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SPECIATION THROUGH COMPETITION: A CRITICAL REVIEW

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Abstract.—We examined causes of speciation in asexual populations in both sympatry and parapatry, providing an alternative explanation for the speciation patterns reported by Dieckmann and Doebeli (1999) and Doebeli and Dieckmann (2003). Both in sympatry and parapatry, they find that speciation occurs relatively easily. We reveal that in the sympatric clonal model, the equilibrium distribution is continuous and the disruptive selection driving evolution of discrete clusters is only transient. Hence, if discrete phenotypes are to remain stable in the sympatric sexual model, there should be some source of nontransient disruptive selection that will drive evolution of assortment. We analyze sexually reproducing populations using the Bulmer's infinitesimal model and show that cost-free assortment alone leads to speciation and disruptive selection only arises when the optimal distribution cannot be matched—in this example, because the phenotypic range is limited. In addition, Doebeli and Dieckmann's analyses assumed a high genetic variance and a high mutation rate. Thus, these theoretical models do not support the conclusion that sympatric speciation is a likely outcome of competition for resources. In their parapatric model (Doebeli and Dieckmann 2003), clustering into distinct phenotypes is driven by edge effects, rather than by frequency-dependent competition.

Key words.—Asexual populations, assortative mating, frequency-dependent selection, overdominance, parapatry, sympatry.

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There has been considerable enthusiasm for sympatric speciation (e.g., Bush 1994; Via 2001; for a review see Coyne and Orr 2004, ch. 4), stimulated by plausible examples (e.g., cichlids, Schliewen et al. 1994; *Rhagoletis pomonella*, Bush 1969; Feder and Berlocher 2002) and by several theoretical studies (e.g., Johnson et al. 1996; Dieckmann and Doebeli 1999; Kondrashov and Kondrashov 1999; Fry 2003). In particular, Dieckmann and Doebeli (1999) showed that in clonal populations distinct clusters can arise due to competition between similar phenotypes, and for sexually reproducing populations, reproductive isolation arises if assortment can evolve. Doebeli and Dieckmann (2003) extended this model to a spatially continuous habitat, and again found that phenotypic clusters arise under a wide range of parameters. In this paper, we relate these simulation results to previous analyses and show that clusters do not arise for the reasons proposed by Dieckmann and Doebeli. In the major part of the paper, we concentrate on the evolution of asexual populations: Dieckmann and Doebeli (1999; Doebeli and Dieckman 2003) argue that disruptive selection arising from competition between similar asexual phenotypes on an ecological gradient acts in a similar way to cause the evolution of biological species with sexual reproduction. We analyze sexually reproducing populations only without spatial structure, using the infinitesimal model (Bulmer 1980).

The idea that competition drives the divergence of species that use different resources was a key element in Darwin's thinking (1859, chs. 3, 4). This emphasis on ecological differentiation as a cause of speciation within broadly continuous populations was challenged by Moritz Wagner, Jordan, and others, who emphasized the importance of geographic isolation for allowing divergence; throughout most of the 20th century the primacy of allopatric speciation was widely accepted (Coyne and Orr 2004, ch. 3). This view was supported primarily by biogeographic evidence—specifically, by the observation that sister species tend to be separated by geographic barriers (Jordan and Kellog 1907; Dobzhansky 1937; Mayr 1942). Recently, Barraclough and Vogler (2000) developed a more sophisticated version of this argument and showed that in birds, insects, and amphibians, there is little overlap between the ranges of the most closely related species. In mammals, similar patterns are seen, though in some groups such as bats, the ranges of sister species do overlap, suggesting parapatric or possibly sympatric speciation (B. Fitzpatrick and M. Turelli, unpubl. ms.).

Parapatric speciation, in which selection maintains divergence within continuously distributed populations, received new attention in the late 20th century. This was stimulated both by theoretical demonstrations that weak disruptive selection could cause sharp divergence in parapatry (Haldane 1948; Fisher 1950; Endler 1973, 1977; Slatkin 1973, 1975; Caisse and Antonovics 1978) and by empirical studies of clines (e.g., McNeilly and Antonovics 1968; Endler 1977; White 1978). It is clear that any population genetic mechanism that causes divergence between separate populations can also be effective in parapatry, provided the population is spread over a wide enough area (Barton and Hewitt 1981). Moreover, range changes can lead to an association between barriers and divergence, even if the barriers themselves were not necessary for divergence and even if the differences evolved in parapatry. Thus, we have a good understanding of the many ways in which species can evolve both in allopatry and parapatry (Turelli et al. 2001; Gavrilets 2004).

In contrast, sympatric speciation, in which a single randomly mating population splits into two or more reproductively isolated species, has been seen as unlikely, for two reasons. First, the disruptive selection that leads to the evolution of reproductive isolation tends to eliminate variation, so that just one type dominates. Second, gene flow and re-

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combination oppose divergence, particularly if independent genes (or characters) must be established in nascent species for assortative mating to evolve. Several influential studies have revealed how strong disruptive selection and assortment have to be to allow speciation in a single population (Maynard Smith 1966; Udovic 1980; Felsenstein 1981a; Gavrilets 2003; for a review see Gavrilets 2004, chs. 9, 10). Dieckmann and Doebeli (1999; Doebeli and Dieckmann 2003) deal with this issue using an elegant model (first analyzed by Roughgarden 1972) in which individuals compete for resources that are distributed continuously along a one-dimensional axis. Even though resources vary smoothly, Dieckmann and Doebeli (1999; Doebeli and Dieckmann 2003) find that discrete phenotypes emerge. In a sexual population, their model allows assortative mating based on either the ecological trait or on a marker trait to evolve, such that reproductively isolated species arise. An attractive feature of this model is that disruptive selection appears to emerge from a continuous unimodal resource distribution, rather than being imposed arbitrarily. Moreover, after scaling the model involves only two parameters: the strength of competition and the width of the resource distribution, both measured relative to the strength of stabilizing selection.

Doebeli and Dieckmann (2003) extended their model to a two-dimensional habitat, in which the optimal trait value varies linearly in space along one spatial axis. In this model of parapatric speciation, discrete clusters of phenotypes can evolve, an outcome attributed by Doebeli and Dieckmann (2003) to competition between similar phenotypes. Surprisingly, clustering evolved most readily at an intermediate slope of the environmental gradient and occurred under a broad range of parameters. We provide a different interpretation of these patterns, in which we try to separate the several causes of clustering that may be at work in these models. We begin with detailed analysis of clustering in asexual models. We reveal that the clustering in Doebeli and Dieckmann (2003) cannot be due to competition, because it occurs even in the limit of no competition (see figs. 3a,c and 4a,c of Doebeli and Dieckmann 2003). Rather, we show that it arises because the densities of similar phenotypes are only weakly coupled, so that large differences can be generated by weak perturbations—in this case, due to edge effects.

We then consider the effects of competition, beginning with a single population (as in Dieckmann and Doebeli 1999). Roughgarden (1972) showed analytically that the equilibrium solution for an asexual population is one where density matches resources perfectly and all phenotypes have the same fitness. The clustering observed by Dieckmann and Doebeli (1999) in their sympatric clonal model is an artifact of their ''adaptive dynamics'' algorithm, which only allows variants of very small effect, and so only approaches equilibrium very slowly (for a review of adaptive dynamics, see Waxman and Gavrilets 2005). Furthermore, we consider how gene flow across a one-dimensional habitat interacts with competition in the asexual populations and show that in the parapatric model the clumping is determined by edge effects due to low maladaptive gene flow for marginal phenotypes. The Roughgarden/Dieckmann-Doebeli model is strongly density-dependent, such that in the limit of low effective density, there is no selection. Therefore, we also examine a logistic model with density-independent selection and find that clustering can occur even with no constraints on phenotypes.

We analyze speciation with asexual reproduction, because the main results of Dieckmann and Doebeli (1999; Doebeli and Dieckmann 2003) are similar for the sexual and asexual cases: the crucial issue is to understand how clustering of phenotypes can arise from a model in which there is a continuous range of resources, corresponding to a continuous range of phenotypes. In the main part of the paper, analyzing an asexual model, we use the term ''speciation'' loosely, to refer to the evolution of distinct phenotypic clusters. Of course, speciation (in the sense of reproductively isolated populations) is only well defined for sexual populations. However, it is essential to start with a clear understanding of how disruptive selection can arise. More generally, clustering is interesting in itself, both in understanding how asexual organisms evolve (Maynard Smith and Szathmary 1995) and how species are distributed across resources (Bulmer 1974; May 1974; Slatkin 1974, 1978; Roughgarden 1976; Milligan 1985).

Finally, we provide a brief analysis of causes of speciation in sexually reproducing populations using the infinitesimal model (Bulmer 1980). We start with the model with no assortment and show that disruptive selection in the Dieckmann and Doebeli (1999) model is caused by a constraint on the phenotypic range, preventing evolution of optimal genetic variance. We show that cost-free normalization of assortment alone leads to strong divergence, which is prevented by ecological selection. The solution using Bulmer's infinitesimal model agrees well with numerical results of the multilocus symmetric model, where all biallelic genotypes are equally probable (e.g., Barton and Shpak 2000). Detailed analysis of the evolution of a quantitative trait under ecological selection on a limited phenotypic range is to be the subject of a forthcoming paper, which makes use of the symmetric or hypergeometric model (Barton and Shpak 2000).

Results

The Basic Model

We model a population with density $\psi[x, z, t]$, which has a distribution of phenotypes, z, and is distributed over a one-dimensional habitat, with position x at time t. We concentrate on equilibria, in which case we omit the time variable. Individuals reproduce continuously in time and disperse according to migration approximated by diffusion with variance σ_d^2 per unit time. For most of our analysis, offspring are identical to their parents, representing either asexual reproduction or the dynamics of separate species. (In the latter case, species might vary phenotypically, in which case the parameters of the competition function should be adjusted appropriately.) The net rate of reproduction (i.e., Malthusian fitness) is $r[x, z]$. This fitness declines with effective local population density, $\tilde{\psi}$ (population density weighed by the intensity of competition between similar phenotypes; details below) and declines with the deviation from an optimum phenotype, which changes linearly in space ($z_{opt} = bx$). The special case of a single population simply has zero range.

Our analysis is entirely deterministic. This simplifies numerical calculations and allows some analytical results. There

is no suggestion that Dieckmann and Doebeli's results rely on stochastic effects, although because there was no exploration of the effects of population density in their individual-based simulations, it is difficult to be sure.

We consider models that vary in two ways. First, stabilizing selection may be density dependent or density independent. Dieckmann and Doebeli (1999; Doebeli and Dieckmann 2003) follow Roughgarden (1972) and set

$$r = r_0\left(1 - \frac{\tilde{\psi}}{K}\right), \quad \text{where}$$
$$K[x, z] = K_0 \exp\left[-\frac{(z - bx)^2}{2\sigma_k^2}\right].$$

Thus, carrying capacity, K, decreases away from the optimum phenotype at bx. Note that in this model, selection is entirely density dependent: as density approaches zero, all phenotypes approach the same growth rate, r_0. However, the Gaussian function ensures that extreme phenotypes suffer extreme mortality even at low density ($r \sim -r_0\tilde{\psi}\ \exp[(z - bx)^2/2\sigma_k^2]$), which becomes extremely large for large $|z - bx|$. An alternative logistic model is to combine density-independent stabilizing selection with linear density dependent regulation:

$$r = r_0\left(1 - \frac{\tilde{\psi}}{K}\right) - \frac{(z - bx)^2}{2\sigma_k^2}.$$

Second, the effective local density may depend only on local population size ($\tilde{\psi}[x] = \int_{-\infty}^{\infty} \psi[x, z]\, dz$), or it may depend on competition with similar phenotypes and with individuals that are nearby in space. In this more general case, $\tilde{\psi}[x, z] = \int_{-\infty}^{\infty} G[\delta x, \delta z]\psi[x + \delta x, z + \delta z]\, d\delta x\, d\delta z$, or more compactly, $\tilde{\psi} = G*\psi$, where $*$ indicates convolution. The smoothing function G is Gaussian with variance σ_s^2, σ_c^2 for space and phenotype, respectively.

