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Three Modes of Adaptive Speciation in Spatially Structured Populations

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abstract: Adaptive speciation with gene flow via the evolution of assortative mating has classically been studied in one of two different scenarios. First, speciation can occur if frequency-dependent competition in sympatry induces disruptive selection, leading to indirect selection for mating with similar phenotypes. Second, if a subpopulation is locally adapted to a specific environment, then there is indirect selection against hybridizing with maladapted immigrants. While both of these mechanisms have been modeled many times, the literature lacks models that allow direct comparisons between them. Here we incorporate both frequency-dependent competition and local adaptation into a single model and investigate whether and how they interact in driving speciation. We report two main results. First, we show that individually, the two mechanisms operate under separate conditions, hardly influencing each other when one of them alone is sufficient to drive speciation. Second, we also find that the two mechanisms can operate together, leading to a third speciation mode in which speciation is initiated by selection against maladapted migrants but completed by within-deme competition in a distinct second phase. While this third mode bears some similarity to classical reinforcement, it is considerably faster, and both newly formed species go on to coexist in sympatry.

Keywords: parapatric speciation, adaptive speciation, assortative mating, frequency-dependent selection, reinforcement, local adaptation.

Introduction

In recent years, conceptual discussion has emerged on whether the old geographic classification of speciation processes as allopatric, parapatric, or sympatric is still useful (Dieckmann et al. 2004; Butlin et al. 2008; Fitzpatrick et al. 2009; Mallet et al. 2009). Until recently, theoreticians mainly studied sympatric models with initial panmixia (for reviews, see Via 2001; Bolnick and Fitzpatrick 2007) and allopatric models without gene flow, at least during an initial phase (for a review, see Servedio and Noor 2003). This traditional approach has been criticized for two main reasons. First, fully sympatric and allopatric speciation represent limiting cases of parapatric speciation, which comprises all intermediate levels of gene flow from very weak to very strong. Second, and more fundamentally, the geographic setting alone typically tells us very little about the mechanism of speciation.

Many researchers have therefore argued for a classification scheme that focuses on mechanisms rather than on geography. In one scenario, speciation occurs as an unselected by-product of (neutral or adaptive) divergence. An example is the evolution of postzygotic isolation due to the accumulation of Dozhansky-Muller incompatibilities (e.g., Gavrilets 2000; Orr and Turelli 2001; Kondrashov 2003). Alternatively, speciation (or reduced gene flow between incipient species) can be a direct target of selection; in a broad sense of the term, these cases are referred to as "adaptive speciation." In contrast to speciation occurring as a by-product, adaptive speciation usually works through the buildup of prezygotic barriers to gene flow (cf. Dieckmann et al. 2004; Doebeli and Dieckmann 2005; Weissing et al. 2011). An important and well-studied special case of adaptive speciation, which is also our focus here, is the adaptive evolution of assortative mating.

In spatially structured populations, assortative mating can evolve via two main mechanisms, which differ in the source of disruptive selection and in how polymorphism is maintained in a population. The first mechanism is driven by competition (Rosenzweig 1978). Many models of competitive speciation assume a sympatric setting (e.g., Dieckmann and Doebeli 1999; Doebeli and Dieckmann 2005; Doebeli et al. 2007; Ito and Dieckmann 2007; Otto et al. 2008; Pennings et al. 2008; Ripa 2009; Rettelbach et al. 2011), but there are also parapatric models (e.g., Meszéna et al. 1997; Day 2001; Doebeli and Dieckmann 2003; Heinz et al. 2009; Payne et al. 2011; Fazalova and Dieck-

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mann 2012). Speciation can occur in these models if frequency-dependent competition induces disruptive selection such that extreme phenotypes have an advantage over intermediates. If hybrids between the extreme phenotypes have such an intermediate (and thus, unfit) phenotype, then there is indirect selection for mating with similar phenotypes, potentially leading to prezygotic isolation.

The second mechanism for the evolution of assortative mating is driven by gene flow. This mechanism requires some initial divergence of subpopulations in a spatially structured environment, for example, due to local adaptation. Selection for assortative mating results when these subpopulations exchange migrants and matings with maladapted immigrants produce unfit hybrids. The classical scenario is reinforcement after secondary contact following initial divergence during an allopatric phase (e.g., Kirkpatrick 2000; Mizera and Meszéna 2003; van Doorn et al. 2009; Thibert-Plante and Hendry 2009). Such initial divergence, however, does not require allopatry and can even evolve with random mating across demes (e.g., in a Levene model; Kisdi and Priklopil 2011).

While these two mechanisms and the resulting paths to adaptive speciation have been treated separately in various model-based analyses, the combined effect of both mechanisms in a single model has never been analyzed. In this study, we use a simple deterministic model, which is well understood in the sympatric case and in which the different selective forces can easily be distinguished. Both classical models appear as limiting cases, and all levels of mixture between them can be analyzed. We find that the two mechanisms work in separate parameter regions, hardly influencing each other in parameter regions where a single mechanism alone is sufficient to drive speciation. Instead, we identify a third parameter region where both mechanisms combine in a characteristic chronological succession. We therefore conclude that adaptive speciation can proceed via three distinct spatiotemporal modes.

Model

We study a sexually reproducing diploid population. Our model builds on that by Pennings et al. (2008), which in turn is based on earlier models by Roughgarden (1972), Christiansen and Loeschcke (1980), Dieckmann and Doebeli (1999), and Matessi et al. (2001). While all of these earlier models considered a fully sympatric setting, our model incorporates spatial structure by considering two demes that are connected by gene flow. We examine two versions of our model that differ in their genetic assumptions. The first version, with fixed allelic effects for the divergent trait, is described below. A second version, in which allelic effects can evolve freely, is analyzed in appendix A.

Ecological Assumptions

Individuals have two traits of interest: an ecological trait X that controls specialization on certain types of a resource and a mating-preference trait that determines the degree of female choosiness. The population is subdivided into two demes that exchange migrants. Individuals mate and compete within demes, resulting in frequency-dependent, "soft" selection (Levene 1953; see also Ravigné et al. 2004, 2009). Migration occurs after birth, before selection and mating, and is symmetric between demes.

The ecological phenotype X is subject to two sources of natural selection, resulting from (1) differential intrinsic resource availability and (2) frequency-dependent resource competition among individuals with similar phenotypes. Accordingly, we assume that different ecological phenotypes specialize on different types of resources and thus have different carrying capacities. More precisely, we assume that the carrying-capacity function K is quadratic in X. Selection can be stabilizing, directional, or disruptive. For stabilizing (or, depending on X and θ, directional) selection, we use a unimodal distribution,

$$K(\mathrm{X}) = K_{\max}\left[1 - \frac{(\theta - \mathrm{X})^2}{2\sigma_{\mathrm{K}}^2}\right]. \tag{1}$$

For disruptive selection, K is instead given by an analogous U-shaped function,

$$K(\mathrm{X}) = K_{\min}\left[1 + \frac{(\theta - \mathrm{X})^2}{2\sigma_{\mathrm{K}}^2}\right]. \tag{2}$$

For stabilizing selection (eq. [1]), θ is the phenotype with maximal carrying capacity, while for disruptive selection (eq. [2]), it is the phenotype with minimal carrying capacity. Terms $K_{\max}$ and $K_{\min}$ denote the maximal and minimal carrying capacities, respectively, and σ_{K} scales the width of the carrying-capacity function. Selection is strong if σ_{K} is small. In the following, we use the parameter $k = 1 - K(\theta + 1)/K(\theta)$ to measure this strength of the selection component resulting from the shape of K. Selection is stabilizing if $k > 0$ and disruptive if $k < 0$. The phenotype with maximal or minimal carrying capacity may differ between demes. We assume that the two demes are symmetric; for example, if the optimum in deme 1 is θ, then it is $-\theta$ in deme 2.

As a second source of natural selection, individuals experience competition from other individuals. The amount of competition experienced by phenotype X can be expressed via an ecologically effective population size that weighs all potential competitors with phenotypes Y according to their competitive impact $\gamma(|\mathrm{X} - \mathrm{Y}|)$ on phenotype X,

$$C(\mathrm{X}) = \sum_{Y} \gamma(|\mathrm{X} - \mathrm{Y}|)N(\mathrm{Y}), \tag{3}$$

where the sum extends over all possible phenotypes, $N(\mathrm{Y})$ is the number of individuals with phenotype Y, and $\gamma(|\mathrm{X} - \mathrm{Y}|)$ measures the strength of competition between X and Y. We assume that γ is a Gaussian function of the phenotypic distance with mean 0 and variance σ_{C}^2,

$$\gamma(|\mathrm{X} - \mathrm{Y}|) = \exp\left[-\frac{(\mathrm{X} - \mathrm{Y})^2}{2\sigma_{\mathrm{C}}^2}\right]. \tag{4}$$

Competition induces frequency-dependent disruptive selection. When σ_{C} is large, individuals also compete with relatively different phenotypes; that is, their individual niche is wide. When σ_{C} is small, individuals mainly compete with their own phenotype, making the resultant negative frequency-dependent selection strong. Analogously to k, we use the parameter $c = 1 - \gamma(1)/\gamma(0) = 1 - \gamma(1)$ to measure the strength of this frequency dependence. The competition function and the carrying-capacity function combine to yield a phenotype-specific mortality of logistic type,

$$d(\mathrm{X}) = \frac{C(\mathrm{X})}{K(\mathrm{X})}. \tag{5}$$

In addition, individuals are affected by sexual selection due to assortative mating. Choosy females prefer males with an ecological phenotype X similar to their own (X thus takes the role of a "magic trait" that underlies both natural and sexual selection; Servedio et al. 2011). We assume that the mating probability ν between a female with phenotype X and a male with phenotype Y depends on their phenotypic distance according to a Gaussian shape,

$$\nu(|\mathrm{X} - \mathrm{Y}|) = \exp\left[-\frac{(\mathrm{X} - \mathrm{Y})^2}{2\sigma_{\mathrm{m}}^2}\right] = (1 - m)^{(\mathrm{X}-\mathrm{Y})^2}, \tag{6}$$

where $m = 1 - \nu(1)$ is the probability that a female rejects a male with a phenotypic distance of 1. Accordingly, m can take values between 0 and 1. A female with $m = 0$ is not choosy at all; if adopted by all females, then this implies random mating at the population level. Conversely, a female with $m = 1$ mates exclusively with males of her own ecological phenotype; if adopted by all females, then this implies complete reproductive isolation at the population level. Only females are choosy, and the mating genotype is not expressed in males. Based on these mating probabilities, we can assign a mating success $\phi(\mathrm{X})$ to different ecological phenotypes. In particular, the mating success of phenotype X is the average of the corresponding female and male mating successes, $\phi(\mathrm{X}) = [\phi_{\mathrm{f}}(\mathrm{X}) + \phi_{\mathrm{m}}(\mathrm{X})]/2$, with

$$\begin{aligned} \phi_{\mathrm{f}}(\mathrm{X}) &= \sum_{Y} N_{\mathrm{m}}(\mathrm{Y})\nu(\mathrm{X}, \mathrm{Y})Q(\mathrm{X}), \\ \phi_{\mathrm{m}}(\mathrm{X}) &= \sum_{Y} N_{\mathrm{f}}(\mathrm{Y})\nu(\mathrm{Y}, \mathrm{X})Q(\mathrm{Y}). \end{aligned} \tag{7}$$

