partial^2 f(z', z)}{\partial z'^2}\right|_{z'=z} = A(z), \tag{8}$$

$$\left.\frac{\partial^2 f(z', z)}{\partial z' \partial z}\right|_{z'=z} = D(z). \tag{9}$$

As is well known (e.g., Geritz et al. 1998), an equilibrium $\hat{z}$, with $s(\hat{z}) = 0$, is convergence stable in the primary trait space if $A(\hat{z}) + D(\hat{z}) < 0$, and the sign of $A(\hat{z})$ indicates whether there is disruptive selection at $\hat{z}$, that is, whether $\hat{z}$ is a local fitness minimum or a local fitness maximum.

For the extended trait space X, it will be convenient to express a trait as having three components, $x = (\zeta, \eta, \rho)$, with

$$\begin{aligned} \zeta &= q_1 z_1 + q_2 z_2, \\ \eta &= \frac{z_2 - z_1}{2}, \\ \rho &= 1 - 2q_1. \end{aligned} \tag{10}$$

Thus, ζ is the mean primary trait, η is half the difference between the primary traits in the mixture, and ρ parameterizes the probabilities q_1 and q_2. For $\eta = 0$, the extended trait corresponds to a phenotypic monomorphism, and we want derivatives of invasion fitness $F(x', x)$ at such a point. For the selection gradient, we get the component in the ζ direction as

$$\left.\frac{\partial F(x', x)}{\partial \zeta'}\right|_{\eta'=\eta=0, \zeta'=\zeta=z} = S_\zeta(z) = s(z), \tag{11}$$

whereas the other two components, S_η and S_ρ, are 0 (appendix), so S at a phenotypic monomorphism is determined by the gradient s in the primary space. We also need a number of second-order derivatives for the elements of the Jacobian and Hessian matrices in equations (5) and (6). We get

$$\left.\frac{\partial^2 F(x', x)}{\partial \zeta'^2}\right|_{\eta'=\eta=0, \zeta'=\zeta=z} = A(z), \tag{12}$$

$$\left.\frac{\partial^2 F(x', x)}{\partial \zeta' \partial \zeta}\right|_{\eta'=\eta=0, \zeta'=\zeta=z} = D(z), \tag{13}$$

which are the same as the corresponding derivatives (8) and (9) in the primary trait space. Furthermore, apart from

$$\left.\frac{\partial^2 F(x', x)}{\partial \eta'^2}\right|_{\eta'=\eta=0, \zeta'=\zeta=z} = 4q_1' q_2' B(z), \tag{14}$$

all other second derivatives needed for equations (5) and (6) are 0 (appendix). It is worth noting that $B(z)$ in derivative (14) is the only quantity not determined by derivatives in the primary trait space that we need for the question of genetic versus stochastic polymorphism.

Branching and Randomization Disruptivity

For an equilibrium primary trait $\hat{z}$, the second derivative (8) gives the local curvature of invasion fitness regarded as a function of the mutant primary trait z'. When the second derivative is positive, $\hat{z}$ is a local fitness minimum, and we may use $A(\hat{z})$ as a measure of the strength of disruptive selection. If the equilibrium is convergence stable, evolutionary branching may take place (Metz et al. 1996; Geritz et al. 1998), and $A(\hat{z})$ is then a measure of the strength of selection for a local genetic dimorphism. For this reason, I will refer to $A(\hat{z})$ as the branching disruptivity at the equilibrium. The terminology will also be used for the same quantity in equation (12), which has a similar interpretation. Now, for a pure-strategy equilibrium in the space of extended strategies, the second derivative (14) measures the strength of selection for a local randomized dimorphism (i.e., a mutant strategy with nonzero η'). The strength depends on q_μ' and is greatest for a 50-50 randomization. I will refer to $B(\hat{z})$ as the randomization disruptivity at the equilibrium. The randomization disruptivity is the value of the second derivative (14) for

$q_1' = q_2' = 0.5$ and represents a natural comparison with the branching disruptivity (because, for $A = B$, Taylor expansion to second order shows that invasion fitness for each component of a local genetic dimorphism with primary traits $z_1' = -\eta'$ and $z_2' = \eta'$ will be the same as invasion fitness for a local randomized dimorphism with η', given equal weights on z_1' and z_2' in both cases). The branching and randomization disruptivities play a crucial role in the classification of equilibria.

