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The Maynard Smith model of sympatric speciation

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Abstract

The paper entitled "Sympatric speciation," which was published by John Maynard Smith in 1966, initiated the development of mathematical models aiming to identify the conditions for sympatric speciation. A part of that paper was devoted to a specific two-locus, two-allele model of sympatric speciation in a population occupying a two-niche system. Maynard Smith provided some initial numerical results on this model. Later, Dickinson and Antonovics (1973) and Caisse and Antonovics (1978) performed more extensive numerical studies on the model. Here, I report analytical results on the haploid version of the Maynard Smith model. I show how the conditions for sympatric and parapatric speciation and the levels of resulting genetic divergence and reproductive isolation are affected by the strength of disruptive selection and nonrandom mating, recombination rate, and the rates of male and female dispersal between the niches.
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1. Introduction

John Maynard Smith has made a number of extremely important contributions to evolutionary biology, many of which have also found various applications well outside evolutionary biology (see other papers in this issue). One of his earlier influential works was the paper entitled "Sympatric speciation" published by American Naturalist in 1966.

There were two major stimulas for the paper in a few preceding years. The first was a series of laboratory experiments with *Drosophila melanogaster* by Thoday and his colleagues (Millicent and Thoday, 1961; Thoday and Boam, 1959; Thoday and Gibson, 1962) that showed rapid emergence of very strong reproductive isolation. These experiments suggested that sympatric speciation can proceed in a straightforward way. The second was the publication of Ernst Mayr's (1963) monumental volume

"Animal speciation and evolution" which forcefully argued against the importance of sympatric speciation in nature. By the mid-1960s, solid theoretical work on sympatric speciation was needed to help clarify the arguments and conflicting evidence on sympatric speciation.

The first part of the paper analysed the conditions for the maintenance of genetic variation in a two-deme version of the Levene model (Levene, 1953) which Maynard Smith generalized for the case of habitat choice by females. The second part was devoted to the following question: given that genetic variation is stably maintained under disruptive selection, can reproductive isolation evolve between the two "extreme" morphs, so that no low-fitness intermediates are produced even when different individuals freely encounter each other?

Maynard Smith discussed four possible mechanisms that can lead to the evolution of reproductive isolation in the presence of gene flow: (1) "habitat selection," by which he meant the tendency of organisms to mate in the same niche where they were born, (2) "pleiotropism," by which he meant the situations in which the alleles that adapt the individuals to local conditions also control mating behavior, (3) "modifier genes," by which

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he meant the establishment of selectively neutral genes that cause pleiotropism in the genes underlying local adaptation, and (4) "assortative mating genes," by which he meant genes that cause assortative mating regardless of the genotype at the selected loci. I note that the majority of the later theoretical research on sympatric speciation has been done along these four lines first discussed by Maynard Smith. Maynard Smith also proposed a specific model of sympatric speciation via the mechanism of "assortative mating genes" which will be the focus of this paper.

In the years since 1966 sympatric speciation went through cycles of increased enthusiasm and skepticism accompanied by a continuous growth in both empirical (reviewed by Coyne and Orr, 2004) and theoretical (reviewed by Gavrilets, 2004) knowledge. Unfortunately, most of the theoretical work remains based on numerical simulations which significantly limits the generality of theoretical conclusions (Gavrilets, 2003). Given the exploding growth of interests in speciation, in general, and in sympatric speciation, in particular, and given the controversies that keep remain associated with sympatric speciation (as is apparent from a comparison of major conclusions on sympatric speciation of Coyne and Orr, 2004 and Gavrilets, 2004 and those of Dieckmann et al., 2004) there is a growing need of a solid quantitative theory of speciation. Although recently a number of simple models of sympatric speciation have been solved analytically (Gavrilets, 2004), obtaining analytical results still remains a major theoretical challenge. Below I present analytical results for a simplified version of the model introduced by Maynard Smith. A part of these results were briefly introduced in Gavrilets (2004, Chapter 10).

I will define sympatric speciation as the emergence of new species from a population where mating is random with respect to the birthplace of the mating partners (Gavrilets, 2003, 2004). Note that, implicitly, mating is allowed to be nonrandom with respect to, for example, genotype, phenotype, and culturally inherited traits. This definition is actually implied in most existing mathematical models of sympatric speciation.

2. The Maynard Smith model

Consider a diploid population with discrete nonoverlapping generations inhabiting two distinct niches. Assume that density-dependent factors operating independently within each niche maintain population sizes at constant levels that do not depend on the average fitness of the populations. This is the case of soft selection (e.g. Christiansen, 1975; DeMeeus et al., 1993; Wallace, 1968).

Let individuals differ with regard to the disruptive selection (DS) locus with alleles **A** and **a** controlling fitness (viability) in a local environment according

to the following scheme:

	AA	**Aa**	**aa**
in niche 1	1	1	$1 - s_1$
in niche 2	$1 - s_2$	$1 - s_2$	1

Here the coefficients s_1 and s_2 measure the strength of viability selection within each niche ($0 \leqslant s_1, s_2 \leqslant 1$). That is, the dominant allele **A** is advantageous in niche 1, whereas the recessive allele **a** is advantageous in niche 2. Maynard Smith assumed that surviving adults form a single randomly mating population. After mating, each female lays a fraction $(1 + H)/2$ of her eggs in the niche where she was raised and lays the remaining fraction $(1 - H)/2$ in the other niche ($0 \leqslant H \leqslant 1$). The coefficient H measures the degree of habitat selection by females. $H = 0$ implies no habitat selection, as in the Levene model (Levene, 1953; Gavrilets, 2004, pp. 235–240). $H = 1$ implies complete habitat selection. Note that, alternatively, one can think of mating as taking place *within* the niches, with males migrating randomly between the niches prior to mating and females either staying in the niche where they were born or going to the other niche with probabilities $(1 + H)/2$ and $(1 - H)/2$, respectively.