Here $N_{\mathrm{m}}(\mathrm{Y})$ and $N_{\mathrm{f}}(\mathrm{Y})$ represent the numbers of males and females, respectively, with phenotype Y. In our numerical analyses, we always assume a 1 : 1 sex ratio ($N_{\mathrm{m}} = N_{\mathrm{f}} = N/2$). Term $Q(\mathrm{X})$ is a normalizing factor that scales the mating activity of females. We obtain this factor by assuming that choosiness is not costly for females ($\phi_{\mathrm{f}}(\mathrm{X}) = 1$ for all X), which leads to

$$Q(\mathrm{X}) = \frac{1}{\sum_{\mathrm{Y}} N_{\mathrm{m}}(\mathrm{Y})\nu(\mathrm{X}, \mathrm{Y})}. \tag{8}$$

While this guarantees that all females have equal mating success, the same is not true for males. Instead, female choosiness induces sexual selection against males with rare phenotypes (Doebeli and Dieckmann 2003; Pennings et al. 2008).

Based on the mortality in equation (5) and the mating success in equation (7), we define the fitness $W(\mathrm{X})$ of a phenotype X as,

$$W(\mathrm{X}) = \phi(\mathrm{X}) \exp\left(\rho[1 - d(\mathrm{X})]\right), \tag{9}$$

with ρ denoting the intrinsic growth rate. The phenotypic distribution $N(\mathrm{X})$ after selection is thus given by

$$\tilde{N}(\mathrm{X}) = N(\mathrm{X}) \exp\left(\rho[1 - d(\mathrm{X})]\right), \tag{10}$$

and the number $B(\mathrm{X})$ of births of individuals with phenotype X is given by

$$B(\mathrm{X}) = \sum_{\mathrm{Y,Z}} \tilde{N}_f(\mathrm{Y})\tilde{N}_m(\mathrm{Z})\nu(|\mathrm{Y} - \mathrm{Z}|)Q(\mathrm{Y})R_{\mathrm{YZ}\to\mathrm{X}}, \tag{11}$$

where $R_{\mathrm{YZ}\to\mathrm{X}}$ is the probability that parents with phenotypes Y and Z produce offspring with phenotype X. All mated individuals are assumed to have the same number of offspring. We assume nonoverlapping generations, so $B(\mathrm{X})$ equals $N(\mathrm{X})$ for the next generation, before migration. Subsequently, a fraction μ of this offspring migrates to the other deme. To summarize, our model features three different components of selection:

1. Frequency-independent natural selection is described by the parameters k and θ, which determine the carrying-capacity function. This fitness component can be stabilizing ($k > 0$), disruptive ($k < 0$), or directional (if the phenotypic range is appropriately restricted; see below). We can interpret this function as reflecting a distribution of resources. In particular, disruptive selection ($k < 0$) describes a situation with two resources. For $\theta \neq 0$, one resource is more abundant in deme 1 and the other is more abundant in deme 2.

2. Negatively frequency-dependent natural selection due

to competition between similar phenotypes is governed by the parameter c. It is this source of selection that allows local coexistence of different phenotypes (i.e., maintenance of polymorphism), which is a prerequisite for competitive speciation. If $c = 0$, then all individuals compete equally strongly with one another. In this case, only a single phenotype/allele can prevail within a deme. Intermediate values of c typically induce frequency-dependent disruptive selection: intermediate phenotypes then suffer competition from two sides and thus have lower fitness than the extreme phenotypes, which are partially protected from competition. Very large values of c can enable a coexistence of intermediate and extreme phenotypes. In the following, we will therefore say that depending on the magnitude of c, frequency-dependent competition creates either two or three ecological niches.

3. The final source of selection in our model is sexual selection on males due to assortative mating. Since female choosiness favors males with common phenotypes, this type of sexual selection is positively frequency dependent (Pennings et al. 2008). The strength of choosiness is evolvable and is measured by the variable m.

Genetic Assumptions

We assume that the ecological trait X is determined by a single diploid locus with two alleles, whose effects are additive and lie symmetrically around 0. Above, we have defined the carrying-capacity function to possess the same symmetry. By considering symmetric allelic effects, we thus assume that the ecological trait evolves in accordance with this environmental symmetry. We relax this assumption in appendix B. The effects of the ecological alleles x are -0.5 and 0.5, such that the ecological phenotypes X are -1, 0, and 1. Note that with these phenotypic values, frequency-independent selection is stabilizing or disruptive if $\theta < 0.5$ and directional if $\theta > 0.5$.

Like the ecological trait, the mating trait is also determined by a single diploid locus without dominance: it can take values from 0 (random mating) to 1 (complete isolation). In our numerical analyses, the mating alleles have values $m = 0, 1/6, 2/6, 3/6$ and follow a stepwise mutation model with a mutation probability of $u = 10^{-5}$ per generation.

To analyze our model, we numerically iterate a version of equation (11), in which phenotypes are replaced by genotypes and the effects of segregation, free recombination, and mutation are captured by the function R (from eq. [11]). The initial conditions are $m = 0$ and nearly equal frequencies of the ecological alleles (one allele has frequency 0.51 in one deme and 0.48 in the other deme to avoid artifacts arising from symmetric initial conditions).

Model Parameters

As specified above, our model features 10 parameters. For the long-term evolutionary dynamics, however, this number can be reduced. First, K_{max}, K_{min}, and ρ scale the population dynamics but do not affect the evolutionary dynamics. Second, u and the number of alleles for m scale the speed of evolution but do not affect its long-term outcome (as long as u is small enough and the number of alleles large enough). Third, the allelic values for X can be chosen as unity without loss of generality. Our model thus has four essential parameters: μ, θ, k, and c. Below we give a comprehensive analysis of the evolutionary dynamics by varying these parameters.

Results

In the following, we describe our results for the model version with fixed allelic effects. Results for the model version with evolvable effects are summarized in appendix A; they confirm and refine our findings for the fixed-effect model.

Figure 1 shows how evolutionary outcomes vary with model parameters. The colored regions comprise parameter combinations for which complete reproductive isolation evolves. As detailed below, we find three different spatiotemporal modes of adaptive speciation, which we will refer to, respectively, as migration-independent competitive speciation, migration-driven ecological speciation, and migration-induced competitive speciation. Speciation in the migration-independent competitive mode (fig. 1, red regions) takes place within a single deme due to intrademe competition. Speciation in the migration-driven ecological mode (fig. 1, green regions) occurs between demes, due to selection against mating with maladapted immigrants (this is similar to classical reinforcement but without a preceding allopatric phase). Finally, speciation in the migration-induced competitive mode (fig. 1, blue regions) begins between demes due to selection against mating with immigrants and is completed within demes due to intrademe competition. Before further explaining these three modes of speciation, we briefly summarize results for those parts of the parameter space in which speciation does not occur. For a detailed description of these cases, see Pennings et al. (2008) and Rettelbach et al. (2011).

No Speciation

Random Mating. Whenever the heterozygotes (with phenotype 0) have the highest fitness, no degree of choosiness evolves. Under stabilizing selection ($\theta < 0.5$ and $k > 0$), this is the case if frequency-independent selection is strong

