Adaptive Dynamics in Allele Space: Evolution of Genetic Polymorphism by Small Mutations in a Heterogeneous Environment

Author(s): Eva Kisdi and Stefan A. H. Geritz

Source: *Evolution*, Vol. 53, No. 4 (Aug., 1999), pp. 993-1008
Published by: Society for the Study of Evolution
Stable URL: http://www.jstor.org/stable/2640805
Accessed: 21-10-2015 00:59 UTC

EVOLUTION
INTERNATIONAL JOURNAL OF ORGANIC EVOLUTION
PUBLISHED BY
THE SOCIETY FOR THE STUDY OF EVOLUTION

Vol. 53 August 1999 No. 4

*Evolution*, 53(4), 1999, pp. 993–1008

# ADAPTIVE DYNAMICS IN ALLELE SPACE: EVOLUTION OF GENETIC POLYMORPHISM BY SMALL MUTATIONS IN A HETEROGENEOUS ENVIRONMENT

ÉVA KISDI[1,2,3] AND STEFAN A. H. GERITZ[2]
[1]*Department of Genetics, Eötvös University, 1088 Budapest, Múzeum krt. 4/A, Hungary*
[2]*Department of Zoology, University of Maryland, College Park, Maryland 20742*

*Abstract.*—We demonstrate how a genetic polymorphism of distinctly different alleles can develop during long-term frequency-dependent evolution in an initially monomorphic diploid population, if mutations have only small phenotypic effect. As a specific example, we use a version of Levene's (1953) soft selection model, where stabilizing selection acts on a continuous trait within each of two habitats. If the optimal phenotypes within the habitats are sufficiently different, then two distinctly different alleles evolve gradually from a single ancestral allele. In a wide range of parameter values, the two locally optimal phenotypes will be realized by one of the homozygotes and the heterozygote, rather than by the two homozygotes. Unlike in the haploid analogue of the model, there can be multiple polymorphic evolutionary attractors with different probabilities of convergence. Our results differ from the population genetic models of short-term evolution in two aspects: (1) a polymorphism that is population genetically stable may be invaded by a new mutant allele and, as a consequence, the population may fall back to monomorphism, (2) long-term evolution by allele substitutions may lead from a population where polymorphism is not possible into one where polymorphism is possible.

*Key words.*—Adaptive dynamics, ESS, evolutionary branching, frequency dependent selection, genetic polymorphism, Levene model, soft selection.

Received April 28, 1998. Accepted February 15, 1999.

Frequency-dependent selection arises under a wide variety of ecological situations such as multiple-niche environments, resource competition, and predation. On a phenotypic level, frequency dependent selection is often analyzed by searching for evolutionarily stable strategies (ESSs; Maynard Smith 1982) or for evolutionarily stable coalitions of coexisting strategies (Brown and Vincent 1987a,b, 1992; Brown and Pavlovic 1992) as the possible final states of evolution. Models focusing on the dynamics of how traits change in time, rather than on the static outcome only, most often investigate directional evolution of continuous traits (e.g., Hofbauer and Sigmund 1990; Marrow et al. 1992, 1996; Abrams et al. 1993a; Vincent et al. 1993; Matsuda and Abrams 1994a,b; Dieckmann et al. 1995; Dieckmann and Law 1996; Abrams and Matsuda 1997; Law et al. 1997). These models are largely compatible with quantitative genetic models (Charlesworth 1990; Iwasa et al. 1991; Taper and Case 1992; Abrams et al. 1993a,b; Taylor 1996; Taylor and Day 1997).

The attractors of directional evolution need not be ESSs (Eshel 1983; Taylor 1989; Nowak 1990; Christiansen 1991; Abrams et al. 1993a; Vincent et al. 1993; Eshel et al. 1997). Assuming clonal inheritance, Metz et al. (1996) and Geritz et al. (1997, 1998) have shown that the non-ESS attractors are evolutionary branching points, at which two coexisting strategies evolve from a single ancestral strategy by small mutational steps (similar results were obtained independently by Eshel et al. 1997).

The evolution of polymorphism at a branching point proceeds as follows. As long as the monomorphic population is away from the branching point, a mutant somewhat nearer the branching point can invade and replace the resident. When directional evolution has arrived at the neighborhood of a branching point, the invading mutant does not replace the former resident anymore; instead the two phenotypically similar strategies form a protected dimorphism. Once dimorphic, the population undergoes disruptive evolution, during which the two strategies grow further and further apart. The emergence of two widely distinct strategies by small evolutionary steps in an initially monomorphic population can be envisaged as branching on the evolutionary tree.

Protected polymorphism and disruptive selection near a branching point have been noted in a number of specific models (Christiansen and Loeschcke 1980; Van Tienderen

[3] Present address: Department of Mathematics, University of Turku, FIN-20014 Turku, Finland; E-mail: eva.kisdi@utu.fi.

and De Jong 1986; Christiansen 1991; Brown and Pavlovic 1992; Abrams et al. 1993a). Specific examples of evolutionary branching have been investigated by Metz et al. (1992, 1996), Doebeli (1996a), Doebeli and Ruxton (1997), Meszéna et al. (1997), Geritz et al. (1998, 1999), Doebeli and Dieckmann (in press), Geritz and Kisdi (in press), Mathias and Kisdi (in press), and Meszéna and Metz (in press). The evolutionary attractors corresponding to fitness minima found, for example, by Cohen and Levin (1991), Ludwig and Levin (1991), Vincent et al. (1993), Doebeli (1996b), and Law et al. (1997) are evolutionary branching points.

An important limitation of the adaptive dynamics framework used by Metz et al. (1996) and Geritz et al. (1997, 1998) for modeling evolutionary branching is the assumption of haploid or clonal inheritance. In this paper we investigate how this framework can be applied to diploid sexually reproducing organisms. Because in diploid populations alleles (rather than phenotypes) undergo mutation and are passed from one generation to the next, we model evolution in allele space by keeping track of the full allelic composition of the population. Evolutionary branching in allele space produces two alleles with distinctly different phenotypic effects, via a series of small mutations in an initially monomorphic population. Genetic polymorphisms of more than two alleles may also develop by repeated branching.

Specifically, we address the following questions. First, does genetic polymorphism evolve by evolutionary branching under similar ecological conditions where phenotypic adaptive dynamics leads to branching? Second, what are the qualitative differences between evolution in haploid and diploid populations? To tackle these questions, we develop a continuous version of Levene's soft selection model (Levene 1953) as an example because of its relative simplicity and because of its well-known population genetics (e.g., see Roughgarden 1979; Maynard Smith 1989). The model of Levene (1953) as well as related models (reviewed by Felsenstein 1976; Hedrick et al. 1976) assume a fixed number of possible alleles (e.g., only two), such that each allele is present in the initial population. Here we assume an initially monomorphic population and a continuum of potential allele types. New alleles arise by mutations of small phenotypic effect. Fitnesses are defined as functions of the phenotype, and the phenotype, in turn, is determined by the alleles additively.

The difference between the classic Levene-type models and our approach is best captured by the notion of internal and external stability (Eshel 1996 and references therein). The classic models investigate short-term evolution in terms of allele frequency changes and internal stability of population genetic equilibria of a given number of alleles. In contrast, we model long-term evolution by allowing new mutant alleles to appear in the population, which may go to fixation, or may coexist with previously present alleles. Long-term evolutionary dynamics may lead to evolutionary branching and thereby to the development of genetic polymorphism with distinctly different alleles through a series of small mutations. The final outcome is an externally stable state, that is, an evolutionarily stable polymorphism that cannot be invaded by any mutant. An analogous, but haploid, model has been investigated by Geritz and Kisdi (in press).

After the description of the model, we first investigate evolution assuming particular values for the model parameters, by generalizing the adaptive dynamics framework of Metz et al. (1996) and Geritz et al. (1997, 1998) to diploid sexual populations. This illustrates evolutionary branching in allele space and reveals the possibility of several evolutionarily stable polymorphisms under the same model parameters. Second, we perform a bifurcation analysis to see how the number and properties of evolutionary attractors depend on the parameters of the model. Third, we show that if there are multiple polymorphic attractors, the probability of evolving to one or another may be markedly different. Fourth, we compare our results with previous investigations of the Levene model, in particular with Hoekstra et al. (1985). Adaptive dynamics in haploid and in diploid populations are contrasted in the Discussion.

## The Model

Consider a population of an annual organism in an environment consisting of two habitats or patches. Within each patch, there is first a period of selection, followed by nonselective competition. During selection, the probability of survival is a Gaussian function of a continuous phenotypic trait, $x$. The patches have different optimal phenotypes, $m_1$ and $m_2$, respectively, but have the same width of the fitness function, $\sigma$. The survival probability of an individual with phenotype $x$ is thus:

$$f_1(x) = \alpha_1 \exp\left(-\frac{(x - m_1)^2}{2\sigma^2}\right) \tag{1a}$$

in patch 1, and

$$f_2(x) = \alpha_2 \exp\left(-\frac{(x - m_2)^2}{2\sigma^2}\right) \tag{1b}$$

in patch 2, where $\alpha_1$ and $\sigma_2$ are arbitrary constants of proportionality. Without loss of generality, we assume that $m_1 = -d/2$ and $m_2 = d/2$, where $d$ is the difference between the optimal phenotypes.

During the competition phase, a fixed number of adults survive within each patch, such that a fraction $c_1$ of the adult population is recruited from the first patch and the remaining fraction $c_2 = 1 - c_1$ from the second patch ("soft selection"; Levene 1953). Offspring are produced by random mating in the entire population and are distributed over the patches randomly.

The trait under selection, $x$, is determined by a single locus with a continuum of possible alleles. Each allele is represented by the phenotype of the corresponding homozygote. To avoid excessive notation, we use the same symbol, $x$, to denote an allele and to denote the phenotype of an individual homozygous for this allele. The alleles act additively on the phenotype, that is, the phenotype of a heterozygote with alleles $x_j$ and $x_k$ is $(x_j + x_k)/2$. Notice that this does not imply additive fitnesses, because the fitness functions (eqs. 1a,b) are nonlinear.

The alleles may undergo mutations of small but finite (i.e., not infinitesimally small) phenotypic effect. In the analysis of the model, we assume that evolution is mutation limited, that is, mutations occur infrequently such that a mutant allele

either has been excluded or has spread and the population has reached its equilibrium by the time the next mutant comes along. The latter assumption greatly facilitates the analysis, but it is clearly unrealistic: One would expect some variation to be maintained by mutation-selection balance. To test the robustness of the model, we performed simulations of the evolutionary process, in which we relaxed the assumption of mutation limitation. As we demonstrate below, the model predictions are robust with respect to variation due to mutations.

## Adaptive Dynamics

In this section we investigate the dynamics of evolution for particular parameter values ($d/\sigma$ and $c_1$). To facilitate the analysis, we first recapitulate the standard equations for allele frequency change in the Levene model (see Levene 1953; Roughgarden 1979, p. 232). If alleles $x_1, \ldots, x_n$ are present with frequencies $p_1, \ldots, p_n$, the frequency of allele $x_j$ changes from one generation to the next according to:

$$\Delta p_j = p_j\left[c_1\frac{\sum_{k=1}^{n} p_k U_{jk}^{(1)}}{\bar{U}^{(1)}} + c_2\frac{\sum_{k=1}^{n} p_k U_{jk}^{(2)}}{\bar{U}^{(2)}} - 1\right], \qquad (2)$$

where $U_{jk}^{(i)} = f_i[(x_j + x_k)/2]$ is the fitness of genotype $x_jx_k$ in patch $i$ ($i = 1, 2$), and $\bar{U}^{(i)} = \Sigma_j \Sigma_k p_j p_k f_i[(x_j + x_k)/2]$ is the average fitness within patch $i$. The marginal fitness of allele $x_j$ across the entire population is:

$$c_1\frac{\sum_{k=1}^{n} p_k U_{jk}^{(1)}}{\bar{U}^{(1)}} + c_2\frac{\sum_{k=1}^{n} p_k U_{jk}^{(2)}}{\bar{U}^{(2)}}. \qquad (3)$$

### *Evolution in Monomorphic Populations*

Consider a rare mutant allele, $y$, in a resident population that is otherwise monomorphic for allele $x$. As long as the frequency of the mutant allele is negligible, the marginal fitness of the mutant simplifies from equation (3) to:

$$W_x(y) = c_1\frac{f_1\left(\frac{x+y}{2}\right)}{f_1(x)} + c_2\frac{f_2\left(\frac{x+y}{2}\right)}{f_2(x)}. \qquad (4)$$

If $W_x(y)$ is greater than one, the mutant allele can spread; otherwise it is excluded from the population.

