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Evolutionary Branching and Sympatric Speciation in Diploid Populations

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Interim Report **IR-99-048**

Evolutionary Branching and Sympatric Speciation in Diploid Populations

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IIASA STUDIES IN ADAPTIVE DYNAMICS NO. 39

The Adaptive Dynamics Network at IIASA fosters the development of new mathematical and conceptual techniques for understanding the evolution of complex adaptive systems.

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The pivotal role of evolutionary theory in life sciences derives from its capability to provide causal explanations for phenomena that are highly improbable in the physico-chemical sense. Yet, until recently, many facts in biology could not be accounted for in the light of evolution. Just as physicists for a long time ignored the presence of chaos, these phenomena were basically not perceived by biologists.

Two examples illustrate this assertion. Although Darwin's publication of "The Origin of Species" sparked off the whole evolutionary revolution, oddly enough, the population genetic framework underlying the modern synthesis holds no clues to speciation events. A second illustration is the more recently appreciated issue of jump increases in biological complexity that result from the aggregation of individuals into mutualistic wholes.

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É. Kisdi
S.A.H. Geritz

Introduction

The formal framework for modelling adaptive dynamics of continuous traits introduced by Metz et al. (1996) and Geritz et al. (1997, 1998) is particularly suitable for modelling evolutionary branching, i.e., the gradual evolution of two phenotypically distinct strategies by small mutations in an initially monomorphic population. A population can undergo evolutionary branching only at particular trait values, called branching points, which are attractors of the monomorphic adaptive dynamics but which nevertheless lack evolutionary stability. In the neighbourhood of a branching point, an invading mutant may coexist with the former resident, forming a protected dimorphism of two phenotypically similar strategies. Next, the two coexisting strategies undergo divergent coevolution, and become phenotypically distinct (Metz et al., 1992, 1996; Eshel et al., 1997; Geritz et al., 1997, 1998).

Evolutionary branching may be interpreted as morphological speciation. The adaptive dynamics framework, however, assumes clonal inheritance, for which the species concept is not well defined. The possible connection between evolutionary branching and speciation depends on whether branching occurs in diploid outbreeding populations, and whether reproductive isolation evolves between the emerging branches.

In diploid populations, alleles are the heritable units which are transmitted from one generation to the next and which undergo mutation. The adaptive dynamics framework of Metz et al. (1996) and Geritz et al. (1997, 1998) can be directly generalized to alleles in diploid populations if a fitness measure is attached to alleles rather than to genotypes or phenotypes (called marginal fitness in population genetics). Evolution then can be described in allele space rather than in trait space. During evolutionary branching, a single allele gives rise to two diverging allele lineages: The outcome of evolutionary branching is thus genetic polymorphism of two alleles with distinctly different phenotypic effects (Kisdi and Geritz, 1999). On the phenotypic level, however, evolutionary branching in diploid populations differs from the clonal model because of the presence of heterozygotes.

In this paper, we first set up an adaptive dynamic model with single locus Mendelian genetics instead of clonal inheritance. Next, we investigate whether evolutionary branching may promote the evolution of reproductive isolation by assortative mating or by spatial segregation. Since numerical analyses and simulations are possible only if ecological details are specified, we use a specific model, i.e., the continuous version of Levene's (1953) soft selection model as an example. The clonal counterpart of this model was used

by Geritz and Kisdi (*in press*); see Brown and Pavlovic (1992), Meszéna et al. (1997) and Geritz et al. (1998) for related clonal models. We shall demonstrate the following points:

(1) As long as the population is monomorphic, the diploid and the clonal models give identical results. In particular, evolutionary branching occurs in allele space under exactly the same ecological conditions as in a clonal population.

(2) In polymorphic populations, adaptive dynamics in a diploid, randomly mating population can be substantially different from the corresponding clonal population.

(3) Evolutionary branching in a diploid population initially results in a protected genetic polymorphism where heterozygotes are at a selective disadvantage. Selection against heterozygotes may be strong, which facilitates the evolution of assortative mating.

(4) Heterozygote inferiority may be only temporary on an evolutionary timescale: As the polymorphic population continues to evolve, it may leave the part of allele space where heterozygotes are selected against. In this case, there is only a limited time during which assortative mating may evolve.

(5) Assortative mating controlled by an independent locus readily evolves during evolutionary branching if heterozygote inferiority is strong or penetrance (the degree of reliability in mate choice) is high. Our numerical results indicate that neither unrealistically strong selection nor very high penetrance is needed. Once partial reproductive isolation is achieved, the evolutionarily stable attractor of the diploid population is very similar to that of the clonal population.

(6) When disruptive selection is due to the spatial heterogeneity of the environment such as in the Levene model, then there is selection for smaller migration between the environmental patches, which also leads to reproductive isolation. Even with moderate decrease in migration, the diploid population evolves to approximately the same evolutionarily stable attractor as a clonal population does.

Adaptive dynamics with diploid Mendelian genetics

Throughout this paper, we consider the evolution of a single continuous trait. The trait is determined by a single autosomal locus with a continuum of possible alleles. The alleles act additively on the phenotype. For the convenience of notation, we denote an allele by x if individuals homozygous for this allele have trait value x; heterozygotes with alleles x and y have phenotype $(x+y)/2$. The trait evolves by mutations of small (but not infinitesimally small) phenotypic effect. New mutations occur infrequently, such that the previous mutant has spread or has been excluded, and the population has reached its population genetical equilibrium, before the next mutant comes along. The rate and size of mutations is assumed to be independent of the trait value.

Our working example will be a version of Levene's (1953) soft selection model with the following ecological assumptions. The environment consists of two habitats or patches. In each patch, the probability of survival (f_i) is given by a Gaussian function of the trait value, i.e.,

$$f_i(x) = \alpha_i \exp\left[\frac{-(x - m_i)^2}{2\sigma^2}\right] \qquad (1)$$

(i=1,2). The patches have different optimal phenotypes (m_i), but do not differ in the width of the Gaussian function (σ). Without loss of generality, the within-patch optimal phenotypes are set to m_1=-d/2 and m_2=d/2, where d is the difference between the optima. After selection, there is 'contest' competition within the patches such that always a fixed number of adults is recruited from each patch. First we assume that adults mate at random in the entire population, and that the offspring are randomly distributed between the patches; later we shall modify this assumption when we investigate the evolution of reproductive isolation and the evolution of migration. A detailed analysis of this model is given by Kisdi and Geritz (1999). Here we shall confine ourselves to the symmetric case, i.e., when half of the adult population is recruited from each patch.

First consider a monomorphic resident population. A mutant allele can invade if its logarithmic fitness is positive. Since the mutant allele is initially rare, it occurs almost exclusively in heterozygotes such that its fitness is approximately equal to the fitness of the heterozygote phenotype (explicit expressions for the mutant's fitness in the Levene model are given in Box 1). A successfully invading mutant allele always replaces the previously established allele, resulting in directional evolution of the trait value, until either an evolutionarily stable trait value or a branching point is reached (Metz et al., 1996; Geritz et al., 1997, 1998).

In the Levene model, it is straightforward to find and characterize the monomorphic evolutionary singularities (see Kisdi and Geritz (1999) for the calculations). There is always a single evolutionary singularity at x^*=0, which is evolutionarily stable if the two patches are not too different (d/σ<2), but otherwise it is a branching point. The clonal counterpart of the Levene model yields exactly the same result. Evolutionary branching thus occurs in a monomorphic population under the same ecological circumstances, and at the same trait value, whether the population is diploid or clonal. Van Dooren (*in press*) gives a general proof of correspondence between clonal and diploid adaptive dynamics in monomorphic populations.