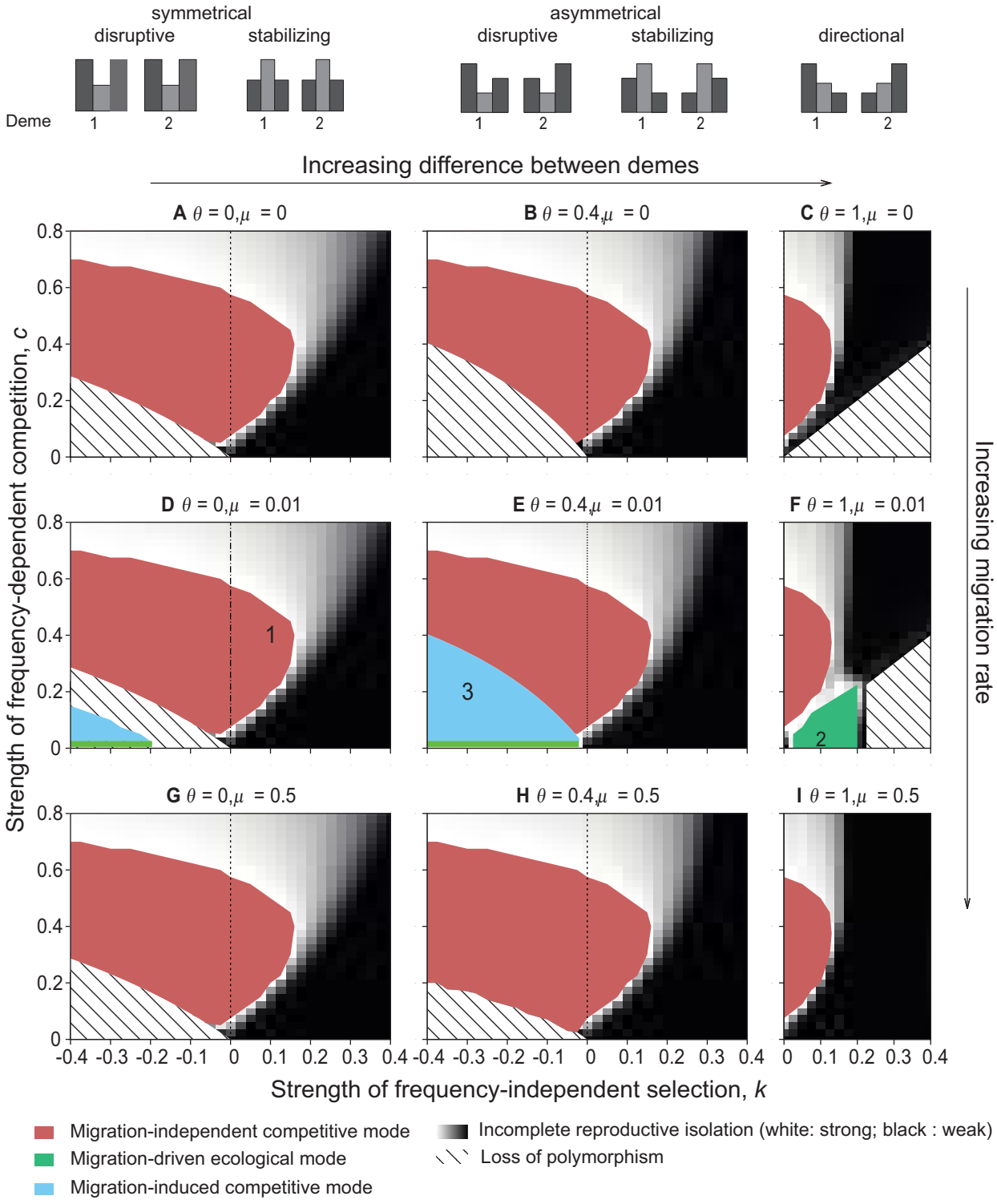

Figure 1: Overview of evolutionary outcomes resulting from varying the four essential parameters. Colored areas refer to the three modes of adaptive speciation discussed in the main text (red = migration-independent competitive mode; green = migration-driven ecological mode; blue = migration-induced competitive mode), with the numbers marking the parameter combinations used in figure 2. Hatched areas indicate loss of polymorphism, while shaded areas correspond to incomplete reproductive isolation, ranging from very light gray (nearly complete isolation) to black (random mating). Bar diagrams illustrate the carrying-capacity function for the three ecological

(large k) and frequency-dependent competition is weak ($k > c$; fig. 1, black area with high k in first and second columns). With directional selection ($\theta > 0.5$), heterozygote advantage can arise either because frequency-independent selection is strong but frequency-dependent competition is also strong (high k and high c) or because the intermediate phenotype is favored as a generalist strategy in the presence of strong migration (fig. 1*I*, black area).

Partial Isolation. Under strongly frequency-dependent selection (high c), each phenotype occupies its own niche, and choosiness evolves to intermediate values until the heterozygotes are rare enough to no longer experience a fitness disadvantage through competition (fig. 1, gray regions).

Loss of Polymorphism. Speciation fails if the polymorphism at the ecological locus is lost. Without migration (i.e., an isolated deme, $\mu = 0$; fig. 1, top row), this happens if frequency-dependent competition is too weak (c is low) to counteract the effect of frequency-independent disruptive ($k < 0$) or directional ($\theta > 0.5$) selection. This happens for $c < 1 - (1 - \theta^2 k)/[1 - (\theta - 1)^2 k]$ (fig. 1*A*, 1*B*, hatched areas) and for $c < 1 - \exp(-k)$ (fig. 1*C*, hatched area), respectively. For $\theta > 0$, if the population becomes monomorphic, then it is always for the locally adapted allele, while for $\theta = 0$, one or the other allele becomes fixed, depending on initial conditions.

When two demes are connected by rare migration (e.g., $\mu = 0.01$; fig. 1, middle row), spatial heterogeneity favors local adaptation and, thus, divergence. As shown in figure 1*E*, 1*F*, the loss of polymorphism at low c seen in the sympatric case ($\mu = 0$) is prevented at higher θ. For $\theta = 0$, it still depends on initial conditions which allele becomes fixed in each deme, as mentioned above. At low migration rates, it is therefore possible that different alleles nearly fix in the two demes, thus maintaining the population-level polymorphism. Stronger frequency-dependent selection (larger c) tends to equalize the allele frequencies across demes, such that typically the same allele goes to fixation in both demes. This can be seen in the monomorphic stripe in figure 1*D* (hatched area); there c is not large enough to support polymorphism within demes, but it is large enough to equalize allele frequencies in the two demes such that the overall polymorphism is lost. For $c = 0$, $\theta = 0$, and small migration rate ($\mu < 0.06$), we obtain numerical results that confirm the condition for the stability of the polymorphism found by Karlin and McGregor (1972): the polymorphism is locally stable if $k < -6\mu$ (results not shown).

For frequent migration ($\mu > 0.2$; fig. 1, bottom row), local adaptation becomes impossible, and the polymorphism is again lost at low c.

Three Spatiotemporal Modes of Adaptive Speciation

Migration-Independent Competitive Mode. Frequency-dependent competition of intermediate strength creates two ecological niches; once these are occupied, intermediate phenotypes are disfavored. This leads to indirect selection against mating between the opposite homozygotes (as such, a mating would produce unfit heterozygous offspring) and, therefore, to the evolution of assortative mating. This is the classical competitive-speciation scenario (Rosenzweig 1978), which has been thoroughly studied in models of sympatric speciation (e.g., Dieckmann and Doebeli 1999; Pennings et al. 2008).

Indeed, competitive speciation is the only mechanism in our model that works in sympatry ($\mu = 0$). Our results for higher migration rates show that this mode operates largely independently of spatial structure, as can be seen from comparing the red areas in figure 1. In other words, migration has little effect because (essentially sympatric) speciation happens in parallel in the two demes. The corresponding evolutionary dynamics (fig. 2, first row) show that the ecological alleles reach a frequency of 0.5 very quickly. Subsequently, choosiness increases (fig. 2*A*), and the frequency of heterozygotes declines to zero. In this manner, speciation is completed on a timescale of a few thousand generations.

Migration-Driven Ecological Mode. Spatial heterogeneity is the only relevant source of divergent selection when selection is directional within demes ($\theta > 0.5$) and frequency-dependent competition is not strong enough to cause within-deme disruptive selection. Without migration, the allele $x = -1$ fixes in deme 1 and the allele $x = 1$ fixes in deme 2 (fig. 1*C*, hatched area). With weak migration (and, thus, a high probability that offspring stay in their natal deme), selection disfavors mating with immigrants, and choosiness increases accordingly. With strong migration, the generalist, intermediate phenotype

phenotypes. For $\theta > 0.5$ (right column), selection is directional across the range of possible phenotypes for any choice of k, so only the part for $k > 0$ is shown (the part with $k < 0$ would be redundant). We show three representative migration rates μ. The migration-driven ecological speciation mode and migration-induced competitive speciation mode can be observed for migration rates of up to approximately 0.2. Note that $\mu = 0$ and $\mu = 0.5$ correspond to full allopatry and full sympatry, respectively. In these cases, only migration-independent competitive speciation can occur. (If, in addition, $\theta = 0$, there is no spatial structure, which is why *A* and *G* are alike.)