The first question to ask is whether spatially heterogeneous selection maintains genetic variation in the DS locus. For simplicity, assume that the sizes of the two populations are the same. Then, as shown by Maynard Smith (1966), if $H = 0$ (i.e. females lay eggs in the two niches at random), genetic variation is maintained if

$$-1 < \frac{s_2 - s_1}{s_1 s_2} < 1.$$

If $H = 1$ (females always lay eggs in the niche where they themselves were raised), then genetic variation is maintained if

$$-\frac{3}{2} < \frac{s_2 - s_1}{s_1 s_2} < 3.$$

These inequalities show that maintaining genetic variation requires sufficiently strong selection (i.e. large s_1 and s_2); increasing the degree of habitat choice by females H increases the range of conditions resulting in stable polymorphism.

At the polymorphic equilibrium the locally advantageous alleles (i.e. allele **A** in niche 1 and allele **a** in niche 2) have higher frequencies than the locally deleterious alleles (i.e. allele **a** in niche 1 and allele **A** in niche 2). However, because mating is random, offspring with locally deleterious genotypes will be constantly produced at relatively high frequencies which will reduce the average fitness of each population.

Assume next that there is also a nonrandom mating (NM) locus with alleles **B** and **b** controlling assortative mating. Let the rate of recombination between the DS and NM locus be r ($0 \leqslant r \leqslant \frac{1}{2}$). We will suppose that mating occurs according to the O'Donald model with dominance (O'Donald, 1960, 1980; Gavrilets, 2004, pp. 287–290). That

is, individuals **BB** and **Bb** have the same phenotype which is recognizably different from the phenotype of individuals **bb**. With probability α, an individual mates with another individual that has the same phenotype ($0 \leqslant \alpha \leqslant 1$). With probability $1 - \alpha$, the individual is engaged in random mating.

For the special case of $H = 1$ (i.e. complete habitat selection by females), $\alpha = 1$ (i.e. no mating between genotypes **BB**/**Bb** and **bb** is allowed), and $r = \frac{1}{2}$ (i.e. the loci are unlinked), Maynard Smith presented a numerical example illustrating that an initial difference in the frequencies of NM alleles between the niches gets progressively amplified. One NM allele gets associated with the locally advantageous allele **A** in niche 1, and the other NM allele gets associated with the locally advantageous allele **a** in niche 2. As a result of this, the individuals raised in a niche will tend to mate together. The eventual outcome of the evolutionary dynamics is the increase in frequencies of two reproductively isolated genotypes **AA**/**BB** and **aa**/**bb** (or **AA**/**bb** and **aa**/**BB**) and the complete disappearance of all intermediate genotypes. This outcome is naturally interpreted as sympatric speciation. [Note that although in Maynard Smith's simulations females always lay eggs in the niche where they were born, speciation is sympatric according to our definition because males move randomly between the niches.] Although Maynard Smith did acknowledge that the parameter combination he used was very favorable for speciation but unrealistic, he thought that more realistic conditions would merely slow down the speciation process but not prevent it completely. According to his 1966 paper, the crucial process in sympatric speciation is the establishment of a stable polymorphism under disruptive selection rather than the subsequent evolution of reproductive isolation, which he believed was likely (p. 649).

Thorough numerical analysis of the Maynard Smith model was undertaken by Dickinson and Antonovics (1973) as a part of their study of a number of different models. They assumed that the alleles at the DS locus act additively (so that in each niche, the fitness of heterozygotes is the average of the fitnesses of homozygotes), and allowed for a limited migration of males between the niches at rate m. The females did not migrate. This model was designed to reflect plant situations, where there is pollen transfer between sessile organisms and seed flow is negligible. Dickinson and Antonovics numerically studied the progress towards speciation during the first 400 generations as a function of the strength of selection for local adaptation s (which was assumed to be equal in both niches), the strength of assortative mating α, and the male migration rate m (see their Fig. 5). The progress toward speciation was measured by the difference Δ_{NM} in the frequency of an NM allele between the niches. Depending on parameter values, the system evolved to a state with no differentiation in the NM locus (i.e. $\Delta_{NM} = 0$) or to a state with some differentiation in the NM locus (i.e. $|\Delta_{NM}| > 0$). The first outcome is promoted by large migration, weak

selection, and weak assortative mating. The second outcome is promoted by low migration, strong selection, and strong assortative mating. This outcome is interpreted as (a step towards) speciation that is sympatric or parapatric, depending on whether male migration rate is $m = \frac{1}{2}$ or $m < \frac{1}{2}$.

Overall, Dickinson and Antonovics (1973) confirmed Maynard Smith's conclusion that sympatric speciation is theoretically possible. However, they also showed that the maintenance of genetic variation under disruptive selection does not necessarily lead to the evolution of reproductive isolation. Some additional conditions have to be satisfied as well. In particular, for sympatric speciation to occur (i) assortative mating has to be strong enough and (ii) the joint strength of selection and assortative mating has to be large enough.

Later, Caisse and Antonovics (1978) extended the two-deme results of Dickinson and Antonovics (1973) to the case of 10 demes arranged along a line. In one version of their model, the rate of male migration between demes decreases exponentially with the distance between them. The other version assumes that males migrated only between the neighboring demes (i.e. as in the stepping-stone model). The strength of selection acting on the DS locus increased linearly from deme 1 to deme 10 or undertook a step change between the two deme in the middle of the array (see Endler, 1977 for a review of earlier work on spatially heterogeneous selection). Most of the results given by Caisse and Antonovics (1978) are for unlinked loci and $\alpha = 0.9$, that is, very strong assortative mating. Slow convergence towards equilibria and the relatively short duration of the simulations (typically 400 generations) make interpretation of their results difficult because there is no guarantee that they correspond to equilibrium rather than transient states. Overall, however, their results appear to be quite compatible with those of Dickinson and Antonovics. In particular, strong selection ($s > 0.1$) and high levels of assortative mating ($\alpha > 0.4$) are necessary for a divergence at the NM locus.

3. The haploid version of the Maynard Smith model

To get a better understanding of the interactions between disruptive selection and nonrandom mating, let us consider a haploid version of the Maynard Smith model. Let the DS locus control viability according to the following symmetric scheme:

	A	**a**
viability in niche 1	1	$1 - s$
viability in niche 2	$1 - s$	1,

$$(1)$$

where coefficient s measures the strength of selection within each niche ($0 \leqslant s \leqslant 1$). Note that this type of spatially heterogeneous selection always maintains genetic variation at the DS locus with both alleles having frequency $\frac{1}{2}$ in both niches. Let the NM locus control assortative mating

according to the O'Donald model. That is, with probability α, an individual mates with another individual that has exactly the same allele (**B** or **b**) at the NM locus. With probability $1 - \alpha$, the individual mates irrespective of genotype.