Evolutionary branching gives rise to a genetically polymorphic population, where the individual branches continue to evolve by mutations and allele substitutions. Figure 1 shows the possible courses of evolution after evolutionary branching in the diploid Levene model. If the two patches are only moderately different, then the population evolves to a unique evolutionarily stable polymorphism of two alleles (Figure 1a). This attractor is symmetrical in the sense that the two alleles are at equal distances on either side of the branching point. The phenotypes of the homozygotes are near the within-patch optima (±d/2), while the intermediate heterozygotes are not well adapted to either patch. If the difference between the patches is greater, then there are two asymmetric attractors (Figure 1b), where one (or the other) homozygote and the heterozygote are near the within-patch optima. With very large difference between the patches, all three attractors exist (Figure 1c). The three cases shown in Figure 1a-c will be referred to as examples (A), (B), and (C), and will be used throughout this paper. Example (B) is illustrated by a simulated evolutionary tree in Box 3.

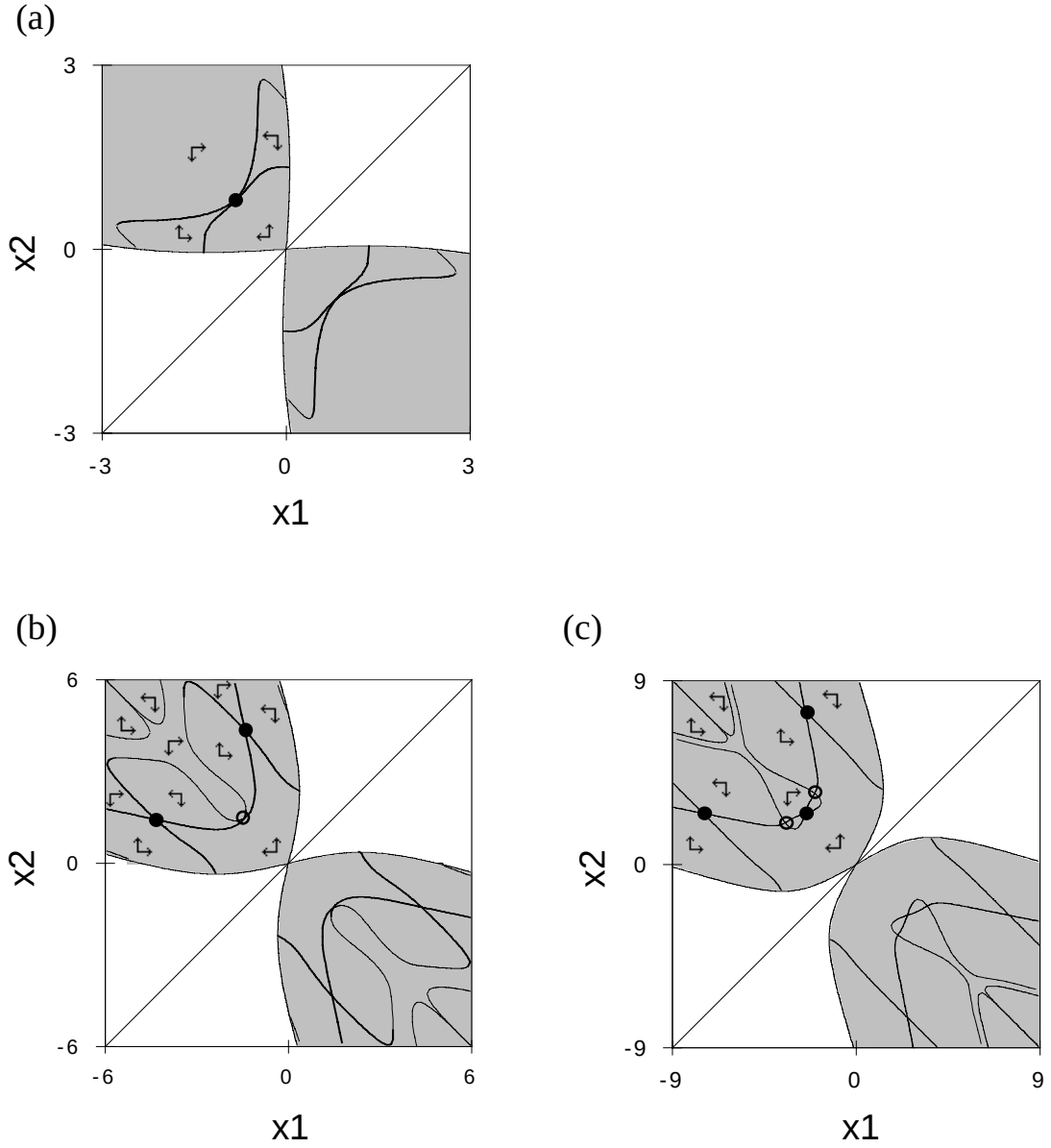

Figure 1. Evolution in polymorphic populations. Horizontal (vertical) arrows show the direction of evolution of allele x_1 (x_2), i.e. whether larger or smaller mutants can substitute the original allele, within the area where the two alleles form a protected polymorphism. Thick and thin lines denote evolutionarily stable and unstable isoclines, respectively (see Geritz et al., 1998). Filled circles indicate the evolutionary attractors, open circles are evolutionary saddle points. The plots are always symmetric in the main diagonal (x_1=x_2) since the order of labeling the resident alleles is arbitrary. For convenience, we may denote the smaller allele by x_1 and thus restrict the analysis to the upper half of the plot above the main diagonal. The symmetry in the second diagonal (x_2=-x_1) is due to the symmetry of the model (i.e., equal patch sizes). The polymorphic population has *(a)* a single symmetric attractor at (x_1, x_2) = (-0.81, 0.81) for d/σ=2.25; *(b)* two asymmetric attractors at (-1.43, 4.35) and (-4.35, 1.43) separated by a saddle for d/σ=3; *(c)* three attractors, one symmetric (-2.5, 2.5) and two asymmetric [(-2.5, 7.5) and (-7.5, 2.5)] separated by two saddles for d/σ=5. All attractors are evolutionarily stable polymorphisms.

Mutation-limited evolution of the polymorphic population follows a stochastic broken line trajectory on the plots of Figure 1: An allele substitution may occur either in the x_1-branch or in the x_2-branch, leading to a small step of random length in a horizontal or in a vertical direction, respectively. Since the trajectory is stochastic, in case of multiple attractors it is not unequivocally determined to which attractor the population will evolve. In example (B) with two asymmetric attractors, the probability of evolving to one or to the other after evolutionary branching at $x_1=x_2=x^*=0$ is the same due to the symmetry of the model. In example (C), the symmetric attractor is reached with the highest probability, because the evolutionary trajectories tend to stay near the -45 degree line.

In contrast to the diploid model, there is always a unique evolutionarily stable dimorphism with two strategies near the within-patch optima in the clonal version of the Levene model (Geritz and Kisdi, *in press*). Although for monomorphic populations the clonal and the diploid models behave identically, in polymorphic populations the two models are significantly different. The reason for this is that the fitness of a mutant invading a polymorphic resident population is determined in a different way (Box 1): In a clonal population, a mutant uniquely determines its own phenotype and interacts with two resident phenotypes only, whereas in a diploid population a mutant allele occurs in two kinds of heterozygotes with different phenotypes, and these heterozygotes interact with three resident phenotypes (i.e., the two homozygotes and the heterozygote of the resident alleles).

Heterozygote inferiority in protected polymorphism

Evolutionary branching in diploid populations leads in the first place to a genetic polymorphism, but not to separate species: Speciation occurs only if reproductive isolation develops between the two homozygotes such that the heterozygotes disappear. In the next two sections, we investigate whether this may happen by evolution of assortative mating.