The analysis can be greatly simplified by scaling distance relative to migration, phenotypic density to carrying capacity, and time to maximum growth rate. Precisely, $T = r_0 t$, distance to $X = (x/\sigma_d)\sqrt{2r_0}$, phenotypes to $Z = z/\sigma_k\sqrt{r_0}$; on this scaling, the gradient is $B = (b\sigma_d)/(r_0\sigma_k\sqrt{2})$. The dimensionless quantity B^2 can be interpreted as the loss of fitness (relative to the baseline growth rate r_0) of individuals who move one dispersal distance along the cline, and hence deviate by $b\sigma_d$ from the optimum phenotype. The scaled equations are:

$$\partial_T\psi = \partial_{X,X}\psi + R\psi, \tag{1a}$$

where

$$R = \left[1 - \frac{(Z - BX)^2}{2} - \tilde{\psi}\right], \quad \text{logistic model} \tag{1b}$$

$$R = \left(1 - \tilde{\psi}\ \exp\left[\frac{(Z - BX)^2}{2}\right]\right),$$

Roughgarden/Dieckmann-Doebeli model. (1c)

After scaling, the model has only three parameters: the gradient, B, and the two parameters, σ_s, σ_c, of the smoothing function, which relates the effective density $\tilde{\psi}$ to the actual density, ψ. Here, σ_s describes spatial and σ_c phenotypic variance of frequency-dependent competition. Even in the asexual model, assignment is not arbitrary, as spatial location (X) has low heritability (high migration rate). However, we will see that the size of the spatial and phenotypic range also influences the outcome. Note, that the difference between $1 - Z^2/2$ and $\exp[-Z^2/2]$ (carrying capacity) is negligible for the small values of Z that are common at equilibrium.

We extend the model to include mutation approximated by diffusion with variance σ_{mut}^2 and occurring on a fraction μ of the population. The rate of increase in genetic variance of a haploid population due to mutation is thus $\mu\sigma_{\text{mut}}^2$, and the dimensionless quantity measuring mutation rate is $U = (\mu/r_0)(\sigma_{\text{mut}}^2/2\sigma_k^2)$. This can be interpreted as the load due to mutational variance around the optimum, relative to the time scaled by baseline growth rate r_0. The full equation including mutation is thus:

$$\partial_T\psi = \partial_{X,X}\psi + R\psi + U\partial_{Z,Z}\psi. \tag{2}$$

In numerical simulations of a finite range, the proportion of the population affected by mutation and reallocated by the edge effect to the outermost character value is a nonlinear function of the variance of (Gaussian) mutation. Therefore, $\mu\sigma_{\text{mut}}^2$ cannot be collapsed into one parameter in the numerical approximation on a finite range (similarly, for migration in space).

Nonspatial Models

Frequency-dependent competition, no spatial structure

We begin by assessing the effects of competition between similar phenotypes, and in the first instance, consider a single population with no spatial structure. That is, effective density is defined as $\tilde{\psi} = G*\psi$, where the smoothing function G is Gaussian with variance σ_c^2; $1/\sigma_c^2$ determines the intensity of competition between similar phenotypes. First, we consider Roughgarden's (1972) model of density-dependent selection (eq. 1c), which is the same as Dieckmann and Doebeli's (1999) model of an asexual population. For a stable equilibrium to exist, then at each point either density must be zero and growth rate negative ($\psi = 0$, $r < 0$) or growth rate must be zero and stable to perturbations ($\psi > 0$, $r = 0$, $\partial_\psi r < 0$). Roughgarden (1972) gave the general solution for cases where density is positive. The condition for growth rate to be zero is that $\tilde{\psi} = K[Z]$, that is, $G(Z, \sigma_c^2)*\psi(Z) = \exp[-Z^2/2]$, where the asterisk represents a convolution. Taking Fourier transforms of both sides and using the fact that the Fourier transform of a convolution is a product leads to:

$$\hat{\psi} = \frac{\hat{K}}{\hat{G}}, \tag{3a}$$

and follows:

$$\psi = \frac{1}{\sqrt{1 - \sigma_c^2}} \exp\left[-\frac{Z^2}{2(1 - \sigma_c^2)}\right] \quad \text{for } 0 < \sigma_c^2 < 1, \tag{3b}$$

where $\hat{}$ denotes a Fourier Transform (Roughgarden 1972). If the competition function is too broad ($\sigma_c^2 > 1$), then no solution with positive density exists; instead, a single phenotype dominates ($\psi = \sqrt{2\pi\sigma_z^2}\delta[Z]$; $\delta[Z]$ denoting Dirac's delta function).

Dieckmann and Doebeli (1999) do not discuss this equi-

librium solution. They present simulations based on an adaptive dynamics algorithm, which shows a pattern of evolutionary branching into discrete phenotypes when $\sigma_z^2 < 1$. Their algorithm begins with a single type at $Z = 0$ ($\psi \sim \delta[Z]$). The growth rate of a phenotype Z is thus $R[z] = (1 - \exp[-Z^2/2\sigma_c^2]\exp[Z^2/2])$, which is positive for $Z \neq 0$ when $\sigma_z^2 < 1$. Variants of the original type will therefore invade for both $Z < 0$ and $Z > 0$, and so two phenotypes can be established, at Z_-, Z_+ say. Variants of Z_- will invade for $Z < Z_-$, since $\partial_Z r[Z] < 0$ at Z_-, and so this branch will evolve smaller and smaller values until a point is reached when $\partial_Z r[Z] = 0$; similarly, the upper branch will diverge in a positive direction. Eventually, further branch points will be reached and the pattern will repeat. Thus, their solution consisting of two phenotypes in the clonal model is transient and given a longer time, additional phenotypes will emerge.

This algorithm has its roots in a game-theory approach, in which the stability of equilibria is investigated by examining the fate of invading alleles (for reviews of adaptive dynamics and discussion of its limitations, see Waxman and Gavrilets 2005). From a population genetic point of view, it can be seen as a model of evolution in the limit of very low genetic variance, maintained by mutations of small effect. However, concentrating on the early stages of such a process can be misleading. Eventually, phenotype space will fill up as the solution of equation (3) is approached; as it does so, selection becomes weaker and weaker. However, as phenotype space fills up, disruptive selection becomes very weak, and the process becomes exceedingly slow (Barton and Polechová 2004). The Gaussian solution will be approached much faster if mutations of large effect occur. In a finite population, the distribution will remain patchy, because of the chance reproductive success of individual phenotypes (cf. Felsenstein 1975). The way in which the population approaches equilibrium depends strongly on the pattern of mutation and reproduction (Barton and Polechová 2004). If the coupling between similar phenotypes due to mutation is low, the asexual population can consist of discrete phenotypes for rather a long time, as the Gaussian carrying capacity can be closely matched by the effective density $\tilde{\psi}$ that is generated by a few discrete spikes.

In general, the solution obtained from $\hat{\psi}\hat{G} = \hat{K}$ may be invalid, because it implies a negative density. In such cases, there must be regions in which $\psi = 0$ and $r < 0$. This is the case in the Roughgarden model if frequency-dependent selection is weak, $\sigma_c^2 > 1$, and more generally in the logistic model (eq. 1b), where the disadvantage of distance from optimal phenotype is not density dependent. Gyllenberg and Meszéna (2005) show that the continuous solution of the Roughgarden model without mutation is structurally unstable, so that in the absence of mutation, arbitrarily small perturburtion can prevent coexistence of different phenotypes.

Figures 1A and B show examples of the equilibrium distribution in the logistic model, together with the growth rate. The bimodal shape of the growth rate curve reflects the disruptive selection on phenotype that emerges when $\sigma_c^2 < 1$ in the logistic model. Figure 1C shows how the number of coexisting phenotypes increases as σ_c^2 decreases. As competition decreases, the phenotypic space fills up and the strength of disruptive selection (as measured by the curvature of the growth rate) decreases.

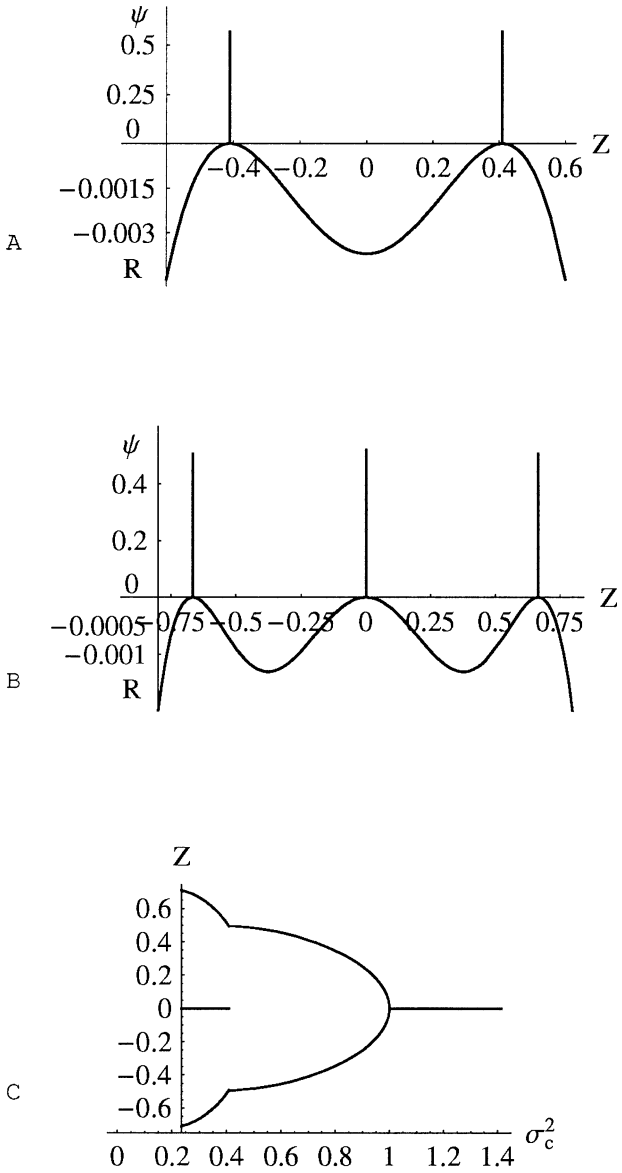

FIG. 1. Logistic model without spatial structure: (A) Upper graph shows the equilibrium density of phenotypes for $\sigma_c^2 = 0.7$. There are two clusters at $Z \sim \pm 0.4$, their abundance ψ is displayed in the uppert part of the graph, the lower part shows the growth rate of invading phenotypes. This is never positive, so that the equilibrium is stable. (B) For $\sigma_c^2 = 0.3$, three phenotypes can stably coexist. (C) The equilibrium distribution of phenotypes as a function of frequency-dependent competition, σ_c^2. There are bifurcations at $\sigma_c^2 = 1, 0.41, 0.24 \ldots$; as variance decreases, phenotypic space fills up.

Sexual populations

We have seen that in Roughgarden's model of competition, an asexual population will ultimately evolve a Gaussian distribution of phenotypes, which precisely matches the Gaussian distribution of resources, so that all phenotypes have equal fitness at equilibrium. Because an additive trait in a

randomly-mating sexual population closely approximates a Gaussian distribution, one expects that the same phenotypic distribution will be reached in the sexual case (Slatkin 1979). Why then do Doebeli (1996), Dieckmann and Doebeli (1999), and Doebeli and Dieckmann (2000) find that sexual populations experience disruptive selection, and hence may evolve assortative mating, leading to discretely clustered phenotypes?

Two factors allow the evolution of assortment in these sexual models. First, there is a transient phase during which trait variance is low, so that the population experiences disruptive selection and assortment is favored. If the population were in the regime where the approximations of adaptive dynamics apply (i.e., near-fixation of a single phenotype and extremely rare mutation to alleles of small effect), then it would evolve arbitrarily slowly. Thus, there could be time for an evolutionarily stable level of assortment to evolve, corresponding to the current degree of disruptive selection on the ecological trait. However, this explanation does not apply to Dieckmann and Doebeli's (1999) simulations of sexual populations, because these involve a high mutation rate and consequent rapid increase in variance of the ecologically selected trait. This should lead to a correspondingly rapid weakening of selection, preventing evolution of assortment.