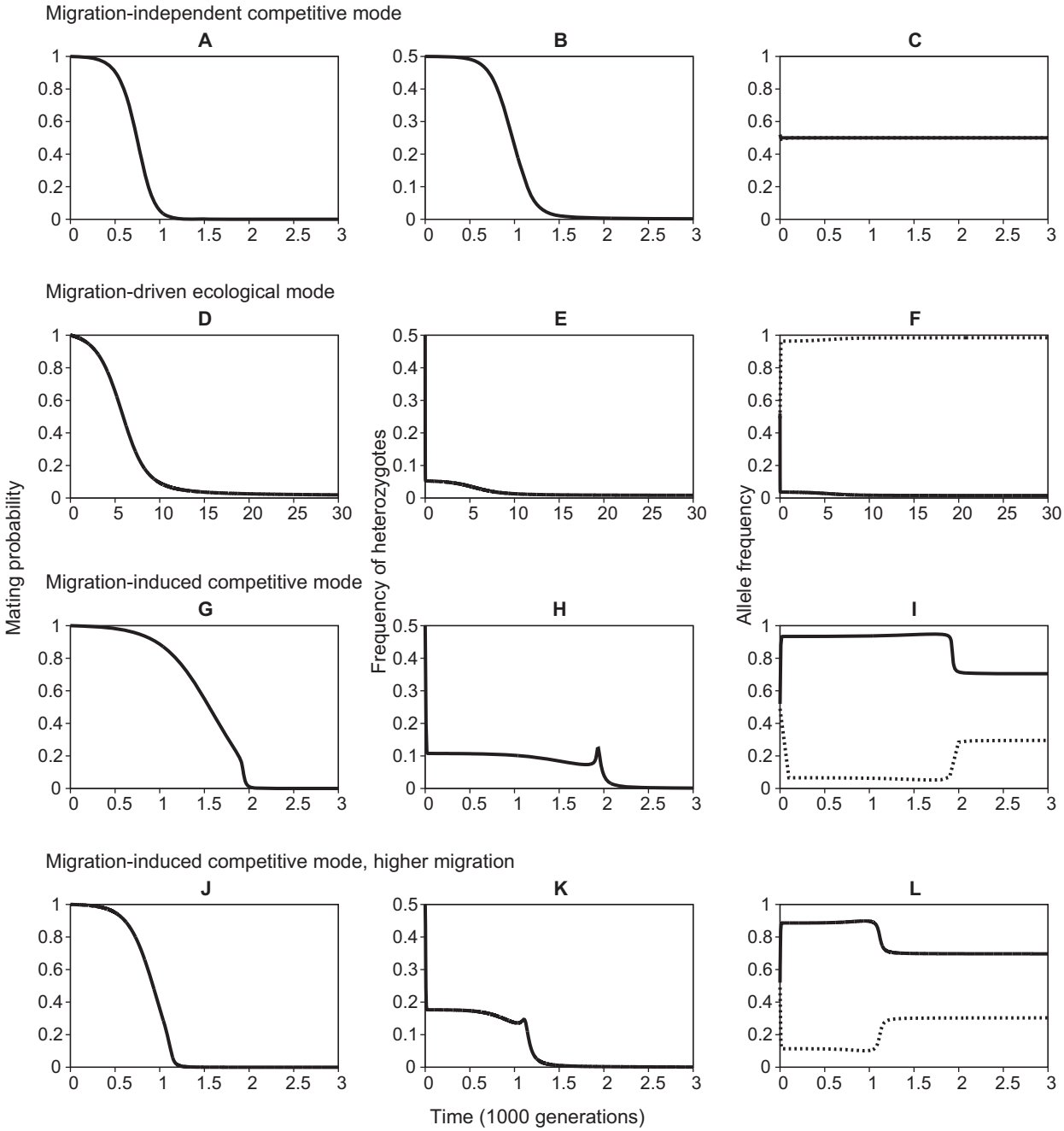

Figure 2: Evolutionary dynamics for the three different spatiotemporal modes of speciation in terms of the mating probability between different homozygotes (first column); the frequency of heterozygotes (second column); frequency of the $x = 1$ allele (third column) in deme 1 (solid lines) and deme 2 (dotted lines). Note the longer timescale for the migration-driven ecological mode. For the migration-induced competitive mode, comparison between *G–I* (lower migration rate) and *J–L* (higher migration rate) illustrates the influence of the migration rate on the speed of this mode's initial migration-dependent phase. *A–C*, "1" in fig. 1*D*: $\mu = 0.01$, $\theta = 0$, $k = 0.1$, $c = 0.4$; *D–F*, "2" in fig. 1*F*: $\mu = 0.01$, $\theta = 1$, $k = 0.1$, $c = 0$; *G–I*, "3" in fig. 1*E*: $\mu = 0.01$, $\theta = 0.4$, $k = -0.3$, $c = 0.2$; *J–L*: $\mu = 0.02$, $\theta = 0.4$, $k = -0.3$, $c = 0.2$.

has an advantage, and no speciation occurs (especially for low k, for which directional selection is weak anyway).

This is a reinforcement-like scenario (fig. 1*F*, green region), which would play out very similarly under secondary contact (i.e., if μ were initially set to 0; cf. Servedio and Noor 2003). It is well known that the evolution of high levels of assortative mating can be extremely slow under this mechanism, and full isolation may never be reached (see "Discussion" for details). Indeed, in our model, choosiness does not become strong enough to complete speciation over tens of thousands of generations (~1% heterozygotes remain). Frequency-dependent competition has a negligible net effect on this mechanism because it works in two opposite directions. On the one hand, increasing c increases the viability and frequency of immigrants, which in turn increases selection for assortative mating. On the other hand, higher c also increases the fitness of hybrids, which in turn decreases selection for assortative mating. The green area in figure 1*F* is separated from the red area by a small strip, where frequency dependence is strong enough to favor rare types but not strong enough to cause divergence within one deme. The evolved choosiness in this parameter region is clearly lower than in the parameter regions allowing for speciation.

Speciation via this migration-driven mode can occur even in the absence of spatial heterogeneity, provided frequency-independent disruptive selection is symmetric and different alleles initially are sufficiently prevalent in both demes. As shown in the "Loss of Polymorphism" section, this is possible for weak migration, negative k, and $c = 0$ (fig. 1*D*, 1*E*, green lines). Choosiness can then evolve due to selection against hybrid heterozygotes, even though immigrant and resident homozygotes may possess equal fitness.

The migration-independent and migration-driven modes described above are classical routes to sympatric speciation and parapatric speciation, respectively, which are already well known from previous studies. Our analysis shows that these modes occur in separate parameter regions. This indicates that for such speciation dynamics, the underlying mechanisms—competition and spatial heterogeneity—do not interfere with each other. There is, however, a third parameter region, in which both mechanisms act in concert, thereby creating a new mode of speciation.

Migration-Induced Competitive Mode. For a bimodal resource distribution ($k < 0$) and frequency-dependent competition ($c > 0$), two niches exist in each deme, such that two reproductively isolated species can locally coexist. In the absence of spatial structure, however, this ecological dimorphism tends to be unstable under random mating. This is because a rare allele finds itself (relatively) more often in unfit heterozygous genotypes and will be driven to extinction unless frequency dependence of competition is sufficiently strong. In contrast, with spatial structure, local extinction is avoided and speciation becomes possible. Note that spatial heterogeneity ($\theta > 0$) is not necessary but makes the speciation process more robust to initial conditions.

The third row of figure 2 shows a situation with weak migration. Speciation proceeds in two phases. In a first, migration-dependent phase, each deme is dominated by one of the two alleles. The locally rare allele is maintained only by migration-selection balance. Selection acts against mating with immigrants because heterozygous offspring have reduced fitness. As choosiness increases, the risk of such maladaptive mating decreases, as does the fitness disadvantage of the immigrants because they also mate more frequently among their own phenotype. Once a threshold value of choosiness is reached, the immigrants are able to maintain themselves. They form a self-contained colony that no longer depends on recurrent immigration; the second local niche has become available to them. In the course of the underlying evolutionary dynamics, this moment is signaled by a sudden steep increase in the frequency of the locally rare allele (fig. 2*I*, 2*L*).

At this point, the second, migration-independent phase of the speciation process sets in. The increased size of the immigrant "colony" leads to an increased number of matings between extreme phenotypes, which induces a sharp peak in the frequency of heterozygote hybrids (fig. 2*H*, 2*K*). Since heterozygotes continue to be unfit, selection against these matings increases sharply, leading to a further increase in choosiness. This, in turn, increases the size of the immigrant colony, which further reinforces selection for choosiness. Through this positive feedback, complete reproductive isolation evolves rapidly.

Thus, the first, migration-dependent phase of the speciation process works similarly to standard between-deme ecological speciation. The length of this phase decreases with increasing migration rate (fig. 2, cf. third and fourth rows). The second, migration-independent phase resembles a competitive-speciation scenario, in which both ecological phenotypes are present at high frequency, leading to a fast increase in reproductive isolation. In this phase, speciation will go to completion even if migration is completely stopped (i.e., in sympatry; results not shown).

Some aspects of a two-phase pattern in the evolution of assortative mating can be observed also in parts of the migration-independent parameter range (fig. C1 in app. C, upper left panel). However, in this case, both phases are driven by local competition. The characteristic property of the migration-induced competitive mode is that its first phase relies on immigration, and speciation cannot occur without this trigger.

Discussion

We have analyzed the evolution of assortative mating in a model with both within-deme frequency-dependent competition and between-deme environmental heterogeneity. While both factors can cause speciation, they do not act synergistically across a large region of parameter space. They may, however, combine in a characteristic chronological order in the presence of a third source of disruptive selection, that is, a bimodal carrying-capacity function.

We have thus demonstrated the existence of three distinct spatiotemporal modes of adaptive speciation—a migration-independent mode, which depends on within-deme competition; a migration-driven mode, which is based on ecological differences among demes; and a new migration-induced mode, in which partial isolation evolves as in the migration-driven mode—but speciation is completed due to competition within demes, as in the migration-independent mode. In all three modes, speciation is adaptive; that is, assortative mating evolves as an adaptation against the production of unfit hybrid offspring. However, the three modes differ markedly in the geographical patterns during and after speciation (fig. 2) and in their dependence on spatial structure and migration rates.

Three Modes of Adaptive Speciation

Whether or not the two considered driving forces of adaptive speciation—between-deme heterogeneity and within-deme competition—interact synergistically depends on the shape of the resource distribution as described by the carrying-capacity function. If the resource distribution within demes is unimodal or flat, then the two driving forces cannot complement each other. There is thus no smooth transition between the corresponding speciation modes (note that the respective parameter regions in figs. 1 and A1 are not contiguous). The reason is that migration-driven ecological speciation and migration-independent competitive speciation are based on different ecological niche structures.

In the migration-driven mode, speciation proceeds if each deme contains a single niche and these niches are ecologically incompatible. Evolution of assortative mating then proceeds through selection on residents to avoid mating with maladapted immigrants. Adding weak frequency-dependent competition to such a scenario has little effect, as long as it does not create a second local niche. (Consequently, the boundaries of the corresponding parameter region in fig. 1 are independent of c as long as $c < 1 - \exp(-k)$).

In contrast, speciation in the migration-independent competitive mode proceeds if strong frequency-dependent competition creates two niches in each deme. The evolution of assortative mating is then driven by local interactions between residents. Adding migration and spatial heterogeneity to such a competitive speciation scenario has little impact on the parameter region allowing speciation, at least in the model with fixed allelic effects (fig. 1). The reason is that in this model, both demes contain the same phenotypes (albeit at different frequencies), so immigration does not fundamentally change selection within demes.

This also remains true for evolving allelic effects (app. A). In this case, however, migration has an indirect effect on speciation by influencing the phenotypic distance between the homozygotes (fig. A1). Weak migration drives these homozygotes further apart (to reduce competition with immigrants), thus lowering competition and inhibiting speciation. In contrast, strong migration brings them closer together (to increase fitness of migrants), thus increasing competition and facilitating speciation. Still, if speciation occurs, then it is clearly driven by within-deme interactions.

Whereas competition and spatial heterogeneity do not combine synergistically with a unimodal resource distribution, this changes with a bimodal distribution. In this case, there are two niches in each deme, but one of these niches cannot be occupied as long as assortative mating is weak and immigrants suffer a high cost of hybridization. In the migration-induced competitive mode, the first phase of speciation resembles the migration-driven ecological mode; that is, assortative mating increases due to selection on residents against mating with immigrants. As assortative mating becomes stronger, immigrants hybridize less frequently, and occupation of the second niche becomes possible. Once this happens, speciation continues and is completed separately within each deme.