During evolutionary branching, disruptive selection occurs such that intermediate phenotypes are selected against. In a diploid population with additive genetics, the phenotypically intermediate heterozygotes are at a disadvantage. Consequently, homozygotes achieve a higher fitness if they mate with their own kind, and avoid the production of inferior heterozygote offspring. Evolutionary branching thus creates a selective environment that favors assortative mating within the branches.

Two factors promote speciation by assortative mating. First, the genetic polymorphism that arises by evolutionary branching in a diploid population is protected, even though the heterozygotes are at a disadvantage (Pimm, 1979; Udovic, 1980; Wilson and Turelli, 1986). It is the very presence of both homozygote genotypes which renders the heterozygotes inferior. Should one allele become rare for any reason, the corresponding homozygotes would disappear, and the heterozygotes would not be intermediate anymore. The frequency-dependent genotypic fitnesses would change in favor of the heterozygotes, and the rare allele would increase in frequency. In models assuming that heterozygote fitness does not increase when one of the homozygotes becomes rare, extinction of the rare allele is a likely outcome (e.g., Spencer et al., 1986; Liou and Price, 1994).

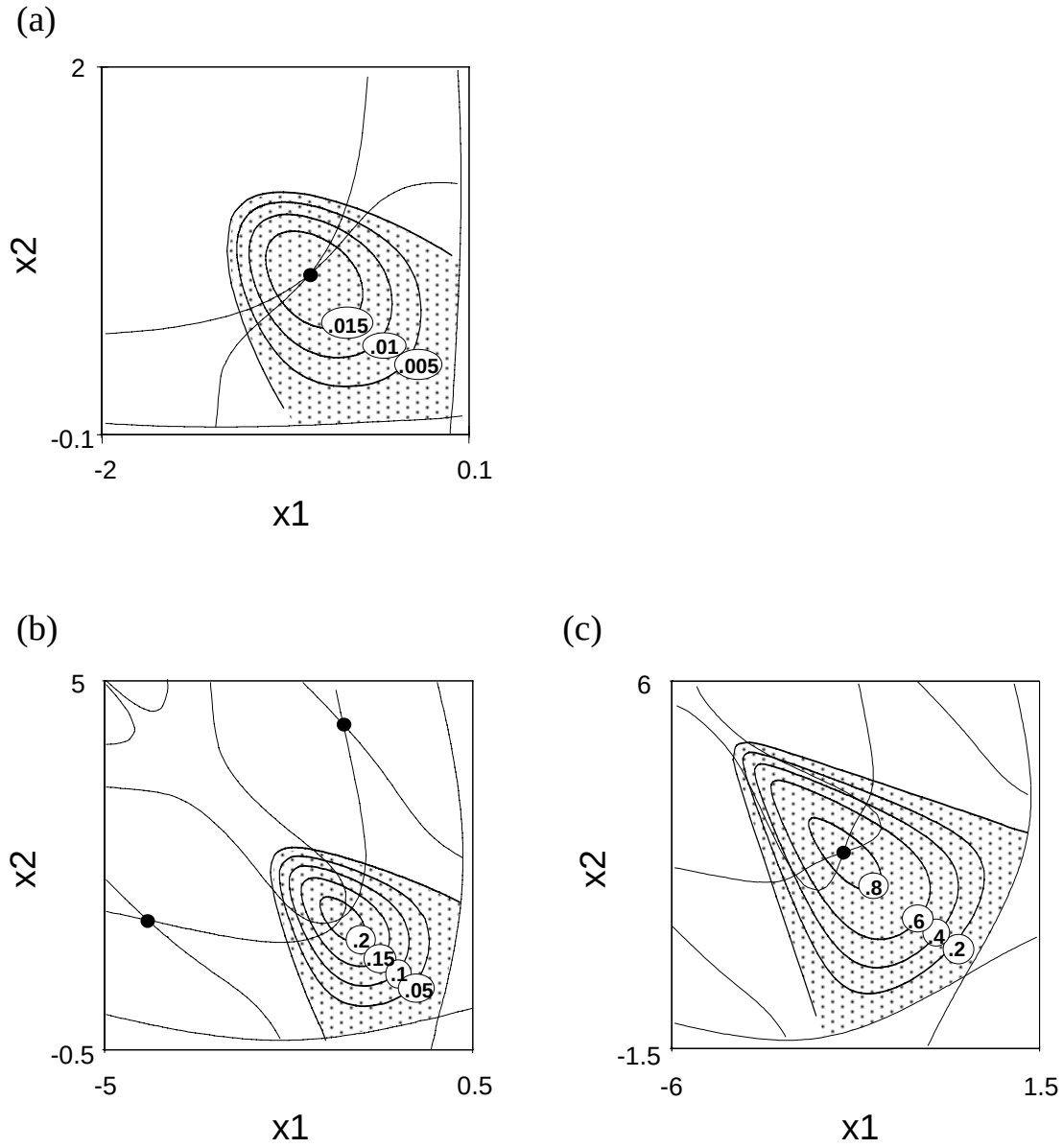

Figure 2. Selection against heterozygotes. Inside the dotted area, the heterozygotes are inferior, outside this area the heterozygotes are superior. The thick contour lines correspond to different values of F_{as}. Isoclines (thin lines) and evolutionary attractors (filled circles) are shown for orientation (enlarged from Figure 1). *(a)* d/σ=2.25; *(b)* d/σ=3; *(c)* d/σ=5.

Second, selection against heterozygotes may be strong during evolution of the polymorphic population. In the Levene model, we measured the degree of heterozygote inferiority by the deficiency of heterozygotes after selection compared to the Hardy-Weinberg equilibrium of zygotes,

$$F_{as} = 1 - \frac{H_{as}}{2pq} \qquad (2)$$

where H_{as} is the equilibrium frequency of heterozygotes among adults. (The subscript refers to "after selection"; before selection, the population is in Hardy-Weinberg equilibrium if mating is random.) Figure 2a-c shows the contour lines of F_{as} for examples (A), (B), and (C), respectively.

Near the branching point ($x_1=x_2=x^*=0$) fitness differences are small, thus the disadvantage of heterozygotes is weak. As the branches evolve further apart, heterozygote inferiority increases. If the difference between the patches (d/σ) is moderate, then the disadvantage of heterozygotes is minor even at the symmetric evolutionary attractor (Figure 2a), because the intermediate heterozygotes still have a relatively high fitness in both patches. With greater difference between the patches, the evolutionary attractors are asymmetric. Heterozygotes are not inferior at the asymmetric attractors (they are nearly optimal in one patch). On its way to one of the asymmetric attractors, the population will thus leave the area of heterozygote inferiority (Figure 2b). Before reaching the attractor, however, the polymorphic population will pass through the region where selection against heterozygotes is the strongest (Figure 3): There is a period in time during which heterozygote inferiority reaches considerably high values. If the patches are very different, then there is again a symmetric attractor to which the population most probably evolves. Contrary to example (A), heterozygotes are now strongly selected against at the symmetric attractor, since the within-patch fitness peaks are further apart such that intermediate heterozygotes have very low fitness in both patches.

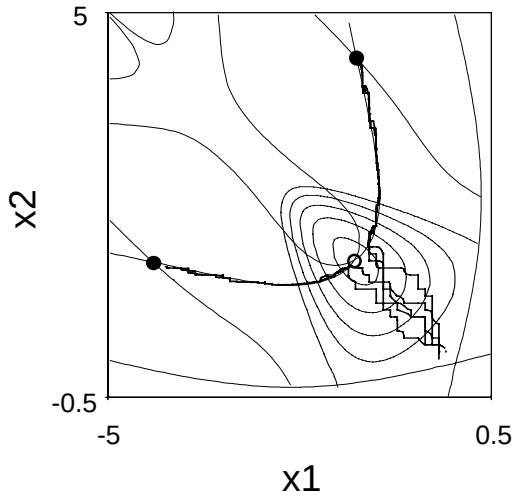

Figure 3. Five simulated stochastic evolutionary trajectories superimposed on Figure 2b. The population evolves from the branching point ($x_1=x_2=x^*=0$) towards one or the other attractor (filled circles) through the vicinity of the evolutionary saddle point (open circle), where heterozygote inferiority is the strongest. The isoclines and the contourlines of F_{as} are shown by thin lines for orientation. The trajectories were obtained by plotting the mean phenotypes (x_1, x_2) within the two branches of simulated evolutionary trees.