Classification of Equilibria

For an equilibrium point in the primary trait space, the selection gradient $s(\hat{z})$ is 0. At a corresponding point $\hat{x} = (\hat{z}, 0, \hat{\rho})$ in the extended trait space, we see from equation (11) that all three components, S_ζ, S_η, and S_ρ, of the selection gradient are 0, so $\hat{x}$ is an equilibrium in the extended space. From equations (5), (6), and (12)–(14), the Jacobian and Hessian matrices at $\hat{x}$ have the nonvanishing elements

$$\mathbf{J}_{\zeta\zeta} = A(\hat{z}) + D(\hat{z}),\ \mathbf{J}_{\eta\eta} = 4\hat{q}_1\hat{q}_2 B(\hat{z}), \tag{15}$$

$$\mathbf{H}_{\zeta\zeta} = A(\hat{z}),\ \mathbf{H}_{\eta\eta} = 4\hat{q}_1\hat{q}_2 B(\hat{z}), \tag{16}$$

with all other matrix elements being 0. From the treatment above, regarding evolution in multidimensional trait spaces, properties of the Jacobian matrix indicate whether $\hat{x}$ is convergence stable in the extended space and properties of the Hessian matrix indicate whether there is stabilizing or disruptive selection at $\hat{x}$.

Let us consider a point $\hat{z}$ that is convergence stable in the primary trait space, and let us use a brief notation like A for $A(\hat{z})$. From equations (8) and (9), the point is convergence stable in the primary trait space when $A + D < 0$. For A negative, the point is uninvadable in the primary trait space, whereas for A positive it is a branching point. Looking now at the corresponding $\hat{x} = (\hat{z}, 0, \hat{\rho})$ in the extended space, with $-1 < \hat{\rho} < 1$, a classification in terms of branching and randomization disruptivities appears in figure 1 (see appendix for details). When A and B are both negative, the point $\hat{x}$ is uninvadable and strongly convergence stable in the extended space and is thus a CSS, since the Hessian and Jacobian matrices are negative semidefinite. When A is positive, the strategy $\hat{x}$ can be destabilized through evolutionary branching along the ζ direction, and when B is positive, it can be destabilized through the evolution of a nonzero η.

In simple situations, in the sense that invasion fitness of a mutant trait is a function of the mutant's arithmetic average success in interactions with residents, it turns out that $A = B$ will hold at an equilibrium $\hat{x}$ (see appendix), which is illustrated by the dashed line in figure 1. For these simple situations, either A and B are both negative, so that $\hat{x}$ is a CSS, or they are both positive, so that branching and the evolution of randomization can occur.

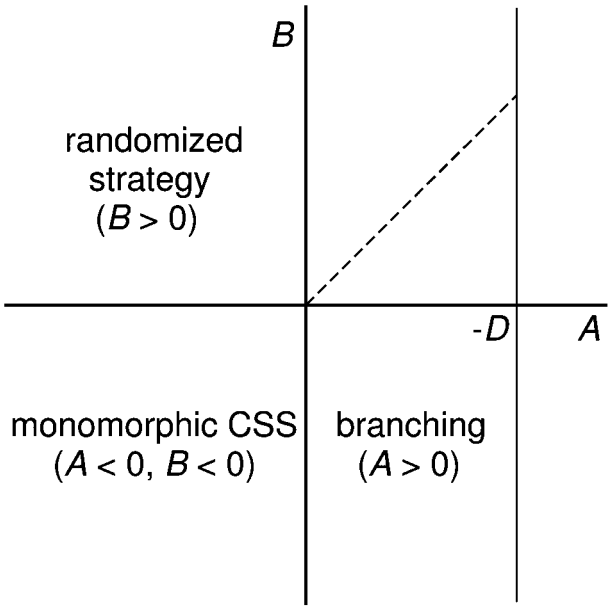

Figure 1: Criteria for continuous stability, evolutionary branching, and the evolution of a randomized strategy for a phenotypically monomorphic equilibrium $\hat{x} = (\hat{z}, 0, \hat{\rho})$ in the extended trait space. The axes give the branching disruptivity $A(\hat{z})$ and the randomization disruptivity $B(\hat{z})$, defined in equations (12) and (14). The point $\hat{z}$ is convergence stable in the primary trait space when $A + D < 0$, that is, to the left of the thin vertical line (it is assumed that $D < 0$). If A and B are both negative, $\hat{x}$ is a continuously stable strategy; if A is positive, there can be evolutionary branching; and if B is positive, a randomized strategy (with nonzero $\eta = (z_2 - z_1)/2$) can evolve. On the dashed line, branching and randomization disruptivities are equal.