Assume that migration takes place after selection but before mating and that the sizes of the populations are the same and constant (i.e. soft selection). Let males migrate between the niches with probability m per generation, and females with probability f per generation. Note that parameter f corresponds to parameter H in the original Maynard Smith model. In the special case numerically studied by Maynard Smith, females always lay eggs in the niche they were born ($f = 0$) whereas males disperse randomly ($m = \frac{1}{2}$).

In this model, there are four different genotypes and two different niches. Thus, the population genetic state has to be characterized by $2 \times (4 - 1) = 6$ independent variables. In spite of this complexity, analytical progress is possible by utilizing the symmetry of the model.

3.1. Dynamic equations

Let the frequencies of four genotypes **AB**, **Ab**, **aB**, and **ab** among offspring in the ith niche be $z_{i,1}, z_{i,2}, z_{i,3}, z_{i,4}$. Assume that the viability of the jth genotype in the ith niche is $w_{i,j}$ ($i = 1, 2; j = 1, 2, 3, 4$). Let us define indicator variables η_i showing the presence or absence of allele **B** at genotype i: $\eta_1 = \eta_3 = 1, \eta_2 = \eta_4 = 0$. Let us also define indicator variables δ_{ij} showing whether genotypes i and j have the same allele at the NM locus. The values of δ_{ij} for different pairs of genotypes can be represented by a matrix:

$$\delta = \begin{pmatrix} 1 & 0 & 1 & 0 \\ 0 & 1 & 0 & 1 \\ 1 & 0 & 1 & 0 \\ 0 & 1 & 0 & 1 \end{pmatrix}.$$

The genotype frequencies among surviving offspring in niche i are

$$z_{i,j}^s = \frac{w_{i,j}}{\overline{w}_i} z_{i,j}, \tag{2}$$

where $\overline{w}_i = \sum_j w_{i,j} z_{i,j}$ is the average fitness in niche i.

After migration, the genotype frequencies in mating females are

$$x_{1,j} = (1 - f)z_{1,j}^s + f z_{2,j}^s, \tag{3a}$$

$$x_{2,j} = (1 - f)z_{2,j}^s + f z_{1,j}^s, \tag{3b}$$

and the genotype frequencies in mating males are

$$y_{1,j} = (1 - m)z_{1,j}^s + m z_{2,j}^s, \tag{4a}$$

$$y_{2,j} = (1 - m)z_{2,j}^s + m z_{1,j}^s. \tag{4b}$$

Note that the frequency of allele **B** among mating males in niche i is

$$Y_i = y_{i,1} + y_{i,3}, \tag{5}$$

and that of alleles **b** is $1 - Y_i$.

The frequency of matings in niche i between females with genotype k and males with genotype l can be written as

$$P_i(k \times l) = x_{i,k} y_{i,l} \left(1 - \alpha + \alpha \frac{\delta_{kl}}{Y_{\eta_l}}\right). \tag{6}$$

This expression implies that each female mates randomly with probability $1 - \alpha$ and assortatively with probability α. Here variable Y_{η_l} is the population frequency of the allele present at the NM locus of males with genotype l.

Genotype frequencies among offspring born in niche i in the next generation are

$$p_{i,j}' = \frac{1}{\overline{W}} \sum_{k,l} P_i(k \times l) R(k, l \to j), \tag{7}$$

where $R(k, l \to j)$ is the probability that $k \times l$ mating results in offspring with genotype j (e.g. Nagylaki, 1992, Eq. 8.9; see also Bürger, 2000), and $\overline{W}$ is the normalizing factor (such that $\sum_j p_{i,j}' = 1$).

To analyse the dynamic equations (2)–(7) it is convenient to use a transformation proposed by Karlin and Feldman (1970). Specifically, let

$$u_{i,1} = z_{i,1} + z_{i,2} + z_{i,3} + z_{i,4}, \tag{8a}$$

$$u_{i,2} = z_{i,1} - z_{i,2} + z_{i,3} - z_{i,4}, \tag{8b}$$

$$u_{i,3} = z_{i,1} + z_{i,2} - z_{i,3} - z_{i,4}, \tag{8c}$$

$$u_{i,4} = z_{i,1} - z_{i,2} - z_{i,3} + z_{i,4} \tag{8d}$$

be new variables describing the genetic state of the population in niche i. Note that $u_{i,1} = 1$, that the frequencies of alleles **A** and **B** are

$$z_{i,A} = z_{i,1} + z_{i,2} = \frac{1 + u_{i,3}}{2}, \tag{9a}$$

$$z_{i,B} = z_{i,1} + z_{i,3} = \frac{1 + u_{i,2}}{2}, \tag{9b}$$

and that linkage disequilibrium is

$$D_i = z_{i,1} z_{i,4} - z_{i,2} z_{i,3} = \frac{u_{i,4} - u_{i,2} u_{i,3}}{4}. \tag{9c}$$

In this model, the population always evolves to an equilibrium state where the locally advantageous allele has a higher frequency than the locally deleterious allele. With regard to other features, there are two qualitatively different regimes. In the first regime, the population evolves to a line of neutrally stable equilibria at which

$$u_{2,1} = u_{2,2} = U_2, \tag{10a}$$

$$u_{3,1} = -u_{3,2} = U_3, \tag{10b}$$

$$u_{4,1} = -u_{4,2} = U_2 U_3, \tag{10c}$$

where U_2 is an arbitrary number between -1 and 1, and U_3 is a positive solution of a certain quadratic equation. The above conditions imply that the frequencies of the alleles at the NM locus in both niches are the same (and arbitrary), the frequencies of the locally advantageous alleles (i.e. allele **A** in niche 1 and allele **a** in niche 2) are the same and uniquely defined by parameters, and that both local populations are at linkage equilibrium.

In the second regime, the population evolves to one of two "speciation equilibria" with strong differentiation in both loci between the niches and linkage disequilibrium within each niche:

$$u_{2,1} = -u_{2,2} = V_2, \tag{11a}$$

$$u_{3,1} = -u_{3,2} = V_3, \tag{11b}$$

$$u_{4,1} = u_{4,2} = V_4, \tag{11c}$$

where V_3, V_4 are positive and their values as well as the absolute value of V_2 are uniquely defined by parameters. The difference between the equilibria is which NM allele (**B** or **b**) is associated with the locally advantageous DS allele.