Evolution of assortative mating

In this section, we investigate the evolution of a simple assortative mating mechanism in the Levene model (see Udovic, 1980 and Geritz and Kisdi, *in prep.* for further analysis of

this model). Assume that mate choice is determined by a single locus with two alleles, *B* and *b*, where *B* is dominant over *b*. Mating occurs preferentially between individuals with the same phenotype such that the dominant phenotype (*BB* and *Bb*) and the recessive phenotype (*bb*) correspond to two partially isolated mating groups. The strength of assortative mating is measured by the penetrance (Π): Mating occurs within the group with probability Π, while mating is random with probability 1-Π. Spring-flowering and autumn-flowering varieties of plant species, or flowers with different colours visited by different pollinators may be examples of such mating groups.

Mating within the groups yields reproductive isolation between the branches of the evolutionary tree if linkage disequilibrium develops between the primary locus undergoing branching and the secondary locus controlling mate choice. If one allele of the secondary locus, say *B*, is associated with x_1 and *b* is associated with x_2, then the x_1x_1 homozygotes tend to belong to the (*BB*+*Bb*) mating group while x_2x_2 homozygotes tend to be in the *bb* mating group. The two homozygotes are therefore partially reproductively isolated, and the x_1x_2 heterozygotes are less frequent than in case of random mating. Without linkage disequilibrium, the alleles of the secondary locus are neutral and do not influence the evolution of the primary locus.

Figure 4 summarizes the population genetical equilibria as determined by numerical simulations assuming Π=0.9 and free recombination between the primary and secondary loci. If the difference between the two patches (d/σ) is not large enough (example (A)), then selection is too weak to maintain linkage disequilibrium (Figure 4a). With sufficiently large d/σ (examples (B) and (C)), however, there is stable linkage disequilibrium for some allele pairs (x_1, x_2). The area of protected polymorhisms can be divided into three regions (Figure 4b,c):

(i) Region *LE*, where the only population genetical equilibrium is with linkage equilibrium;

(ii) Region *LE+LD*, where linkage equilibrium is still stable, but where in addition there are two stable population genetical equilibria with linkage disequilibrium as well (the two linkage disequilibria correspond to whether *B* is associated with x_1 or with x_2);

(iii) Region *LD*, where linkage equilibrium is unstable, and only the two equilibria with linkage disequilibrium are stable.

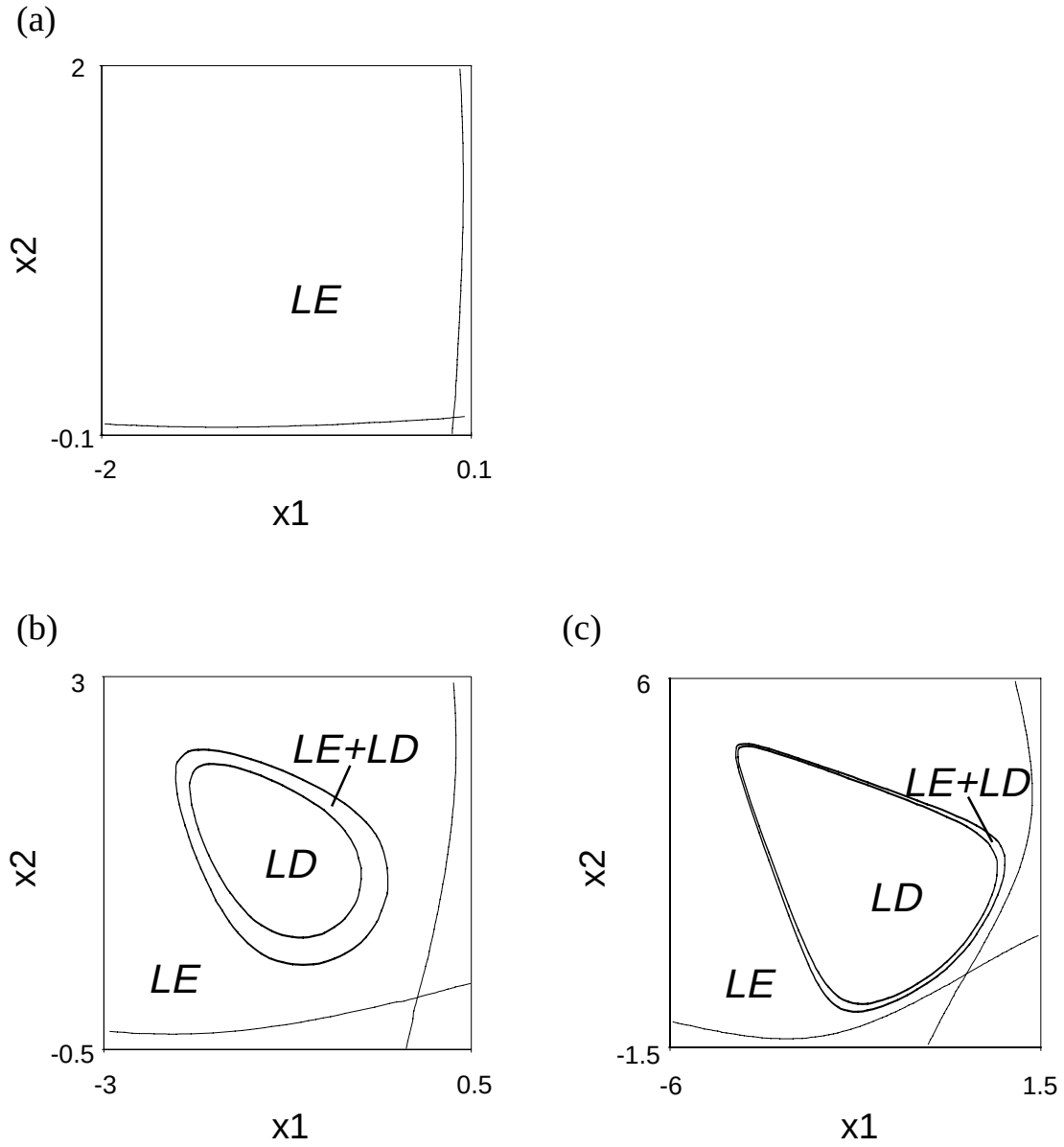

Figure 4. Population genetical equilibria of the resident population. *LE*: linkage equilibrium is stable, *LD*: there is a pair of stable equilibria with linkage disequilibrium, *LE+LD*: both linkage equilibrium and linkage disequilibrium are stable. *(a)* d/σ=2.25; *(b)* d/σ=3; *(c)* d/σ=5.

Linkage disequilibrium can be maintained if there is sufficiently strong selection against heterozygotes on the primary locus. Linkage disequilibrium is stable if heterozygote inferiority is stronger than $F_{as}^{(D)} = 0.07$ assuming Π=0.9 and free recombination (i.e., the border lines between part *LE* and part *LE+LD* in Figures 4b,c coincide with the F_{as}=0.07 contourlines in Figures 2b,c). Linkage equilibrium is unstable if $F_{as} > F_{as}^{(E)} = 0.12$. These thresholds do not depend on d/σ. Increasing penetrance decreases both $F_{as}^{(D)}$ and $F_{as}^{(E)}$ such that both become zero when Π=1; decreasing rate of recombination decreases $F_{as}^{(D)}$ but does not influence $F_{as}^{(E)}$.