The second factor, and we believe the most important, is that if the range of the character is limited, no population can achieve the Gaussian equilibrium distribution of phenotypes, and so it remains under disruptive selection. For example, Dieckmann and Doebeli (1999, their fig. 1b) assume that the trait is controlled by five loci, and is confined to the range $|z| \le Z_m = 0.5$; $\sigma_k^2 = 1$, $\sigma_c^2 = 0.16$. For these parameters, the optimal equilibrium is a Gaussian with variance $\sigma_k^2 - \sigma_c^2 = 0.84$. However, with a range constrained on $|z| \le 0.5$, the maximum possible variance is $Z_m^2 = 0.25$, when the two extreme phenotypes coexist at equal frequency. A diploid population at linkage equilibrium, with n biallelic loci of additive effect, has variance $Z_m^2/2n$, which in this example is only 0.025. Thus, a sexual population experiences strong disruptive selection and will tend to evolve assortative mating. Even with complete assortment, such that the population clusters into two species with the most extreme possible phenotypes, the population will still have less than optimal variance, and so there will still be some disruptive selection.

To understand how nonrandom mating evolves, it is helpful to consider the limiting case of asexual reproduction, in which the trait is constrained within $|z| \le Z_m$. When the possible range is narrower than a critical value ($Z_m < Z_m^*$), there is a stable equilibrium with just the two extreme phenotypes present; in the above example, $Z_m^* = 0.39$ (numerical solution: growth rate is zero for the two marginal phenotypes, smaller than zero otherwise). For a somewhat wider range ($Z_m^* < Z_m < Z_m^{**}$), an equilibrium with three phenotypes present is stable (here, $Z_m^{**} = 0.69$). As the possible range of the character increases, more and more phenotypes can be maintained and the continuous Gaussian distribution is approached.

Next, consider a sexual population. Depending on the number of genes involved (n), and the maximum trait range ($-Z_m < z < Z_m$), there are three possible cases. First, if $Z_m^2/2n > \sigma_k^2 - \sigma_c^2$, then the sexual population can closely approximate the neutral Gaussian distribution of phenotypes while at linkage equilibrium. Any combination of allele frequencies that satisfies $\Sigma_i\ (p_i - \tfrac{1}{2}) = 0$ and $\Sigma_i\ 2p_iq_i(Z_m/n)^2 = \sigma_k^2 - \sigma_c^2$ (where p_i denotes the frequency of allele 1 at locus i, q_i frequency of allele 0) will satisfy this requirement, though there will be extremely weak selection on allele frequencies that arises because the binomial distribution of discrete genotypes differs slightly from the continuous Gaussian. Second, if $Z_m^2 > \sigma_k^2 - \sigma_c^2 > Z_m^2/2n$ then a population at linkage equilibrium with equal allele frequencies will have a lower than optimal variance, but can achieve the optimal distribution with sufficient linkage disequilibrium. Some intermediate level of assortment may then evolve. Again, there may be many feasible equilibria. For example, the population might have equal allele frequencies at all loci, and partial assortment. Alternatively, two or more reproductively isolated subpopulations might evolve, each at linkage equilibrium with variance $Z_m^2/2n$ but overall with variance $\sigma_k^2 - \sigma_c^2$. Many combinations of assortment and allele frequencies could generate the same phenotypic distribution. Finally, if $\sigma_k^2 - \sigma_c^2 > Z_m^2$, then the population will still experience disruptive selection even when the two extreme phenotypes coexist without interbreeding. The examples in Dieckmann and Doebeli (1999), which are given for up to seven loci, fall into this parameter range. These simulations of sexual populations are therefore fundamentally different from the asexual models in the same papers; in the latter, the adaptive dynamics algorithm ensures that all possible phenotypes can be reached. In the model of sexually reproducing population, frequency-dependent selection on a trait with restricted phenotypic range results in nontransient disruptive selection, which drives the evolution of distinct phenotypes. The importance of strictly limited phenotypic range was not explicitly noted by Dieckmann and Doebeli (1999). Rather, they assumed disruptive selection arises in a similar way as in the clonal model.

Infinitesimal model of sexual populations

With large numbers of loci or with rare alleles of large effect, the potential range of the trait is very much larger than the range seen within a population at linkage equilibrium. The long sustained responses of most traits to artificial selection suggests that this is the typical situation. We are then likely to be in the second regime, in which the optimal variance can be achieved if the trait variance is inflated by linkage disequilibrium. (Note that with large numbers of loci, linkage disequilibria between any pair of loci can be weak, and yet the overall trait variance can be greatly increased.) We can understand this case by examining the infinitesimal model (Bulmer 1980). At the phenotypic level, this simply assumes that offspring follow a Gaussian distribution with mean at the midparent value and a fixed within-family variance σ_f^2. With random mating, the population rapidly approaches a Gaussian trait distribution, with variance $\sigma_g^2 = 2\sigma_f^2$. This can be justified in the limit of very many unlinked additive loci, when selection on the trait causes very slow changes in allele frequency at individual loci. The cumulative effect may change the mean significantly, but changes in within-family variance can be neglected in the short term.

This model allows the trait distribution to become non-Gaussian under selection and/or assortative mating (Bulmer 1980; Turelli and Barton 1994): such distortions are due to linkage disequilibrium and so disappear rapidly once selection and assortment are removed.

The infinitesimal model is likely to be an accurate approximation for our purposes, because we will primarily be considering equilibria maintained by disruptive selection. Then, allele frequencies are likely to change slowly through selection on the within-family variance; if the latter is at its maximum, then allele frequencies remain constant. The key assumption is then that the variance of the trait within families is independent of the parental values. Plainly, if parents have extreme genotypes, offspring have zero variance and this assumption fails. However, if the range of the character is much wider than the phenotypic distribution, the infinitesimal model will be accurate. We will check our analytical predictions against numerical calculations for a symmetric model, in which all genotypes in the same phenotypic class (determined by a number of + or − alleles) are equally probable (Barton and Shpak 2000).

Following Dieckmann and Doebeli (1999), we first consider a single trait, which determines both assortment and survival. The trait distribution $\psi(x)$ follows:

$$\partial_t \psi = -r\frac{(G*\psi)}{K}\psi + r\int \psi(z')\psi(z'')\tilde{A}(z', z'') \times \exp\left[-\frac{[z-(z'+z'')/2]^2}{2\sigma_f^2}\right] dz'\, dz''. \quad (4)$$

The first term represents density- and frequency-dependent mortality, with effective density given by a smoothing over G, which is a Gaussian with variance σ_c^2. The second term represents the birth of individuals with phenotype z, from parents who had phenotypes z', z''. The function $\tilde{A}(z', z'')$ could represent any kind of selection on fertility, but here, represents assortative mating proportional to $A(z', z'') = \exp[-[y/(2\sigma_A^2)](z', -z'')^2]$. The parameter y represents the strength of assortment; for the moment we assume that this is the same for all mating pairs. σ_A measures the typical mismatch between parents. Clearly, results depend only on the parameter combination y/σ_A^2.

Dieckmann and Doebeli (1999) and Doebeli and Dieckmann (2000) normalize the assortment function such that all phenotypes have the same reproductive output. The normalization coefficient d, imposing disruptive selection so that the assortment is cost-free, can be found analytically given that both trait distribution and assortment are Gaussian (Appendix 4). It is inversely dependent on γ, a measure of the strength of assortment, relative to the genetic variance ($\gamma = y\sigma_g^2/\sigma_A^2$).

The probability of mating between two parents who differ by two genetic standard deviations (i.e., $|x' - x''| = 2\sigma_g$) is reduced by $e^{-2\gamma}$; thus, γ larger than ~ 1 implies extremely strong assortment. It will also be convenient to define $\gamma^* = (y\sigma_f^2)/\sigma_A^2$, which measures the strength of assortment relative to the within-family variance, σ_f^2. If γ^* is large, then there will be substantial reproductive isolation between different members of the same family: the probability of cross-mating between siblings that differ by two standard deviations is reduced by $e^{-2\gamma}$*.

Moore (1979) showed that assortative mating is likely to be accompanied by a cost that will lead to a quick loss of genetic variance. Kirkpatrick and Nuismer (2004) argue that sexual selection will eliminate rare phenotypes, and that this stabilizing selection impedes the evolution of assortment. They examine the effects of pure Gaussian assortment $A(x', x'')$, and also of holding the reproductive success of one sex constant (referred to as the ''plant'' and ''animal'' models, respectively). The latter normalization still leads to sexual selection on male reproductive success.

It is important to realize that the normalization of assortment used by Dieckmann and Doebeli (1999) and Doebeli and Dieckmann (2000) is stronger than either the plant or animal models considered by Kirkpatrick and Nuismer (2004): it introduces a disruptive selection $\exp[+(ydx^2)/(2\sigma_A^2)]$, which precisely compensates for the stabilizing sexual selection caused by the raw Gaussian function, $A(x', x'')$. This model can be justified biologically if there is strict monogamy, so that all individuals find a partner. Alternatively, it can be seen as a formal procedure, which isolates the effects of indirect selection on assortment, just as one postulates modifiers of recombination that have no direct effect on fitness. (Kirkpatrick and Nuismer [2004] give numerical results for nonselective mating, using a model of monogamy from Kirkpatrick et al. [1990], which assumes that as the breeding season proceeds, females choose from a dwindling pool of unmated males. This model is difficult to analyze exactly, though Kirkpatrick et al. [1990] gave a Gaussian approximation to it.)

We begin by examining the effects of ecological selection (first term in eq. 4), and then analyze the consequences of pure assortment (second term in eq. 4). Finally, we combine these two processes. We obtain analytical results for the infinitesimal model that assume a Gaussian equilibrium and check these against numerical solutions of the symmetric model.

Ecological selection: random mating.—Suppose that $K = K_0\exp[-x^2/(2\sigma_k^2)]$, G is Gaussian with variance $\sigma_c^2 < 1$; we show in Appendix 4, that there is a Gaussian solution for ψ of mass C with variance σ_g^2:

$$\frac{1}{\sigma_g^2} + \frac{1}{\sigma_g^2 + \sigma_c^2} - \frac{1}{\sigma_k^2} = \frac{1}{\frac{\sigma_g^2}{2} + \sigma_f^2}. \quad (5)$$

The properties of the Gaussian equilibrium are described in Figure 2. Figure 2A depicts the dependence between genetic variance σ_g^2 and within-family variance σ_f^2 in different selection regimes. If the variance in the absence of selection, $\sigma_g^2 = 2\sigma_f^2$, is equal to the variance favored by selection, $\sigma_k^2 - \sigma_c^2$, then at equilibrium $\sigma_g^2 = \sigma_k^2 - \sigma_c^2 = 2\sigma_f^2$, and all phenotypes have equal fitness. This null point is given in Figure 2A by the points at which the curves showing the equilibrium genetic variance, σ_g^2, cross the line $2\sigma_f^2$. If $2\sigma_f^2 < \sigma_k^2 - \sigma_c^2$, then at equilibrium $2\sigma_f^2 < \sigma_g^2 < \sigma_k^2 - \sigma_c^2$ and the population experiences disruptive selection (left of curves in

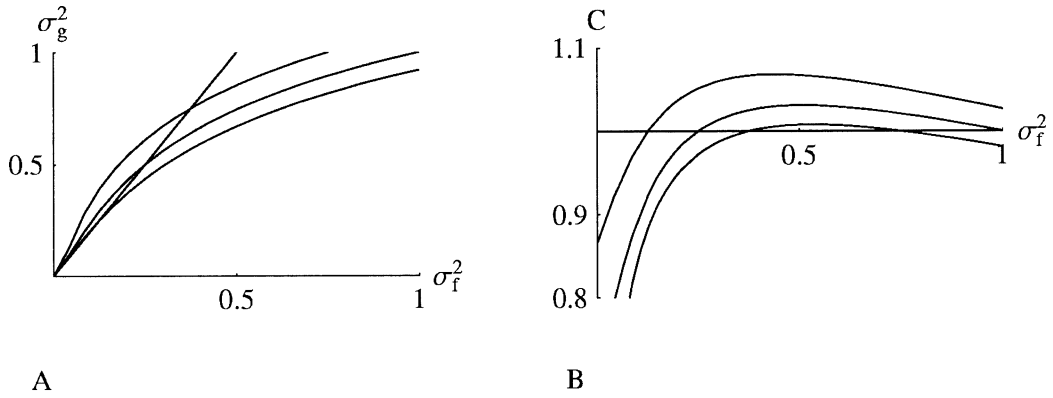

FIG. 2. (A) The equilibrium genetic variance, assuming a Gaussian solution and random mating. The straight line shows the variance with no selection. The three curves are for $\sigma_c^2 = 0.25, 0.5, 0.75$ (top to bottom). Where the curves cross the line $\sigma_g^2 = 2\sigma_f^2$, there is no selection at equilibrium. Below this point, there is disruptive selection, and the variance is lower than the null value; above this point, there is stabilizing selection. (B) The corresponding density, scaled relative to $K_0\sqrt{2\pi}$ (σ_c^2 increasing from bottom to top).