We will start analysing system (2)–(7) with the simplest case. Some details of the derivations are outlined in Appendix. A *Maple* file with more details can be downloaded from the author's web page.

3.2. Basic case: no female migration, random male migration, and no linkage

Assume that females never migrate ($f = 0$), males disperse randomly between the niches (i.e. $m = \frac{1}{2}$) and the loci are unlinked (i.e. $r = \frac{1}{2}$). This case corresponds to the one analysed by Maynard Smith (1966).

At the line of equilibria (10), U_2 is an arbitrary number between -1 and 1, and U_3 is given by a positive solution of a quadratic equation

$$U_3^2 + \frac{2-s}{2s} U_3 - \frac{1}{2} = 0. \tag{12}$$

The frequency of the locally advantageous allele at the DS locus is $(1 + U_3)/2$. This frequency grows from $\frac{1}{2}$ to $\frac{3}{4}$ as the strength of selection for local adaptation s increases from 0 to 1. The difference in the frequencies of an DS allele between the niches is equal to U_3. This difference grows from 0 to $\frac{1}{2}$ as s increases from 0 to 1 (see Fig. 1(a)).

The line of equilibria exists always. It is locally stable if

$$\alpha < \alpha_c \equiv \frac{1}{2} + \frac{\sqrt{(3s)^2 + 4(1-s)} - 3s}{4}, \tag{13}$$

that is, if the intensity of assortative mating is smaller than a critical value α_c. Fig. 1(b) illustrates the dependence of α_c on s.
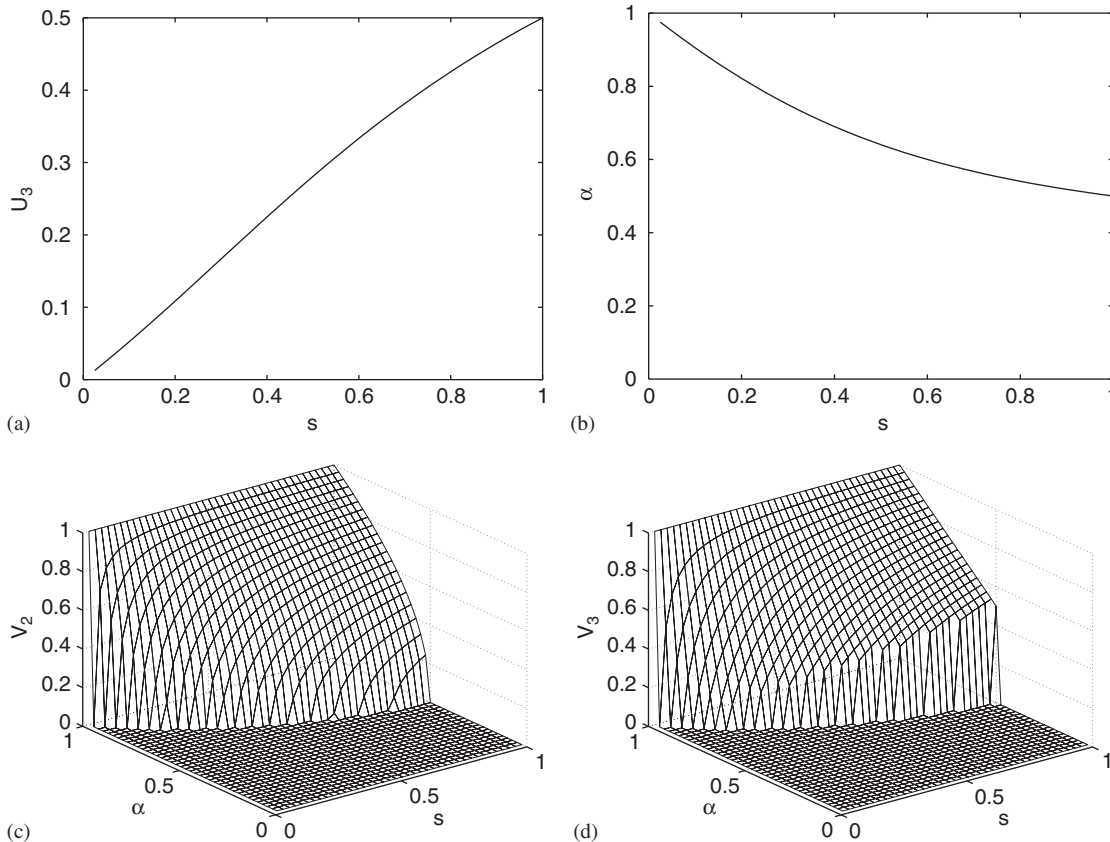

Fig. 1. Equilibria in the basic model. (a) The value of U_3 at the line of equilibria. (b) The critical value of α for the existence of a pair of speciation equilibria. (c) The values of V_3 at a speciation equilibrium. (d) The values of V_2 at a speciation equilibrium.

At the speciation equilibria (11),

$$V_2 = \pm\sqrt{\frac{(1+\alpha)(2\alpha^2 + 3s\alpha - 2\alpha - s)}{4\alpha s}}, \tag{14a}$$

$$V_3 = \frac{s+\alpha-1}{s}, \tag{14b}$$

$$V_4 = V_2. \tag{14c}$$

The speciation equilibria are feasible if α is larger than the critical value α_c defined by Eq. (13). That is, the speciation equilibria exist only when the line of equilibria (10) is unstable. Numerical simulations suggest that no other equilibria are stable in this model. That is, for $\alpha < \alpha_c$ the system evolves to the line of equilibria whereas for

$$\alpha > \alpha_c, \tag{15}$$

the system evolves to a speciation equilibrium.