A convenient way to summarize which mutant allele can invade a monomorphic resident population is to construct a so-called pairwise invasibility plot, which indicates for each pair of resident and mutant alleles whether $W_x(y)$ is greater or smaller than one (Van Tienderen and De Jong 1986; Metz et al. 1992, 1996; Kisdi and Meszéna 1993, 1995; Geritz et al. 1997, 1998). The top panels of Figure 1 show the pairwise invasibility plots obtained by evaluating equation (4) for all pairs of resident and mutant alleles ($x$ and $y$) for two different sets of parameter values. In the dotted areas $W_x(y) > 1$ such that the mutant can invade, whereas in the clear areas $W_x(y) < 1$ such that the mutant dies out. Because the resident allele neither spreads nor vanishes ($W_x[x] = 1$), the main diagonal ($y = x$) is one of the border lines between the "invasion" ($W_x[y] > 1$) and "noninvasion" ($W_x[y] < 1$) areas.

If a mutant allele invades, it may either substitute the original resident or it may form a polymorphism with the original allele. A pair of alleles forms a protected polymorphism if both of them can spread when rare, that is, if

$$W_x(y) > 1 \quad \text{and} \quad W_y(x) > 1. \qquad (5)$$

These pairs can be visualized by taking the mirror image of the pairwise invasibility plot along its main diagonal and superimposing the mirror image on the original: The area where the "invasion" parts of the mirror image and the original overlap gives the set of possible protected polymorphisms (Fig. 1, bottom panels). All two-allele polymorphisms in this model are protected (see Appendix 2).

If mutations are of small phenotypic effect ($|y - x| \leq \delta$), a mutant cannot form a polymorphism with the resident unless the population is in the neighborhood of $x^*$, the so-called evolutionarily singular allele (Fig. 1). As long as the population is away from $x^*$, invasion implies allele substitution and recurrent allele substitutions result in directional evolution. In both examples shown in Figure 1, directional evolution proceeds toward the singular allele $x^*$.

Geritz et al. (1998) provide a full classification of possible types of evolutionary singularities together with the dynamic characteristics of evolution taking place nearby. For the present purposes, we consider only two properties of the singularities: (1) convergence stability, that is, whether the singularity is an attractor of evolution proceeding by a series of allele substitutions (Eshel 1983; Taylor 1989; Christiansen 1991); and (2) local evolutionary stability, that is, whether a population monomorphic for the singular allele is resistant to invasions by nearby mutants.

The top panel of Figure 1a shows a pairwise invasibility plot when the two patches are similar in their respective optimal phenotypes ($d/\sigma = 1$, $c_1 = 0.5$); $x^* = 0$ is an evolutionarily stable allele, because there is no mutant that could invade a population that is monomorphic for $x^*$. Notice that in the neighborhood of $x^*$ there are pairs of alleles that can form protected polymorphisms (Fig. 1a, bottom panel), so that a population evolving towards $x^*$ may become polymorphic before it reaches the singularity. However, this polymorphism can be invaded by alleles still nearer to $x^*$ and therefore will eventually be resolved at the evolutionarily stable allele (Eshel et al. 1997; Geritz et al. 1998).

If the optimal phenotypes within the two patches are more different, the convergence stable singularity lacks evolutionary stability, that is, it is a branching point. In Figure 1b ($d/\sigma = 3$, $c_1 = 0.5$), a population that is monomorphic for $x^* = 0$ can be invaded by both smaller and larger mutants and the mutant allele forms a protected polymorphism with the original resident. The population therefore necessarily becomes polymorphic in the neighborhood of $x^*$. In this polymorphic population, only mutants outside the two resident alleles can invade and invasion is followed by the elimination of the allele in the middle. Repeated invasions of mutants thus give rise to a series of polymorphisms of two increasingly distinct alleles (Eshel et al. 1997; Geritz et al. 1998). Simulated evolutionary trees with branching are shown in Figure 2.

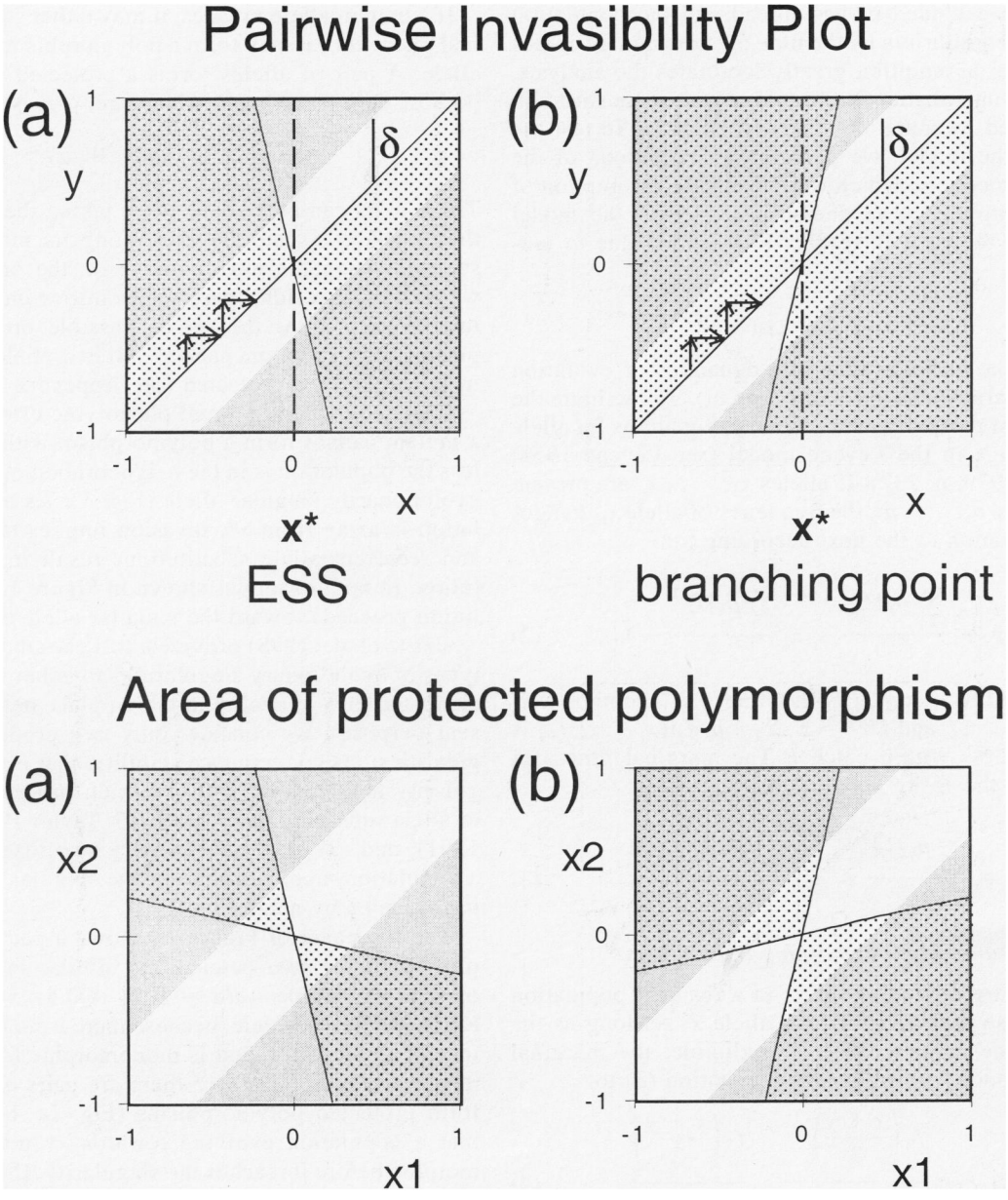
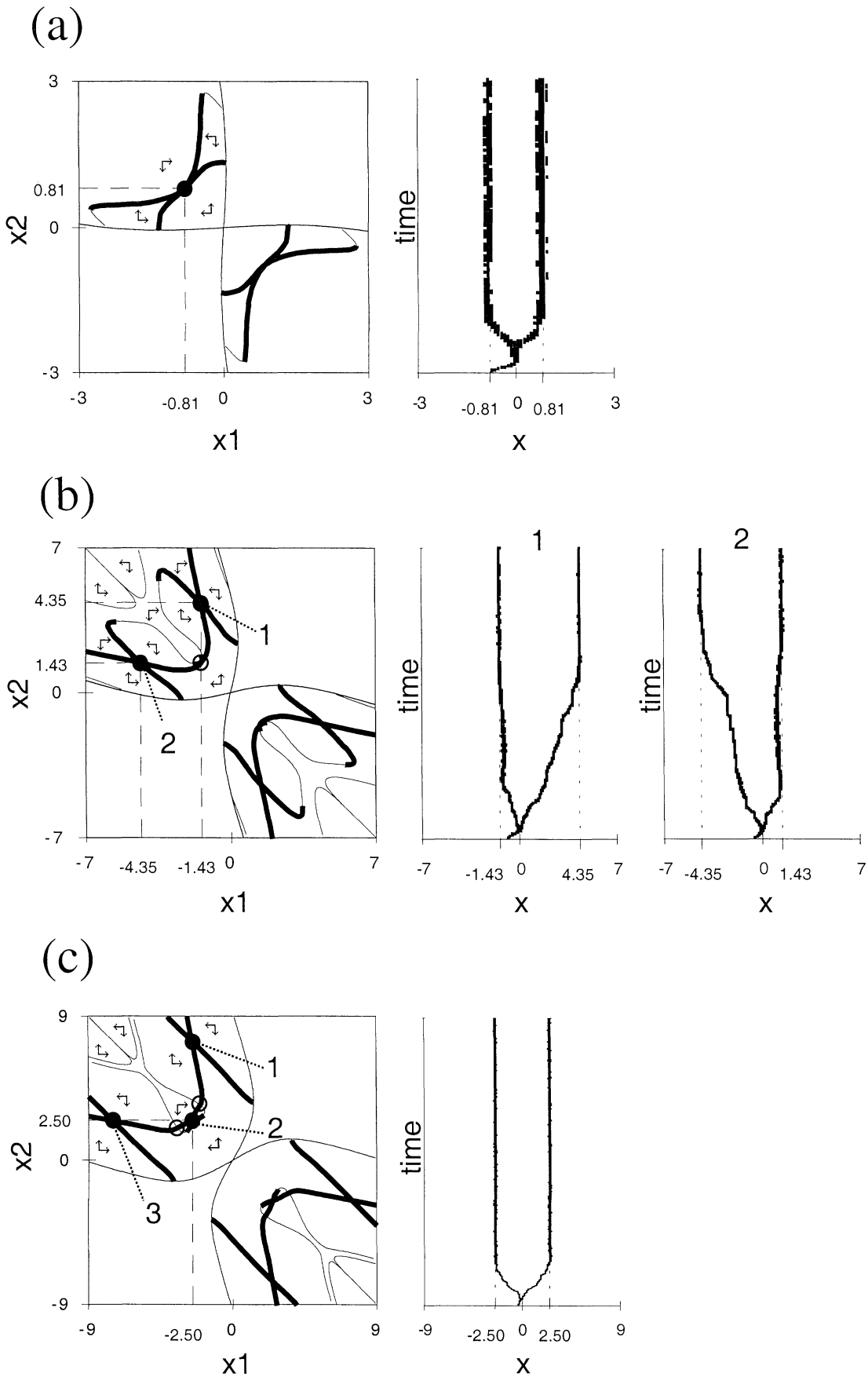



FIG. 1. Pairwise invasibility plots (top) and the area of protected polymorphism (bottom) for two sets of parameters. Inside the dotted areas of the pairwise invasibility plots the mutant allele ($y$) can invade the resident ($x$), otherwise the mutant dies out. The arrows indicate the substitution of the original resident by its mutant. The light band along the diagonal shows which mutants are feasible with small mutations ($|y - x| \leq \delta$). In the bottom panels, dotted areas denote the allele pairs ($x_1$, $x_2$) which form a protected polymorphism. (a) $d/\sigma = 1$, $c_1 = 0.5$; $x^* = 0$ is a convergence stable ESS. (b) $d/\sigma = 3$, $c_1 = 0.5$; $x^* = 0$ is convergence stable, but evolutionarily unstable, that is, it is a branching point.