Fig. 2A). However, if $\sigma_k^2 - \sigma_c^2 < 2\sigma_f^2$, then at equilibrium $\sigma_k^2 - \sigma_c^2 < \sigma_g^2 < 2\sigma_f^2$, and the population experiences stabilizing selection (right of curves in Fig. 2A). Note that the population density is maximized when genetic variance is greater than the neutral value $\sigma_k^2 - \sigma_c^2$, so that the population under stabilizing selection (Fig. 2B).

Assortative mating.—To understand effects of assortment with cost-free normalization (d), we derive the equilibrium Gaussian solution for assortment with no ecological selection (Appendix 4). With strong cost-free assortment, genetic variance diverges even with no disruptive ecological selection:

$$\frac{\sigma_f^2[2(1 - d\gamma) + \gamma/\gamma^*]}{2(1 - d\gamma)} = \sigma_g^2. \quad (6)$$

It follows, that for the Gaussian solution with positive variance to exist, we need the scaled strength of assortment $\gamma^* = (y\sigma_f^2)/\sigma_A^2$ to be $\gamma^* < ¼$ or $\gamma^* > ½$. In the limit of random mating, $\gamma^* \to 0$, and so we have $\gamma \to 2\gamma^*$ (i.e., $\sigma_g^2 \to 2\sigma_f^2$), as expected. When assortment is strong ($\gamma^* > ¼$, i.e., $\sigma_f^2 > \sigma_A^2/(4Y)$) the variance diverges. This is because with strong assortment, the variance of the midparental value is almost as large as the variance in the population as a whole. Since the variance among the offspring is increased by σ_f^2 under the infinitesimal model, the variance can increase without limit. (Although a positive Gaussian solution exists for $\gamma^* > ½$, numerical recursions show that the variance also diverges in this case.) This divergence is an artifact of the assumption that assortment is cost-free. As the variance increases, the fraction of the population that are likely mates decreases to an indefinitely low value. However, this is precisely compensated by the normalization coefficient, d.

The model of nonselective assortative mating has already been analyzed by Wright (1921), who showed that (for biallelic loci of additive effect) assortative mating can indeed result in a large increase of genetic variance. The same formula is derived in Crow and Felsenstein (1968) and Bulmer (1980); see Felsenstein (1981b), Gavrilets (2004, ch. 9; pp. 301–309) for reviews.

Ecological selection combined with assortative mating on the same trait.—Finally, we combine ecological selection with nonrandom mating by matching the expressions for the changes due to death and birth. For the genetic variance σ_g^2 we obtain (Appendix 4):

$$\frac{1}{\sigma_g^2} + \frac{1}{\sigma_g^2 + \sigma_c^2} - \frac{1}{\sigma_k^2} = \frac{1}{\sigma_g^2}\frac{4\gamma}{4\gamma^* + 2\gamma - 1 + \sqrt{1 + 4\gamma^2}}. \quad (7)$$

This equilibrium can be compared with the equilibrium under assortment alone (eq. 6), which diverges for $\gamma^* > ¼$. Figure 3A shows that divergence is suppressed by ecological selection, which favors an intermediate variance.

Figure 3A shows how the equilibrium variance is increased by assortative mating. With no ecological selection, assortative mating causes the genetic variance to diverge, for sufficiently large within-family variance (here, $\sigma_f^2 > 0.5$; thick upward curve). Cost-free assortment itself can drive evolution of reproductive isolation (Fig. 3A, upward curve), as cross-matings will gradually vanish. Ecological selection favors an intermediate variance, and so prevents this divergence (Fig. 3A, bottom downward curve). In this example, assortative mating produces only a modest increase in variance and has little effect on the density (Fig. 3B). (Note that in this example, $\gamma^* = y\sigma_f^2/\sigma_A^2$, which equals 0.25 for $\sigma_f^2 = 0.5$. Then, siblings that differ by two standard deviations [$2\sigma_f$] have a mating probability reduced by $e^{-2\gamma^*}$.)

Comparison with the symmetric model.—We now compare this analysis of the infinitesimal model with numerical recursions of a discrete genetic model. We assume a haploid population, in which n loci carry two alleles, of equal and additive effect α on the trait. The trait is confined to the range $\pm Z_m = \pm n\alpha/2$. The maximum possible variance of a population at linkage equilibrium is $n\alpha^2/4$; the corresponding within-family variance averages half this ($n\alpha^2/8$), but varies with parental genotypes. The maximum variance, with complete linkage disequilibrium, is $Z_m^2 = n^2\alpha^2/4$; thus, as the number of loci increases, assortative mating has greater scope for increasing the variance by maintaining positive linkage disequilibria. Following Doebeli (1996), we use the symmetric model, which assumes that all haploid genotypes with the same number of + alleles have equal frequency. Thus, we only need to follow $n + 1$ genotypic classes, rather than the 2^n distinct haploid genotypes. Numerical recursions are

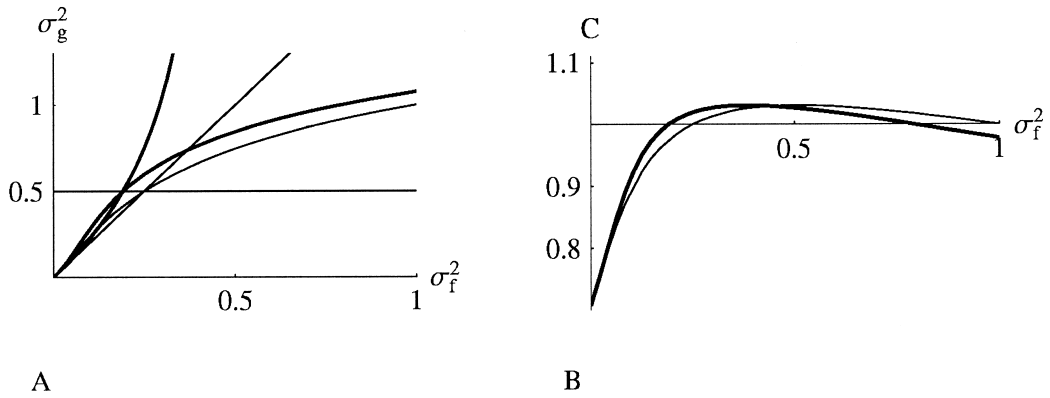

FIG. 3. The combined effects of ecological selection and assortative mating on a single trait, under the infinitesimal model. (A) The equilibrium genetic variance, σ_g^2, plotted against within-family variance, σ_f^2. The straight line shows the variance with random mating alone ($\sigma_g^2 = 2\sigma_f^2$). The thin lower curve shows the effect of ecological selection ($\sigma_c^2 = 0.5$), with random mating. (These two lines are included in Fig. 2A.) The two thick curves to the left show how assortative mating increases the genetic variance ($y = 10$, $\sigma_A^2 = 20$). The upward curve shows the equilibrium variance that would be obtained with no selection (eq. 6). In this model of assortment, that variance tends toward ∞ for $\sigma_f^2 > \sigma_A^2/4y = ½$. The downward curve shows the equilibrium with both ecological selection and assortative mating (eq. 7): now, ecological selection prevents divergence of the variance. The horizontal line shows the equilibrium under ecological selection alone, $\sigma_g^2 = 1 - \sigma_c^2 = 0.5$. Below this line, there is disruptive selection, and above it stabilizing selection. (B) The density, scaled relative to $K_0\sqrt{2\pi}$, under ecological selection. Thin line, random mating; thick line, assortative mating. Parameters are as in (A).

extremely fast, because the distribution of offspring from given parents can be stored in an $n \times n \times n$ table; the limiting step is the normalization required to ensure that all genotypes have the same mating success, despite Gaussian assortment.

Figure 4 shows that with 12 loci, the genetic variance is closely approximated by the infinitesimal model (compare curve with upper series of points); even with six loci, agreement is quite good. What is important is that the trait should be free to span the full range of values allowed by selection. In Figure 4, we keep the average within-family variance fixed at $\sigma_f^2 = 0.375$. Thus, the range with three loci is only ± 1.5, and so the within-family variance differs greatly between genotypes that are common in the population (e.g., offspring of $+++/---$ matings are genetically identical, whereas $+--/--+$ have maximum variance). With 12 loci, the trait range increased to ± 3. Therefore, most genotypes are intermediate, so that within-family variance is approximately constant across genotypes, as assumed by the infinitesimal model.

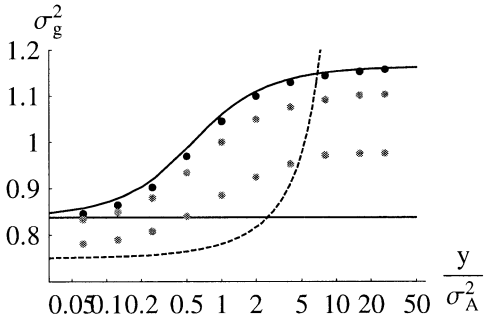

FIG. 4. The increase in genetic variance, σ_g^2, with the strength of assortment, y/σ_a^2; within family variance is $\sigma_f^2 = 0.375$, and competition is $\sigma_c^2 = 0.1$. The upper solid curve shows the genetic variance under the infinitesimal model. Points show the comparison with the haploid symmetric model, with $n = 3$, 6, and 12 loci (bottom to top). With discrete loci, allelic effects, α, are scaled so that the average within-family variance in the base population is fixed at $\sigma_f^2 = 0.375 = n\alpha^2/8$. Hence, the maximum possible range of the trait is $\pm Z_m = n\alpha/2 = 1.5$, 2.12, 3.0 for three, six, 12 loci. The maximum possible variance, with complete linkage disequilibrium, is $Z_m^2 = n^2\,\alpha^2 = 2.25$, 4.5, 9.0 for three, six, 12 loci. The upward dashed curve shows the variance with assortative mating alone: this diverges at $y/\sigma_A^2 > 4\sigma_f^2 = 1.5$. The horizontal dashed line shows the equilibrium variance with random mating, $\sigma_g^2 = 0.838$. This lies between the value for a population at linkage equilibrium ($2\sigma_f^2 = 0.75$) and the value favored by ecological selection ($\sigma_g^2 = 1 - \sigma_c^2 = 0.9$). Note that with strong assortment, genetic variance is higher than that favored by ecological selection, so that the population experiences stabilizing selection. Numerical recursions of the symmetric model were continued for $t = 100$, by which time equilibrium had been approached closely. The continuous time model was approximated by using a time-step $\Delta t = 0.1$.

Our numerical recursions suggest that the population always converges to the Gaussian equilibria given above, provided that ecological selection is included and provided that the trait range is sufficiently wide. (As noted above, the variance can diverge under assortment alone.) However, we have not checked that the symmetric equilibria are stable (Barton and Shpak 2000): under stabilizing selection, the variance may evolve to a lower value than shown here, because particular genotypes can approach fixation. (For example, a population fixed for $++--$ would have zero variance, whereas a population containing equal frequencies of $++--$, $+-+-$, . . . , $--++$, as assumed by the symmetric model, would have high variance.) We will examine this issue in a subsequent paper.