Following Maynard Smith (1966) and Dickinson and Antonovics (1973), one can characterize the resulting degree of reproductive isolation by the difference in the frequencies of an NM allele between the niches. At the speciation equilibria this difference is equal to $|V_2|$. Fig. 1(c) illustrates the level of between-niche divergence in the NM locus achieved at equilibrium. One can see that once the condition for sympatric speciation is satisfied, the equilibrium value of $|V_2|$ approaches 1 very rapidly with

increasing s and α. Fig. 1(d) illustrates the level of between-niche divergence in the DS locus achieved at speciation equilibria. At the speciation equilibria the difference in the allele frequencies in the DS locus is equal to $|V_3|$. Note that at a speciation equilibrium $|V_3|$ can reach the maximum possible value of 1 whereas at the line of equilibria the maximum value was $\frac{1}{2}$.

The critical value α_c decreases from 1 to $\frac{1}{2}$ as s grows from 0 to 1. That is, sympatric speciation requires sufficiently strong assortative mating and selection for local adaptation. The population switches from the first regime to the second "because" this results in increasing its average fitness. In this model, the gain in the average fitness can be written as $(\alpha - \alpha_c)/8$. Quantitatively similar behavior is observed in some other models of sympatric speciation (Gavrilets, 2004). However, in other models (e.g. Gavrilets, 2005; Kirkpatrick and Ravigné, 2002; Kisdi and Geritz, 1999) whether or not the population evolves to the speciation equilibrium depends also on initial conditions.

3.3. Effects of linkage

The results can be generalized for the case of linked loci. It is well appreciated that close linkage increases both the plausibility and the level of genetic divergence between sympatric populations (e.g. Felsenstein, 1981; Gavrilets,
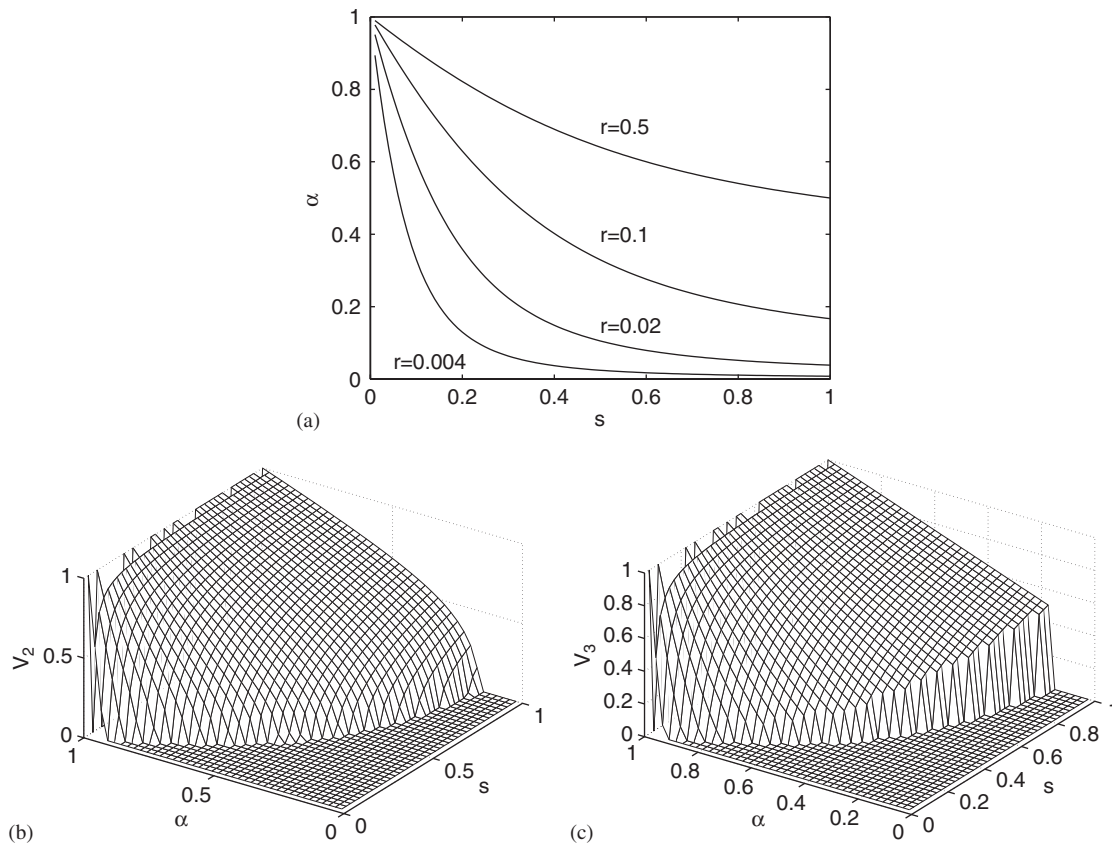

Fig. 2. Effects of recombination rate r on the possibility of sympatric speciation. (a) Critical value of α for four different values of r. (b) The value of V_2 at speciation equilibria with $r = 0.1$. (c) The value of V_3 at speciation equilibria with $r = 0.1$.

2004; Udovic, 1980). Fig. 2 illustrates these effects for the Maynard Smith model. In particular, Fig. 2(a) shows that increasing recombination rate r dramatically increases the range of combinations of s and α leading to sympatric speciation. Figs. 2(b) and (c) show that once conditions for sympatric speciation are satisfied the level of divergence in both the NM and DS loci grows quickly.

3.4. Effects of female migration

Let $m = \frac{1}{2}$ but f and r be between 0 and $\frac{1}{2}$. Note that this is still the case of sympatric speciation (because males disperse randomly). On the line of equilibria U_3 is given by a positive solution of the quadratic equation

$$U_3^2 + (1 + 2f)\frac{2-s}{2s}U_3 - \frac{1}{2} + f = 0. \qquad (16)$$

As before U_3 also gives the difference in the frequency of an allele in the DS locus between the niches (see Fig. 3(b)). Increasing female migration rate f decreases the degree of divergence between the niches in the DS locus with U_3 approaching zero as f approaches $\frac{1}{2}$. Note that U_3 does not depend on the recombination rate.

The effects of female migration rate f on the possibility of sympatric speciation are illustrated in Fig. 3(a). The range of parameter values resulting in sympatric speciation

quickly shrinks with increasing f. Figs. 3(c) and (d) show that increasing female migration also decreases the resulting levels of divergence in both loci.