→

FIG. 2. Isocline plots and corresponding simulated evolutionary trees. In the isocline plots, horizontal (vertical) arrows indicate in which direction the allele $x_1$ ($x_2$) evolves. At the $x_1$ ($x_2$) isoclines, the direction of the horizontal (vertical) arrows changes. Thick isoclines are evolutionarily stable, thin isoclines are evolutionarily unstable. Filled circles denote convergence stable polymorphisms, open circles are saddle points. The evolutionary trees show the alleles present in simulations (time span of $3 \times 10^5$ generations). $c_1 = 0.5$; (a) $d/\sigma = 2.25$; (b) $d/\sigma = 3$; (c) $d/\sigma = 5$.



*Evolution in Polymorphic Populations*

To model evolution after evolutionary branching has taken place, we need to generalize the analysis for polymorphic populations. Consider a population with two distinctly different resident alleles, $x_1$ and $x_2$, in population genetic equilibrium. From equation (3), the marginal fitness of a rare mutant allele, $y$, is:

$$W_{x_1,x_2}(y) = c_1 \frac{p_1 f_1\left(\frac{y + x_1}{2}\right) + p_2 f_1\left(\frac{y + x_2}{2}\right)}{\bar{U}^{(1)}} + c_2 \frac{p_1 f_2\left(\frac{y + x_1}{2}\right) + p_2 f_2\left(\frac{y + x_2}{2}\right)}{\bar{U}^{(2)}}, \quad (6)$$

where $p_1$ and $p_2$ are the equilibrium allele frequencies of $x_1$ and $x_2$, respectively, and

$$\bar{U}^{(i)} = p_1^2 f_i(x_1) + 2p_1p_2 f_i\left(\frac{x_1 + x_2}{2}\right) + p_2^2 f_i(x_2)$$

is the average survival probability during trait-dependent selection in patch $i$ ($i = 1, 2$).

Evolution can be described solely in terms of alleles present only if the marginal fitness of a mutant allele is unequivocally determined by the types of the resident alleles, $x_1$ and $x_2$. In polymorphic populations, this requires that there is a unique stable equilibrium of allele frequencies, $p_1$ and $p_2$, for each possible pair of resident alleles. In general, the diploid two-allele, two-patch Levene model with arbitrary genotypic fitnesses may have up to three polymorphic population genetic equilibria, two of which can be stable. In Appendix 2, however, we derive sufficient conditions under which there is at most one polymorphic equilibrium and show that this condition is always fulfilled if the within-patch fitness is a Gaussian function of an additively determined phenotypic trait. In the present version of the Levene model, therefore, any complication arising from multiple population genetic equilibria need not be considered. Models of adaptive dynamics with several attractors of the resident population were considered by Rand et al. (1994), Doebeli and Ruxton (1997), and Geritz et al. (unpubl.).

For each pair of resident alleles ($x_1$, $x_2$) inside the area of protected polymorphisms, equation (6) determines whether a mutant of $x_1$ or $x_2$ can invade. Like in the monomorphic case, if a mutant similar to a resident allele ($|y - x_i| \leq \delta$) is able to invade, it will usually substitute the corresponding resident allele ($x_i$). Consequently, inside most parts of the area of protected polymorphisms the alleles undergo directional coevolution. The left panels of Figure 2 show the direction where the resident alleles evolve; this can be determined from equation (6) by evaluating for each pair of resident alleles ($x_1$, $x_2$) whether a slightly larger or a slightly smaller mutant of $x_1$ and $x_2$ can invade. The evolution of a polymorphic population can be envisaged as a stochastic broken-line trajectory: When a larger (smaller) mutant of $x_1$ substitutes the original resident allele, the population moves horizontally to the right (left); substitution of $x_2$ by a larger (smaller) mutant corresponds to a vertical step upward (downward). Because the number of consecutive steps into horizontal or vertical direction depends on which resident allele happens to mutate next and the length of each step is random within some small mutation radius ($\delta$), the evolutionary trajectory is subject to sampling stochasticity.

Lines inside the area of protected polymorphisms at which directional evolution of $x_1$ ($x_2$) ceases are called $x_1$ ($x_2$) isoclines (Fig. 2; Geritz et al. 1998). The points of an isocline are analogous to monomorphic singularities. For example, if $x_2$ were fixed and only $x_1$ could mutate, then at the $x_1$ isocline, $x_1$ may be evolutionarily stable or may undergo branching. The intersections of isoclines are polymorphic evolutionary singularities where neither of the two alleles undergoes directional evolution. Such a singularity is an evolutionarily stable polymorphism if both isoclines are evolutionarily stable at their intersection. For a general discussion of convergence stability in polymorphic populations, see Matessi and Di Pascuale (1996).

The left panels of Figure 2 show the isoclines for three values of $d/\sigma$, assuming equal patch sizes ($c_1 = c_2 = 0.5$). Isocline plots are always symmetric in the main diagonal ($y = x$) due to the arbitrary order of labeling the resident alleles. Symmetry in the second diagonal ($y = -x$) is, however, a consequence of equal patch size.

If the difference between the optimal phenotypes in the first and in the second patch is moderate (Fig. 2a; $d/\sigma = 2.25$), there is a single convergence stable and evolutionarily stable polymorphism of two alleles ($-0.81$, $0.81$). The alleles are arranged symmetrically on either side of the branching point ($x^* = 0$). If the difference between the optimal phenotypes is greater, then the isoclines intersect three times (Fig. 2b; $d/\sigma = 3$): There are two convergence stable and evolutionarily stable singularities at ($-1.43$, $4.35$) and ($-4.35$, $1.43$), separated by an evolutionary saddle at ($-1.46$, $1.46$). Although the model is symmetric in terms of relative patch size ($c_1 = c_2 = 0.5$), the alleles of a convergence stable singularity are now asymmetric relative to the branching point ($x^* = 0$). The population may evolve to either convergence stable singularity, depending on the sampling stochasticity of the broken-line trajectories. If the difference between the optimal phenotypes is very large (Fig. 2c; $d/\sigma = 5$), then three convergence stable and evolutionarily stable singularities exist, one symmetrical ($-2.5$, $2.5$) and two asymmetrical ([$-2.5$, $7.5$] and [$-7.5$,$2.5$]).

*Simulation of the Evolutionary Process*

To check the model predictions and, in particular, their robustness with respect to the frequency of mutations, we performed simulations of the evolutionary process (see Appendix 1 for the details). The evolutionary trees in the right panels of Figure 2 show which alleles are present in the simulated populations at various times. In contrast to the assumption made for the analysis of the model, in the simulations mutations occur rather frequently and the population usually does not reach its population genetic equilibrium before a new mutant comes along. Due to recurrent mutations and incomplete allele substitution, there is always some polymorphism within the branches of the evolutionary tree: Each

allele predicted by the analysis is surrounded by a "cloud" of mutants (see Fig. 2a; in Fig. 2b,c, this "cloud" is too narrow to be seen clearly). Nevertheless, this within-branch variability does not confound the predicted course of evolution. The simulated population first evolves to the branching point ($x^* = 0$), undergoes evolutionary branching, and proceeds toward one of the convergence stable and evolutionarily stable polymorphisms, as indicated by the corresponding isocline plot. The model predictions are also fairly robust with respect to the size of mutations.

## Bifurcation Analysis

In this section, we perform a bifurcation analysis of monomorphic and polymorphic evolutionary singularities to investigate the conditions under which evolutionary branching occurs and the characteristics of the resulting evolutionarily stable polymorphisms.

### *Monomorphic Singularities*

The position of the monomorphic evolutionary singularity and its stability properties can be determined analytically (see Appendix 3). There is always a single monomorphic singularity,

$$x^* = c_1 m_1 + c_2 m_2 = (c_2 - c_1)d/2. \quad (7)$$

The singularity corresponds to a generalist phenotype, that is, a type that is intermediate between the optimal phenotypes in the two patches. If the relative sizes of the patches are unequal, the singularity is nearer to the optimum of the larger patch.

The monomorphic singularity is always convergence stable, that is, a monomorphic population first evolves to the generalist phenotype. However, the singularity is evolutionarily stable only if

$$(d/\sigma)^2 < \frac{1}{c_1 c_2}. \quad (8)$$

If this condition is not satisfied, the singularity is a branching point, which gives rise to the evolution of a polymorphism. The difference between the two optimal phenotypes required for evolutionary branching is smallest if the patches are of equal size; the more asymmetric the relative size of the patches, the greater difference between the optimal phenotypes is necessary to get evolutionary branching (Fig. 3).

### *Polymorphic Singularities*

The number, position, and stability properties of polymorphic singularities were determined numerically (Appendix 3). Figure 3 shows the parameter regions with respectively zero, one, two, or three convergence stable singularities. Except for a very narrow parameter range (see below), all polymorphic convergence stable singularities with two alleles are evolutionarily stable as well, that is, there is no further branching to polymorphisms of more than two alleles.

In Figure 3, note that evolutionarily stable polymorphisms can exist even if there is a monomorphic ESS for the same parameter values. Although an initially monomorphic population cannot reach this polymorphism by small mutations,

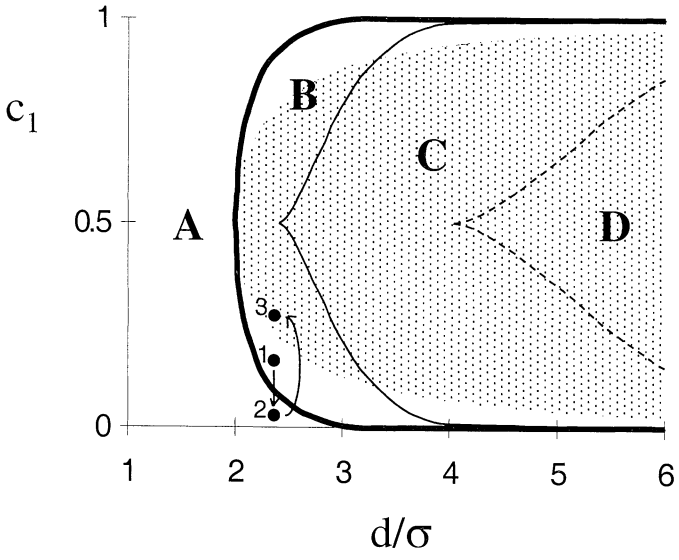



FIG. 3. Bifurcation plot of evolutionary singularities. Inside the dotted area, the monomorphic population has a branching point, outside this area the monomorphic population has an evolutionarily stable allele. The population has no polymorphic singularity in region A (left of the thick line), a single convergence stable as well as evolutionarily stable polymorphism in region B (between the thick line and the thin solid line), two convergence stable and evolutionarily stable polymorphisms separated by an evolutionary saddle in region C (between the thin solid line and the dashed line), and three convergence stable polymorphisms in region D (right of the dashed line). The numbered dots illustrate the evolutionary hysteresis effect (see text for explanation).

it may evolve if the initial population is polymorphic or if a sufficiently different allele is introduced, for example, by occasional interspecific hybridization. The simultaneous existence of an ESS and an evolutionarily stable polymorphism produces an evolutionary hysteresis effect. Assume that the initial population is polymorphic (point 1 in Fig. 3), and that the relative patch size changes due to some change in the environment such that the polymorphism is lost (point 2 in Fig. 3). The population then evolves to the monomorphic ESS. Once the population is monomorphic, the polymorphism cannot be regained by simply restoring the original relative patch size because the monomorphic population is still at an ESS. A substantially greater change in the environment is needed to make the evolution of polymorphism possible by small mutations (point 3 in Fig. 3). This phenomenon is not restricted to the present model, but appears to be a generic feature (Geritz et al. 1999).