Spatial Models

Spatial structure: no frequency-dependent competition

Linear gradient on limited space.—We now turn to considering the behavior of the model in the absence of frequency-dependent competition. That is, the variance of the phenotypic competition function tends to infinity, $\sigma_c^2 \to \infty$ and spatial competition is only local ($\sigma_s^2 \to 0$). Thus, fitness decreases with the total number of individuals of all phe-

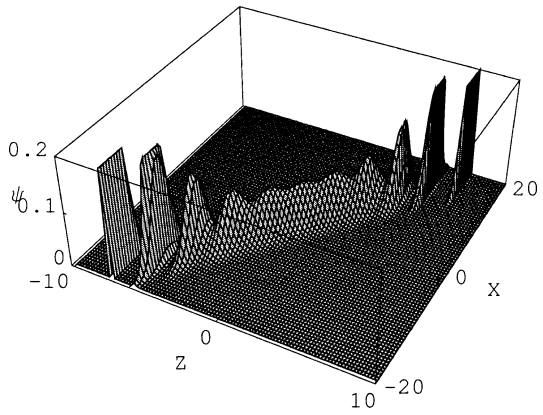

FIG. 5. Numerical solution of the logistic model (eq. 1b) with no competition ($\sigma_c = \infty$, $\sigma_s = 0$), scaled gradient $B = 0.4$; range $|X| < 20$, $|Z| < 10$; grid spacing $\delta X = 0.5$, $\delta Z = 0.2$, $T = 6250$. The spatial range is $-20 < X < 20$.

notypes in the immediate spatial neighborhood. Under the logistic model (eq. 1b), this is similar to the model considered by Kirkpatrick and Barton (1997). However, Kirkpatrick and Barton (1997) approximated a sexual population by assuming a quantitative trait with fixed variance, σ_z^2. Assessing the joint evolution of trait mean and population density Kirkpatrick and Barton (1997) found that unless the gradient is too steep, there exists a perfect solution in which the trait mean follows the optimum $\bar{Z} = BX$. Variation around the optimum reduces the mean fitness, such that if the gradient is too steep, ($B_u = \sigma_z^2/(2\sqrt{2}) + \sqrt{2}$; Kirkpatrick and Barton 1997), the population goes extinct even if the mean is perfectly adapted. However, adaptation can collapse before this upper limit is reached. If the gradient is steeper than a critical value ($B_c = \sigma_z^2/(2\sqrt{2})$; Kirkpatrick and Barton 1997), the perfect solution becomes unstable and the population becomes adapted to a restricted region. (Note that a factor of 2 in the definition of A [Kirkpatrick and Barton 1997, eq. 11a was lost in eq. 18 there, and in Table 17.1 of Barton 2001]. With our scaling, $\sigma_z^2 = 2A$. Also, V_s in those papers corresponds to σ_k^2 here; additive genetic variance V_g corresponds to the phenotypic variance σ_z^2 in the clonal model here.)

Barton (2001) considered a variety of models in which the phenotypic variance could evolve, including a continuum-of-alleles model, which is equivalent to asexual reproduction when only a single locus is considered. In these models, the population could adapt to arbitrarily steep gradients by increasing its genetic variance. (At least, up to the upper limit at which the variance around the optimum is so high that the mean growth rate falls to zero.) In particular, the model of equation (1b) has a perfect solution in which ψ is a Gaussian with variance $\sigma_z^2 = B\sqrt{2}$, a mean that matches the optimum ($\bar{Z} = BX$), and density $N = 1 - B/\sqrt{2}$. (Therefore, the population goes extinct if $B > \sqrt{2}$.)

Figure 5 shows a numerical solution of the logistic model on a finite range. Although the solution fits the Gaussian prediction of Kirkpatrick and Barton (1997) well at the center, there are extreme fluctuations at the edges. Despite the fluctuations, the trait mean changes smoothly over most of the range (Fig. 6A) and the effective (i.e., total) density stays constant and close to the prediction (Fig. 6B). The distribution of any particular phenotype across space is close to the predicted Gaussian distribution (Fig. 6C) apart from the edges. Solutions can be arbitrarily rough along the Z-axis because all that matters is that the total density—integrated over all trait values—matches the carrying capacity. The fluc-

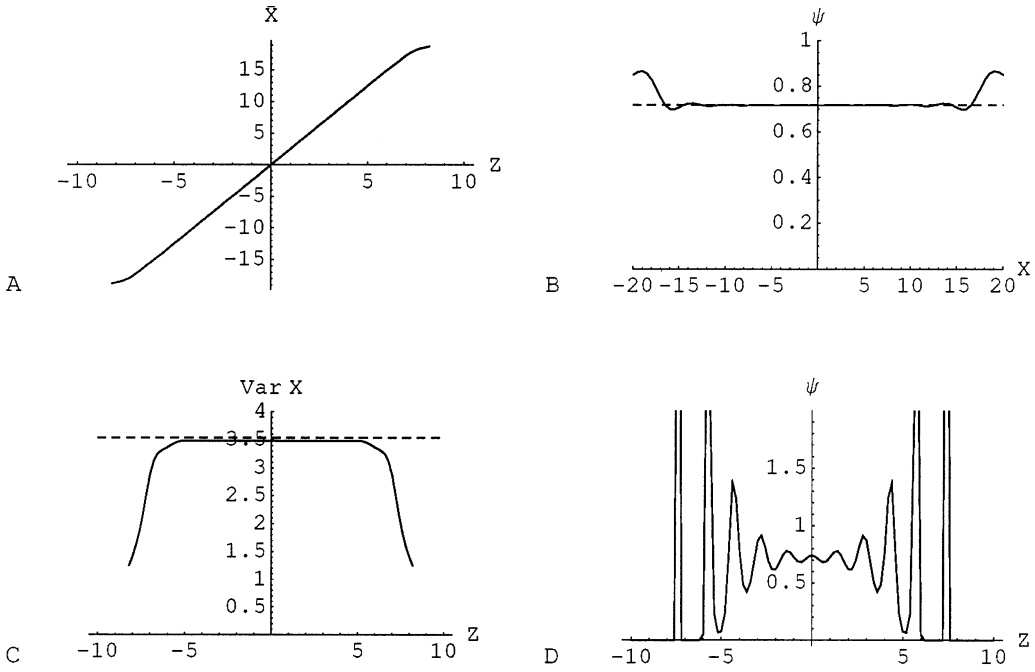

FIG. 6. Details of the solution shown in Figure 5. (A) The mean location of phenotypes $\bar{Z}$. (B) Total population density plotted against distance, X. The dashed line shows the simple prediction for the logistic model ($1 - B/\sqrt{2}$). (C) The variance of location of phenotypes Z. The dashed line shows the prediction, $\sqrt{2}B$. (D) Total abundance of phenotypes Z.

tuations are manifest primarily in the relative abundance of different phenotypes (Fig. 6D).

In this example, strong fluctuations are driven by edge effects. Near the boundaries, there is half as much gene flow from elsewhere, and hence the mean fitness of the local population tends to be higher. This leads, in turn, to higher density at the edges, which suppresses the abundance of phenotypes adapted to regions a little away from the edge, as a higher proportion of these submarginal population is then maladapted. Numerical calculations show that damped oscillations in phenotype abundance propagate in from the boundaries (Fig. 5). Neither the *X*- or *Z*-grid spacing affect the solution of $\psi(Z)$ and $\psi(X)$, showing that it is not an artifact of the use of a discrete grid for the numerical calculations. Increasing the gradient facilitates the edge effect (if $|Z| \geq B|X|$, which always holds in Doebeli and Dieckmann 2003), as it promotes the advantage of marginal phenotypes.

Even when the fluctuations are strong, effective density remains high everywhere (Fig. 6B). Thus, the phenotypic clumping occurs while the population is at high density and should not be influenced by whether selection is density dependent, as in the Roughgarden/Dieckmann-Doebeli model. Indeed, when the spatial and phenotypic range are limited as in the Doebeli and Dieckmann (2003) paper (i.e., $\sigma_k = 1$, $X_m = Z_m = 3.3$), then simultaneous adaptation to the optimal gradient and greater fitness of the marginal phenotype due to edge effects leads to branching and establishment of two stable phenotypes for $B = 1$, without any frequency-dependent competition whatever (Figure 7A). Because these phenomena occur at high effective density, they are similar in the logistic and the Roughgarden/Dieckmann-Doebeli models.

In the absence of mutation, the variance of the discrete clumps that form near the ends decreases indefinitely with time: our numerical results suggest that the equilibrium consists of discrete clumps near the edges. The fluctuations propagate to the center as $t \to \infty$. It is not clear whether the limit is a true continuum or would consist of very finely spaced discrete phenotypes. Adding mutation couples the frequencies of adjacent phenotypes and so tends to broaden the sharp clumps that form near the edges. This effect of mutation is shown in Figure 7B. Note that Dieckmann and Doebeli's choice of mutation rate was low enough that sharp clumps would form, given the range of *X* and *Z* that they used.

The key to understanding the clumping illustrated in Figures 5–7 is that in the absence of mutation, there is no coupling between neighbouring phenotypes. The density-dependence in equation (1) is mediated through the smoothed density, $\tilde{\psi}$, which is an average over all phenotypes. Thus, similar phenotypes can vary greatly in abundance (e.g., Fig. 6D), provided that the overall smoothed density matches the equilibrium solution. Mutation tends to smooth out these fluctuations by coupling the phenotypes, but will have little effect if it is weak. If mutational variance σ^2_{mut} is about 0.1–1% of environmental variance V_e (Lynch and Walsh 1998, ch. 12), then assuming moderate heritability $h^2 = ½$, V_e equals V_g and mutational variance $\sigma^2_{\text{mut}} = (0.001 - 0.01)V_g$. Phenotypic variance σ_z^2 equals $\sqrt{2}B$ in the logistic model, and $\sqrt{2}B + o(B^2)$ in the Roughgarden/Dieckmann-Doebeli model and in a clonal model, $V_g = \sigma_z^2$. We show that mutational variance

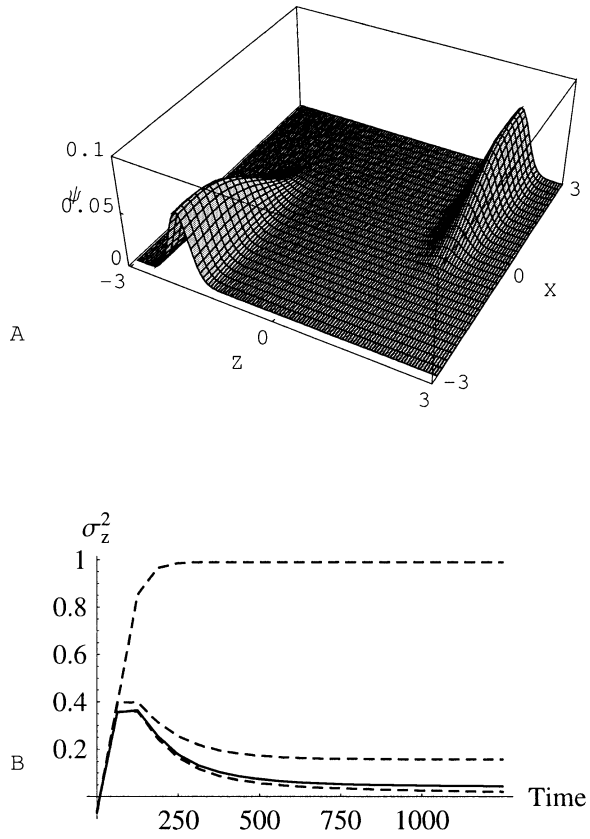

FIG. 7. (A) Solution to the Roughgarden/Dieckmann-Doebeli model (eq. 1c) on the same limited range as was used by Doebeli and Dieckmann (2003), but without frequency-dependent competition ($\sigma_k = 1$, $L = 3$, $B = 1$, $\sigma^2_{\text{mut}} = 0.05$, $\mu = 0.005$, $m = ½$). The simulation was run for a time $T = 625$. (B) The variance of phenotype, *Z*, within one of the two clusters, plotted against time, *T*. Parameters for mutation are $\sigma^2_{\text{mut}} = 0.05$, $\mu = 0.005$ (solid line) and $\mu = 1$, $\sigma^2_{\text{mut}} = 0$, 0.01, 0.04 bottom to top for the three dashed lines. Variance of the phenotype is calculated as an average variance on one-half of the range, thus phenotypic variance σ_z^2 below about one means that clusters are still distinct.

in this range $\sigma^2_{\text{mut}} = (0.001 - 0.01)\sqrt{2}B$, covering estimates of mutation used in Doebeli and Dieckmann (2003), does not substantially suppress the clustering for the ranges of trait and space used by Dieckmann and Doebeli (2003) model (Fig. 7B).