Our results clearly show the limitations of a geographical classification of speciation. All scenarios discussed here are parapatric, according to the definition by Gavrilets (2003). Nevertheless, gene flow plays radically different roles in the three speciation modes. On the one hand, there is almost no effect of gene flow in the migration-independent competitive mode and in the second phase of the migration-induced competitive mode. In both cases, evolution of reproductive isolation is driven by within-deme competition—a classical "sympatric" mechanism. On the other hand, gene flow is vital for the migration-driven ecological mode and in the first phase of the migration-induced competitive mode, which in many aspects are similar to reinforcement. As a consequence, the three modes of speciation lead to entirely different patterns in the distribution of phenotypes (and genotypes) through space and time. On the one hand, the two migration-dependent modes require strong initial spatial differentiation, in contrast to

the migration-independent competitive mode; on the other hand, the two competitive modes lead to coexisting species in sympatry, in contrast to the migration-driven ecological mode.

Model Assumptions and Generality of Results

Our model has been designed as a minimal model to understand the interplay between different sources of selection for the evolution of reproductive isolation. Such models do not attempt to emulate a specific biological system but rather try to uncover general principles. To this end, they make a large number of simplifying assumptions. In our case, this concerns, in particular, the genetics of the ecological trait and the mating trait. We have confirmed the robustness of our results to the most severe genetic constraints, that is, the assumption of fixed allelic effects (app. A). Another unrealistic assumption that could influence the outcome is the symmetry of fitness functions and migration rates. Indeed, Servedio and Kirkpatrick (1997) found that reinforcement can be sensitive to asymmetric migration. Therefore, in additional numerical analyses (app. B), we specifically tested whether asymmetric migration affects the migration-induced competitive speciation mode. We find that even a strong asymmetry does not change our qualitative results. The only exception is strictly unidirectional migration (a continent-island model), where speciation cannot be completed because females on the continent do not evolve assortative mating (and, hence, female immigrants keep hybridizing).

Our symmetry assumption for the fitness function implies that the fitness of phenotype 1 in deme 1 is the same as the fitness of phenotype 2 in deme 2 and vice versa. Again, additional numerical analyses show the robustness of our results (app. B). In particular, migration-induced competitive speciation still occurs in one of the demes if there is disruptive selection in this deme only and directional selection in the other deme (with maximum fitness at the lower fitness peak of the first deme).

In this study, we did not assume costs for assortative mating. Costs that are related to finding a mating partner (such as limited trials or predation risk during search) should have a very weak effect on the migration-dependent mechanisms. The populations are nearly monomorphic, and the resident females should have no problem finding a proper partner. In the migration-independent phase of the migration-induced competitive mechanism, such search costs could make it more difficult for the rare phenotype to establish because they increase positive frequency dependence (Kopp and Hermisson 2008).

Another simplifying assumption is the use of a "magic trait," that is, a trait under disruptive selection that also contributes to mate choice. Servedio et al. (2011) argue that such magic traits may not be rare in nature. In the alternative scenario, in which the trait under divergent selection differs from that determining mate choice, reproductive isolation can evolve only if linkage disequilibrium between the two traits develops. For a single sympatric population, this can be difficult (Felsenstein 1981; Gavrilets 2005), although Dieckmann and Doebeli (1999) have shown that speciation is in principle possible even in this case. If gene flow is limited because of spatial structure, then the formation of linkage disequilibria is simplified. As a consequence, migration-induced competitive speciation may be easier than migration-independent competitive speciation in a two-trait scenario.

Finally, our models assume a discrete deme structure. Competitive speciation in a model of an environmental cline in continuous space has previously been studied by Doebeli and Dieckmann (2003). Since there is no clear difference between "residents" and "immigrants" in a cline model, the distinction between migration-independent and migration-dependent modes of speciation becomes less applicable. Nevertheless, we find several correspondences. The parameter region in which speciation along the cline is possible can be roughly divided into two regions (fig. 4*c* in Doebeli and Dieckmann 2003). A first migration-independent region, where speciation is possible without spatial heterogeneity, requires sufficiently strong local frequency dependence but is virtually unaffected by migration rates and by the slope of the environmental gradient. In contrast, a second migration-dependent region is almost unaffected by local frequency dependence; instead, it merely relies on an environmental gradient of intermediate slope and on not too strong migration. This is similar to our model, in which the migration-driven ecological mode requires an intermediate distance of the fitness maxima (for too large θ, the migration-driven mechanism cannot work because immigrants immediately die; fig. 1). Since the local resource distribution is always unimodal in the model by Doebeli and Dieckmann (2003), a migration-induced competitive speciation mode cannot exist. Whether such a mechanism also occurs in continuous space requires further study.

How Likely Is Speciation through Each of the Three Modes?

In the past, there has been a lot of discussion about the plausibility and relevance of mechanisms that explain speciation through the adaptive evolution of assortative mating. Since both sympatric competitive speciation and parapatric ecological speciation (especially reinforcement) have been amply reviewed elsewhere (Via 2001; Servedio and Noor 2003; Coyne and Orr 2004; Dieckmann et al. 2004; Gavrilets 2004; Bolnick and Fitzpatrick 2007), we focus on a com-

parison of these modes of speciation with the new migration-induced competitive mode identified here.

One argument against sympatric speciation due to competition is that negative frequency-dependent selection must be quite strong. This is true, in particular, for the evolution of assortative mating from low initial levels of choosiness. The principal reason is that female choosiness induces sexual selection, which is positively frequency dependent (implying a cost of rarity) and thus favors intermediate "hybrid" phenotypes while they are still common. This may create a barrier against speciation at intermediate levels of choosiness, which was first described by Matessi et al. (2001) and later studied in detail by Pennings et al. (2008). At first sight, it seems as though this problem could perhaps be solved by assuming a bimodal resource distribution, as this further reduces the fitness of heterozygotes. However, for weak negative frequency dependence (low *c*), another problem appears: frequency-independent disruptive selection at the phenotypic level creates rare-allele disadvantage due to underdominance at the ecological locus, which can lead to a loss of polymorphism. As seen in figure 1, the parameter region in which speciation is prevented by this effect can be substantial. However, in the presence of spatial heterogeneity, speciation can be salvaged by the migration-induced mechanism (cf. fig. 1*B* and 1*E*).

A "race between speciation and extinction" (i.e., loss of polymorphism) has also been cited in arguments against reinforcement (Paterson 1978; Spencer et al. 1986; Liou and Price 1994; Coyne and Orr 2004). This is exactly what happens within demes in the first phase of our migration-independent competitive speciation mode. That is, the race against extinction is quickly lost locally, but both incipient species are preserved globally due to spatial heterogeneity. Once assortative mating is strong enough, the polymorphism also becomes stable within demes.

A somewhat related situation has recently been modeled by Aguilée et al. (2013). These authors studied speciation in a dynamic landscape characterized by the repeated splitting and merging of habitats (thus alternating allopatric, parapatric, and sympatric conditions). Purely sympatric speciation was deliberately prevented by a combination of small population size and an unfavorable genetic architecture, with multiple unlinked loci determining the ecological trait. Speciation was possible, however, via repeated cycles of divergence in allopatry, followed by reinforcement in parapatry and sympatry.

Another classical criticism against reinforcement (Moore 1957; Spencer et al. 1986) is that it is self-defeating. This is because, with increasing choosiness, the rate of hybridization decreases, and hence the selection pressure for a further increase in choosiness declines to zero. If reinforcement takes place in a parapatric scenario, then the hybridization rate is further limited by the migration rate, which must not be too high or else speciation will fail due to swamping. In addition, maladapted immigrants are under selection for disassortative mating, since under locally directional selection, hybrids have higher fitness than the "wrong" homozygotes (Servedio 2004). As a consequence, the evolution of increased choosiness becomes very slow once high levels of assortment have been reached, and it may stop altogether before full prezygotic isolation is attained. Indeed, a recent study by Bank et al. (2011) found that reinforcement will lead to complete isolation only if it evolves from a low level of assortment in a single major step. These earlier findings are fully consistent with the results presented here (fig. 2*B*). However, as may be seen from figure 2, the evolution of assortative mating is about an order of magnitude faster in the migration-induced competitive mode than in the migration-driven ecological mode. Eventually, the selection pressure goes to zero too, but only at a much higher level of assortment. This is because, contrary to reinforcement-like scenarios, the frequency of encounters between the extreme homozygotes increases as speciation proceeds. Also, there never is selection for disassortative mating, because the extreme homozygotes have higher fitness than the hybrids. As a consequence, complete isolation is reached in all cases.

We can conclude that speciation via the migration-induced competitive mode avoids some of the main difficulties of both pure competitive speciation and reinforcement-like parapatric speciation. In a nutshell, it uses the migration-driven mechanism as long as the latter is powerful at low choosiness and then switches to the competitive mechanism at high choosiness, when underdominance is less of an issue and weak negative frequency dependence is sufficient to maintain the polymorphism.

But how likely is it that the conditions required for migration-induced competitive speciation are met in nature? This mode depends on a combination of three key factors: spatially heterogeneous selection, (weak) local frequency-dependent selection, and local frequency-independent disruptive selection against hybrids. In our model, selection against hybrids arises from a U-shaped or bimodal carrying-capacity function, which might reflect the availability of different types of resource. One may envisage the homozygous genotypes as specialists for two discrete resources with an unequal distribution across demes (i.e., one resource is more common in deme 1 and the other in deme 2). Hybrids are not well adapted to either resource and are outcompeted by the specialists. This scenario is a generalization of the classical (parapatric) scenario with only a single resource (and, hence, stabilizing selection) in each deme. Hybrid disadvantage may also arise from ecological factors unrelated to resource use or from in-

trinsic incompatibilities. These alternative interpretations require no changes to our model and the reported results.

The first phase of migration-induced competitive speciation depends on a frequency-dependent disadvantage for rare genotypes, even though coexistence is possible for two reproductively isolated species. Empirical evidence for the plausibility of such a scenario comes from the study of interspecific reproductive interference (i.e., heterospecific interactions during mate acquisition that have negative fitness consequences for at least one species and are caused by incomplete species recognition; see Gröning and Hochkirch 2008 for a review). Even though these studies typically focus on interference between species that are already fully isolated, the results are likely to be relevant for incipient species and intraspecific polymorphisms as well. Most importantly, these studies show that reproductive interference can have severe fitness consequences and can lead to the extinction of rare species due to "sexual exclusion" (Kuno 1992; Hochkirch et al. 2007; Thum 2007; Kishi et al. 2009). Some authors have argued that sexual exclusion may be more important for structuring communities than competitive exclusion (Gröning and Hochkirch 2008 and references therein) and that it may explain the nonoverlapping geographical distributions of some closely related species (Thum 2007). Hybrid inferiority leading to positive frequency dependence has also been invoked as a theoretical explanation for the formation of mosaic hybrid zones (M'Gonigle and FitzJohn 2009) and for understanding the coexistence of ecologically fully equivalent species in parapatry or mosaic sympatry (M'Gonigle et al. 2012).