The position of the polymorphic evolutionary singularities as a function of relative patch size are shown in Figure 4 for three values of $d/\sigma$. For moderate values of the difference between the optimal phenotypes ($2 < d/\sigma < 2.41$), there is a single convergence stable polymorphism for all values of $c_1$ (Fig. 4a). If the two patches have approximately the same size ($c_1 \approx 0.5$), then the two homozygote phenotypes are near the within-patch optima, whereas the intermediate heterozygote is suboptimal in both patches. However, this arrangement is not robust with respect to relative patch size. If the patches differ substantially in size, then one homozygote is almost optimal in the large patch and the other homozygote

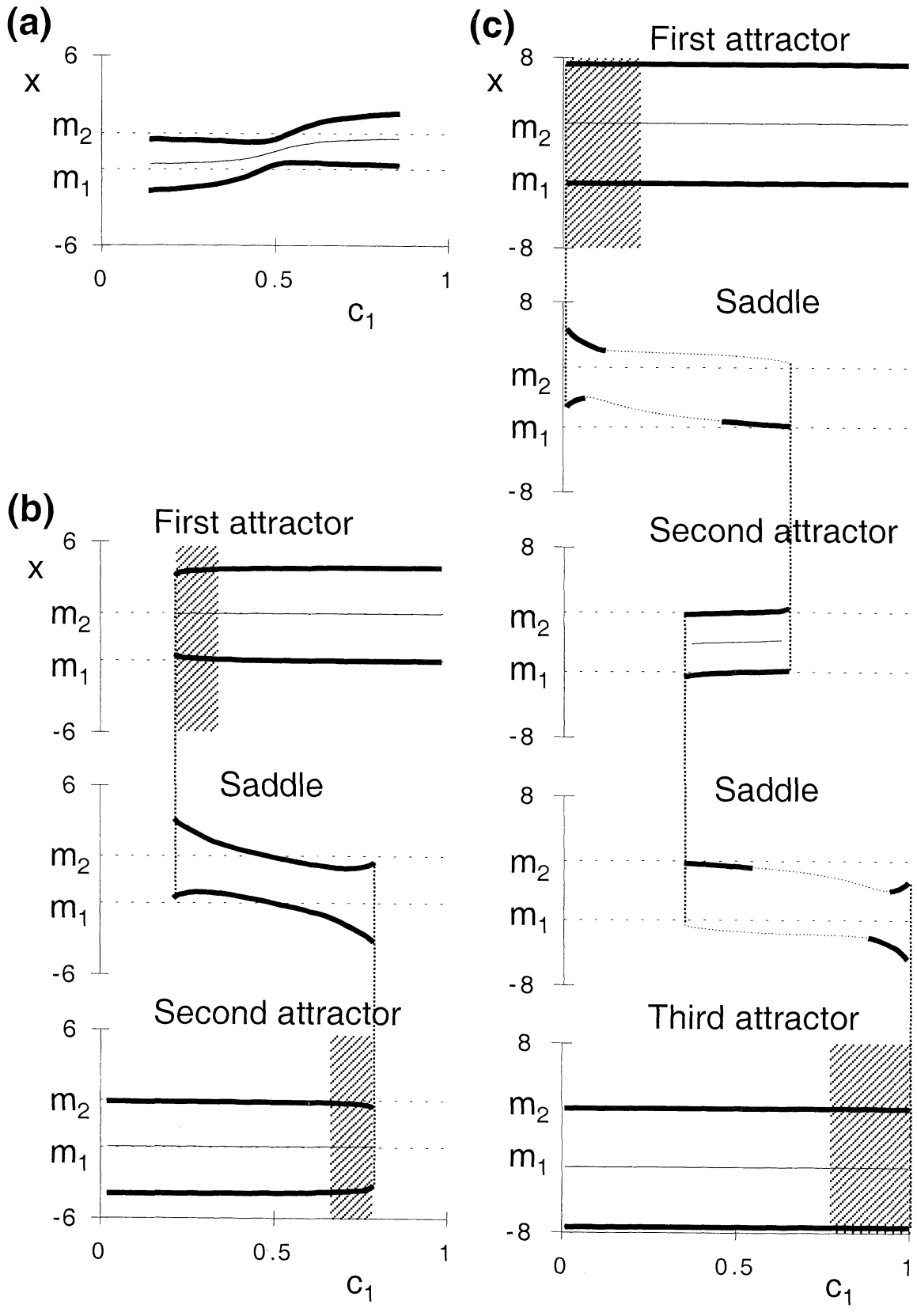



FIG. 4. The position of evolutionary singularities as a function of relative patch size. Thick lines represent the evolutionarily stable alleles or, equivalently, the homozygote phenotypes; thin dotted lines correspond to evolutionarily unstable singular alleles; thin solid lines show the phenotype of the heterozygote in the convergence stable polymorphisms. Different singularities are shown in separate plots, vertical dotted lines indicate fold bifurcations. In the shaded parts the singularity cannot be reached from the branching point. (a) $d/\sigma = 2.25$; (b) $d/\sigma = 3$; (c) $d/\sigma = 5$.

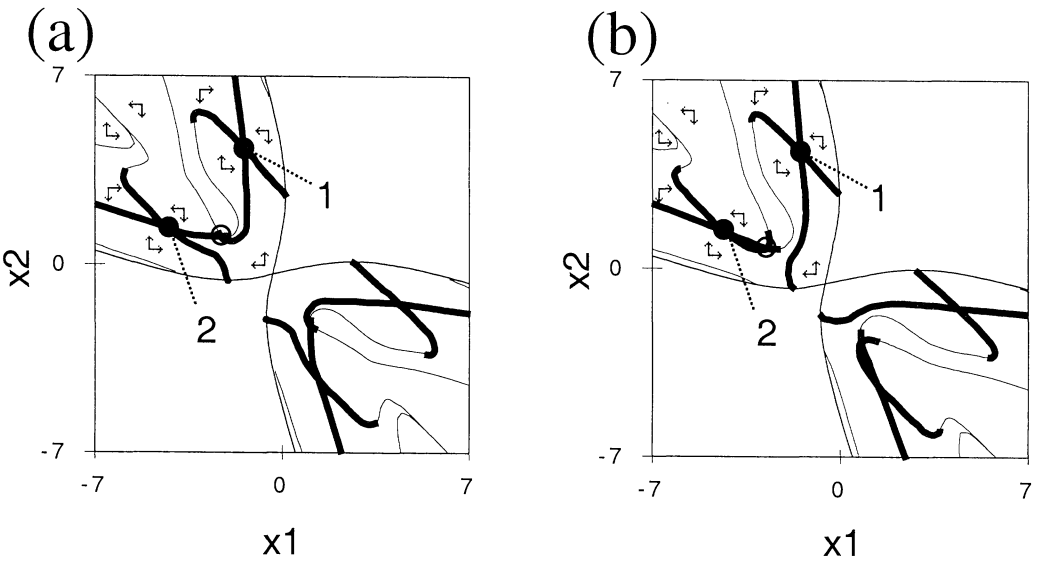



FIG. 5. Asymmetric isocline plots for $d/\sigma = 3$ (notations as in Fig. 2). (a) $c_1 = 0.63$; (b) $c_1 = 0.7$.

has an extreme phenotype such that the intermediate heterozygote is almost optimal in the small patch. For lower values of $d/\sigma$, the "almost optimal" phenotypes are closer to each other and differ more from $m_1$ and $m_2$. This is so because a slight difference from the optimum does not decrease the within-patch fitness significantly (the fitness function is flat at its top) but does increase fitness in the other patch.

If the difference between the optimal phenotypes is greater ($2.41 < d/\sigma < 4.03$), there are two convergence stable polymorphic singularities, the position of which remains approximately constant over a wide range of relative patch size (Fig. 4b). At the first convergence stable singularity, one homozygote is almost optimal in the first patch and the heterozygote is almost optimal in the second patch; the roles are reversed at the second convergence stable singularity. These arrangements appear in the asymmetric evolutionary trees of the symmetric model in Figure 2b. At sufficiently unequal patch sizes, however, only one polymorphic singularity exists such that a homozygote is almost optimal in the large patch and the heterozygote is almost optimal in the small patch.

For very large values of the difference between the optimal phenotypes ($d/\sigma > 4.03$) there are three convergence stable singularities (Fig. 4c). Two of them (the first and the third in Fig. 4c) are similar to those in Figure 4b, except that they exist for virtually the whole range of relative patch size. At the remaining convergence stable singularity in the middle (the second one in Fig. 4c), the two homozygotes are almost optimal in the two patches, whereas the heterozygote has low fitness in both patches. This convergence stable singularity lacks evolutionary stability for two very narrow ranges of relative patch size ($0.349 < c_1 < 0.352$ and $0.648 < c_1 < 0.651$ for $d/\sigma = 5$, which is too narrow to be shown in Fig. 4c) just before it disappears through a fold bifurcation; all other convergence stable polymorphic singularities are evolutionarily stable. Simulations of the evolutionary tree show that in these narrow parameter ranges the population undergoes secondary branching at the polymorphic singularity, resulting in three alleles. Soon after secondary branching, however, one allele goes extinct, and the population evolves to the first ($0.648 < c_1 < 0.652$) or to the third convergence stable singularity ($0.348 < c_1 < 0.352$).

## ADAPTIVE DYNAMICS WITH MULTIPLE EVOLUTIONARY ATTRACTORS

As we have seen in the previous sections, the model has two or three convergence stable polymorphic singularities in a wide range of parameters. Recall that evolution in our model proceeds by finite steps along broken-line trajectories subject to sampling stochasticity, thus the course of evolution is not determined unequivocally. What is then the probability that a population, after evolutionary branching, will evolve to one or another polymorphic singularity?

Consider first the case of two convergence stable singularities such as in Figure 2b. If the patches have exactly the same size ($c_1 = c_2 = 0.5$) and the population enters the area of protected polymorphisms exactly at the branching point, due to symmetry, the population evolves with equal probabilities to the first or to the second convergence stable singularity. However, if the population evolves to the branching point from a distance, then it becomes polymorphic slightly before reaching the branching point, that is, as soon as a mutant is inside the area of protected polymorphisms. The convergence stable singularity that is nearer to the initial polymorphic population is reached with higher probability.