Torus.—We demonstrate that fluctuations are indeed generated by edge effects by simulating the model on the surface of a torus. Then, there are no edges along either the spatial or phenotypic axes, the optimum still changes linearly but wraps around the torus in a spiral. We describe the formal procedure of simulating a torus in Appendix 1. The solution stays smooth even when there is no mutation in the model, provided we start from a smooth distribution centered on the spiraling optimum (Fig. 8A). This contrasts with the strong clustering that evolves when exactly the same model is run on a square geometry with edges, where the fluctuations arise due to the direct advantage of phenotypes at the edges.

However, numerical solution starting from a narrow Gaussian around $Z = 0$ shows that even on the torus, Gaussian equilibrium can get approximated by several clusters provided the coupling between phenotypes is low in relation to

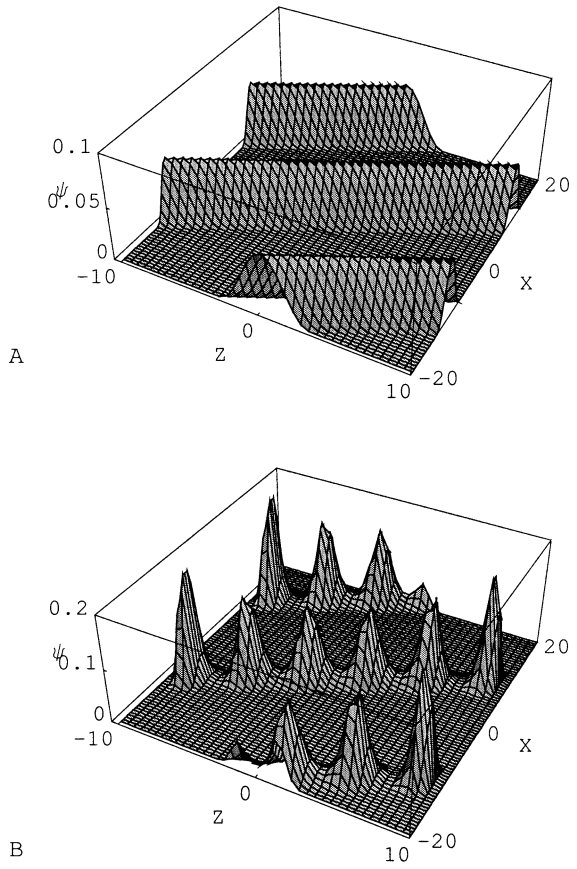

FIG. 8. (A) On a torus, the smooth solution is always stable provided there is moderate coupling between the phenotypes. In the model with no competition ($\sigma_c = \infty$, $\sigma_s = 0$), with migration ($m =$ ½, stepping-stone model), the solution is stable for mutation with variance $\sigma^2_{mut} = 0.01$. Scaled gradient $B = 1$, range $|X| < 19.75$, $|Z| < 9.75$; grid spacing $\delta X = 0.5$, $\delta Z = 0.5$, $T \to \infty$. (B) The density can form spikes when coupling is very low. Specifically, for mutation set to zero, the solution consists of discrete spikes ($T = 1250$, other parameters as in A). Starting from homogenous solution (A), spikes do not develop even with no mutation.

the migration (see Fig 8B: sharp clusters emerge if $\sigma^2_{mut} \leq 10^{-4}$). With no coupling (mutation), Roughgarden's continuous solution is structurally unstable (Gyllenberg and Meszéna 2005). We suggest that with low mutation, the evolution of discrete clusters is driven by the instability of the continuous solution, though we have not performed the analysis of stability of Roughgarden model with low but finite coupling. As the remaining selection for nearby phenotypes is very low once a few clusters get established, clusters remain stable for very long times (Barton and Polechová 2004). Unlike the edge effects, these clusters are not driven by the impossibility of migration outside the spatial range resulting in an advantage of marginal phenotypes and emerge only with very low coupling.

Frequency-dependent competition with spatial structure

Finally, we analyze the full parapatric model, where both spatial (X, σ_s^2) and phenotypic (Z, σ_c^2) Gaussian smoothing determine the effective density $\tilde{\psi} = \psi * G$. Doebeli and Dieckmann (2003) extend their model to parapatry by considering an optimal character value, which varies along one axis of a two-dimensional habitat. We model the system without the second independent axis, so that density varies only with one spatial axis, X, and one phenotypic axis, Z, with the optimal value at $Z_{opt} = BX$. This reduction seems reasonable, because the solution in Doebeli and Dieckmann (2003) is uniform in Y, and clusters emerge along the gradient of optimal phenotype.

Phenotypic clusters, triggered by edge effects, get suppressed due to frequency-dependent competition, which results into high effective density of a narrow phenotypic cluster. Over a longer range, the solution is homogeneous away from the edges. We can find the Gaussian approximation to the homogenous solution using the method of variations (Appendix 2), setting the population density $\psi(X) = A \exp[-(\alpha x^2)/2]$ and effective density $\tilde{\psi}(X) = A \exp[(-x^2/2)\alpha/[1 - \alpha(\sigma_s^2 + \sigma_c^2/2)]]$. The variance of competition function in terms of influence on spatial character, can be represented as $\sigma_s^2 + \sigma_c^2/2$, because both spatial and phenotypic frequency-dependent competition functions that weight the impact of population density, are Gaussian, $Z^2_{opt} = B^2X^2$ (see description above eq. 1). By optimizing the parameters A and α, we can obtain the variance of the phenotype for both logistic and the Roughgarden/Dieckmann-Doebeli model. We find that for weak to moderate gradients, the Gaussian approximations agree well with numerical solutions (Figs. A2, A3). Phenotypic variation increases as the gradient becomes steeper because migration from place to place brings in different phenotypes (effect of mutation is considerably smaller). Phenotypic variance increases more slowly than in the model without frequency-dependent competition. This is because the impact of frequency-dependent competition becomes weaker as the gradient steepens, as the overall frequency-dependent competition acting on a phenotype has variance of $B^2\sigma_s^2 + \sigma_c^2$. Both numerical solutions and Gaussian approximations of phenotypic density converge to the value expected without frequency-dependent competition as the gradient steepens. For the solution to be positive, gradient can be arbitrarily steep in the Roughgarden/Dieckmann-Doebeli model, whereas in the logistic model, the maximum possible gradient in the absence of frequency-dependent selection is $B = \sqrt{2}$). Thus, in the logistic model, the convergence is faster than in the Roughgarden/Dieckmann-Doebeli model (Figs. A2, A3).

By continuity with the spatially independent model ($B =$ 0), we expect the equilibrium solution in the logistic model to consist of discrete phenotypes when the competition function is narrow: for a sufficiently small gradient, the solution should consist of two peaks, which shift smoothly along the cline ($B = 0.02$, $\sigma_c^2 = 0.7^2$, Fig. 9A). However, as the gradient steepens, the variance of overall frequency-dependent competition acting on a phenotype increases and migration couples the phenotypes. The resulting solution is unimodal, even for quite shallow gradients ($B = 0.2$, $\sigma_c^2 = 0.7^2$, Fig. 9B). In numerical solutions on a narrow range, clustering along the gradients interferes with clustering due to the edge effect, which leads to fluctuating phenotype with positions of maxima just perpendicular to the trait axis (Figs. 9A, B).

Clustering as a result of edge effects is facilitated as the gradient steepens, because the advantage of reduced mixing

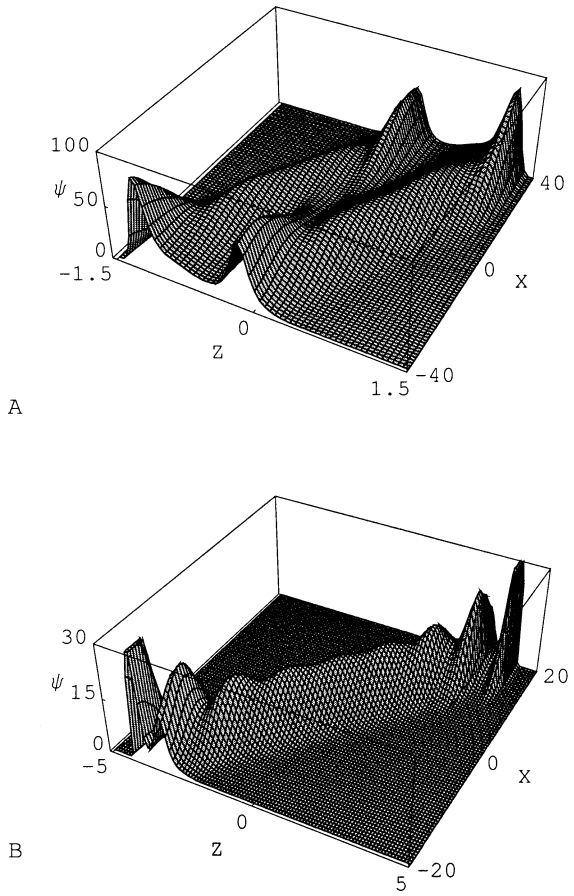

FIG. 9. (A) Numerical solution of the logistic model, for a shallow gradient ($B = 0.02$, $\sigma_c^2 = 0.7^2$). (B) A steeper gradient ($B = 0.2$) suppresses the clustering and gives a unimodal distribution.

of the marginal phenotypes is relatively higher. Doebeli and Dieckmann (2003) state that clumping is caused by a narrow scaled frequency-dependent competition σ_c/σ_k (figs. 3, 4a,c of Doebeli and Dieckmann 2003). However, clumping occurs even in the limit with no competition ($\sigma_c \to 0$) as can be seen in Doebeli and Dieckmann (2003, figs. 3, 4a,c). We do not either have a precise explanation for the decreased tendency to clumping revealed by Doebeli and Dieckmann (2003, figs 3, 4b,c) for a steep gradient, $a(\sigma_s/\sigma_k) > 1$ (in our notation, $b\ \sigma_s > 1$). In Doebeli and Dieckmann (2003), the range of $|X|$ is equal to $|Z|$ but the original gradient, a, is set to vary between zero and one. Thus, a decreased tendency to clumping cannot emerge due to missing phenotypic space for the optimum determined by the gradient. Also, unlike the sympatric case, in the parapatric Roughgarden/Dieckmann-Doebeli's model, there is no threshold of strength of frequency-dependent competition (σ_c, σ_s) versus strength of stabilizing selection (σ_s) when the phenotypic distribution would approach zero variance at a given locality. Clearly, if clumping occurs due to the edge effect, then sharp clusters are suppressed by narrow frequency-dependent competition σ_c, σ_s; whereas increase in σ_k has negligible effect. Therefore, the scaled measures σ_c/σ_k, σ_s/σ_k are not necessarily sufficient to determine the outcome.

DISCUSSION

Dieckmann and Doebeli (1999) and Doebeli and Dieckmann (2000, 2003) have simulated competition between similar phenotypes. They show that phenotypic clustering arises in both asexual and sexual populations and in both sympatry and parapatry. In the sexual case, the consequent disruptive selection generates associations with traits that cause assortative mating, leading to the origin of biological species. Our analysis of this family of models shows that clustering arises for a variety of reasons, so that the superficial similarity between the different models is misleading. Our key points follow. For a single asexual population, clustering is transient, and in any case leads to very weak disruptive selection. In the sexual case, clustering and disruptive selection arise because the trait range is restricted. With unlimited range, the infinitesimal model leads to simple analytical solutions. These show that assortative mating alone drives divergence, which is opposed by competitive selection. In parapatry, clustering does not require competition. Instead, it arises because adjacent phenotypes are weakly coupled, so that edge effects and other perturbations can readily produce rough phenotypic distributions.