Genetic Variation as a Cue for Phenotype Determination

The branching and randomization disruptivities characterize how selection would act on a phenotypic dimorphism with primary traits z_1 and z_2 that are close to an equilibrium $\hat{z}$. When the branching disruptivity is positive and greater than the randomization disruptivity (in the region below the dashed line in fig. 1), selection tends to favor genetic over random phenotype determination. The interpretation is that it is adaptively advantageous for an individual to use its heritable genotype as a cue for phenotype determination. It then follows that an individual's genotype contains information relevant to phenotype determination in a similar way as when information in an environmental cue is taken advantage of in adaptive phenotypic plasticity. On the other hand, if the randomization disruptivity is greater than the branching disruptivity (to

the left of the dashed line in fig. 1), it is better for an individual to randomize than to employ such a genetic cue.

Examples

An overview of the analysis of three examples appears in figure 2, based on the criteria using the branching and randomization disruptivities in figure 1. To investigate whether these criteria correctly predict the evolution of alternative phenotypes from a monomorphic state, I have run a number of individual-based simulations, using both single-locus asexual individuals and multilocus diploid individuals. The examples suggest that spatial variation in conditions, together with less-than-random dispersal of individuals over habitats, tends to favor branching over the evolution of randomization, whereas both large-scale temporal fluctuations in conditions and competition between relatives are circumstances that may favor the evolution of randomization over branching.

Spatially Variable Environment

The model is described in the previous section on spatial environmental variation. Invasion fitness is given by the logarithm of the leading eigenvalue of the population projection matrix for a mutant population that is small compared to a resident population. From the invasion fitness and its derivatives (appendix), we find that the points $\hat{x} = (z_0, 0, \hat{\rho})$ are equilibria and that

$$A = \frac{1-m}{m}\frac{\delta^2}{\sigma^4} - \frac{1}{\sigma^2},$$
$$B = \frac{\delta^2}{\sigma^4} - \frac{1}{\sigma^2}, \tag{17}$$
$$D = \frac{m-1}{m}\frac{\delta^2}{\sigma^4}.$$

Since $A + D = -1/\sigma^2$ is negative, the point z_0 is convergence stable in the primary trait space. Values of A and B are illustrated in figure 2 for random dispersal over patches ($m = 0.5$) and for a lower probability of migration ($m = 0.2$). Both branching and randomization disruptivities increase as the separation between patch optima (2δ) increases. For $m = 0.5$, A and B are equal, but for restricted migration, A is greater than B, and branching may be favored over the evolution of randomization.

To gain an intuitive understanding of why branching could be favored over randomization, note that for a genetically polymorphic population with $m < 0.5$, an individual's genotype may be regarded as a probabilistic cue