Unequal patch size also changes the probability of reaching one or the other convergence stable singularity. Moderate asymmetry in the relative patch size ($0.34 < c_1 < 0.66$ for $d/\sigma = 3$) moves the saddle point into an asymmetric position relative to the branching point (compare Fig. 5a with the symmetric case shown in Fig. 2b). The population is more likely to evolve to the convergence stable singularity that is on the same side of the saddle as the branching point. For example, the second convergence stable singularity can be reached from the branching point in Figure 5a ($c_1 = 0.63$) only if, by chance, more (or larger) mutations occur in $x_1$

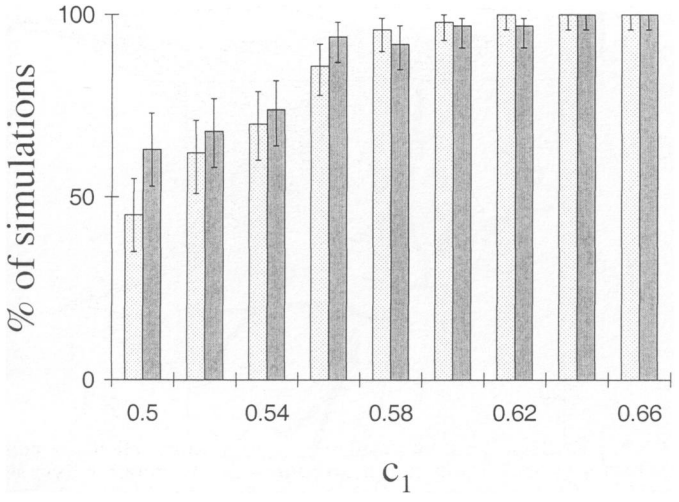



FIG. 6. Percentage of simulations evolving to the first singularity ("1" in Fig. 5) as a function of relative patch size. Simulations starting below the branching point ($x_{ini} = -1$) are shown by the light columns, simulations starting above the branching point ($x_{ini} = 1$) are represented by the dark columns. The error bars show the 95% confidence intervals of the measured percentages. $d/\sigma = 3$, mutation stepsize 0.1, 100 simulations for each column. Notice that the scale of the horizontal axis runs only from 0.5 to 0.66.

than in $x_2$. However, if mutations are small and many mutations are necessary to reach a singularity, such a sampling error has only low probability.

If the asymmetry of patch sizes is more pronounced ($0.22 < c_1 < 0.34$ or $0.66 < c_1 < 0.79$ for $d/\sigma = 3$), then there are still two singularities that are locally convergence stable, but one of them cannot be reached by a population that enters the area of protected polymorphism near the branching point. In Figure 5b ($c_1 = 0.7$), the evolutionary trajectory necessarily hits the $x_1$ isocline, and thereafter proceeds along this isocline upward to the first convergence stable singularity. In Figure 4, shaded parts mark the convergence stable singularities that are isolated from the branching point.

To assess the actual probabilities of arriving at the first versus at the second convergence stable singularity, we run a series of simulations starting with a monomorphic population either above or below the branching point for a range of relative patch sizes and $d/\sigma = 3$ (Fig. 6). If the relative patch sizes are equal ($c_1 = 0.5$), then simulations starting above the branching point reach the first singularity significantly more often than simulations starting below the branching point ($\chi^2 = 6.52$, df = 1, $P < 0.02$) because a population approaching the branching point from above (below) enters the area of protected polymorphisms slightly nearer to the first (second) singularity. If the first patch is larger than the second patch by just a few percent, the probability of evolving to the first convergence stable singularity markedly increases, until the second singularity becomes unreachable from the branching point (at $c_1 = 0.66$ in Fig. 6). The difference between simulations starting below and above the branching point quickly disappears as the size of the patches becomes unequal, such that the direction of evolution is governed by the asymmetry of the isocline plot rather than by the starting point.

Three convergence stable singularities occur only for very large differences between the optimal phenotypes in the first and in the second patch. Unfortunately, in this case the simulations require much larger computational capacity (see Appendix 1), such that a statistical survey similar to the one shown in Figure 6 was not feasible. If the patches are of equal size (Fig. 2c), the population entering the area of protected polymorphism at the branching point ($x^* = 0$) proceeds by small evolutionary steps most probably in the vicinity of the second diagonal ($x_2 = -x_1$) and therefore arrives at the middle convergence stable singularity (the second one in Fig. 2c). Because the two saddles direct the trajectories from the branching point toward the middle convergence stable singularity, the population will reach this singularity with highest probability even if the patch sizes are not equal (Fig. 7a). If the patch sizes are so much different that the second singularity does not exist (Fig. 7b), then the population is more

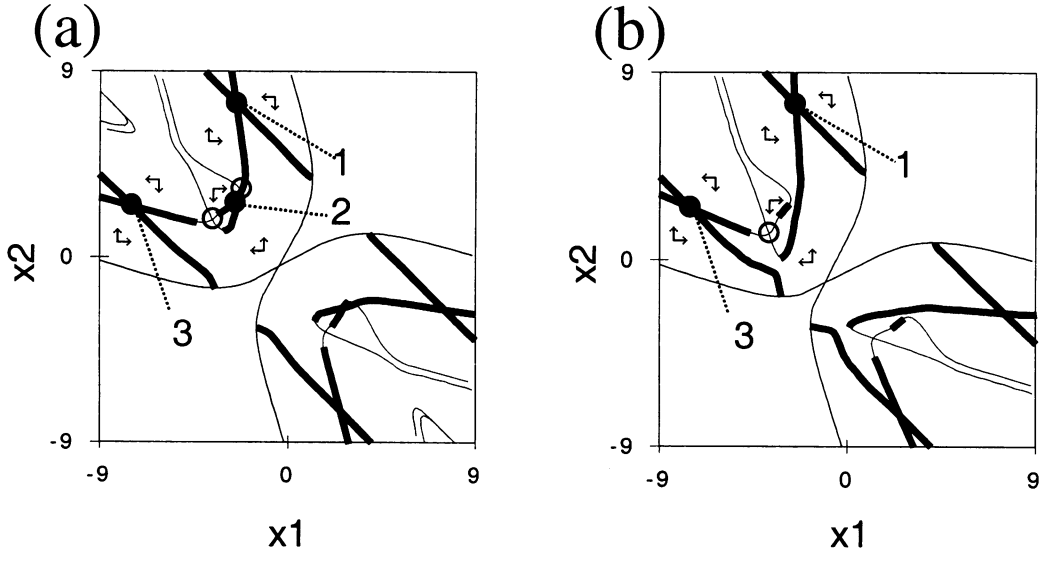



FIG. 7. Asymmetric isocline plots for $d/\sigma = 5$ (notations as in Fig. 2). (a) $c_1 = 0.6$; (b) $c_1 = 0.75$.

likely to stay on the side of the remaining saddle where the branching point is. Compared to Figure 7a, the probability of arriving at the first singularity thus markedly increases in Figure 7b. In case of strongly different patch sizes one convergence stable singularity becomes isolated from the branching point (shaded parts in Fig. 4c).

## Comparison with Previous Models of Short-Term Evolution

In this section, we compare our results on long-term evolution with the classic population genetic analyses of short-term evolution in the Levene model (reviewed by Felsenstein 1976; Hedrick et al. 1976; see also Maynard Smith and Hoekstra 1980; Hoekstra et al. 1985). There are two types of differences between short-term and long-term evolution of genetic polymorphism that we shall investigate in turn: (1) a protected polymorphism indicated by a short-term evolutionary model may be invaded by a new mutant allele that takes over the whole population, thereby the polymorphism may be lost in long-term evolution; and (2) if polymorphism is not possible in the initial population, long-term evolution by allele substitutions may lead to a different population where polymorphism becomes possible.

The first possibility can easily be demonstrated. In our version of the Levene model, protected polymorphism is always possible in short-term evolution, even if a monomorphic ESS is the only possible final outcome (Fig. 1, bottom panels; Appendix 3). However, protected polymorphisms are generally not stable against new mutations: A polymorphic population evolves by allele substitutions as shown, for example, in Figure 2. If there is no evolutionarily stable polymorphism (parameter region A in Fig. 3), then any initially polymorphic population will eventually fall back to monomorphism during its long-term evolution and will evolve to the monomorphic ESS. If there is one (or several) evolutionarily stable polymorphism as well as a monomorphic ESS for the same parameter values (nondotted parts of regions B,C,D in Fig. 3), then an initially polymorphic population will attain an evolutionarily stable polymorphism or will fall back to monomorphism depending on the starting point. Long-term persistence of polymorphism is guaranteed only if there is no monomorphic ESS, that is, if the monomorphic population has a branching point (dotted region in Fig. 3).

The possibility of long-term evolution from a population where polymorphism is not possible toward alleles that can form polymorphisms is particularly intriguing. Several authors have pointed out that the maintenance of genetic polymorphism in heterogeneous environments is not robust when selection is weak: To maintain two alleles, the ratio of patch sizes must lie within a narrow range (Maynard Smith 1966; Maynard Smith and Hoekstra 1980; Hoekstra et al. 1985). Obviously, selection is weak if different alleles determine similar phenotypes. In our model, two similar alleles form a polymorphism at the beginning of evolutionary branching; nevertheless, evolutionary branching happens in a wide range of relative patch size (Fig. 3).

The comparison of these seemingly conflicting results is somewhat hindered by the different assumptions of the models. Generally, two alleles form a protected polymorphism in the Levene model if

$$\sum_{i=1}^{2} \frac{c_i}{U_{11}^{(i)}/U_{12}^{(i)}} > 1 \quad \text{and} \quad \sum_{i=1}^{2} \frac{c_i}{U_{22}^{(i)}/U_{12}^{(i)}} > 1$$

(Levene 1953). These conditions contain four free parameters (two homozygote fitnesses relative to the heterozygote fitness in both patches). Most population genetic models use simplifying assumptions to reduce the number of parameters, such as equal strength of selection in the two patches and constant degree of dominance (Hoekstra et al. 1985) or complete dominance (Maynard Smith 1966). These assumptions are not satisfied in our model: We assumed that the genotypic fitnesses ($U_{jk}^{(i)}$) are determined by the phenotypes through equations (1a,b), and the phenotypes, in turn, are determined by the alleles additively. The relative strength of selection in the two patches as well as the degree of dominance in fitness depend on the alleles present and are therefore not independent of one another.

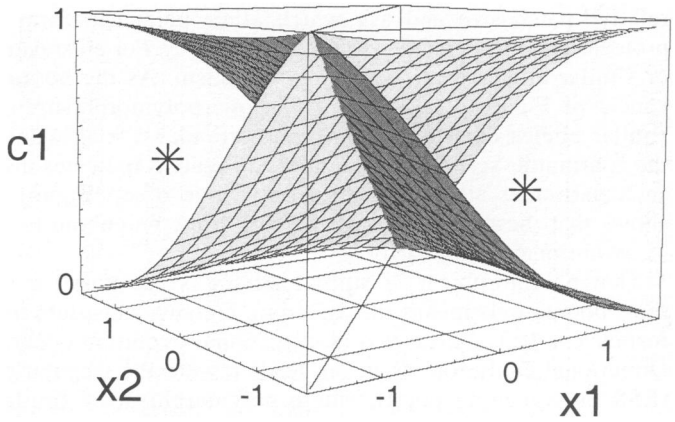



FIG. 8. Range of relative patch size ($c_1$) for which alleles $x_1$ and $x_2$ form a protected polymorphism ($d/\sigma = 3$). To obtain polymorphism, $c_1$ has to be in between the two surfaces, inside the volumes indicated by stars. For example, $x_1 = 0$ and $x_2 = -1$ are maintained in a protected polymorphism if $c_1 > 0.4$ (above the dark surface) and $c_1 < 0.87$ (below the light surface). The intersection of the two surfaces lies above the main diagonal $x_1 = x_2$. This implies that a given pair of similar alleles may form a polymorphism only if $c_1$ is chosen from a very narrow range. However, the intersection of the surfaces spans the whole range of $c_1$ (0,1) along the main diagonal. This means that there exists polymorphisms of similar alleles for any $c_1$, but at different locations along the main diagonal. These locations correspond to the evolutionary singularities (see text). Horizontal cross-sections of this figure show the area of protected polymorphism for various values of $c_1$ (cf. Fig. 1, bottom panel).

Hoekstra et al. (1985) found the range of relative patch size where polymorphism is possible with various selection coefficients (see their Fig. 1). Analogously, from equation (5) we can determine the range of relative patch size ($c_1$) where a given pair of alleles, $x_1$ and $x_2$, forms a protected polymorphism (Fig. 8). As shown in Figure 8, this range is very narrow if $x_1$ and $x_2$ are similar to one another. This is in agreement with the previous results: Weak selection does not maintain polymorphism unless relative patch size is "fine tuned."