Immediately after branching, fitness differences are small and therefore the population is in linkage equilibrium. As long as the population stays in linkage equilibrium, the evolution of the primary trait is not affected by the secondary locus, i.e., evolution proceeds according to Figure 1. In example (A), heterozygote inferiority never reaches the threshold of F_{as} necessary to obtain linkage disequilibrium (cf. Figure 2a): Although a protected polymorphism evolves, mating remains random with respect to the primary trait. In examples (B) and (C), however, heterozygote inferiority increases during evolution such that linkage equilibrium looses its stability (cf. Figures 2b,c and 3). As linkage disequilibrium develops, the branches of the evolutionary tree become partially reproductively isolated.

The population switches from linkage equilibrium to linkage disequilibrium already somewhere inside region *LE+LD*. The place of this switch depends on the initial frequency of allele *b*: If *b* is common, then linkage disequilibrium appears near the outer boundary of part *LE+LD*, i.e., almost immediately when heterozygote inferiority becomes strong enough to maintain linkage disequilibrium. If *b* is rare, however, then the population switches to linkage disequilibrium only near the inner boundary of *LE+LD*, where selection on the primary locus is stronger. The reason for this difference is the following. If *b* is rare, then most *b* alleles are in the large (*BB+Bb*) mating group, where recombination destroys the association between *b* an the primary allele: As a result, strong selection is necessary to maintain linkage disequilibrium and to spread *b*. If *b* is common, however, then the (*BB+Bb*) mating group is small. The rare allele *B* occurs exclusively in (*BB+Bb*), where *B* is common and consequently where the primary allele associated with *B* is overrepresented: Recombination is therefore less effective in destroying the association between the alleles. *B* and *b* are neutral as long as the primary locus is monomorphic or the population is in linkage equilibrium. The frequency of allele *b* before the population switches to linkage disequilibrium may be determined by mutation equilibrium between *B* and *b*, or may vary randomly by genetic drift.

As soon as the population switches to linkage disequilibrium, mating becomes nonrandom with respect to the primary locus, and this influences the fitness of a mutant allele. Generally, the initial dynamics of a mutant in the primary locus depends on whether it first co-occurs with *B* or *b*. However, if the invading mutant allele *y* is similar to a resident allele x_i, then the linkage between *y* and *B/b* converges fast to the linkage between x_i and *B/b* relative to the slow increase of *y*. The fitness of *y*, therefore, can be approximated assuming that *y* co-occurs with the resident alleles with the same frequency as x_i in equilibrium (see Box 2 for the analytic expression of fitness with nonrandom mating).

Figure 5 shows the evolution of a polymorphic population after linkage disequilibrium has been established. There is a single, symmetric, evolutionarily stable attractor in both examples (B) and (C). In the evolutionarily stable population, the two homozygotes are nearly optimal in the two patches, similarly to the evolutionarily stable dimorphism of the clonal model. Due to assortative mating, the two homozygotes are partially reproductively isolated: There are 63.1 per cent less heterozygotes compared to Hardy-Weinberg equilibrium among offspring before selection in example (B), and 88.6 per cent less in example (C). In Box 3, we present simulated evolutionary trees to demonstrate the effect of assortative mating on the evolutionary dynamics of the primary locus.

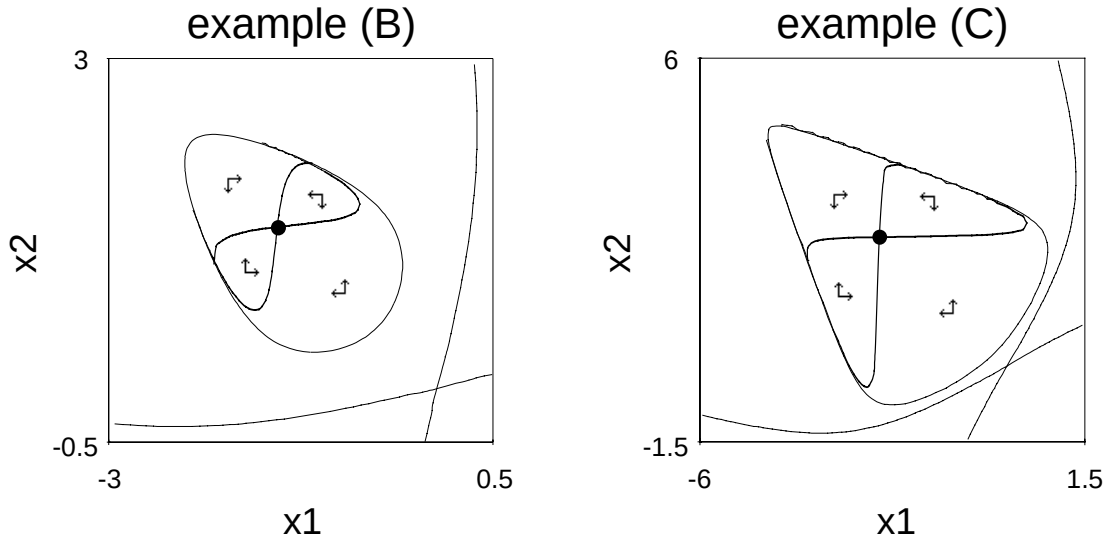

Figure 5. Evolution under linkage disequilibrium. The isoclines are drawn in the area where linkage disequilibrium is stable (*LE+LD* and *LD* in Figure 4) using the fitness function derived in Box 2. Notations as in Figure 1. Left panel: d/σ=3 (example (B)); right panel: d/σ=5 (example (C)).

Evolution of migration

In spatially heterogeneous environments, an alternative way to achieve reproductive isolation between locally adapted lineages is to decrease migration between the patches. In polymorphic populations, each allele is most frequent in the patch where it is most fit. It is therefore selectively advantageous for each allele to stay rather than to migrate: Mutants with lower migration rate always spread, independently of whether heterozygotes are inferior, and independently of linkage between the primary locus and the locus that controls migration (Balkau and Feldman, 1973). Restricted migration facilitates the evolution of locally adapted homozygotes and decreases the frequency of heterozygotes, and therefore influences the dynamics of evolution in a somewhat similar manner as assortative mating.

In the Levene model, assume that adults do not form a single mating pool, but mating is random within the patches and only a fraction m of adults migrates to the other patch before mating. The case of complete mixing (random mating in the entire population) corresponds to m=0.5. Figure 6 shows the evolution of polymorphic populations with reduced migration for example (B) (see Figure 1b for m=0.5). With moderate migration, a symmetric evolutionary attractor appears next to the two asymmetric attractors (Figure 6a); the stochastic evolutionary trajectory starting at the branching point ($x_1=x_2=x^*=0$) most probably leads to this symmetric attractor. With lower levels of migration, the asymmetric attractors are not reachable from the branching point at all (Figure 6b).