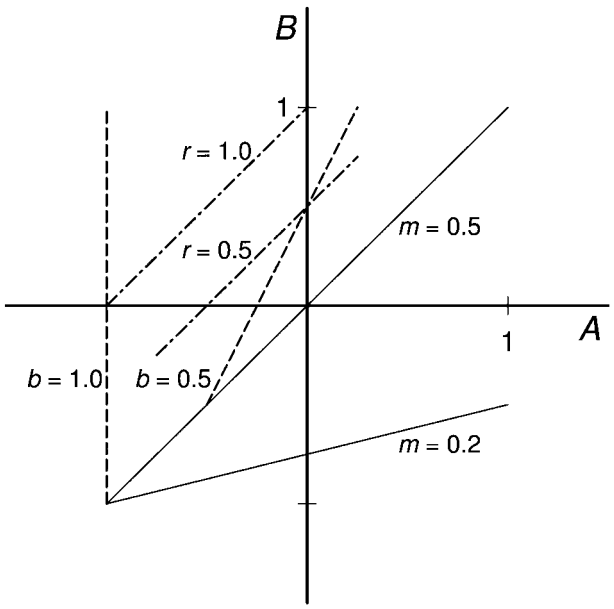

Figure 2: Lines illustrate branching and randomization disruptivities at phenotypically monomorphic equilibria $\hat{x} = (\hat{z}, 0, \hat{\rho})$ for the examples in the text. The solid lines refer to the example with spatially variable environment and show A and B as the parameter δ varies (determining the separation between primary trait optima in the two patches) for two different values of the probability m of migration ($0 < \delta^2 < 2$ for $m = 0.5$ and $0 < \delta^2 < 0.5$ for $m = 0.2$; $\sigma^2 = 1$). The dashed lines refer to the example with temporally variable environment and show A and B as the parameter δ varies (determining the separation between primary trait optima for recruits in the two temporal conditions) for two different values of the proportion b recruited each season ($0 < \delta^2 < 2$ for $b = 1$ and $0 < \delta^2 < 3$ for $b = 0.5$; $\sigma^2 = 1$). The dashed-and-dotted lines refer to the example with competition between relatives and show A and B as the parameter γ varies (giving the sharpness of the peak of the resource distribution) for two different values of the coefficient of relatedness r ($0 < \gamma < 1$ both for $r = 1$ and $r = 0.5$; $\sigma^2 = 1$).

of the coming selective environment because selection in the previous generations will have led to differences in genotype frequencies between the patches. For random dispersal over patches, the genotype would be an uninformative cue, whereas for the biologically unrealistic case of $m > 0.5$, the genotypic cue would be "misleading" by instead decreasing the chance that an individual develops the right phenotype for the coming selective environment.

For random dispersal over patches, branching and the evolution of randomization are equally possible, and one might expect that the mutations appearing will determine the outcome. This is illustrated by the individual-based simulations in figures 3 and 4. If mutational increments in the average primary trait $\zeta = q_1 z_1 + q_2 z_2$ tend to be larger than the increments in $\eta = (z_2 - z_1)/2$, corresponding to a positive genetic correlation between z_1 and z_2, genetic polymorphism is the more likely outcome (fig. 3*A*),

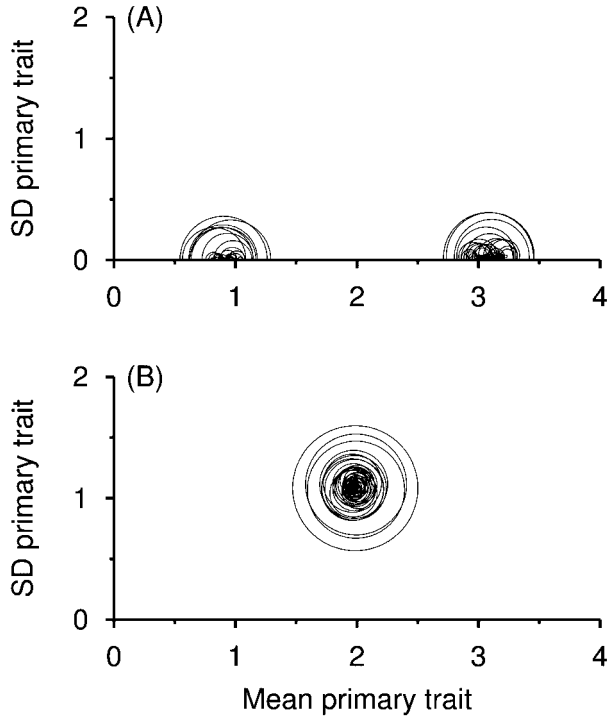

Figure 3: Bubble plots of genotype distributions in simulations for a spatially variable environment, with parameters such that both branching and evolution of a randomization are possible ($m = 0.5$, $z_0 = 2$, $\delta = 1.25$, $\sigma = 1$, leading to $A = B = 0.5625$). Individuals are haploid with a locus coding for $x = (\zeta, \eta, \rho)$. The mean primary trait, ζ, is on the horizontal axis, and the standard deviation, $s_z = [(1 - \rho^2)\eta^2]^{1/2}$, of the distribution given by x is on the vertical axis. A genotype is represented by a bubble with center at (ζ, s_z) and area proportional to the genotype frequency. *A*, Mutational increments in ζ have a wider distribution (SD = 0.05) than increments in η (SD = 0.005), leading to the evolution of genetic polymorphism in the form of two clusters of genotypes, each coding for an essentially fixed primary trait. *B*, The distribution of mutational increments is instead somewhat wider for η than for ζ (SD = 0.025 for η and 0.01 for ζ), leading to the evolution of a single cluster of genotypes coding for randomized strategies. For each simulation, the population size was 10^4, the mutation rate 0.001, and the starting point a genotypically monomorphic population at $\hat{x} = (z_0, 0, 0)$. The situation after 4×10^5 generations is shown.

whereas a negative genetic correlation between z_1 and z_2 favors the evolution of randomization (fig. 3*B*). Similar conclusions hold for situations with more complex genetics than the asexual case in figures 3 and 4. For instance, figure 5 illustrates how a genetic polymorphism for the two-patch model can be implemented in diploid organisms through a nonlinear genotype-phenotype mapping, corresponding to a threshold trait (Roff 1996). In figure 5, most of the genetic variation is present as a major effect at one of the loci, as can be inferred from the trimodal distribution of additive effects (fig. 5*A*), but polygenically determined threshold traits are also feasible mechanisms for genetic determination of the phenotype (simulations not shown).