Let us now change our perspective such that we fix the

relative patch size and ask which allele pairs can form a protected polymorphism given this value of $c_1$. For most pairs of similar alleles there is no polymorphism. As the bottom panels of Figure 1 and Figure 8 show, polymorphisms of similar alleles exist only for some particular alleles within the continuous set of potential alleles. Figure 8 indicates that such particular alleles exist for any value of $c_1$; Figure 1 shows that these particular alleles are in the neighbourhood of evolutionary singularities.

That polymorphism of similar alleles is not possible in most populations means that invading mutants substitute the former resident alleles, that is, directional evolution occurs. Directional evolution, however, leads toward the singularity (ESS or branching point), where polymorphism of similar alleles becomes possible. The fine tuning necessary for the evolution of polymorphism is thus done by directional evolution. The final fate of the emerging polymorphism depends on the evolutionary stability of the singularity: Near an ESS, stabilizing selection ultimately leads to the monomorphic ESS, but near a branching point, the polymorphism is preserved. As disruptive selection leads to more different alleles, selection becomes stronger and the requirements for polymorphism become less restrictive.

## Discussion

### *Evolution of Genetic Polymorphism in Heterogeneous Environments*

The maintenance of genetic polymorphism in heterogeneous environments under soft selection is a classical result of population genetics (Levene 1953; Felsenstein 1976; Hedrick et al. 1976). In this paper, we investigated how such a polymorphism may arise in the first place and how it will evolve once established, when mutations of only small phenotypic effect occur. To this end we studied the adaptive dynamics of a diploid panmictic population in a two-patch environment with soft selection under the assumption of mutation-limited evolution.

We found that a monomorphic population first always evolves to a generalist phenotype. The generalist utilizes both patches, although in case of unequal patch sizes its phenotype is nearer to the optimal phenotype in the larger patch. If the difference between the optimal phenotypes in the two patches is small, then the generalist is evolutionarily stable. However, if the difference is large enough, then the population undergoes evolutionary branching and gradually evolves to an evolutionarily stable genetic polymorphism with two distinct alleles. For a wide range of parameter values, one of the homozygotes and the heterozygote become specialists for the two patches, whereas the other homozygote has low fitness in both patches. Many models of multiple niche polymorphism assume that the two homozygotes have the highest fitness in the first and in the second patch, respectively. In the present model, however, such a polymorphism is reached by gradual evolution only in two separate parameter ranges, that is, if the difference between the optimal phenotypes is moderate and the patches are not too different in size or if the difference between the optimal phenotypes is very large.

In the present analysis, we assumed that the within-patch fitnesses are Gaussian functions of the phenotype (equations (1a,b); general formulas are given in Appendix 3). Non-gaussian fitness functions may lead to significantly different patterns of evolution. For example, adding sufficiently large constants to the right hand sides of equations (1a,b) may result in several monomorphic evolutionary singularities instead of the single singularity given by equation (7). Assuming that the within-patch fitness functions are symmetric and have identical shape, evolutionary branching is possible for at least some values of the relative patch size, if the fitness functions are convex at their intersection (Appendix 3). For Gaussian fitness functions, the minimal difference required for branching thus is $d/\sigma = 2$ (cf. Fig. 3). If the fitness functions are more peaked, however, then the minimal difference is smaller.

Similarly to Levene (1953), we assumed that the number of adults recruited from each patch is constant (''soft selection''). Constant recruitment from each patch requires high fecundity, otherwise there may be not enough individuals left after selection to fill the patch (this requirement is especially strong if the patches are very different ($d/\sigma$ is large), such that a phenotype adapted to one patch has hardly any survivors in the other patch). If the number of recruited adults is not constant, then a single specialist may be evolutionarily stable even if the unexploited patch is large (Meszéna et al. 1997). The reason for this is that if the population is specialized for one patch, then only a few individuals survive in the other patch. An allele slightly better adapted to the second patch and less to the first patch does not spread because only a small fraction of adults is recruited from the patch where this allele is favored. However, if the number of recruited adults is independent of the phenotype, as in case of soft selection, then two specialists evolve via evolutionary branching. Soft selection thus facilitates the exploitation of different environments.

In our model, we did not consider the possibility of a single genotype having different phenotypes in different patches, that is, phenotypic plasticity. In a quantitative genetic model of soft selection, Via and Lande (1985) have shown that given sufficient genetic variation and without cost of plasticity, evolution leads to the optimal reaction norm, that is, to a genotype that produces the optimal phenotype in each patch. Once the optimal reaction norm has evolved, selection is stabilizing such that there is no increase in genetic variance over that maintained by mutations (Via and Lande 1987). Van Tienderen (1991) investigated directional evolution in a similar model with cost attached to plasticity. One would expect that with very high cost, plasticity is practically ruled out and the results should be comparable to our ones. Indeed, Van Tienderen (1991) found a single attractor under soft selection. Assuming a very high cost of deviating from a constant phenotype across the patches and assuming equal width of the fitness functions, his attractor coincides with the single convergence stable singularity in our model (eq. 7). Van Tienderen assumed constant genetic variance and did not investigate the increase of genetic variability due to disruptive selection that arises when the singularity is a branching point (for the relation between evolutionary stability and the stability of genetic variance in a general multilocus model, see Taylor and Day 1997).

### *Adaptive Dynamics in Diploid Populations*

Comparison of the present model with its haploid counterpart (Geritz and Kisdi, in press) shows that the adaptive dynamics of a diploid population is much richer. In case of monomorphic populations the haploid and diploid models are similar; in particular, the conditions for evolutionary branching are identical. However, there are qualitative differences between haploid and diploid adaptive dynamics in polymorphic populations. After branching, the haploid population always evolves to a unique evolutionarily stable coalition of two specialist phenotypes. In diploid populations, there can be up to three convergence stable polymorphisms, often with markedly different probabilities of reaching them.

A modeling approach related to ours was used by Christiansen and Loeschcke (1980) to study the evolution of resource exploitation. They assumed only a limited set of alleles covering a small range of phenotypes and concluded that only the outermost alleles remain when the population is near a phenotype that we call branching point. However, they did not follow further the evolution of polymorphic populations.

In the model analysis we assumed that mutations are small and occur infrequently. However, the simulations show that the model is fairly robust with respect to both the size and rate of mutations. A crucial assumption in our approach is that of the finite mutational steps. If mutations were infinitesimally small, we would recover the differential equation-based adaptive dynamics models. However, the latter models are unable to capture evolutionary branching (Geritz et al. 1998). The probabilistic nature of arriving at one or the other convergence stable polymorphism is also a consequence of finite mutations.

Another simplifying assumption of the model is that alleles have additive effects. With partial dominance or overdominance, the marginal fitness $W_x(y)$ becomes nondifferentiable at $y = x$. The graphical analysis based on pairwise invasibility plots remains unchanged, but the analytic approach described in Appendix 3 can no longer be applied. In a monomorphic resident population, partial dominance does not affect the singular alleles or their stability properties and hence does not affect evolutionary branching, provided there is a monotonous dominance hierarchy among different allele types, that is, of any two alleles, the larger one is either always (partially) dominant or always (partially) recessive. For polymorphic populations, however, the results may be different depending on the degree of dominance (see Van Dooren, in press). With complete dominance, we effectively recover the results of the clonal model for both monomorphic and polymorphic populations.

The assumption of a single locus controlling a quantitative trait is only a first step in modeling adaptive dynamics in diploid populations. Although by no means common, a single locus may have major effect on a continuous trait. In the African finch *Pyrenestes o. ostrinus*, Smith (1993) found that the width of the lower mandible was effectively determined by a single locus with two alleles. The two alleles correspond to two peaks of the estimated fitness function, that is, they appear to form an evolutionarily stable polymorphism.

Preliminary simulations of the present model with two unlinked loci resulted in evolutionary branching in both loci, producing four gamete types. After evolutionary branching, however, one allele went extinct again and then the locus remained monomorphic, unless the difference between the optimal phenotypes was very large. At the evolutionarily attractor there were two gamete types phenotypically equivalent to the two alleles of the convergence stable singularity in the one-locus model. If the difference between the optimal phenotypes was very large, then both loci remained polymorphic. But with extremely large differences between the optimal phenotypes, such a strong linkage disequilibrium developed between the loci that the population virtually contained only two gamete types, which again were phenotypically equivalent to the convergence stable singularity of the one-locus model. The final outcome of the two-locus simulations thus resembled the one-locus model in a wide range of parameters. It is not yet known to what extent the results of the two-locus simulations generalize to multilocus traits.

Note that we assumed a more flexible type of genetic variation than the one used in most multilocus population genetic models. The latter models usually consider a given number of alleles (e.g., two) per locus, each allele with only small phenotypic effect. In our model, however, a continuum of potential alleles was assumed for each locus. Although the immediately available mutations were near the current resident alleles at any time, there was no limit on how far a locus may evolve by repeated allele substitutions: The phenotypic effect of a single locus was not constrained a priori to be small. However, it is biologically plausible that a single allele may not have an arbitrarily large effect on a continuous trait. When we sufficiently constrained the range of available alleles in the two-locus simulations, both loci stayed polymorpic. Evolutionary branching thus gave rise to a genetic variation similar to the one assumed in multilocus genetic models.

Evolutionary branching in haploid populations could be regarded as morphological speciation. In diploid outbreeding populations evolutionary branching in allele space produces protected polymorphism, but not phenotypically distinct lineages. Heterozygotes and recombinants with intermediate phenotypes are selected against during evolutionary branching (for heterozygote disadvantage in protected polymorphisms, see Udovic 1980; Wilson and Turelli 1986). However, random mating and recombination restore the intermediate types in each generation, and thus prevent the evolution of a bimodal phenotypic distribution. Phenotypically distinct lineages may evolve only if assortative mating develops, that is, if the population undergoes speciation. Although the evolution of assortative mating is highly controversial, recent theoretical studies (e.g., Diehl and Bush 1989; de Meeüs et al. 1993; Doebeli 1996a; Johnson et al. 1996; Kawecki 1996, 1997; Doebeli and Dieckmann, in press) as well as empirical evidence (e.g., Coyne and Orr 1989, 1997; Johannesson et al. 1995; Noor 1995; Schluter and Nagel 1995; Saetre et al. 1997; Galis and Metz 1998; Nagel and Schluter 1998; Rundle and Schluter 1998) seem to support the possibility.

### Acknowledgments

We are grateful to G. Meszéna and J. A. J. Metz for many discussions on adaptive dynamics and to T. Day, who spent

considerable effort to help us to connect the present results to previous models. A discussion with J. Hofbauer led us to the proof in Appendix 2. P. Abrams and T. Van Dooren made valuable comments on an earlier version of the manuscript. A substantial part of this research was done while ÉK received a scholarship from Collegium Budapest, Institute for Advanced Study in Budapest, Hungary, and enjoyed the wonderful working conditions provided by this institute. This work was supported by the Hungarian Science Foundation, OTKA (T 019272), and by a Dutch-Hungarian cooperation grant (NWO-OTKA 048-011-003-02).

## Literature Cited

Abrams, P. A., and H. Matsuda. 1997. Fitness minimization and dynamic instability as a consequence of predator-prey coevolution. Evol. Ecol. 11:1–20.

Abrams, P. A., H. Matsuda, and Y. Harada. 1993a. Evolutionarily unstable fitness maxima and stable fitness minima of continuous traits. Evol. Ecol. 7:465–487.

Abrams, P. A., H. Matsuda, and Y. Harada. 1993b. On the relationship between quantitative genetic and ESS models. Evolution 47:982–985.

Brown, J. S., and N. B. Pavlovic. 1992. Evolution in heterogeneous environments: effects of migration on habitat specialization. Evol. Ecol. 6:360–382.