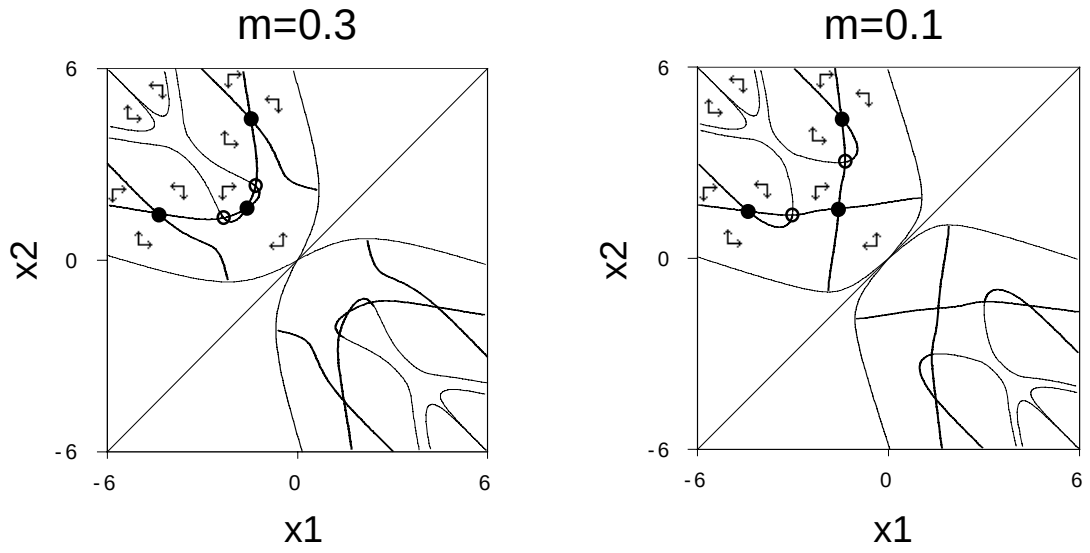

Figure 6. Evolution with restricted migration. Notations as in Figure 1. Left panel: m=0.3; right panel: m=0.1; d/σ=3 for both plots (example (B)).

The possible courses of evolution of the primary trait depend on the relative speed of evolution of the two loci. If migration evolves fast, i.e., if it attains low values soon after evolutionary branching, then the population evolves to the symmetric attractor (Figure 6b). At the symmetric attractor, the two homozygotes are optimal in the two patches, and since low migration ensures reproductive isolation, speciation is completed. If, however, the evolution of migration is very slow, then the population may reach one of the asymmetric attractors of Figure 1b before migration decreases appreciably. For convenience, assume that the attractor where the x_1 homozygotes are optimal in the first patch and the heterozygotes are optimal in the second patch has been reached. As migration decreases, the population will stay at the asymmetric attractor. At very low levels of migration, however, the asymmetric attractor looses its evolutionary stability, and x_1 undergoes evolutionary branching. The upper branch originating from x_1 and the x_2-branch gradually converge to the optimum in the second patch, until a single allele replaces both of them. The lower branch originating from x_1 remains optimal in the first patch. Eventually, therefore, the population reaches the symmetric attractor where the two homozygotes are optimal in the two patches. This happens, however, by an evolutionary detour through three-allele polymorphisms. Since for this scenario migration must evolve very slowly, the population will spend considerable time at the asymmetric attractor before it turns into a branching point and evolution to the symmetric attractor becomes possible.

In examples (A) and (C), the symmetric attractor exists already with complete mixing (m=0.5), therefore decreasing migration does not change qualitatively the evolution of the primary trait. As in example (B), the asymmetric attractors of example (C) become unreachable from the branching point if migration is small.

Discussion

In this paper, we investigated a model where genetic polymorphism is maintained by environmental heterogeneity, and examined whether reproductive isolation may evolve that turns the within-species genetic variability into between-species diversity. There is extensive literature both on multiple niche polymorphism and on the evolution of reproductive isolation. Most models, however, assume a fixed number of pre-defined alleles in the primary locus and investigate whether they may form a protected polymorphism, or whether reproductive isolation may be established in a population containing the pre-defined alleles. Contrary to this static picture, we assumed gradual evolutionary change in the primary locus: Which alleles will be present in the population is determined by which mutants spread and which alleles are driven to extinction by selection. Applying the adaptive dynamics framework of Metz et al. (1996) and Geritz et al. (1997, 1998) to diploid populations, we first investigated the conditions under which genetic polymorphism may come about by phenotypically small mutations, and identified the evolutionarily stable polymorphisms that cannot be invaded by any further mutant allele. Second, we examined the evolution of reproductive isolation by assortative mating and by reduced migration in the polymorphic populations resulted from the evolution of the primary locus, and the effect of these on the further evolution of the primary locus.

During evolutionary branching, intermediate heterozygotes have lower fitness than homozygotes such that assortative mating is advantageous. Though the evolution of assortative mating is highly controversial, some recent 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) seems to support the possibility. It greatly depends on the type of assortative mating whether or not it can be established during evolutionary branching. It is useful to distinguish three groups of possible assortative mating mechanisms (Felsenstein, 1981; Rice and Hostert, 1993):

(1) If the primary trait determines mate choice directly or pleiotropically (e.g. body size, emergence time or flowering time), then evolutionary branching immediately results in reproductive isolation (Templeton, 1981; Rice, 1984; Smith, 1988; Rice and Salt, 1990; Rice and Hostert, 1993; Bush, 1994). Sexual selection facilitates the evolution of reproductive isolation if there is disruptive selection on male characters (Lande, 1981, 1982; Galis and Metz, 1998).

(2a) If mate choice is determined by a separate locus, and there is a single allele that ensures assortative mating if present in all individuals, then this allele is readily spread even by weak selection ('one-allele' mechanisms sensu Felsenstein, 1981). Reduced migration (Balkau and Feldman, 1973), and increased 'choosiness' in mate choice based on the primary trait (Maynard Smith, 1966; Seger, 1985; Doebeli, 1996; Dieckmann and Doebeli, 1999) are examples for this possibility.

(2b) If mate choice is determined by a separate locus such that different alleles must be present in different branches in order to have assortative mating ('two-allele' mechanisms), then selection against heterozygotes must be sufficiently strong in order to overcome the homogenizing effect of recombination (Dickinson and Antonovics, 1973; Udovic, 1980; Felsenstein, 1981; Seger, 1985). As we have demonstrated, however, evolution of the primary trait may lead to strong heterozygote inferiority and thus to the establishment of assortative mating even in this case. Once partial reproductive isolation is achieved,

additional assortative mating mechanisms can evolve easier, because there are less heterozygotes and thus recombination occurs less frequently (Johnson et al., 1996). There is also selection for increased penetrance and tighter linkage (Udovic, 1980), perfecting reproductive isolation.

Reproductive isolation may also evolve before divergence in the primary trait (Palumbi, 1992; Henry, 1994; Turner and Burrows, 1995; Galis and Metz, 1998). If penetrance is complete in our assortative mating model (Π=1), then the population splits up into two reproductively perfectly isolated mating groups or sibling species (*BB* and *bb*, respectively) before evolutionary branching happens in the primary locus. As mutations limit the speed of evolution and they occur randomly, one or the other sibling species evolves faster by chance, and dooms the slower species to extinction. By mutations in the secondary locus, however, the extinct species may reappear. As soon as evolutionary branching begins, the two alleles of the primary locus get associated with the two sibling species such that the branches are reproductively isolated from the onset.

Continuous traits are usually influenced by many loci, with possibly many potential alleles in each locus. In this paper, we considered the extreme case of a single primary locus (or a single group of tightly linked loci) with many potential alleles. For directional evolution in monomorphic populations, quantitative genetic models and the adaptive dynamics approach are in good agreement (e.g., Abrams et al., 1993). Evolutionary branching with many loci determining the primary trait, however, is hindered by recombination in randomly mating populations: as the many intermediate genotypes are re-created in each generation, the phenotypic distribution of the population remains unimodal despite disruptive selection (Doebeli, 1996; Dieckmann and Doebeli, 1999). Reproductive isolation by assortative mating, however, restricts recombination and restores evolutionary branching.