While each single condition required for migration-induced competitive speciation appears plausible by itself, all three are needed to enable speciation through this mode, limiting its scope accordingly. Since the resultant geographical pattern is the same as for migration-independent competitive speciation, it will be possible to distinguish these two modes empirically only if information about the speciation history is available.

Summarizing, our study shows how locally disruptive selection may (or may not) interact synergistically with spatially heterogeneous selection in the evolution of prezygotic isolation. The migration-induced competitive speciation mode adds a new facet to adaptive speciation theory, further increasing the range of ecological settings in which speciation through the evolution of assortative mating is a realistic possibility.

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APPENDIX A

Evolving Allelic Effects

The simple model with two fixed alleles, which was analyzed in the main text, assumes that the ecological trait can vary only over a limited phenotype range. Since a restricted trait space enhances competition among phenotypes, this assumption implies a strong genetic constraint that may artificially promote an increase in choosiness (Polechová and Barton 2005). Importantly, the constraint is not a result of the single-locus nature of the model. In fact, by adding more loci, the constraint may even get stronger, because more loci generate more intermediate phenotypes and the average distance among phenotypes decreases (Rettelbach et al. 2011). Our simple model also assumes discrete alleles at the mating locus. As shown by Rettelbach et al. (2011), large steps in the evolution of assortative mating make speciation easier.

To demonstrate the robustness of our results, we therefore use a model that specifically relaxes the key constraints but still allows for comprehensive analysis. For the ecological trait, we assume evolvable allelic effects to allow for an unrestricted trait space. At the mating locus, we allow for the evolution of assortative mating in infinitesimal steps. A similar model has been studied by Ripa (2009) for a sympatric scenario. The evolutionary outcome in this model strongly depends on the relative rates of evolution, that is, on how fast choosiness evolves relative to the ecological trait. This is because larger choosiness and a larger distance between phenotypes can be seen as alternative ways to avoid competition: either get rid of the intermediate phenotypes or create a niche for them by getting out of their way.

Model

In the following, we describe a model in which both the mating trait and the allelic effects at the ecological locus

can evolve gradually. For the ecological locus, this means that the phenotypic distance between the two homozygote phenotypes can increase or decrease (e.g., due to evolution at a modifier locus; cf. Kopp and Hermisson 2006), but they always remain symmetric around $\mathrm{X} = 0$, which is also the phenotype of the heterozygotes. At the mating locus, m can gradually change between 0 and 1.

The ecological assumptions are the same as in the core model, the only difference being the definition of $K(\mathrm{X})$ in the case of disruptive selection. Since a U-shaped carrying-capacity curve (as used in the main text) would cause the allelic effects to evolve toward infinity, we instead define K as the maximum of two quadratic curves,

$$K(\mathrm{X}) = \max\left\{K_{\max}\left[1 - \frac{(\theta + 1 - \mathrm{X})^2}{2\sigma_K^2}\right], K_{\max}\left[1 - \frac{(\theta - 1 - \mathrm{X})^2}{2\sigma_K^2}\right]\right\}. \tag{A1}$$

To study the evolution of allelic effects, we use evolutionary invasion analysis (Geritz et al. 1998). That is, we consider the dynamics of a rare mutant modifier allele in a resident population at demographic and genetic equilibrium, which is determined by iterating equation (11) in the main text for 100,000 generations. Whether the frequency of such a rare modifier allele grows or shrinks is determined by the sign of its invasion fitness. Let x denote the allelic effect at the ecological locus (i.e., the two homozygotes have phenotypes x and $-\mathrm{x}$, respectively). Assume a mutant modifier allele whose carriers have allelic effect x_{mut}, whereas the resident population has effect x_{res}. The mutant allele spreads to both demes and to all genetic backgrounds (here, ecological genotypes). We are interested in the dynamics of a vector containing the frequencies of all these mutant genotypes. Within a single deme, the mutant dynamics is determined by a matrix $\mathbf{D}$ with elements

$$\mathbf{D}(i, j) = W(j)\sum_h W(h)N(h)\,(\nu(j, h)Q(j) + \nu(h, j)Q(h))R_{jh\to i}. \tag{A2}$$

This is the number of mutants with genotype i produced by matings between (male or female) mutants with genotype j and all possible resident genotypes (index h) after selection (cf. eq. [11]). Let $\mathbf{D}_1$ and $\mathbf{D}_2$ be the mutant matrices for demes 1 and 2, respectively. The mutant dynamics in the whole population is then governed by the matrix

$$\mathbf{D} = \begin{bmatrix} (1-\mu)\mathbf{D}_1 & \mu\mathbf{D}_2 \\ \mu\mathbf{D}_1 & (1-\mu)\mathbf{D}_2 \end{bmatrix}. \tag{A3}$$

The invasion fitness of the mutant is given by the dominant eigenvalue of $\mathbf{D}$. Taking the first derivative of this eigenvalue with respect to x_{mut} at the point $x_{\mathrm{mut}} = x_{\mathrm{res}}$ gives the invasion fitness gradient (Geritz et al. 1998). If the invasion fitness gradient is positive (negative), then the allelic effect x will increase (decrease). A similar analysis can be done for the mating trait m (i.e., the effect of the allele at the mating locus).

Using this approach, the evolutionary equilibria of x and m can be determined by following the selection gradient in both traits. The selection gradient vanishes at the intersection of the evolutionary isoclines for x and m, which in turn are given by the combinations of trait values x and m for which selection for one of the traits vanishes. The convergence stability of an evolutionary equilibrium can be determined by examining the directions of the selection gradient in the four areas between the intersecting isoclines. If there are multiple stable equilibria, then the evolutionary outcome may depend on the relative speeds at which the two traits evolve. Since a full analysis of evolutionary equilibria, their stability, and basins of attraction is complex (e.g., Ripa 2009), here we focus on a simple but realistic special case and assume that evolution starts at $m = x = 0$ and that x evolves much faster than m. This means that evolution quickly converges to the x isocline and then proceeds along this line until reaching a stable equilibrium. We focus on this limiting case mainly because it represents the opposite extreme of the model with fixed allelic effects. Note that direct selection on the ecological trait will usually be stronger than indirect selection on the mating trait, lending some plausibility to this limit (cf. Ripa 2009).

Results

The long-term evolution of the mating probability is illustrated in figure A1. At first glance, these results look quite different from those for fixed effects (fig. 1). The reason is that with evolving effects, a change in the parameter k can be compensated for by evolution of the trait value x such that it is mainly the ratio of k and c that matters. Therefore, figure A1 is dominated by diagonals. Nevertheless, we find the same three modes of speciation as in the model with fixed allelic effects.

Migration-Independent Competitive Mode

If x evolves faster than m, speciation in the migration-independent competitive mode is only possible in a limited parameter range (fig. A1, red areas). Instead, in many cases, x evolves to a value that is large enough to significantly reduce competition between homozygotes and heterozygotes. For example, with fixed $x = 1$, speciation is