Brown, J. S., and T. L. Vincent. 1987a. Coevolution as an evolutionary game. Evolution 41:66–79.

———. 1987b. A theory for the evolutionary game. Theor. Popul. Biol. 31:140–166.

———. 1992. Organization of predatorprey communities as an evolutionary game. Evolution 46:1269–1283.

Charlesworth, B. 1990. Optimization models, quantitative genetics, and mutation. Evolution 44:520–538.

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

Christiansen, F. B., and V. Loeschcke. 1980. Evolution and intraspecific exploitative competition. I. One locus theory for small additive gene effects. Theor. Popul. Biol. 18:297–313.

Cohen, D., and S. A. Levin. 1991. Dispersal in patchy environments: the effects of temporal and spatial structure. Theor. Popul. Biol. 39:63–99.

Coyne, J. A., and H. A. Orr. 1989. Patterns of speciation in *Drosophila*. Evolution 43:362–381.

———. 1997. "Patterns of speciation in Drosophila", revisited. Evolution 51:295–303.

De Meeûs, T., Y. Michalakis, F. Renaud, and I. Olivieri. 1993. Polymorphism in heterogeneous environments, evolution of habitat selection and sympatric speciation: soft and hard selection models. Evol. Ecol. 7:175–198.

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

Dieckmann, U., P. Marrow, and R. Law. 1995. Evolutionary cycling in predator-prey interactions: population dynamics and the Red Queen. J. Theor. Biol. 176:91–102.

Diehl, S. R., and G. L. Bush. 1989. The role of habitat preference in adaptation and speciation. Pp. 345–365 *in* D. Otte and J. A. Endler, eds. Speciation and its consequences. Sinauer, Sunderland, MA.

Doebeli, M. 1996a. A quantitative genetic model for sympatric speciation. J. Evol. Biol. 9:893–909.

———. 1996b. An explicit genetic model for ecological character displacement. Ecology 77:510–520.

Doebeli, M., and U. Dieckmann. Evolutionary branching with multilocus genetics. *In* U. Dieckmann and J. A. J. Metz, eds. Adaptive dynamics in context. Cambridge Univ. Press, Cambridge. *In press.*

Doebeli, M., and G. D. Ruxton. 1997. Evolution of dispersal rates in metapopulation models: branching and cyclic dynamics in phenotype space. Evolution 51:1730–1741.

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

———. 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., U. Motro, and E. Sansone. 1997. Continuous stability and evolutionary convergence. J. Theor. Biol. 185:333-343.

Felsenstein, J. 1976. The theoretical population genetics of variable selection and migration. Annu. Rev. Genet. 10:253–280.

Galis, F., and J. A. J. Metz. 1998. Why are there so many cichlid species? Trends Ecol. Evol. 13:1–2.

Geritz, S. A. H., and É. Kisdi. Adaptive dynamics and evolutionary branching in mutation-limited evolution. *In* U. Dieckmann and J. A. J. Metz, eds. Adaptive dynamics in context. Cambridge Univ. Press, Cambridge. *In press.*

Geritz, S. A. H., J. A. J. Metz, É. Kisdi, and G. Meszéna. 1997. Dynamics of adaptation and evolutionary branching. Phys. Rev. Lett. 78:2024–2027.

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

Geritz, S. A. H., E. van der Meijden, and J. A. J. Metz. 1999. Evolutionary dynamics of seed size and seedling competitive ability. Theor. Popul. Biol. 55:324–343.

Hedrick, P. W., M. E. Ginevan, and E. P. Ewing. 1976. Genetic polymorphism in heterogeneous environments. Annu. Rev. Ecol. Syst. 7:1–32.

Hoekstra, R. F., R. Bijlsma, and J. Dolman. 1985. Polymorphism from environmental heterogeneity: Models are only robust if the heterozygote is close in fitness to the favoured homozygote in each environment. Genet. Res. Camb. 45:299–314.

Hofbauer, J., and K. Sigmund. 1990. Adaptive dynamics and evolutionary stability. Appl. Math. Lett. 3:75–79.

Iwasa, Y., A. Pomiankowski, and S. Nee. 1991. The evolution of costly mate preferences. II. The "handicap" principle. Evolution 45:1431–1442.

Johannesson, K., E. Rolan-Alvarez, and A. Ekendahl. 1995. Incipient reproductive isolation between two sympatric morphs of the intertidal snail *Lyttorina saxatilis*. Evolution 49:1180–1190.

Johnson, P. A., F. C. Hoppenstaedt, J. J. Smith, and G. L. Bush. 1996. Conditions for sympatric speciation: A diploid model incorporating habitat fidelity and nonhabitat assortative mating. Evol. Ecol. 10:187–205.

Kawecki, T. J. 1996. Sympatric speciation driven by beneficial mutations. Proc. R. Soc. Lond. B Biol. Sci. 263:1515–1520.

———. 1997. Sympatric speciation via habitat specialization driven by deleterious mutations. Evolution 51:1751–1763.

Kisdi, É., and G. Meszéna. 1993. Density dependent life history evolution in fluctuating environments. Pp. 26–62 *in* J. Yoshimura and C. Clark, eds. Adaptation in a stochastic environment. Lecture Notes in Biomathematics Vol. 98. Springer Verlag, Berlin.

———. 1995. Life histories with lottery competition in a stochastic environment: ESSs which do not prevail. Theor. Popul. Biol. 47: 191–211.

Law, R., P. Marrow, and U. Dieckmann. 1997. On evolution under asymmetric competition. Evol. Ecol. 11:485–501.

Levene, H. 1953. Genetic equilibrium when more than one ecological niche is available. Am. Nat. 87:331–333.

Ludwig, D., and S. A. Levin. 1991. Evolutionary stability of plant communities and the maintenance of multiple dispersal types. Theor. Popul. Biol. 40:285–307.

Marrow, P., R. Law, and C. Cannings. 1992. The coevolution of predator-prey interactions: ESSs and Red Queen dynamics. Proc. R. Soc. Lond. B Biol. Sci. 250:133–141.

Marrow, P., U. Dieckmann, and R. Law. 1996. Evolutionary dynamics of predator-prey systems: an ecological perspective. J. Math. Biol. 34:556–578.

Matessi, C., and C. Di Pasquale. 1996. Long-term evolution of multilocus traits. J. Math. Biol. 34:613–653.

Mathias, A., and É. Kisdi. Evolutionary branching and coexistence of germination strategies. *In* U. Dieckmann and J. A. J. Metz, eds. Adaptive dynamics in context. Cambridge Univ. Press, Cambridge. *In press.*

Matsuda, H., and P. A. Abrams. 1994a. Timid consumers: Self-extinction due to adaptive change in foraging and antipredator effort. Theor. Popul. Biol. 45:76–91.
———. 1994b. Runaway evolution to self-extinction under asymmetrical competition. Evolution 48:1764–1772.
Maynard Smith, J. 1966. Sympatric speciation. Am. Nat. 100:637–650.
———. 1982. Evolution and the theory of games. Cambridge Univ. Press, Cambridge.
———. 1989. Evolutionary genetics. Oxford Univ. Press, Oxford.
Maynard Smith, J., and R. F. Hoekstra. 1980. Polymorphism in a varied environment: how robust are the models? Genet. Res. Camb. 35:45–57.
Meszéna, G., and J. A. J. Metz. The role of effective environmental dimensionality. *In* U. Dieckmann and J. A. J. Metz, eds. Adaptive dynamics in context. Cambridge Univ. Press. *In press.*
Meszéna, G., I. Czibula, and S. A. H. Geritz. 1997. Adaptive dynamics in a 2-patch environment: a toy model for allopatric and parapatric speciation. J. Biol. Syst. 5:265–284.
Metz, J. A. J., R. M. Nisbet, S. A. H. Geritz. 1992. How should we define "fitness" for general ecological scenarios? Trends Ecol. Evol. 7:198–202.
Metz, J. A. J., S. A. H. Geritz, G. Meszéna, F. J. A. Jacobs, and J. S. van Heerwaarden. 1996. Adaptive dynamics, a geometrical study of the consequences of nearly faithful reproduction. Pp. 183–231 *in* S. J. van Strien and S. M. Verduyn Lunel, eds. Stochastic and spatial structures of dynamical systems. North Holland, Amsterdam, The Netherlands.
Nagel, L., and D. Schluter. 1998. Body size, natural selection, and speciation in sticklebacks. Evolution 52:209–218.
Noor, M. A. 1995. Speciation driven by natural selection in *Drosophila*. Nature 375:674–675.
Nowak, M. 1990. An evolutionary stable strategy may be inaccessible. J. Theor. Biol. 142:237–241.
Rand, D. A., H. B. Wilson, J. M. McGlade. 1994. Dynamics and evolution: Evolutionarily stable attractors, invasion exponents and phenotype dynamics. Phil. Trans. R. Soc. Lond. B Biol. Sci. 343:261–283.
Roughgarden, J. 1979. Theory of population genetics and evolutionary ecology: an introduction. Macmillan, New York.
Rundle, H. D., and D. Schluter. 1998. Reinforcement of stickleback mate preferences: sympatry breeds contempt. Evolution 52:200–208.
Saetre, G. P., T. Moum, S. Bures, M. Kral, M. Adamjan, and J. Moreno. 1997. A sexually selected character displacement in flycatchers reinforces premating isolation. Nature 387:589–592.
Schluter, D., and L. M. Nagel. 1995. Parallel speciation by natural selection. Am. Nat. 146:292–301.
Smith, T. B. 1993. Disruptive selection and the genetic basis of bill size polymorphism in the African finch *Pyrenestes*. Nature 363:618–620.
Taper, M. L., and T. J. Case. 1992. Models of character displacement and the theoretical robustness of taxon cycles. Evolution 46:317–333.
Taylor, P. D. 1989. Evolutionary stability in one-parameter models under weak selection. Theor. Popul. Biol. 36:125–143.
———. 1996. The selection differential in quantitative genetics and ESS models. Evolution 50:2106–2110.
Taylor, P. D., and T. Day. 1997. Evolutionary stability under the replicator and the gradient dynamics. Evol. Ecol. 11:579–590.
Udovic, D. 1980. Frequency-dependent selection, disruptive selection, and the evolution of reproductive isolation. Am. Nat. 116:621–641.
Van Dooren, T. Adaptive dynamics for Mendelian genetics. *In* U. Dieckmann and J. A. J. Metz, eds. Adaptive dynamics in context. Cambridge Univ. Press, Cambridge. *In press.*
Van Tienderen, P. H. 1991. Evolution of generalists and specialists in spatially heterogeneous environments. Evolution 45:1317–1331.
Van Tienderen, P. H., and G. De Jong. 1986. Sex ratio under the haystack model: polymorphism may occur. J. Theor. Biol. 122: 69–81.
Via, S., and R. Lande. 1985. Genotype-environment interaction and the evolution of phenotypic plasticity. Evolution 39:505–522.
———. 1987. Evolution of genetic variability in a spatially heterogeneous environment: effects of genotype-environment interaction. Genet.Res. 49:147–156.
Vincent, T. L., Y. Cohen, and J. S. Brown. 1993. Evolution via strategy dynamics. Theor. Popul. Biol. 44:149–176.
Wilson, D. S., and M. Turelli. 1986. Stable underdominance and the evolutionary invasion of empty niches. Am. Nat. 127:835–850.

Corresponding Editor: H. A. Orr

## Appendix 1

### *Simulated Evolutionary Trees*

The simulation algorithm consisted of two modules, a deterministic iteration of allele frequencies for sufficiently common allele types and an individual-based simulation to account for demographic stochasticity when an allele type is rare. The latter module was included because the chance of loss of new mutant alleles by demographic stochasticity influences the speed of evolution and the relative speed of evolution of $x_1$ and $x_2$, in turn, determines the shape of the evolutionary trajectory in polymorphic populations. The shape of the trajectories is important for the results presented in Figure 6.