3.5. Effects of restrictions on male migration

Let $f = 0$ but m and r be between 0 and $\frac{1}{2}$. This is the case of parapatric speciation. On the line of equilibria the value of U_3 is given by a positive solution of the quadratic equation:

$$U_3^2 + m\frac{2-s}{s}U_3 - (1 - m) = 0. \qquad (17)$$

Obviously, decreasing the rate of male migration m increases the frequency of the locally advantageous allele. This effect is illustrated in Fig. 4(b). Note that u_3 does not depend on the recombination rate.

The pair of speciation equilibria (11) are defined by much more cumbersome equations. Fig. 4(a) illustrates the conditions for existence of these equilibria. Interestingly, the effect of reduced male migration is much less significant than that of reduced recombination (compare Fig. 2(a) with Fig. 2(a)). Figs. 4(c) and (d) illustrate the level of between-niche divergence in the NM and DS loci achieved at equilibrium. One can see that once the conditions for sympatric speciation are satisfied, the equilibrium
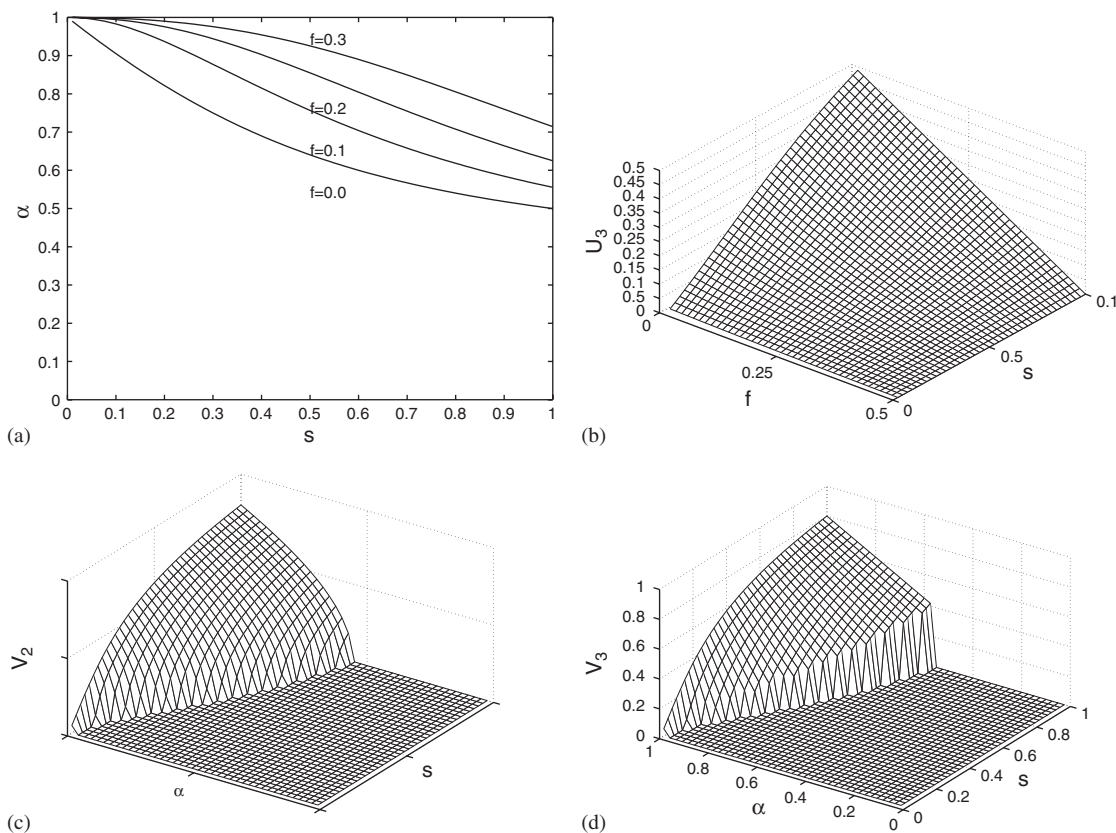

Fig. 3. Effects of female migration rate f on the possibility of sympatric speciation. (a) The critical values of α for the existence of a pair of speciation equilibria for four different values of f. (b) The value of U_3 at the line of equilibria. (c) The value of V_2 at a speciation equilibrium with $f = 0.1$. (d) The value of V_3 at a speciation equilibrium with $f = 0.1$.
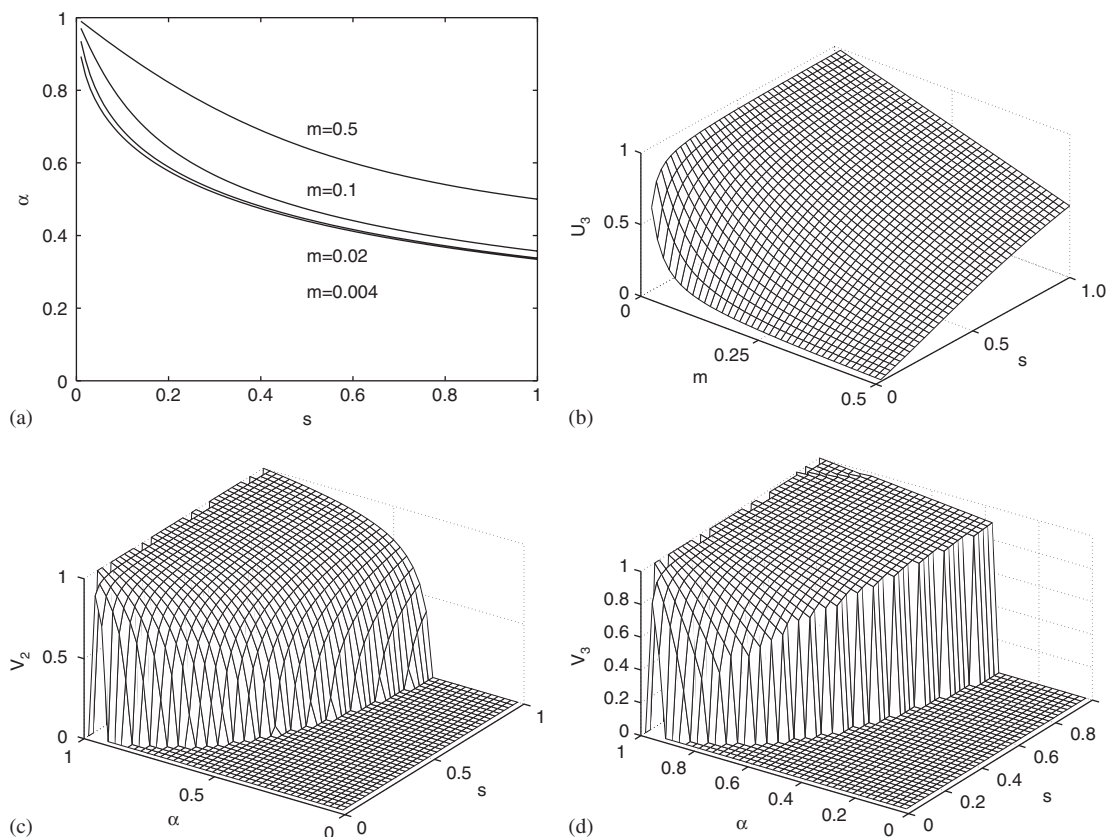
Fig. 4. Effects of male migration rate m on the possibility of parapatric speciation. (a) Critical value of α for four different values of m. The curves corresponding to $m = 0.02$ and $m = 0.004$ are very close to each other. (b) The value of U_3 at the line of equilibria. (c) The value of V_2 at the speciation equilibrium with $m = 0.1$. (d) The value of V_3 at the speciation equilibrium with $m = 0.1$.