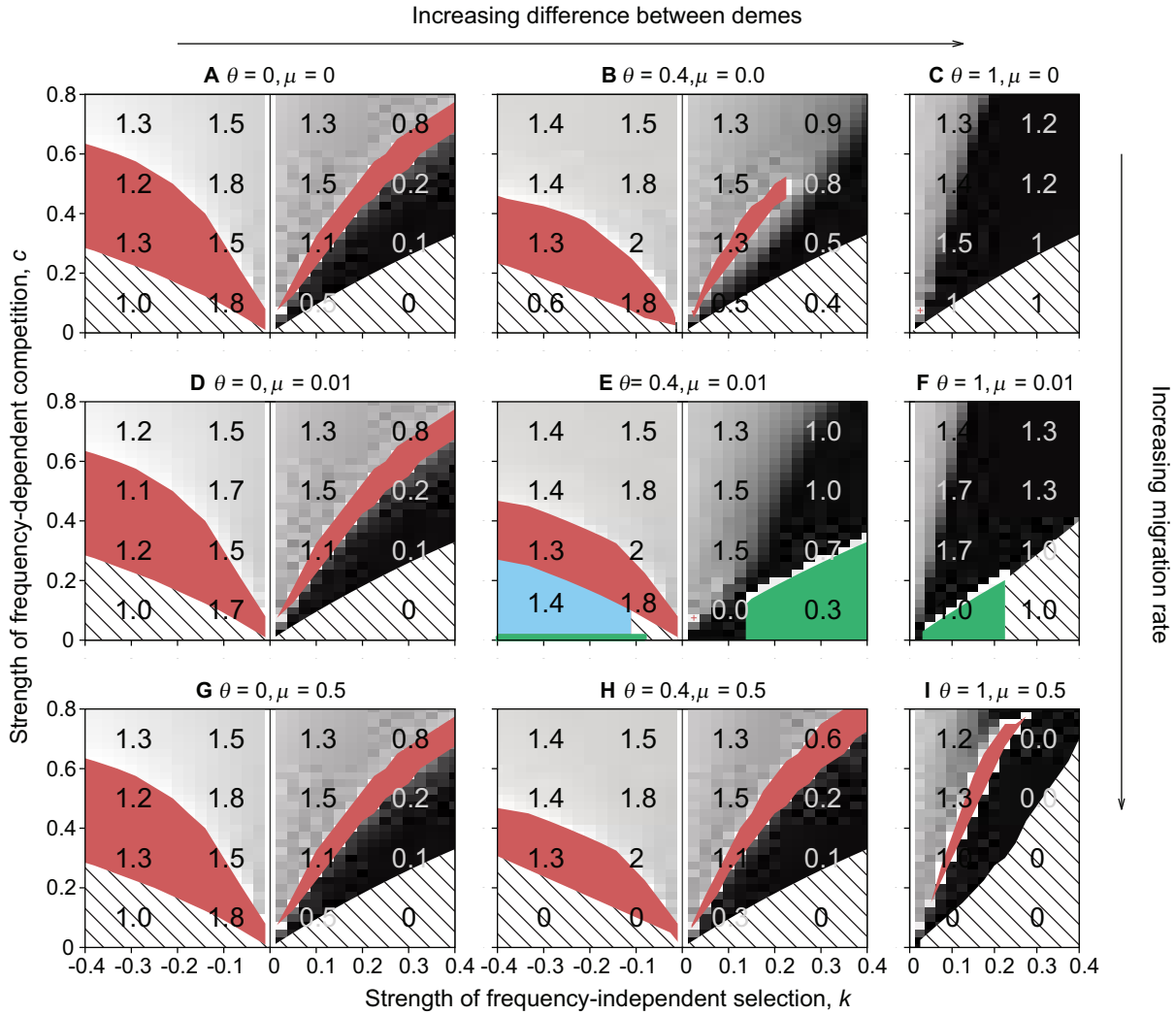

Figure A1: Evolution of assortative mating in the model version with evolving allelic effects if the population starts at random mating and choosiness evolves more slowly than the ecological trait. The plots show the equilibrium mating probability between different homozygotes as determined from invasion fitness analysis, ranging from 0 (white; complete isolation) to 1 (black; random mating). Hatched areas indicate loss of polymorphism. Inset numbers are equilibrium values of the ecological trait (allelic effect x) for the respective parameter combinations. Colors delineate the areas for the different modes of speciation discussed in the main text: red = migration-independent competitive speciation; green = migration-driven ecological speciation; blue = migration-induced competitive speciation. The case $k = 0$ is not shown, because in this case x would evolve to infinity.

possible in fig. 1*E* for $k = 0.1$ and $c = 0.4$, but for the same parameter values in fig. A1*E*, the evolved value of x is 1.6. At this value, the niches of the different phenotypes are hardly overlapping, and only limited choosiness evolves. In the absence of migration, our results are in full agreement with those of Ripa (2009), who provides a detailed analysis.

In contrast to the model with fixed alleles, the parameter range for migration-independent competitive speciation is no longer independent of θ and μ. The effect is an indirect one: spatial structure influences evolution at the ecological locus, which in turn affects the evolution of choosiness. As shown in figure A1, weak migration can hamper the evolution of reproductive isolation through local competition, whereas strong migration can facilitate the process. With weak migration, the locally favored–type experiences increased competition (relative to $\mu = 0$) due to the migrants. As a consequence, the distance between extreme phenotypes increases, and speciation becomes more difficult (cf. fig. A1*B* and A1*E*). In contrast, strong migration prevents the evolution of large values of x, because extreme homozygotes would be very unfit when finding themselves in the wrong

deme (selection for generalists). For example, for $k = 0.3$, $c = 0.7$, and $\theta = 0.4$, x evolves to about 1 for $\mu = 0.01$ (fig. A1*E*) but only to about 0.6 for $\mu = 0.5$ (fig. A1*H*). With the reduced distance between the extreme phenotypes, selection for increased choosiness is strong and complete isolation can evolve.

Migration-Driven Ecological Mode

Adaptive speciation via local adaptation and selection against mating with immigrants is possible for intermediate k and $c < 1 - \exp(-k)$ (fig. A1*E*, A1*F*, green areas). In this case, x evolves to $\approx\theta$. In contrast to the model with fixed effects, speciation fails for small k. Instead, the population becomes practically monomorphic for a generalist genotype with $x \approx 0$, because selection for local adaptation is not strong enough.

Migration-Induced Competitive Mode

As in the model with fixed allelic effects, a two-phased process of migration-induced competitive speciation is observed if the resource distribution is bimodal and frequency-dependent selection is too weak to maintain the polymorphism in the absence of migration (fig. A1*E*, blue area). The process is illustrated in fig. A2*C*. As long as choosiness is low, the frequency of the less fit homozygote in a given deme is very low and the selection gradient for choosiness decreases (corresponding to the migration-dependent phase). With intermediate choosiness, the second niche opens up (because the rare phenotype no longer produces only heterozygote offspring) and the less fit homozygotes increases in frequency (fig. A2*C*, sharp rise of the solid line). At the same point, the choosiness gradient increases (corresponding to the fast migration-independent phase) until choosiness is very high and speciation is (nearly) completed. In contrast to the model with fixed allelic effects, the migration-induced competitive mode does not work for $\theta = 0$ (i.e., no spatial heterogeneity). This is because with gradual evolution of choosiness, the polymorphism at the ecological locus becomes unstable for a small interval of intermediate values of m (Bürger and Schneider 2006; Pennings et al. 2008). With small m, the global polymorphism is maintained, because the rare phenotypes in each deme cannot increase in frequency. With large m, local coexistence is possible within a single deme. For some intermediate m, however, the rare phenotype can increase but local coexistence is not yet possible, leading to global extinction of one of the two alleles. With discrete values for m (as assumed in the main text), this small window of instability is never hit.

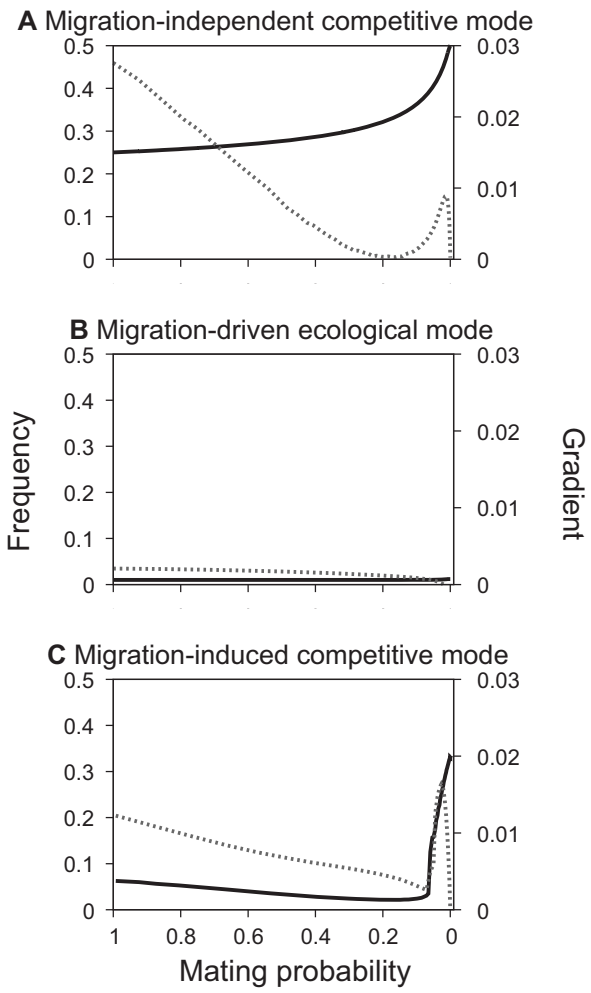

Figure A2: Three modes of speciation in the model version with evolving allelic effects at the ecological locus (i.e., gradual evolution of both x and m). Solid lines show the frequency (in one deme) of the rare homozygote phenotype as a function of the mating probability between different homozygotes. Dotted lines show the strength of selection for assortative mating, as measured by the selection gradient for m (at the equilibrium value of x). *A*, Migration-independent competitive speciation, $\mu = 0.01$, $\theta = 0$, $k = 0.1$, $c = 0.3$. *B*, Migration-driven ecological speciation, $\mu = 0.01$, $\theta = 1$, $k = 0.1$, $c = 0$. *C*, Migration-induced competitive speciation, $\mu = 0.01$, $\theta = 0.4$, $k = -0.25$, $c = 0.05$; the two phases of the process are clearly visible.

No Speciation

As in the model with fixed effects, there is a large parameter range in which speciation does not occur. One notable difference from the previous model is that with evolving effects, no branching occurs in the random mating area with $c < 1 - \exp(-k)$ and small θ (i.e., the population remains monomorphic for a generalist ecological phenotype).