Doebeli (1996) and Dieckmann and Doebeli (1999) demonstrated evolutionary branching in a simulation model with many unlinked loci and two alleles per locus, assuming that mating can be assortative by the primary trait and there is genetic variation in "choosiness" (i.e., in the tendency to choose mates with trait values similar to own). Once the evolution of the primary trait had arrived at the branching point, "choosiness" increased and the population split into two reproductively isolated lineages with divergence in the primary trait. In this way, evolutionary branching could be obtained under the same ecological conditions which lead to branching in the corresponding clonal model. Since 'choosiness' has to increase in both incipient branches for reproductive isolation between them, this scenario is related to the 'one-allele' mechanism (2a) above. Dieckmann and Doebeli (1999) also carried out simulations where mating was assortative by an arbitrary 'marker' trait rather than by the primary trait. In this case, linkage disequilibrium must develop between the primary and marker traits in order to achieve reproductive isolation, hence this scenario bears the same burdens as the 'two-allele' mechanisms (2b) above. Nevertheless, reproductive isolation evolved and evolutionary branching occurred in a considerable part of the parameter space.

We assumed that the alleles of the primary locus act additively on the phenotype. However, dominance is selected for in polymorphic populations (Sheppard, 1958; Wilson and Turelli, 1986), and even in monomorphic populations during allele substitutions (Wagner and Bürger, 1985). Van Dooren (1999) investigated the evolution of dominance in the present version of the Levene model. Assortative mating may evolve only as long as

heterozygotes are phenotypically intermediate and therefore are selected against during evolutionary branching. The evolution of dominance thus may be viewed as a rival mechanism to speciation by assortative mating: With complete dominance, assortative mating becomes selectively neutral. Whether assortative mating or dominance evolves faster depends for which there is more genetic variation in the population. If heterozygote inferiority is weak (d/σ is not too large in the Levene model), then only assortative mating mechanisms that have very high penetrance can get established, which may be not available at all. With stronger selection against heterozygotes, however, lower penetrance is sufficient, hence there may be several assortative mating mechanisms that can spread in the population. Both the evolution of assortative mating and the evolution of dominance may be viewed as possible ways to eliminate the phenotypically intermediate heterozygotes. Once the intermediate phenotype has disappeared due to assortative mating or dominant-recessive inheritance, the adaptive dynamics of the primary locus are identical to the clonal model.

In spatially heterogeneous environments, reproductive isolation may also be achieved by reduced migration (Balkau and Feldman, 1973) or by habitat choice (Diehl and Bush, 1989; de Meeûs et al., 1993; Kawecki, 1996, 1997). Reduced migration or habitat choice is advantageous because it increases the probability that an individual lives in the patch where it is best adapted to. Migration thus decreases even if heterozygotes are not inferior, if assortative mating has already been established, or if the primary trait has dominant-recessive inheritance. Selection for habitat choice is able to overcome recombination in case of 'two-allele' mechanisms (i.e., when carriers of one allele prefer one habitat while carriers of the other allele prefer the other habitat; Diehl and Bush, 1989; Kawecki, 1996, 1997). Costs of migration (such as investment into fruits instead of seeds or the risk of landing in unsuitable habitats for plants, and increased exposure to predators in animals) further strengthen selection for lower migration. Temporal fluctuations in the environment, however, select for increased migration or weaker habitat choice: If the fluctuations are not perfectly correlated over the patches, then migration can ensure survival in locally disastrous years (risk spreading; Levin et al., 1984).

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Box 1. Mutant's fitness in the Levene model with clonal inheritance and with diploid Mendelian genetics under random mating

Consider a rare mutant allele or clonal strategy. Let $< f_i >^{mut}$ be the survival probability of the mutant in patch *i* (*i*=1,2); in case of diploid inheritance, this is the weighted average of the survival probabilities of mutant heterozygotes. After selection, the within-patch frequency of the mutant increases by a factor $< f_i >^{mut} / < f_i >$, where $< f_i >$ is the average survival probability of the resident population. The logarithmic fitness of the mutant is therefore

$$\ln\left[\frac{1}{2}\frac{< f_1 >^{mut}}{< f_1 >}+\frac{1}{2}\frac{< f_2 >^{mut}}{< f_2 >}\right] \tag{B1.1}$$

Monomorphic resident populations

In a monomorphic population where all individuals have phenotype *x*, $< f_i >= f_i(x)$ ($f_i(x)$ is given by Eq. 1). In the clonal model, $< f_i >^{mut} = f_i(y)$; the logarithmic fitness of the mutant is therefore

$$s_x(y) = \ln\left[\frac{1}{2}\frac{f_1(y)}{f_1(x)}+\frac{1}{2}\frac{f_2(y)}{f_2(x)}\right] \tag{B1.2}$$

In a diploid population, a rare allele *y* occurs almost exclusively in heterozygotes with phenotype $(x+y)/2$. The survival probability of the mutant allele in patch *i* is therefore $< f_i >^{mut} = f_i\left(\frac{x+y}{2}\right)$, and the mutant's fitness is

$$s_x(y) = \ln\left[\frac{1}{2}\frac{f_1\left(\frac{x+y}{2}\right)}{f_1(x)}+\frac{1}{2}\frac{f_2\left(\frac{x+y}{2}\right)}{f_2(x)}\right] \tag{B1.3}$$

Polymorphic resident populations

In a clonal population with two resident strategies (x_1 and x_2) $< f_i >= pf_i(x_1) + qf_i(x_2)$, where *p* and *q* are the equilibrium frequencies of x_1 and x_2, respectively. Substituting into B1.1, the mutant's fitness is

$$s_{x_1,x_2}(y) = \ln\left[\frac{1}{2}\frac{f_1(y)}{pf_1(x_1)+qf_1(x_2)}+\frac{1}{2}\frac{f_2(y)}{pf_2(x_1)+qf_2(x_2)}\right] \tag{B1.4}$$

In a diploid population with alleles x_1 and x_2, a rare mutant allele *y* occurs in two heterozygotes with phenotypes $(x_1+y)/2$ and $(x_2+y)/2$, respectively. The survival probability of the mutant allele in patch *i* is the weighted average $< f_i >^{mut} = pf_i\left(\frac{x_1+y}{2}\right)+qf_i\left(\frac{x_2+y}{2}\right)$. The logarithmic fitness of the rare allele is

$$s_{x_1,x_2}(y) = \ln\left[\frac{1}{2}\frac{pf_1\left(\frac{x_1+y}{2}\right)+qf_1\left(\frac{x_2+y}{2}\right)}{< f_1 >}+\frac{1}{2}\frac{pf_2\left(\frac{x_1+y}{2}\right)+qf_2\left(\frac{x_2+y}{2}\right)}{< f_2 >}\right] \tag{B1.5}$$

where $< f_i >= p^2 f_i(x_1) + 2pqf_i\left(\frac{x_1 + x_2}{2}\right) + q^2 f_i(x_2)$ (i=1,2) are the average survival probabilities in the resident diploid population.

Box 2. The fitness of a mutant allele with nonrandom mating

Consider a resident population with alleles x_1 and x_2 in the primary locus, and alleles B_1 and B_2 in the secondary locus. Let y be a rare mutant derived from, and hence similar to, x_1. The linkage between y and the alleles of the secondary locus effectively stabilizes before y significantly increases in frequency. Since y and x_1 are similar, the linkage between y and the secondary locus is similar to the linkage between x_1 and the secondary locus. We calculated the fitness of y assuming that the linkage relationships have stabilized and the probability that y is in a zygote with ordered genotype $yx_jB_kB_l$ is the same as the probability that x_1 is in $x_1x_jB_kB_l$. The latter probability is z_{1jkl}/p_1, where z_{1jkl} is the frequency of $x_1x_jB_kB_l$ zygotes and $p_1 = \sum_{j,k,l} z_{1jkl}$ is the frequency of allele x_1. The equilibrium genotypic frequencies of the resident population were determined by numerical simulation (see Udovic, 1980 for the population genetical recursions). The average survival probability of allele y in patch i is given by

$$< f_i >^{mut} = \sum_{j,k,l} \frac{z_{1jkl}}{p_1} f_i\left(\frac{y+x_j}{2}\right) \qquad \text{(B2.1)}$$