Single Asexual Populations

Dieckmann and Doebeli (1999) argue that competition for a single resource between similar phenotypes leads to disruptive selection in both asexual and sexual populations. This is a crucial difference to other models of sympatric speciation, which always impose some source of disruptive selection. The evolution of clusters contrasts with Roughgarden's (1972) analysis of the same model, which gave a Gaussian equilibrium distribution. The branching shown by Dieckmann and Doebeli (1999) is a transient phase, which arises because their adaptive dynamics approach involves successive invasions by mutations of very small effect. As more types invade the population, the phenotypic distribution better matches the resource distribution, and selection becomes weaker: the approach to the continuous equilibrium identified by Roughgarden (1972) is therefore extremely slow (Fig. 1A; Barton and Polechová 2004). By the same token, the disruptive selection present when even a few phenotypes are present is extremely weak—far too weak to drive assortative mating in a sexual context.

Models of competition for a continuously distributed resource, of the kind studied by Roughgarden (1972), almost never admit a continuous solution: the Gaussian form used by Dieckmann and Doebeli (1999) is a special case. We have shown that with a different form of density-dependence, solutions are discrete, and Gyllenberg and Meszéna (2005) show more generally that Roughgarden's (1972) model is unstable toward small perturbations of fitness. However, this does not alter our main conclusion, because even these solutions involve only weak disruptive selection: the resource distribution can be closely matched by just a few types (e.g., Figs. 1A, B). The situation is different with Levene's (1953) model, which is commonly used for adaptive dynamics analysis, where the number of distinct niches (usually two) determines the number of stable phenotypic clusters (e.g., Kisdi and Geritz 1999).

Single Sexual Population

In Dieckmann and Doebeli's (1999) sexual model, disruptive selection arises because the trait distribution is limited: the lack of competitors outside the phenotypic range gives an advantage to extreme phenotypes. For their parameters, the phenotypic distribution is constrained to be unimodal with random mating, but with possibility of assortment and limited range, it can be bimodal or even multimodal. (For example, Doebeli [1996] shows trimodal solutions for a very similar model—here also, disruptive selection is generated by limited trait range.) Thus, the causes of clustering in the sexual and asexual models are quite different. Moreover, in the sexual case disruptive selection does not emerge from a fully continuous model—an attractive feature of this scheme. Instead, it is imposed by the arbitrary locations of boundaries to the trait. Bürger and Gimelfarb (2004) provide detailed analysis of the effects of stabilizing and frequency-dependent selection on an additive trait with limited variance, using quadratic approximation of the Roughgarden/Dieckmann-Doebeli model. Their analysis shows more polymorphism if maximum variance is greater than the predicted equilibrium (Slatkin 1979, eq. 30), in accordance with our conclusions.

Infinitesimal Model

The overwhelming evidence from artificial selection is that traits can be selected to move many standard deviations outside their original range of variation. Indeed, no limit to selection has been observed in the majority of selection experiments involving large populations (Barton and Keightley 2002). For traits with an indefinite range and determined by large numbers of additive loci, the infinitesimal model provides a convenient model (Bulmer 1980). We use it to derive explicit Gaussian solutions to the model of selection and assortment used by Dieckmann and Doebeli (1999). That allows us to predict the exact variance when the equilibrium solution is Gaussian with no remaining selection; when disruptive selection arises as perfect adaptation is not possible; and when the variance of Gaussian solution increases to infinity, strongly supporting possible speciation. We find that the infinitesimal model shows good agreement with the symmetric model when six or more loci determine the trait (Fig. 4).

We show that with random mating, the equilibrium is continuous unimodal Gaussian (Fig. 2). However, cost-free assortment alone causes the distribution to diverge indefinitely (Fig. 3A, upward curve). Ecological selection prevents this indefinite increase, so that the resulting distribution has intermediate variance (Fig. 3A, thick downward curve). This analysis shows that it is cost-free assortment that makes sympatric speciation so easy: disruptive selection is not required at all. Gavrilets (2004, ch. 9, pp. 309, 328–330) emphasized this point with reference to a variety of models, stressing that adding cost to assortment results in a quick loss of genetic variation in the corresponding locus.

Parapatric Speciation

Speciation in parapatry is hardly controversial, because even weak selection can lead to divergence (e.g., Haldane 1948; Endler 1973; Caisse and Antonovics 1978). The interesting feature demonstrated by Doebeli and Dieckmann (2003) was that discrete clusters emerge, even when the optimal trait value changes linearly. The existence of clusters might make reinforcement of mating isolation easier, though this was not demonstrated.

Doebeli and Dieckmann (2003) explain evolution of the clusters as a consequence of narrow frequency-dependent competition, similarly to the sympatric model of Dieckmann and Doebeli (1999). However, competition is not necessary: clusters arise when genetic variance is limited even with no competition. We show that clusters are generated by edge effects on a limited range, and the same explanation was independently provided by Gavrilets (2004, pp. 247–248). Reduced maladaptive gene flow at the margins provides an advantage to marginal phenotypes; their population density increases, which suppresses nearby phenotypes. These fluctuations then propagate to the center of the range (Fig. 5). Due to weak coupling between phenotypes (i.e., a low mutation rate and clonal reproduction), the solution can consist of a rather large number of narrow discrete clusters. We have shown that, although exactly two clusters are formed under Doebeli and Dieckmann's (2003) parameters, more can form with a wider range. On an infinite range, the analytical solution to the Roughgarden/Dieckmann-Doebeli model is homogeneous and close to a spatial Gaussian. In numerical solutions, where edge effects can be avoided by wrapping the population around a torus, this continuous distribution is locally stable (Fig. 8A). However, starting from a monomorphic population, the population density can be approximated by a few discrete types even on a torus (Fig. 8B). This is similar to the case of sympatry—even when the equilibrium phenotypic distribution is continuous, it can be approximated by several discrete clusters, unless there is sufficiently strong coupling between the phenotypes (via mutation or sexual reproduction). We stress that this clumping occurs only with very low coupling between phenotypes. Hence, this numerical instability could not possibly drive the evolution of discrete clusters in sexual populations. A slight advantage of marginal phenotypes only triggers evolution of distinct phenotypes as assortment is cost-free.

We believe that our analysis of Dieckmann and Doebeli's models clarifies the various processes that were seen in simulations. Broadly, our results support the argument that sympatric speciation is plausible if there is sufficiently strong source disruptive selection, together with a sufficiently labile mating system (Maynard Smith 1966; Felsenstein 1981a; Gavrilets 2003). However, our analysis of the infinitesimal model supports Gavrilets's (2004) review by showing that nonrandom mating alone can lead to divergence without any ecological selection at all. The question is then whether such extremely labile mating systems are likely to be found in nature.

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Appendix 1: Torus

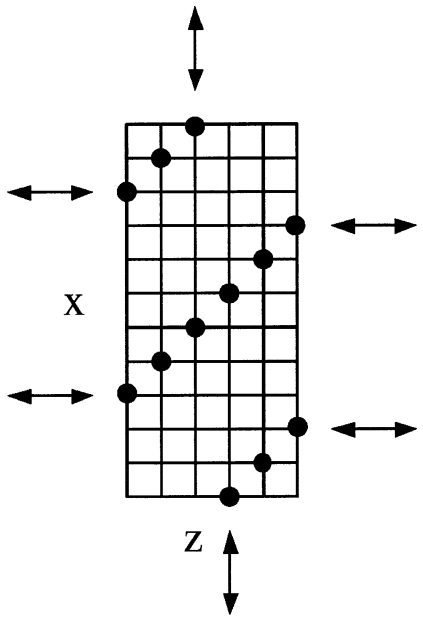

Fig. A1. To simulate the behavior on a torus, we set the fitness to be a sum of Gaussians, with optimum on $Z = BX \pm 2k(Z_m + \delta Z/2)$ and the range to $|Z + \delta Z/2| = B/2|X + \delta X/2|$; $k = 0, 1$. Then the gradient matches, had the two-dimensional surface been twisted in both directions. Individuals (densities) migrating from any place do not experience abrupt changes in fitness, as fitness is precisely matched for the individuals migrating out of the latice, redirected to the opposite margin.

Table A1. Notation of variables used in this paper, Dieckmann and Doebeli (1999), and Doebeli and Dieckmann (2003).

This paper	Dieckmann and Doebeli	
z	u	trait (ecological character) axis
Z	—	scaled trait, $Z = z/\sigma_k\sqrt{r_0}$, in the range $\langle -Z_m, Z_m\rangle$, with spacing δZ in numerical solutions
x	x	spatial axis, the second spatial axis in the Doebeli and Dieckmann (2003) paper, y, is omitted here as the solution is uniform for it
X	—	scaled spatial axis, $X = (x/\sigma_d)\sqrt{2r_0}$, in the range $\langle -X_m, X_m\rangle$, with spacing δX in numerical solutions
b	a	gradient of optimal value, $z_{opt} = bx$
B	—	scaled gradient $B = (b\sigma_d)/(r_0\sigma_k\sqrt{2})$
ψ	N	population density
$\tilde{\psi}$	N_{eff}	effective density, ψ weighed by phenotypic (and spatial) frequency-dependent competition
r_0	r	maximum growth rate
r		growth rate, intrinsic rate of increase (unscaled)
R	—	growth rate defined by the scaled variables
t		time (in generations)
T	—	scaled time, $T = r_0 t$
σ_z^2		variance of the trait
σ_x^2		variance of spatial location
σ_k^2	σ_K^2	variance of carrying capacity (stabilizing selection) for the trait ($\sigma_k^2 \equiv 1$)
σ_c^2	σ_C^2	variance of phenotypic frequency-dependent competition
σ_s^2	σ_s^2	variance of spatial frequency-dependent competition
σ_d^2	σ_d^2	variance of migration
σ_{mut}^2	σ_a^2	variance of mutation
μ	μ_a	probability of mutation
m	—	variance of migration in the stepping-stone model
σ_g^2		genetic variance (variable)
σ_f^2		variance within families (i.e., variance of offspring)
σ_A^2		variance of assortment
y		strength of assortment
γ		strength of assortment relative to genetic variance $\gamma = y(\sigma_g^2/\sigma_A^2)$
γ^*		strength of assortment relative to within-family variance $\gamma = y(\sigma_f^2/\sigma_A^2)$

Appendix 2: Method of Variations

Under asexual reproduction with no mutation, different phenotypes reproduce independently. Interactions between phenotypes occur only via the competition function, G, which determines the effective local density, $\tilde{\psi}$. Thus, the key mathematical problem is to determine the spatial distribution of a given phenotype, Z. This depends solely on its local growth rate, $R[X, Z]$. Numerical calculations show that for a wide range of parameters, this distribution is approximately Gaussian in space. Here, we describe a simple method for finding such a Gaussian approximation. We treat R as dependent on X only: the solution is homogeneous along Z ($Z = BX$); and we omit the dependence of R on ψ as R is zero for any $\psi > 0$ at equilibrium. (We demonstrate numerically that these are sensible approximations; Fig. A1, see also Fig. A2).

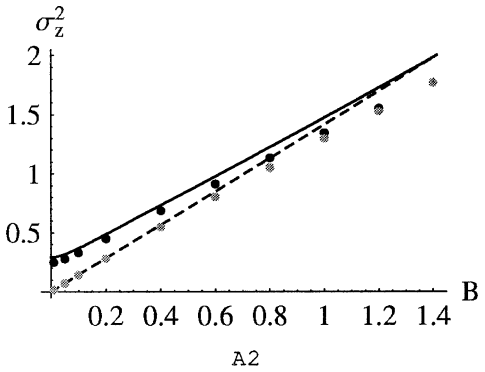

FIG. A2. The trait variance of central phenotypes under the logistic model, plotted against the gradient, B (dots). This can be approximated by the method of variations (solid line), assuming an unimodal Gaussian distribution. Note that for very shallow gradients ($B \leq 0.02$) the true distribution of phenotypes is in fact bimodal (Figs. 5A, 8A). $\sigma_c^2 = 0.7^2$, $\sigma_s^2 = 1$, range $|X| < 40$, $|Z| < 5$; grid spacing $\delta X = 0.5$, $\delta Z = 0.2$, $T = 12.5$. Dashed line shows simple Gaussian prediction $\sigma_z^2 = B\sqrt{2}$ with no frequency-dependent competition (i.e., $\sigma_c \to \infty$, $\sigma_s \to 0$). In the logistic model, the maximum gradient for the solution to be positive is $B = \sqrt{2}$.