I have performed a number of additional simulations, for instance, starting away from the convergence stable $\hat{z}$ in the primary trait space, and the overall impression is that the criteria in figure 1 are useful in predicting the evolutionary outcome. A question of general interest is how strongly a situation with $A > B$ will bias the outcome toward genetic polymorphism. According to the reasoning above, where an individual's genotype was considered a cue of the coming selective environment, one might expect genetic polymorphism to be adaptively superior for $A > B$ and to be adaptively inferior for $A < B$, so that these inequalities would sharply determine the evolutionary outcome. This reasoning has some validity, but in practice there can be quite noticeable effects of the mutational process. As an illustration, when I repeated the simulation in figure 3*B* with the probability of migration decreased from $m = 0.5$ to $m = 0.45$ (so that $A = 0.9097$ and $B = 0.5625$), random phenotype determination still

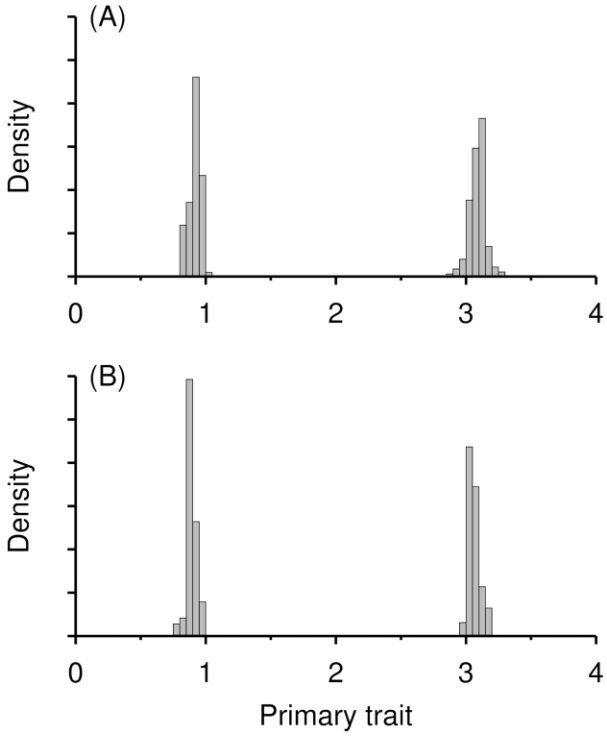

Figure 4: Histograms of realized primary traits for the genotype distributions shown in figure 3. *A*, The bimodal distribution is a consequence of genetic polymorphism, with the two modes corresponding to the genotype clusters in figure 3*A*. The rather similar distribution in *B* is instead a consequence of randomization between two primary traits z_1 and z_2 by genotypes in the single cluster in figure 3*B*.