values of $|V_2|$ and V_3 approach 1 very rapidly with increasing s and α.

3.6. Equal migration of sexes

Finally, let us assume that the male and female migration rates are arbitrary but equal ($f = m$). In this case, on the line of equilibria the value of U_3 is given by a positive solution of the quadratic equation

$$U_3^2 + 2m\,\frac{2-s}{s}\,U_3 - (1 - 2m) = 0. \qquad (18)$$

Obviously, decreasing the rate of migration increases the frequency of the locally advantageous allele.

As for speciation equilibria (11), it turns out that they do not exist. This suggests that the assumption about unequal migration rates is crucial for the possibility of speciation in the haploid version of the Maynard Smith model. (There is of course a possibility that some nonsymmetric equilibria exist and are stable but I was not able to find them.) I do not have an intuitive explanation of this result, however, somewhat similar effects have been observed in other models with sex differences. For example, it is well known that the structure of equilibria is much more complex in one-locus, two-allele models with differential selection in

the sexes than in the standard model with no sex differences (Bodmer, 1965).

4. Discussion

The results on the haploid version of the Maynard Smith model presented above put earlier conclusions, which were based exclusively on numerical simulations, on firmer theoretical grounds. Here I have shown that in the model studied sympatric speciation requires sufficiently strong nonrandom mating and sufficiently strong and spatially heterogeneous selection for local adaptation. Close linkage between the disruptive selection locus and the nonrandom mating locus greatly increases the range of parameter values resulting in sympatric speciation (see Fig. 2(a)). Restrictions on male migration promote (parapatric) speciation but the overall effect is not particularly great (see Fig. 4(a)). The explanation is that decreasing male migration rate decreases the opportunities for hybridization which, in turn, reduces the "incentive" to evolve stronger reproductive isolation. These conclusions are well appreciated in theoretical speciation research (Gavrilets, 2004). A less appreciated feature of the Maynard Smith model is the importance of the assumption that females' movement between the niches is restricted. Allowing for

females to migrate and increasing the rate of female migration quickly decreases the opportunity for speciation (see Fig. 3(a)). Also, speciation in (the haploid version of) the Maynard Smith model appears to require that the rates of male and female migration are different. If one allows for equal migration of both sexes, then speciation does not seem to be possible, even when migration and recombination rates are small and selection and assortative mating are very strong. Therefore, although speciation in the Maynard Smith model is sympatric, spatial subdivision and restrictions on migration are crucial for its success.

An important biological question concerns the levels of reproductive isolation and of gene flow between the emerging clusters of genotypes. Theoretical results (see Figs. 1–4) show that once the conditions for speciation are satisfied, genetic divergence between the clusters quickly approaches the maximum possible value with increasing the strength of selection s and the strength of nonrandom mating α. However, unless s and α are very close to one, hybridization will be ongoing. As a result, no genetic divergence in neutral markers will be maintained between the sympatric or parapatric clusters of genotypes.

Where comparable, the analytical results on the haploid version of the Maynard Smith model appear to be qualitatively similar to the earlier numerical results on the original diploid model (Caisse and Antonovics, 1978; Dickinson and Antonovics, 1973). Whether the differences in male and female migration rates in the diploid model are as important as in the haploid model is currently unknown.

One major implicit limitation of the Maynard Smith model is the absence of costs of being choosy. In the model, all organisms have equal mating success no matter how many (or few) preferred mates exist in their local populations. Costs of being choosy have been recently identified as a major factor opposing sympatric speciation (Gavrilets, 2004) whose importance is similar to that of recombination (Felsenstein, 1981). Another important limitation is the assumption that selection is soft, so that, the contribution of a local population to the overall offspring pool does not depend on the average fitness of the local population. Introducing costs of being choosy and using hard selection instead of soft selection are expected to make the maintenance of genetic variation and sympatric speciation much more difficult.

In the models considered here I assumed that migration takes place after selection. Migration occurring before reproduction/selection will impose a positive correlation between offspring fitness and place of origin, reinforcing the speciation process.

nonrandom mating increases. Intuitively one expects this range to shrink (because each individual locus will be subject to weaker selection whereas recombination will become more effective in destroying within-population differentiation). Finding conditions for speciation in multi-locus models represents a formidable mathematical challenge.

Acknowledgements

Appendix A

A.1. Equilibria in the basic case

Let $f = 0, m = \frac{1}{2}, r = \frac{1}{2}$. I will start by looking for symmetric equilibria satisfying conditions $u_{2,1} = u_{2,2}, u_{3,1} = -u_{3,2}$ and $u_{4,1} = -u_{4,2}$. Dropping the second subscript for simplicity of notation, the equations for a change per generation in u_2 and u_3 are

$$\Delta u_2 = \frac{s(u_4 - u_2 u_3)}{2 - s + s u_3},$$ (19a)

$$\Delta u_3 = -\frac{2s u_3^2 + (2 - s) u_3 - s}{2(2 - s + s u_3)}.$$ (19b)

From the first equation it follows that at equilibrium $u_4 = u_2 u_3$. Assuming this, the equation for u_4 becomes

$$\Delta u_4 = -\frac{u_2(2s u_3^2 + (2 - s) u_3 - s]}{2(2 - s + s u_3)}.$$ (19c)

Thus, at equilibrium u_2 is defined by Eq. (12), u_2 is arbitrary, and $u_4 = u_2 u_3$. This set of conditions defines a line of equilibria (10).