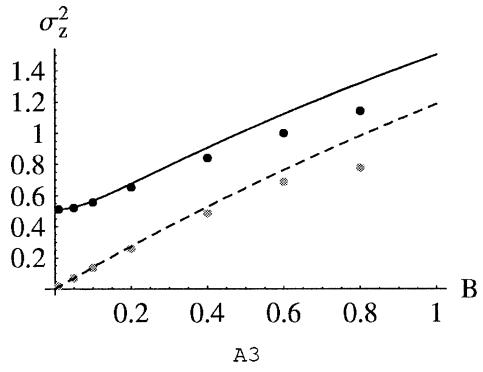

FIG. A3. The trait variance of central phenotypes under the Roughgarden/Dieckmann-Doebeli model ($\sigma_c^2 = 0.7^2$, $\sigma_s^2 = 1$), plotted against the gradient, B. Solution is always unimodal Gaussian. Parameters as in Figure 6. Dashed line shows the method of variations approximation ($\sigma_z^2 = 8/1 + \sqrt{1 + 32/B^2}$), gray circles the numerical results with no frequency-dependent competition (i.e., $\sigma_c \to \infty$, $\sigma_s \to 0$). Numerical solutions for $B = 0.6$ and 0.8 were obtained as $B^2\mathrm{var}(X)$ rather then calculating the phenotypic variance σ_z^2 directly from the numerical solution, which fluctuates due to edge effect.

According to Euler, Lagrange differential equation (e.g., Arfken and Weber 2001), the solution to equation 1 is an extremum of:

$$J = \frac{1}{2}\int_{-\infty}^{\infty} [(\partial_X \psi)^2 - R[X]\psi^2]\, dX. \tag{A1}$$

Suppose that ψ is constrained to take some particular form, described by parameters c. We approximate the solution by finding the extremum of J with respect to these parameters. This approximation is a good one, in the sense that it minimizes the mean square rate of change, $\partial_T J = -\int_{-\infty}^{\infty} (\partial_T \psi)^2\, dX$. The condition for an extremum is:

$$\partial_c J = 0 = -\int_{-\infty}^{\infty} (\partial_{X,X}\psi + R[X]\psi)\partial_c \psi\, dX. \tag{A2}$$

For example, suppose that ψ is a Gaussian, $A \exp[-\alpha(X - Y)^2/2]$. Then, $\partial_{X,X}\psi = \psi[\alpha^2(X - Y)^2 - \alpha]$,

$$0 = -\int_{-\infty}^{\infty} [\alpha^2(X - Y)^2 - \alpha + R[X]]\psi\partial_c\psi\, dX$$

$$= E^*\left\{[\alpha^2(X - Y)^2 - \alpha + R[X]]\frac{\partial_c\psi}{\psi}\right\}, \tag{A3}$$

where

$$E^*[f] \equiv \frac{\int_{-\infty}^{\infty} f\psi^2\, dX}{\int_{-\infty}^{\infty} \psi^2\, dX}.$$

Differentiating with respect to each of the three parameters: $\partial_A\psi = \psi/A$, $\partial_Y\psi = \alpha(X - Y)\psi$, $\partial_\alpha\psi = \psi(X - Y)^2/2$. Thus:

$$0 = \alpha^2 E^*[(X - Y)^2] - \alpha + E^*[R(X)], \tag{A4a}$$

$$0 = E^*[R(X - Y)], \quad \text{and} \tag{A4b}$$

$$0 = \alpha^2 E^*[(X - Y)^4] - \alpha E^*[(X - Y)^2] + E^*[R(X - Y)^2], \tag{A4c}$$

where we have used the fact that $E^*[(X - Y)^k] = 0$ for odd k.

APPENDIX 3: NUMERICAL METHODS

Our basic model (eq. 1) is continuous in both space, X, and phenotype, Z. We approximate this by assuming that individuals can take a discrete set of phenotypes (Z, with range $[-Z_m, Z_m]$ and spacing δZ), and live at discrete points (X, with range $[-X_m, X_m]$ and spacing δX); reproduction occurs with generation time δT. We approximate migration by a stepping-stone model, specified by the probability that individuals move ($\ldots -2, -1, 0, 1, 2 \ldots$) demes, and then reproduce with fitness $\exp(R\delta T)$. This choice approximates $1 + R\,\delta T$ for small δT and ensures that offspring numbers are always positive. The advantage of this simple scheme is that it corresponds to a definite population genetic model and is more robust than more sophisticated interpolation algorithms (e.g., Runge-Kutta).

In the model with no competition, we use nearest neighbor migration, with a fraction $m/2$ being exchanged with each of the two neighboring demes in each time step. In scaled variables, the population density after migration is $\psi_{T+\delta T} = \psi T + (m/2)[\psi_T(X + \delta X) - 2\psi_T(X) + \psi_T(X - \delta X)]$; we choose $m = \frac{1}{2}$ throughout. Local density is measured simply as the density at the lattice point. In the more general model with competition, we approximate a Gaussian distribution both to measure local density and to describe the distribution of migration distances. We begin with a discretized Gaussian distribution $g(X) = \exp[-x^2/(2\sigma_d^2)]$, truncated at $k = 3$ standard deviations. This distribution will not have precisely the desired variance, σ_d^2, and so we correct it to adjust the variance to equal σ_d^2:

$$g^*(X) = g(X)\frac{[s_4 + s_0 vX^2 - s_2(v + X^2)]}{s_0 s_4 - s_2^2}, \tag{A5}$$

where s_j is the jth moment of the trial distribution, g; X has spacing δX; and v is the variance of the untruncated Gaussian. This adjustment has only a small effect if the grid spacing δX is small. Finally, we set the variance of the dispersal distribution to $\sigma_d^2 = 2\delta T$, to be consistent with the scalings $\delta T = r_0\,\delta t$, $\delta X^2 = \delta x^2(2r_0/(\sigma^2)$. Competition from individuals with similar phenotypes or at nearby locations and mutation that moves offspring to a different phenotypic class are handled in the same way as dispersal along the X-axis. Numerical investigations show that changing the lattice spacing (δX, δZ) does not affect the solution, ψ. The order of events in numerical calculations is migration → reproduction → mutation.

APPENDIX 4: INFINITESIMAL MODEL

Normalization of Cost-Free Assortment

To ensure that assortment causes no direct sexual selection requires that we find normalization factors $d(x)$ such that:

$$1 = \int \tilde{A}[z', z'']\psi[z'']\, dz'' \quad \forall z', \tag{A6}$$

where $\tilde{A}[z', z''] = D[z']A[z', z'']D[z'']$. In general, finding the D is not trivial: it requires solution of a multivariate quadratic for D, (eq. A6). However, if the assortment function A and the trait distribution are both Gaussian, then we have:

$$\tilde{A}[z', z''] = \frac{\sqrt{1 + \gamma(1 - d)}}{C} \times \exp\left\{-\frac{Y}{2\sigma_A^2}[(z' - z'')^2 - d(z'^2 + z''^2)]\right\}, \quad \text{(A7)}$$

where

$$C = \int \psi[z'']\, dz'', \qquad d = 1 + \frac{1}{2\gamma} - \sqrt{1 + \frac{1}{4\gamma^2}}, \quad \text{and} \quad \gamma = \frac{y\sigma_g^2}{\sigma_A^2}.$$

Ecological Selection: Random Mating

Suppose that $K = K_0\exp[-x^2/(2\sigma_k^2)]$, G is Gaussian with variance $\sigma_c^2 < 1$. We seek a Gaussian solution for ψ with variance σ_g^2 and mass C. We must have:

$$0 = -\frac{r}{K_0}\frac{C}{2\pi\sqrt{\sigma_g^2(\sigma_g^2 + \sigma_c^2)}}\exp\left[-\frac{z^2}{2}\left(\frac{1}{\sigma_g^2} + \frac{1}{\sigma_g^2 + \sigma_c^2} - \frac{1}{\sigma_k^2}\right)\right] + \frac{r}{\sqrt{2\pi\left(\frac{\sigma_g^2}{2} + \sigma_f^2\right)}}\exp\left[-\frac{z^2}{2\left(\frac{\sigma_g^2}{2} + \sigma_f^2\right)}\right]. \quad \text{(A8)}$$

This implies that a Gaussian equilibrium exists, with variance σ_g^2 and density C, which satisfy:

$$\frac{1}{\sigma_g^2} + \frac{1}{\sigma_g^2 + \sigma_c^2} - \frac{1}{\sigma_k^2} = \frac{1}{\frac{\sigma_g^2}{2} + \sigma_f^2} \quad \text{(A9a)}$$

$$C = K_0\sqrt{\frac{2\pi\sigma_g^2(\sigma_g^2 + \sigma_c^2)}{\frac{\sigma_g^2}{2} + \sigma_f^2}} = K_0\sqrt{2\pi[2\sigma_g^2 + \sigma_c^2 - \sigma_g^2(\sigma_g^2 + \sigma_c^2)]}, \quad \text{for } \sigma_k^2 = 1. \quad \text{(A9b)}$$

Assortative Mating, No Ecological Selection

The distribution of newborn individuals is:

$$\int \psi(z')\psi(z'')\tilde{A}(z', z'')N\left[\frac{(z' + z'')}{2}, \sigma_f^2\right] dz'\, dz'' = \sqrt{\frac{2[1 + \gamma(1 - d)]}{2\pi\sigma_f^2[1 + \gamma(2 - d)][2(1 - d\gamma) + \gamma/\gamma^*]}} \times C\exp\left\{-\frac{z^2}{2\sigma_f^2}\left[\frac{2(1 - d\gamma)}{2(1 - d\gamma) + \gamma/\gamma^*}\right]\right\}, \quad \text{(A10)}$$

where $d = 1 + 1/2\gamma - \sqrt{1 + (1/4\gamma^2)}$, so that all phenotypes have the same fitness, the leading factor in equation (A10) tends to

$$\sqrt{\frac{2(1 - \gamma d)}{2\pi\sigma_f^2[2(1 - d\gamma) + \gamma/\gamma^*]}},$$

which is the same as the coefficient in the exponential term. The distribution is therefore a Gaussian with unit mass, providing a check on the calculation.

For there to be a Gaussian solution in which birth balances death, the variance of the offspring distribution must match the variance of the parent distribution:

$$\frac{\sigma_f^2[2(1 - d\gamma) + \gamma/\gamma^*]}{2(1 - d\gamma)} = \sigma_g^2. \quad \text{(A11)}$$

Using $\sigma_g^2/\sigma_f^2 = \gamma/\gamma^*$,

$$2(\gamma - \gamma^*)(1 - d\gamma) = \gamma, \quad \text{(A12)}$$

which solves to give the equilibrium variance (expressed in terms of $\gamma = y\sigma_g^2/\sigma_A^2$). For pure assortment,

$$d = 1 + \frac{1}{2\gamma} - \sqrt{1 + \frac{1}{4\gamma^2}},$$

and so:

$$\gamma = \frac{2\gamma^*(1 - 2\gamma^*)}{(1 - 4\gamma^*)}. \quad \text{(A13)}$$

Ecological Selection Combined with Assortative Mating on the Same Trait

Combining ecological selection with nonrandom mating by matching the expressions for the changes due to death and birth, we obtain:

$$\frac{1}{\sigma_g^2} + \frac{1}{\sigma_g^2 + \sigma_c^2} - \frac{1}{\sigma_k^2} = \frac{2(1 - d\gamma)}{\sigma_f^2[2(1 - d\gamma) + \gamma/\gamma^*]} \quad \text{(A14a)}$$

and

$$C = K_0\sqrt{2\pi}\sqrt{\frac{2[\gamma(1 - d) + 1]\sigma_g^2(\sigma_g^2 + \sigma_c^2)}{\sigma_f^2[\gamma(2 - d) + 1](2 - 2d\gamma + \gamma/\gamma^*)}}. \quad \text{(A14b)}$$

Substituting for d, assuming pure assortment, the first equation leads to:

$$\frac{1}{\sigma_g^2} + \frac{1}{\sigma_g^2 + \sigma_c^2} - \frac{1}{\sigma_k^2} = \frac{1}{\sigma_g^2}\frac{4\gamma}{4\gamma^* + 2\gamma - 1 + \sqrt{1 + 4\gamma^2}}. \quad \text{(A15)}$$

This equilibrium can be compared with the equilibrium under assortment alone (eq. A11), which diverges for $\gamma^* > ¼$.