The deterministic iteration was based on equation (2). In the individual-based module, each individual allele was paired with an allele chosen randomly from the deterministic part (thus only heterozygotes with a single rare allele were considered) and assigned randomly to patch 1 (patch 2) with probability $c_1$ ($c_2$). Whether the heterozygote survived until reproduction was decided randomly, the probability being determined by its phenotype during the period of selection and by the deterministic population during the period of competition. The number of offspring was binomially distributed, males did not differ from females in their number of offspring. Fecundity had to be sufficiently high such that enough offspring survive the period of selection to recruit the fixed number of adults from each patch: The expected number of offspring was 45 (with variance 4.5) for $d/\sigma \leq 3$ (Figs. 2a,b, 6), but the expected number had to be 4500 (with variance 450) for $d/\sigma = 5$ (Fig. 2c). Each offspring inherited the rare allele at random with probability 0.5. (The probability of assignment to patch 1 or 2 and fecundity cancel out in the deterministic dynamics, but influence demographic stochasticity.)

The dynamics of an allele type was modelled by the individual-based module whenever it was present in less than $N_T$ zygotes. Because of potentially high mortality rates, the threshold had to be set at high values, $N_T = 1000$ for $d/\sigma \leq 3$, and $N_T = 10^5$ for $d/\sigma = 5$; unfortunately, this greatly increased the computational capacity needed for the simulations. When the number of alleles exceeded $N_T$, the allele type was introduced into the deterministic iteration at a frequency $N_T/N_{total} = 0.01$ (implying a total population size of $10^5$ for $N_T = 1000$ and that of $10^7$ for $N_T = 10^5$). Conversely, if the frequency of an allele type dropped below 0.01 in the deterministic module, the allele type was moved into the individual-based module. An allele type was extinct if its number hit zero in the individual-based simulation.

New alleles were generated by mutations of alleles already present and added to the population via the individual-based module. Each allele in the deterministic module mutated with a probability $10^{-5}$ per generation, such that the total number of mutations was Poisson-distributed. The mutants differed from the original allele by a small mutation stepsize $\delta = 0.1$ in a random direction. The evolutionary trees of Figure 2 show the allele types present in the deterministic part (i.e., with a frequency greater than 0.01).

## Appendix 2

### *Unique Equilibrium Allele Frequencies in Two-Allele Polymorphisms*

In this appendix, we prove that under the present model assumptions a polymorphic population with two alleles always has a

unique stable population genetic equilibrium. To do so, we first derive a sufficient condition under which the Levene model has at most one polymorphic equilibrium; then we show that this condition is fulfilled whenever the alleles determine a trait additively and the within-patch fitness is a Gaussian function of this trait.

Consider two alleles, $A_1$ and $A_2$, with frequencies $p$ and $q = 1 - p$, respectively, and denote the fitness of genotype $A_jA_k$ in patch $i$ by $U^{(i)}_{jk}$. Equation (2) can be rewritten as

$$\Delta p = c_1\frac{pq}{2\bar{U}^{(1)}}\frac{d\bar{U}^{(1)}}{dp} + c_2\frac{pq}{2\bar{U}^{(2)}}\frac{d\bar{U}^{(2)}}{dp}$$

$$= \frac{pq}{2}\frac{d}{dp}[c_1 \ln \bar{U}^{(1)} + c_2 \ln \bar{U}^{(2)}], \qquad \text{(A1)}$$

where $\bar{U}^{(i)} = p^2U^{(i)}_{11} + 2pqU^{(i)}_{12} + q^2U^{(i)}_{22}$ is the average fitness within patch $i$ ($i = 1, 2$). At a polymorphic equilibrium, $c_1\ln \bar{U}^{(1)} + c_2\ln \bar{U}^{(2)}$ must have an extremum. If both $\ln \bar{U}^{(1)}$ and $\ln \bar{U}^{(2)}$ are concave at any $p$, then $c_1\ln \bar{U}^{(1)} + c_2\ln \bar{U}^{(2)}$ has at most one extremum, so that there can be at most one polymorphic equilibrium. This will be true if $(d^2/dp^2)\ln \bar{U}^{(i)}$ are both negative for all $p$.

The details of the following derivations have been performed using Mathematica 2.2.3. The second derivative $(d^2/dp^2)\ln \bar{U}^{(i)}$ is of the form $A/B^2$, where $A$ is a mountain parabola as a function of $p$. Therefore, because $B^2 > 0$, $(d^2/dp^2)\ln \bar{U}^{(i)}$ is negative for all $p$ if the maximum of $A$ is negative. The maximal value of $A$ is $\bar{U}^{(i)}_{11}\bar{U}^{(i)}_{22} - [\bar{U}^{(i)}_{12}]^2$, and therefore $(d^2/dp^2)\ln \bar{U}^{(i)}$ is negative for all $p$ if $\bar{U}^{(i)}_{11}\bar{U}^{(i)}_{22} - [\bar{U}^{(i)}_{12}]^2 < 0$. This proves the following:

*Lemma:* If the within-patch genotypic fitnesses of the Levene model satisfy the condition $\bar{U}^{(i)}_{11}\bar{U}^{(i)}_{22}/[\bar{U}^{(i)}_{12}]^2 < 1$ in both patches ($i = 1, 2$), then there is at most one polymorphic equilibrium of two alleles.

Now we substitute $U^{(i)}_{jk} = f_i[(x_j + x_k)/2]$ with $f_i$ as given by equations (1a,b), according to the assumption of additive genetics and Gaussian fitness functions. This yields $\bar{U}^{(i)}_{11}\bar{U}^{(i)}_{22}/[\bar{U}^{(i)}_{12}]^2 = \exp(-\{[(x_1 - x_2)^2]/4\sigma^2\})$, which is less than one whenever $x_1 \neq x_2$. It thus follows that in the present model, there is at most one polymorphic equilibrium. In the area of protected polymorphism, where both trivial equilibria ($p = 0$ and $p = 1$) are unstable, there is a unique stable equilibrium allele frequency. Moreover, if one trivial equilibrium is stable and the other is unstable, then there cannot be any polymorphic equilibrium, that is, nonprotected polymorphisms of two alleles are not possible.

The above proof directly generalizes to more than two patches; extension to more than two alleles, however, is not straightforward. Therefore, this proof does not exclude nonprotected polymorphisms of three alleles and it does not guarantee that during directional evolution in a polymorphic population with two resident alleles invasion of a third allele leads definitely to the substitution of one resident allele. However, we can show that if two alleles of the three are sufficiently similar (i.e., produced by a small mutation), then generically three alleles may be present in equilibrium only near an isocline (Kisdi, unpubl.).

## Appendix 3

### *Analytical Characterization of the Evolutionary Singularities*

#### *Monomorphic Singularities*

To determine the monomorphic evolutionary singularities, recall that if the population is monomorphic for the singular allele $x^*$, then the marginal fitness $W_{x^*}(y)$ of small mutants must either be less than one for all $y \neq x^*$ (ESS) or greater than one for all $y \neq x^*$ (branching point), whereas $W_{x^*}(x^*) = 1$ (Fig. 1). At the monomorphic singularity $W_{x^*}(y)$ thus has an extremum, and $x^*$ is implicitly determined by

$$\left.\frac{\partial W_x(y)}{\partial y}\right|_{y=x=x^*} = \frac{1}{2}\left[c_1\frac{f_1'(x^*)}{f_1(x^*)} + c_2\frac{f_2'(x^*)}{f_2(x^*)}\right] = 0. \qquad \text{(A2)}$$

Substituting the Gaussian fitness functions from equations (1a,b), the above equation can easily be solved to obtain equation (7).

The monomorphic singularity is convergence stable if

$$\left[\frac{\partial^2 W_x(y)}{\partial x\partial y} + \frac{\partial^2 W_x(y)}{\partial y^2}\right]_{y=x=x^*}$$

$$= \frac{1}{2}\left[c_1\frac{f_1''}{f_1} + c_2\frac{f_2''}{f_2} - c_1\left(\frac{f_1'}{f_1}\right)^2 - c_2\left(\frac{f_2'}{f_2}\right)^2\right]_{x^*} < 0 \qquad \text{(A3)}$$

(Eshel 1983). Notice that this condition is fulfilled if the fitness functions are concave or only moderately convex at $x^*$. For Gaussian fitness functions, this condition is equivalent to $1/\sigma^2 > 0$, which is always satisfied.

The monomorphic singularity is an ESS if

$$\left.\frac{\partial^2 W_x(y)}{\partial y^2}\right|_{y=x=x^*} = \frac{1}{4}\left[c_1\frac{f_1''}{f_1} + c_2\frac{f_2''}{f_2}\right]_{x^*} < 0 \qquad \text{(A4)}$$

(Maynard Smith 1982). The monomorphic singularity is evolutionarily unstable, and thus evolutionary branching is possible if the fitness functions are convex at $x^*$; strong convexity, however, makes the singularity convergence unstable (see above). When the Gaussian fitness functions are substituted and $x^*$ is replaced from equation (7), this condition reduces to inequality (8).

In the neighborhood of the monomorphic singularity, there are pairs of alleles that can form a protected polymorphism if

$$\left[\frac{\partial^2 W_x(y)}{\partial x^2} + \frac{\partial^2 W_x(y)}{\partial y^2}\right]_{y=x=x^*}$$

$$= c_1\left(\frac{f_1'}{f_1}\right)^2 + c_2\left(\frac{f_2'}{f_2}\right)^2 - \frac{1}{2}\left[c_1\frac{f_1''}{f_1} + c_2\frac{f_2''}{f_2}\right] > 0 \qquad \text{(A5)}$$

(Geritz et al. 1998). This condition is always fulfilled if equation (A3) holds, that is, if the singularity is convergence stable. With Gaussian fitness functions, the singularity is always convergence stable, therefore there is always an area of protected polymorphism attached to the singularity (Fig. 1, bottom panels).

To get some insight into the bifurcation patterns with non-Gaussian fitness functions, assume only that $f_1$ and $f_2$ have identical shape (i.e., $f_2[x] = f_1[x - d]$), they are symmetric ($f_1[m_1 + x] = f_1[m_1 - x]$), and analytical. Because $f_1$ and $f_2$ must be concave near their maxima, the singularity is an ESS if $d$ is sufficiently small. By symmetry, if evolutionary branching is possible with a certain value of $d$ and $c_1 = c$, then it is also possible with $c_1 = 1 - c$; it follows that branching may occur with the smallest between-patch difference if $c_1 = 0.5$. With $c_1 = 0.5$, the singular point coincides with the point where $f_1$ and $f_2$ intersect, and branching becomes possible when $f_1$ and $f_2$ are sufficiently apart such that they are convex at their intersection. Convergence stability may be lost at large values of $d$ if the fitness function is too convex far from its maximum; but this is not the case with Gaussian fitness functions.

#### *Polymorphic Singularities*

Analogously to equation (A2), a polymorphic evolutionary singularity $(x_1^*, x_2^*)$ is implicitly determined by

$$\left.\frac{\partial W_{x_1,x_2}(y)}{\partial y}\right|_{\substack{y=x_i^*\\ x_1=x_1^*,\, x_2=x_2^*}} = 0 \quad \text{for } i = 1, 2, \qquad \text{(A6)}$$

where $W_{x_1,x_2}$ is given by equation (6). The polymorphic singularity is evolutionarily stable if

$$\left.\frac{\partial^2 W_{x_1,x_2}(y)}{\partial y^2}\right|_{\substack{y=x_i^*\\ x_1=x_1^*,\, x_2=x_2^*}} < 0 \quad \text{for } i = 1, 2 \qquad \text{(A7)}$$

(Geritz et al. 1998). The bifurcation analysis of polymorphic singularities was done by solving equations (A6) numerically for $x_1^*$ and $x_2^*$ using the Newton-Raphson algorithm and checking evolutionary stability by equation (A7) for a range of parameter values.