Next let us look for symmetric equilibria satisfying conditions $u_{2,1} = -u_{2,2}, u_{3,1} = -u_{3,2}$ and $u_{4,1} = u_{4,2}$. Dropping the second subscript for simplicity of notation, the equation for a change per generation in u_2 is

$$\Delta u_2 = \frac{2s u_2 u_3 - s(1 + \alpha) u_4 + (2 - s)(1 - \alpha) u_2}{2[(2 - s + s u_3)]}.$$ (20a)

Solving the right-hand side of the last equation for u_4 and substituting the resulting equation into an equation for a change per generation in u_3, one finds that

$$\Delta u_2 = \frac{s(1 + \alpha)^2 [2s u_3^2 + (2 - s) u_3 - s] + 4\alpha u_2^2 [s(s - 2) u_3 + 2s - 2 - s^2 - 2\alpha s + 2\alpha]}{2[(2 - s + s u_3)] s(1 + \alpha)^2}.$$ (20b)

A critical theoretical question is how the range of parameter values resulting in speciation will be affected if the number of loci underlying local adaptation and

The right-hand side of the last equation can be used to express u_2 is a function of u_3. Substituting the resulting expression for u_2 into an equation for a change per

generation in u_4, one finds that

$$\Delta u_4 = \frac{-2u_2(su_3 + 1 - \alpha - s)(su_3 + 1 - \alpha + \alpha s)}{s(1 + \alpha)(2 - s + su_3)}. \tag{20c}$$

Of the two solutions for u_3 the last equation only is feasible.

Solutions for other parameter values are identified using similar procedures.

A.2. The eigenvalues of the stability matrix when $r = \frac{1}{2}, f = 0, m = \frac{1}{2}$

Let us choose a point on the line of equilibria. Straightforward calculations show that the eigenvalues of the stability matrix at this point do not depend on its coordinates and are

$$\lambda_1 = \frac{4(1 - s)}{[2 - s(1 - u_3)]^2}, \tag{21a}$$

$$\lambda_2 = \frac{(1 - s)(1 + \alpha)}{[2 - s(1 - u_3)]^2}, \tag{21b}$$

$$\lambda_3 = \frac{(1 - s)(1 + \alpha)}{2 - s(1 - u_3)}, \tag{21c}$$

$$\lambda_4 = \frac{2(1 - s)}{[2 - s(1 - u_3)]^2}, \tag{21d}$$

which all are always between 0 and 1,

$$\lambda_5 = \frac{1 + \alpha}{2 - s(1 - u_3)}, \tag{21e}$$

which is always nonnegative, and

$$\lambda_6 = 1. \tag{21f}$$

The line of equilibria is locally stable if $\lambda_5 < 1$ which is the same as $\alpha < 1 - s(1 - u_3)$.

A.3. Speciation equilibria when $m = \frac{1}{2}$

Straightforward calculations show that V_3 is given by a positive solution of the quadratic

$$2s^2 V_3^2 + A_1 V_3 + A_0 = 0, \tag{22a}$$

where

$$A_1 = (2 - s)s[(1 - \alpha)(1 + 2r) + 2f(1 + \alpha)], \tag{22b}$$

$$A_0 = -s^2(1 + \alpha) + 2r(1 - \alpha)[s^2 + 2(1 - s)(1 - \alpha)] + 2f(1 + \alpha)[s^2 + 4r(1 - s)(1 - \alpha)]. \tag{22c}$$

Given V_3, the value of V_2 can be found from

$$V_2^2 = \frac{s(1 + \alpha)^2(1 - 2f)[2sV_3^2 + (2 - s)(1 + 2f)V_3 - s(1 - 2f)]}{4\alpha\{s(2 - s)V_3 + s^2 + 2(1 - s)[1 - \alpha + 2f(1 + \alpha)]\}}. \tag{22d}$$

Finally, given V_3 and V_2,

$$V_4 = \frac{V_2\{2sV_3 + (2 - s)[1 - \alpha + 2f(1 + \alpha)]\}}{s(1 + \alpha)(1 - 2f)}. \tag{22e}$$

The speciation equilibria exist whenever the solutions of the above equations are biologically meaningful (i.e. $0 \leqslant |V_2|, V_3, V_4 \leqslant 1$).

A.4. Speciation equilibria when $f = 0$

Straightforward calculations show that V_3 is given by a positive solution of the quadratic

$$s^2(1 - m)V_3^2 + B_1 V_3 + B_0 = 0 \tag{23a}$$

satisfying $0 \leqslant V_3 \leqslant 1$. Here,

$$B_1 = s(2 - s)m(1 + r - m)(1 - \alpha), \tag{23b}$$

$$B_0 = rm(1 - \alpha)[s^2 + 2(1 - \alpha)(1 - s)] - s^2(1 - m)[1 - m(1 - \alpha)]. \tag{23c}$$

Given V_3, the value of V_2 can be found from

$$V_2^2 = s[1 - m(1 - \alpha)]^2 \frac{sV_3^2 + m(2 - s)V_3 - s(1 - m)}{C_2 u_3^2 + C_1 u_3 - C_0}, \tag{23d}$$

where

$$C_2 = s^2(1 - 2m)^2,$$
$$C_1 = s(2 - s)m[1 - 4m(1 - m)(1 - \alpha)],$$

$$C_0 = s^2(1 - m)\{1 - 4m[1 - m(1 - \alpha)]\} - 4m^2\alpha(1 - \alpha)(1 - s).$$

Finally, given V_3 and V_2,

$$u_4 = \frac{V_2[sV_3 + m(2 - s)(1 - \alpha)]}{s[1 - m(1 - \alpha)]}. \tag{23e}$$

The speciation equilibria exist whenever the solutions of the above equations are biologically meaningful (i.e. $0 \leqslant |V_2|, V_3, V_4 \leqslant 1$).

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