APPENDIX B

Asymmetries between Patches

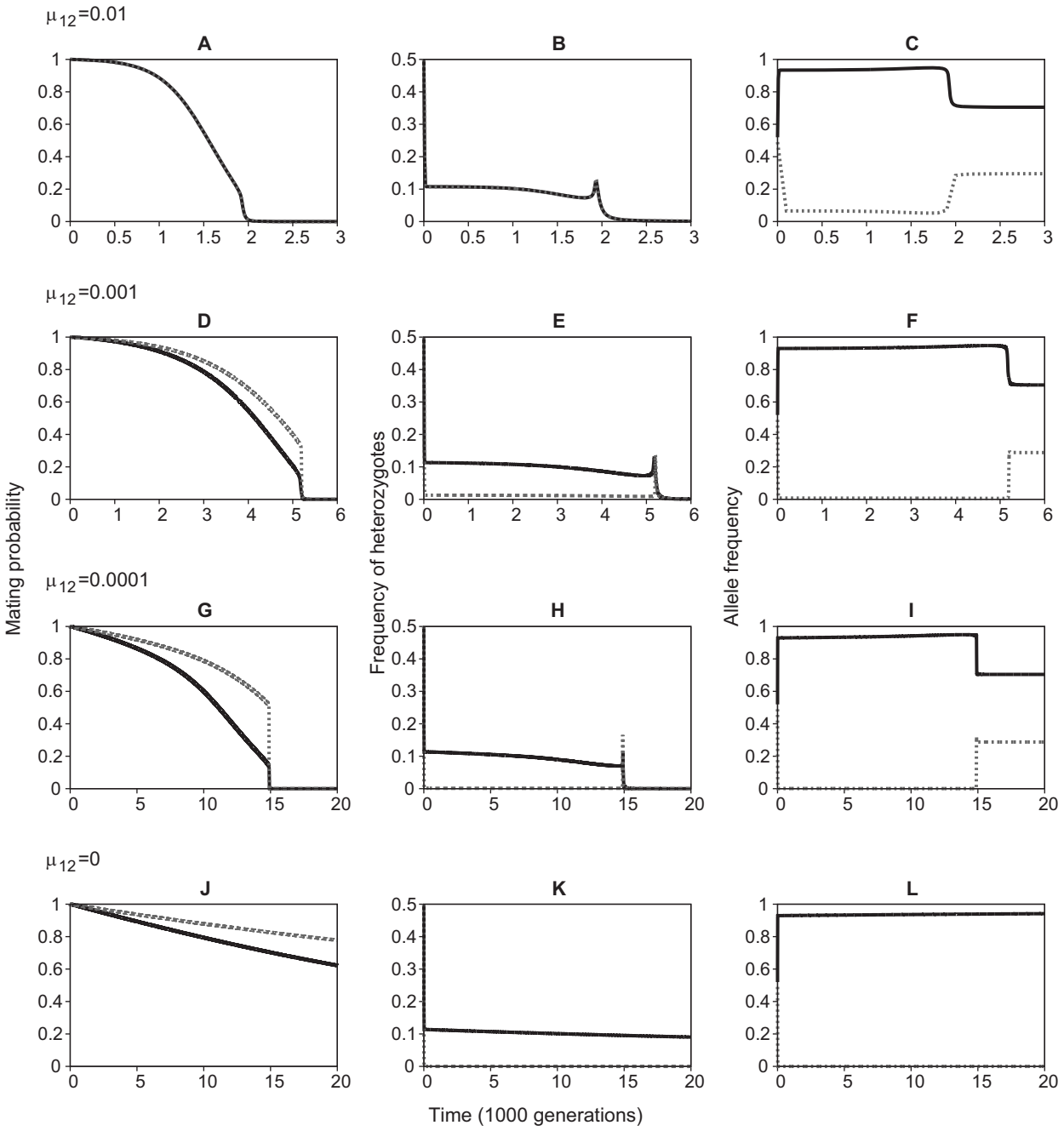

Figure B1: Time courses for asymmetric migration rates. Migration from deme 2 to deme 1 is always $\mu_{21} = 0.1$; migration μ_{12} from deme 1 to deme 2 is as specified. Further parameters are $\theta = 0.4$, $k = -0.3$, $c = 0.2$. Solid lines = deme 1 (the deme with more immigrants than emigrants); dotted lines = deme 2. First column shows mating probability between different homozygotes, second column shows frequency of heterozygotes, third column shows frequency of $x = 1$ allele.

We performed additional simulations to study the effect of asymmetries in the migration rate and in the fitness function. The results are shown in figure B1 (for asymmetric migration) and figure B2 (for asymmetric fitness).

With asymmetric migration (fig. B1, second and third rows), assortative mating evolves first in the deme with many immigrants and later in the deme with few immigrants. As long as there is any migration in both directions, speciation occurs. With one-way migration (i.e., a continent-island model), speciation does not occur within 10,000 generations (note that the timescale in the third row is different). The problem in this case is that for the migration-driven phase, it does not matter where the immigrants come from but that immigrants and hybrids have lower fitness such that there is selection for assortative mating on the residents. However, in the continent population, no assortative mating evolves, and immigrant females from the continent keep hybridizing on the island. Thus, they do not lose their disadvantage of producing unfit hybrids. As a consequence, the migration-independent phase does not take place, and the case reduces to the migration-driven ecological mode.

Asymmetric fitness functions have an influence on the stability of the polymorphism. If one allele has a higher overall fitness, then it is more likely to become fixed. However, in parameter ranges where the polymorphism is stabilized at all, either by frequency dependence or by local adaptation plus low migration, only the shape of the fitness function matters and not the exact values. Additional simulations confirm this. Figure B2 shows that the migration-induced competitive mechanism works even if there is disruptive selection in deme 1 and directional selection in deme 2 (for the phenotype with lower fitness in deme 1). In this case, coexistence of the two species is only possible in deme 1. The migration-driven phase is slowed down relative to the case with disruptive selection in both demes.

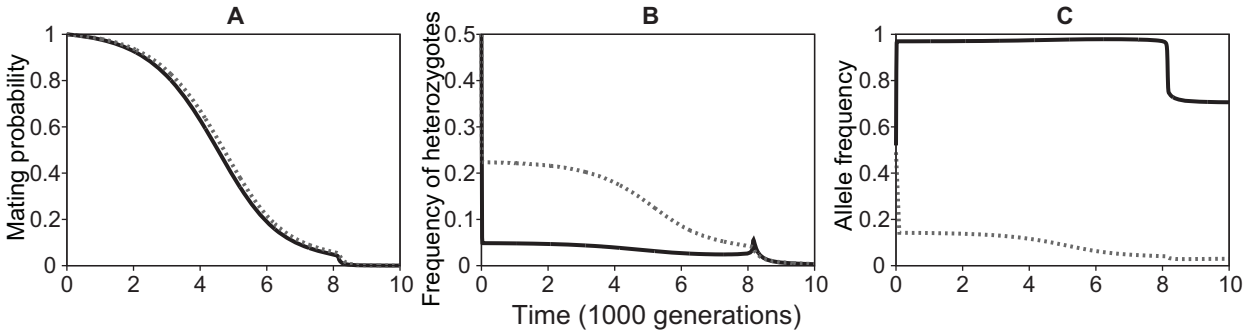

Figure B2: Migration-induced competitive speciation with directional selection in the second deme, with $\mu = 0.01$; $c = 0.1$; deme 1 (disruptive selection): $k = -0.3, \theta = 0.2$; deme 2 (directional selection): $k = 0.2, \theta = 0.6$. Solid lines = deme 1; dotted lines = deme 2. Assortative mating evolves a bit faster in deme 1. The migration-independent phase happens only in deme 1.

APPENDIX C

Examples of Evolutionary Dynamics

In figure C1, we show a variety of temporal patterns underlying the results in figure 1*E*. The borders between different regimes are not always abrupt; for example, aspects of the migration-induced two-phased pattern are also visible in the migration-independent competitive regime. Nevertheless, the borders are well defined because in the migration-induced competitive regime speciation cannot occur within a single population.

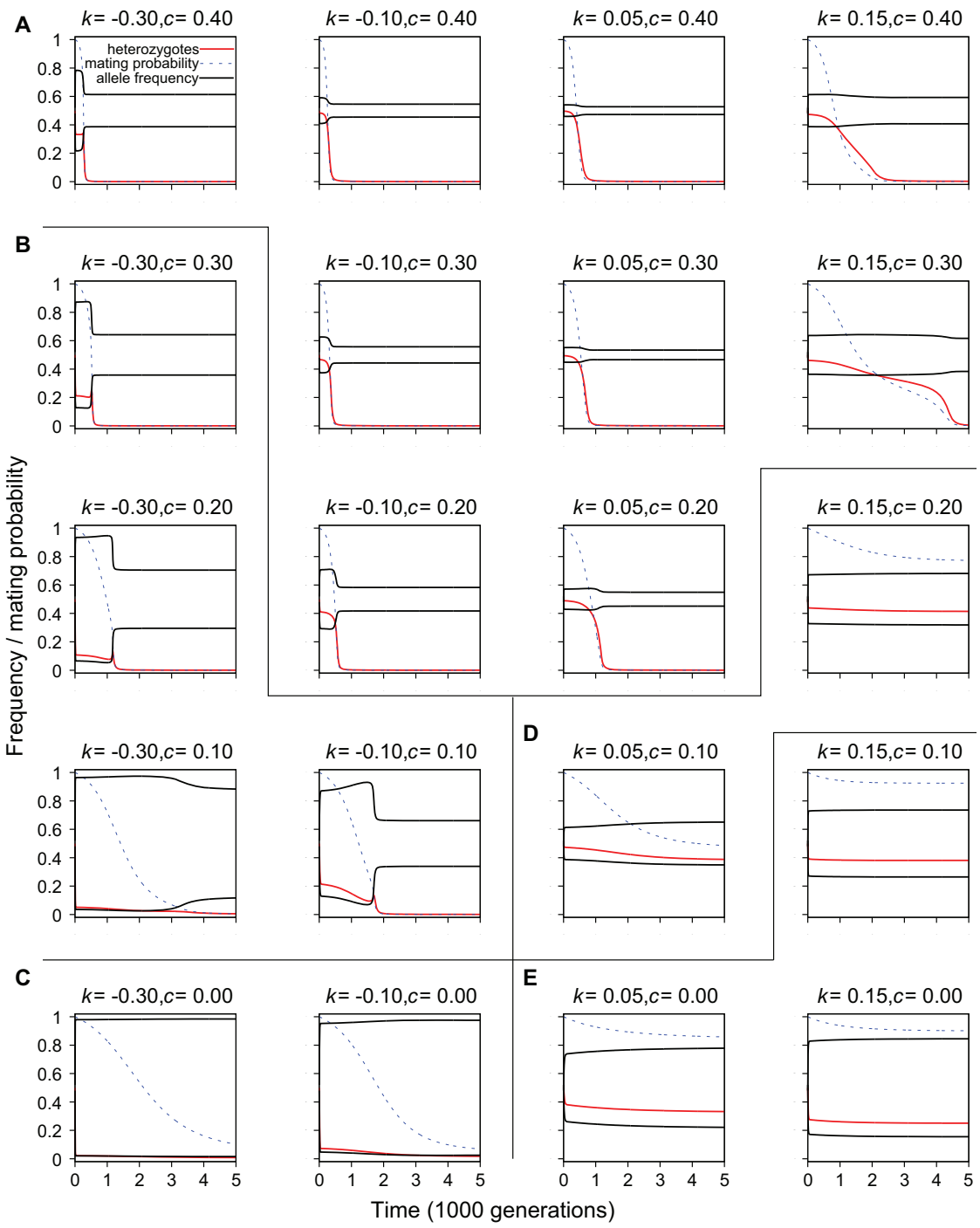

Figure C1: Time courses for different parameters in figure 1*E*. Lines show the borders between the different regimes: black lines = allele frequency; dashed lines = mating probability; red lines = heterozygotes. *A*, Migration-independent competitive speciation. *B*, Migration-induced competitive speciation. *C*, Migration-driven ecological speciation. *D*, Partial isolation. *E*, Random mating.

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