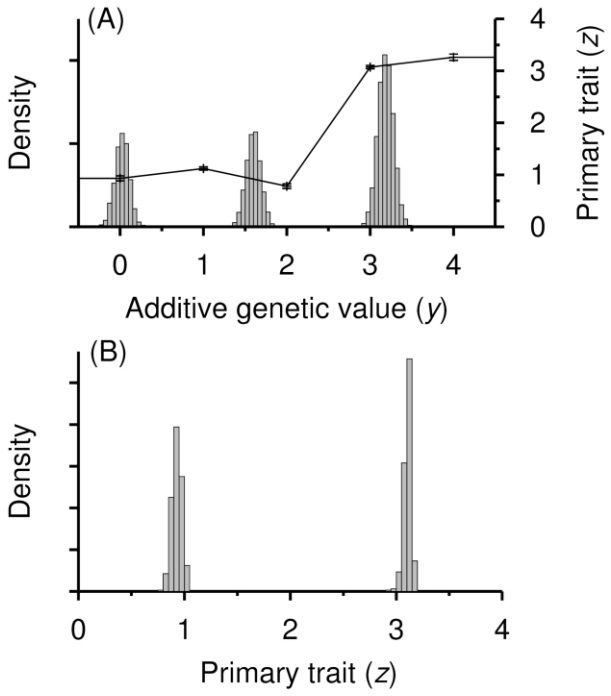

Figure 5: Multilocus genetic polymorphism. A population of 10^4 diploid hermaphroditic individuals, with random mating within two habitat patches, was simulated using the same model parameters as in figures 3 and 4. An individual had five unlinked loci coding for an "additive genetic value" y. An additional unlinked locus coded for five parameters of a "genotype-phenotype mapping," given by the value of the primary trait $z(y)$ for $y = 0, 1, 2, 3, 4$, with z being defined by linear interpolation between these values and constant outside the range. *A*, Histogram of y (*left axis*), together with the average mapping from y to z (*right axis*; the error bars give $\pm$SD of the five parameters). *B*, Histogram of the resulting distribution of z. The starting point was a genetically monomorphic population with each y allele contributing 0.2 (giving a total y of 2), and with $z(y) = y$ for the mapping parameters. The figure shows the situation after 4×10^5 generations.

evolved and remained for around 2×10^5 generations, although it was then gradually replaced by genetic determination.

Temporally Variable Environment

During a season, either of two possible population-wide environmental conditions prevails, each with a probability of one-half. The environment influences survival in a similar way as in the previous example, with $\alpha_i(z)$ in equation (1) giving juvenile survival in condition i as a function of a primary trait z. A positive proportion b of the established adults are replaced by recruited juveniles each season, whereas the remaining adults survive, so for $b < 1$, generations overlap. The model is a simple version of the so-called lottery model, which has been used to study evolution in fluctuating environments (e.g., Chesson and Warner 1981; Seger and Brockmann 1987).

From invasion fitness and its derivatives (appendix), we find that the points $\hat{x} = (z_0, 0, \hat{\rho})$ are equilibria and that

$$A = b(1 - b)\frac{\delta^2}{\sigma^4} - \frac{b}{\sigma^2},$$

$$B = b\frac{\delta^2}{\sigma^4} - \frac{b}{\sigma^2}, \tag{18}$$

$$D = -b(1 - b)\frac{\delta^2}{\sigma^4}.$$

Since $A + D = -b/\sigma^2$ is negative, the point z_0 is convergence stable in the primary trait space. Values of A and B are illustrated in figure 2 for total replacement of the population each season ($b = 1$) and for a case with overlapping generations ($b = 0.5$). The randomization disruptivity increases as the separation between the primary trait optima (2δ) for the two temporal conditions increases. With nonoverlapping generations ($b = 1$), the branching disruptivity is always negative, preventing the evolution of a genetic polymorphism. We also see from equation (18) that the branching disruptivity A will be smaller than the randomization disruptivity B for any $b > 0$, favoring the evolution of a randomized strategy. Nevertheless, for small b and sufficiently large values of δ, evolutionary branching is a possibility, provided that it is mutationally favored. These results were confirmed by simulations and are in general agreement with the conclusions of Seger and Brockmann (1987).

Competition between Relatives

There are a large number of patches, each of which can support a deme of two haploid individuals. An empty patch is colonized by two random dispersers, and the resulting deme may persist for a number of generations, with random genetic representation of the deme members in the next generation, leading to increasing average within-deme relatedness with deme age. A deme ends by the production of dispersing offspring, which go into a dispersal pool and then attempt to colonize empty patches. The expected relatedness, r, between the deme members at the time of production of dispersing offspring is a function of the probability that the deme ends a given season, so r is determined by a model parameter (r will be 0 if a deme is certain to end the first season, and r is larger for smaller probability of ending; see the appendix).

The deme members compete for resources available for the production of dispersing offspring. The number of dispersing offspring of an individual with primary trait z'

sharing the patch with an individual with z is proportional to

$$\alpha(z', z) = g(z')\frac{1}{1 + c(z', z)}, \tag{19}$$

where $g(z')$ is a resource distribution function and $c(z', z)$ a coefficient of competition. The resource distribution,

$$g(z) = \begin{cases} 1 - \frac{1}{2}\gamma(z - z_0)^2 & \text{if } |z - z_0| \le \sqrt{2/\gamma} \\ 0 & \text{otherwise} \end{cases}, \tag{20}$$

has a maximum at primary trait z_0, and the positive parameter γ gives the sharpness of the peak of the distribution. The competition coefficient is given by

$$c(z', z) = \exp\left[-\frac{(z' - z)^2}{2\sigma^2}\right], \tag{21}$$

corresponding to symmetric competition. We can note that $\alpha(z, z) = g(z)/2$, so the deme members share the available resource equally when $z' = z$.