(i=1,2), while the average survival probability of the resident population in patch i is

$$< f_i > = \sum_{h,j,k,l} z_{h,j,k,l} f_i\left(\frac{x_h+x_j}{2}\right) \qquad \text{(B2.2)}$$

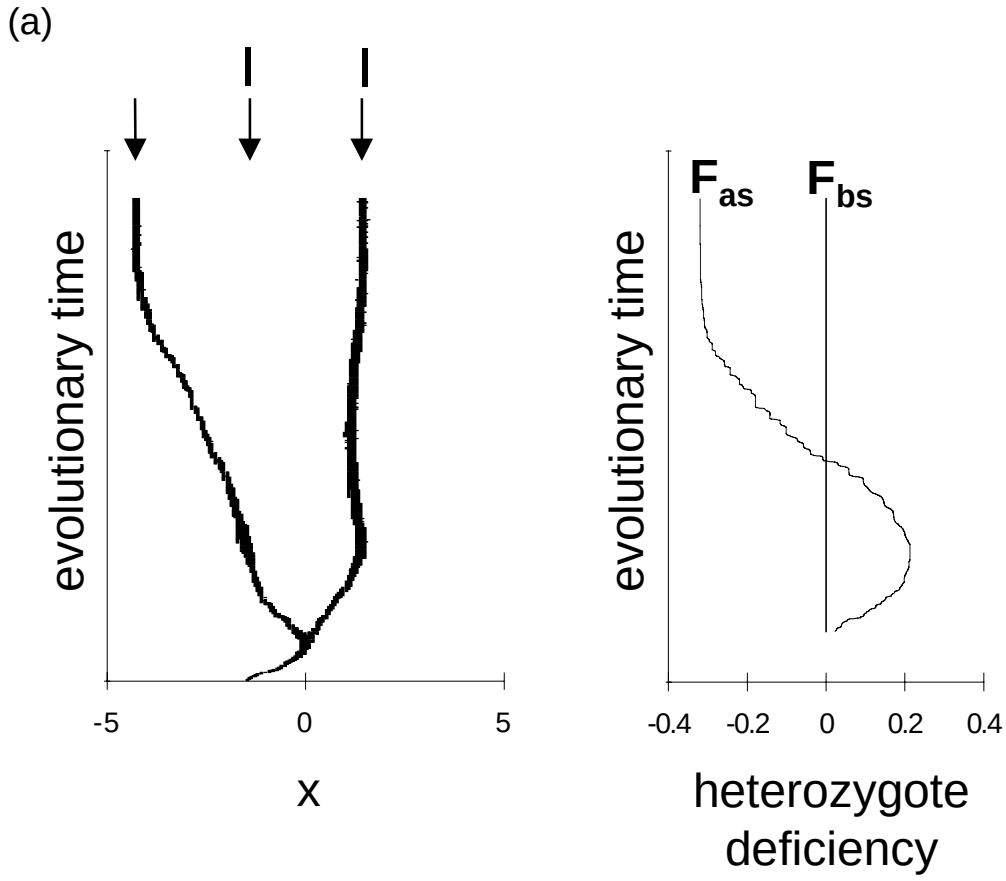
The fitness of the mutant allele can be obtained by substituting (B2.1) and (B2.2) into Eq. B1.1.

Box 3. Simulated evolutionary trees

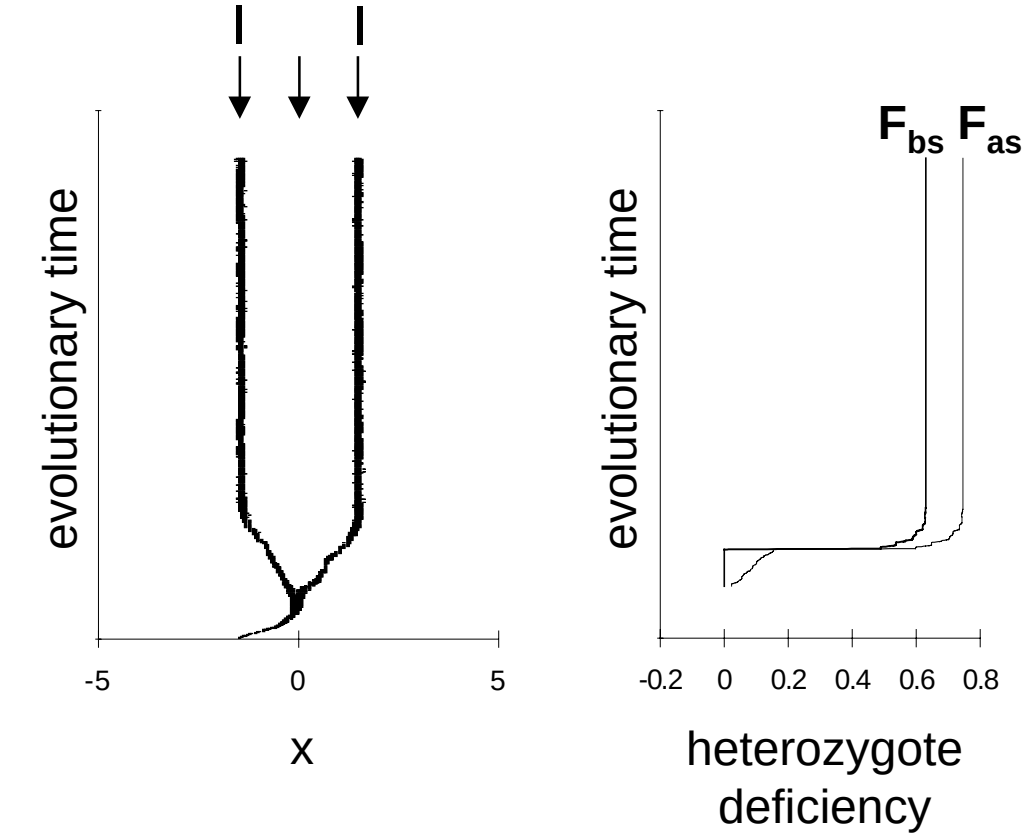
The figures of this box were obtained by direct simulations of the evolutionary process for example (B) (d/σ=3) with (a) random mating and (b) assortative mating (Π=0.9 and free recombination). The evolutionary trees in the left panels show which alleles are present in the primary locus, and the right panels show the deficiency of heterozygotes compared to Hardy-Weinberg equilibrium before (F_{bs}) and after selection (F_{as}).

After evolutionary branching at x^*=0, the randomly mating population (a) evolves to an asymmetric evolutionarily stable attractor, where one of the two homozygote phenotypes and the intermediate heterozygote phenotype (arrows) are near the within-patch optima (bars; cf. Figure 1b). F_{as} initially increases as selection against heterozygotes becomes stronger. Later in evolution, however, F_{as} is negative since heterozyogotes are superior (cf. Figure 3). F_{bs} is always zero because of random mating.

With a secondary locus for assortative mating (b), initially the two loci are in linkage equilibrium such that mating is random concerning the primary locus (F_{bs}=0). However, linkage disequilibrium develops as soon as heterozygote inferiority is strong enough, and assortative mating produces less heterozygotes in the primary locus (F_{bs} becomes positive). The abrupt change in F_{bs} is due to the population genetical bistability shown in Figure 4. The population evolves to the symmetric attractor (cf. Figure 5, left panel). The evolutionarily stable population is phenotypically similar to the clonal model: The two homozygotes are optimal in the two patches, while heterozygotes are to a large extent missing due to assortative mating.



(b)


evolutionary time
-5
0
5
x
evolutionary time
F_{bs} F_{as}
-0.2
0
0.2
0.4
0.6
0.8
heterozygote deficiency