From invasion fitness and its derivatives (appendix), we find that the points $\hat{x} = (z_0, 0, \hat{\rho})$ are equilibria and that

$$\begin{aligned} A &= -\gamma + (1 - r)\frac{1}{2\sigma^2}, \\ B &= -\gamma + (1 + r)\frac{1}{2\sigma^2}, \\ D &= -(1 - r)\frac{1}{2\sigma^2}. \end{aligned} \tag{22}$$

Since $A + D = -\gamma$ is negative, the point z_0 is convergence stable in the primary trait space. Values of A and B are illustrated in figure 2 for the limit of full relatedness ($r = 1$) and for a case with smaller relatedness ($r = 0.5$). From equation (22), the randomization disruptivity is positive when the competitive effects vary more rapidly with the primary trait difference than does the resource distribution (small σ^2 or small γ). We also see that relatedness tends to favor the evolution of randomization over branching ($A < B$). For full relatedness, we have $A < 0$, so that branching is not possible (cf. fig. 2). On the other hand, for unrelated deme members ($r = 0$), the branching and randomization disruptivities are equal (and would appear on the diagonal in fig. 2).

A useful interpretation of the decrease in branching disruptivity with increasing r is that an individual cannot avoid competing with a relative by having a different primary trait when that trait is genetically determined (in a heritable manner), and this phenomenon has been studied by Day (2001). It is also worth noting that relatedness promotes phenotypic polymorphism under competition because an individual helps both itself and its relative by having a trait value that reduces competition. For instance, for parameter values $\gamma > 1/2\sigma^2$, there is never any branching disruptivity, since A will be negative for any $r \ge 0$, so one would perhaps not expect alternative phenotypes to evolve. However, alternative phenotypes can still evolve if r is large enough to make B positive. Thus, trait-mediated competition between relatives promotes the evolution of alternative phenotypes by way of character displacement.

Discussion

Studying the evolution of phenotype determination in terms of the signs and relative magnitudes of the branching and randomization disruptivities, as illustrated in figure 1, has the advantage of bringing genetic and random determination into a single analysis. This was achieved by limiting consideration to situations close to a phenotypically monomorphic equilibrium. The approach can be regarded as an extension of the analysis of evolutionary branching in adaptive dynamics (Metz et al. 1996; Geritz et al. 1998), which gives a unified perspective on the evolution of genetic polymorphism. Such conceptual unification is helpful and important, but when alternative phenotypes have evolved away from a previously monomorphic state, there is in principle a new situation that may require a separate analysis. In addition, phenotypic polymorphism could come about in different ways, such as by modification of a previously existing polymorphism or by mutations of large effect or drastic changes in environments. Nevertheless, the individual-based simulations I performed showed that an analysis in terms of branching and randomization disruptivities can often succeed in predicting the evolutionary outcome.

One important feature of my analysis is that cases are split into those where a genotype can function as an advantageous cue for determination of alternative phenotypes and those where such a genetic cue instead would be disadvantageous compared to random determination, as well as the intermediate cases where genetic and random determination are neutral relative to each other. This kind of division has general validity and makes the point that genetic determination of the phenotype is conceptually parallel to environmental determination, for which issues of the accuracy and other statistical properties of environmental cues are fundamental. In fact, it could be helpful to regard genetic determination of alternative phenotypes as a special case of adaptive phenotypic plasticity.

It is common to think of alternative phenotypes as threshold traits or developmental switches (Roff 1996;

Lynch and Walsh 1998; West-Eberhard 2003), where trait expression is switched when the value of an internal liability passes a threshold. The liability might be the concentration of a hormone, which in turn could be influenced by genes, environmental cues, or random effects. If we ask in which way such a phenotype-determining mechanism can be an adaptation, there is the question of the perfection of alternatives being switched between, on the one hand, and the question of when to switch, on the other hand. For the latter, we can regard random variation in liability as adaptive if suitable phenotype frequencies are produced in this way, and any further improvement in the fit of phenotype to selective circumstances achieved by a switching mechanism is then also a possible adaptation. Environmental cues are, of course, candidates to be employed in such better-than-random switching mechanisms, but, as we have seen, heritable genetic variation is also a candidate. Thus, the alternative phenotypes of a multiple-niche polymorphism, together with the switching mechanism, are elements of an adaptation to spatially varying conditions. For genetic switching, spatial variation in phenotype frequencies from recent selection, unless obliterated by extensive gene flow, will be the source of information in the genetic cue. There is some similarity to the idea of local adaptation when gene flow is limited, but the difference is that the alternative phenotypes and the switching mechanism make up one adaptation to a range of environments rather than that each phenotype separately is an adaptation to a single environment. So, in the context of an evolved switching mechanism, like a nonlinear genotype-phenotype mapping, it becomes natural to view genetic determination of the phenotype as potentially an adaptation, falling within the general framework of phenotypic plasticity.

Several decades ago, ecological geneticists were concerned with providing an adaptive interpretation of genetic polymorphism (e.g., Dobzhansky 1951; Cain and Sheppard 1954; Fisher 1958; Ford 1965, 1971), but this interest seems to have declined in recent times. Possibly, the currently dominating idea of the gene as the fundamental unit of selection (Dawkins 1976) has been responsible for the decline because that idea might seem to be in conflict with the notion that genetic variation could play a role in an adaptive device for the organism. In ecological genetics, a traditional but also controversial view was that genetic polymorphism can be adaptive if it renders an organism efficient over a range of environments (Dobzhansky 1951; Cain and Sheppard 1954), corresponding to the modern concept of multiple-niche polymorphism. When examining this idea, Fisher (1958) traced it to the discussion by Darwin (1859) of divergence of character under natural selection and went on to suggest that a "theory of games," with randomized strategies as one feasible evolutionary outcome, could be a way to understand species interactions, such as those between predators and prey. Fisher's treatment is significant as apparently the first explicit suggestion of an evolutionary game theory. It is also clear that Fisher (1958) interpreted balanced polymorphism as an evolved randomized strategy. Ford (1965) had a similar view of genetic polymorphism as a means to regulate phenotype frequencies, and he argued that it would usually be preferable as a switching device, compared to environmental cues, perhaps because he felt that environmental cues would result in excessive fluctuations in phenotype frequencies. It then appears that traditional ecological genetics did not view genetic determination of the phenotype as essentially different from random determination. It was only with the subsequent analysis of bet hedging that this distinction became clear (Seger and Brockmann 1987), although the interpretation of genetic polymorphism as a special case of phenotypic plasticity, which I have argued for here, was not made.

My treatment did not deal directly with environmental phenotype determination, which is possibly the most widespread and important system, because I wanted a simple and focused treatment of genetic and random determination. Nevertheless, environmental determination could be included in the same kind of analysis. By regarding reaction norms as multidimensional "pure strategies," one can investigate whether some component of such a pure strategy would be exposed to disruptive selection, possibly leading to evolutionary branching and genetic polymorphism or, alternatively, whether a randomized reaction norm might evolve. A similar kind of approach has recently been used to study environmental sex determination (Van Dooren and Leimar 2003; Leimar et al. 2004). Factors such as the accuracy of environmental cues and the degree of correlation between environmental cue values observed by different individuals, together with other aspects of the ecological situation, will determine if some component of a reaction norm would be exposed to branching or randomization disruptivity.

Of the examples I used, the effect of competition between relatives on the evolution of alternative phenotypes and on phenotype determination seems not to have been modeled previously. The conclusion that random determination should be favored was, however, reached by Moran (1992), and Day (2001) found that relatedness decreased branching disruptivity in situations where relatives compete. In practice, compared to purely random determination, other mechanisms based on phenotypic cues like relative size or age might often be more efficient in reducing competition between interacting relatives and could thus be expected instead.

On the basis of the simulations for the examples, it is clear that the mutational process can have a marked in-

fluence on the system of phenotype determination that evolves. For instance, if there is little additive genetic variation in the degree of random phenotype determination, genetic determination might instead be the evolutionary outcome. A similar point was made by Van Dooren and Leimar (2003) and Leimar et al. (2004) in the context of sex determination. The explanation is that genetic and random cues can sometimes serve equally or nearly equally well for phenotype determination. As a consequence, in situations where branching and randomization disruptivities are equal, it seems appropriate to regard a genetic determination of the phenotype as a randomization device. Nevertheless, considering general fitness functions, the theory developed here shows that those situations are marginal. Using heritable genetic variation for phenotype determination will generally either be selectively favored and thus be a case of adaptive genetic determination of the phenotype or instead have a selective cost as compared to random determination.

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