

A quantitative genetic competition model for sympatric speciation

M. Doebeli

*Zoology Institute, University of Basel, Rheinsprung 9, CH-4051 Basel, Switzerland
e-mail: doebeli@ubaclu.unibas.ch*

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Abstract

I use multilocus genetics to describe assortative mating in a competition model. The intensity of competition between individuals is influenced by a quantitative character whose value is determined additively by alleles from many loci. With assortative mating based on this character, frequency- and density-dependent competition can subdivide a population with an initially unimodal character distribution. The character distribution becomes bimodal, and the subpopulations corresponding to the two modes are reproductively separated because mating is assortative. This happens if the resource distribution is unimodal, i.e. even if selection due to phenotypic carrying capacities is not disruptive. The results suggest that sympatric speciation due to frequency-dependent selection can occur in quite general ecological scenarios if mating is assortative. I also discuss the evolution of assortative mating. Since it induces bimodal phenotype distributions, assortative mating leads to a better match of the resources if their distribution is also bimodal. Moreover, in a population with a bimodal phenotype distribution, the average strength of frequency-dependent competition is lower than in a unimodal population. Therefore, assortative mating permits higher equilibrium densities than random mating even if the resource distribution is unimodal. Thus, even though it may lead to a less efficient resource use, assortative mating is favoured over random mating because it reduces frequency-dependent effects of competition.

Introduction

Understanding speciation remains one of the fundamental problems in biology. Perhaps due to a lack of convincing alternatives, the predominant view is that new species arise most often in allopatry through divergent evolution in geographically

isolated populations of the same ancestral species (Mayr, 1963; Coyne, 1992). However, this view has repeatedly been challenged by both empiricists and theoreticians. Since Maynard Smith (1966), many models have been used to describe the conditions under which sympatric speciation is theoretically possible (Dickinson and Antonovics 1973; Rosenzweig, 1978; Pimm, 1979; Gibbons, 1979; Udovic, 1980; Felsenstein, 1981; Kondrashov, 1983a, b, 1986; Rice, 1984; Seger, 1985; Diehl and Bush, 1989; Wilson, 1989; Fialkowski, 1992; Johnson et al., 1996). The usual approach is to assume that there is disruptive selection in a polymorphic population with assortative mating. Various mechanisms for this have been proposed. For example, disruptive selection and polymorphism can be caused by environmental heterogeneity (Maynard Smith, 1966; Felsenstein, 1981; Rice, 1984; Diehl and Bush, 1989) or frequency-dependent selection (Seger, 1985; Kondrashov, 1986). Furthermore, assortative mating may or may not be based on phenotypes under selection (Udovic, 1980; Felsenstein, 1981; Kondrashov, 1986; Johnson et al., 1966). The goal of this paper is to fill some gaps in the existing theory and to show that, on theoretical grounds, sympatric speciation could be quite common. Using a competition model in which the interactions are determined by a quantitative character, it will be shown that quite general ecological scenarios can set the stage for this mode of speciation.

The basic idea for these scenarios is the concept of ecological character displacement (Slatkin, 1980). In two coevolving species, selection can produce a difference in the distributions of a character that affects the strength of competition between individuals. Suppose, for example, that a character such as size is normally distributed with similar means in both species. If resource use is size-dependent, competition for resources will be more intense between individuals of similar size. Frequency-dependence then favours a displacement of the characters, i.e., a divergence of the means in the two species. The displacement is due to the selective forces acting on the distributions of characters that determine ecological dynamics. Obviously, these forces also act on the character distribution within a single population. Thus, a population, with an initially very peaked unimodal distribution could gradually evolve towards a wider and flatter distribution, due to frequency-dependent competition. However, the equilibrium distribution would in general remain unimodal if mating is random. This paper explores the consequences of relaxing the assumption of random mating. It studies the joint effect of assortative mating and frequency-dependent competition on the distribution of a quantitative character that defines the competitive interactions.

Ecological models for quantitative characters often describe the genetics of a character implicitly by assuming that it is normally distributed (e.g., Slatkin, 1980; Taper and Case, 1985). It is difficult to introduce assortative mating in such models, because assortative mating would cause deviations from normality. Therefore, I use a model that describes the genetics of the character explicitly, with many haploid loci and alleles with small effects. In the model, selection acts on phenotypes, and the genetics assumed determine the distribution of offspring phenotypes from the phenotypes of their parents. Since the genetics are explicit, it is straightforward to incorporate assortative mating based on the quantitative character by assuming that

similarity of phenotypes increases their mating probability. The main consequence is that the disruptive mechanism of frequency-dependence is more effective when mating is assortative. Thus, an initially unimodal character distribution can become a bimodal distribution, similar to ecological character displacement between species. Since mating is assortative, phenotypes corresponding to the different humps have a low probability of mating with each other. Although belonging to the same population, the two subunits given by the two modes of the character distribution are in effect reproductively isolated. Thus the splitting of the character distribution can be viewed as the initial phase of speciation.

The results suggest that mate choice based on a quantitative character determining ecological interactions can induce sympatric speciation. In some previous models (Maynard Smith, 1966; Seger, 1985; Kondrashov, 1986; Fialkowski, 1992; Johnson et al., 1966) sympatric speciation was studied by looking at the evolution of mate choice. Speciation was said to have occurred when a gene for particular types of assortative mating was fixed. The present approach is slightly different. A certain degree of assortative mating is assumed, and speciation occurs because the distribution of the character that determines mating choice splits into two modes with only little gene flow between them.

But the evolution of assortative mating can also be studied. If the phenotypic carrying capacities are bimodal, a bimodal character distribution allows a better exploitation of the resources. Therefore, assortative mating evolves to a degree that induces sympatric speciation. However, the match with the carrying capacity curve is not the sole determinant of the course of evolution. With frequency/dependence, different phenotype distributions lead to different average effects of competition. Because the average strength of competition is lower in bimodal than in unimodal populations, assortative mating is favoured over random mating even if the resource distribution is unimodal. Thus, even in this case degrees of assortative mating inducing sympatric speciation can evolve, because assortative mating dampens the effect of frequency-dependent competition.

The model

We start with a 1-dimensional dynamical model for a population with discrete generations

$$N_{t+1} = N_t \cdot f(N_t), \quad (1)$$

where N_t is the density of the population at time t , and where competition is implicitly modeled by the density-dependent fitness function

$$f(N) = \frac{\lambda}{1 + (aN)^b} \quad (2)$$

(Bellows, 1981). The parameter λ is the intrinsic growth rate of the population, while the parameter b reflects the competitive process that leads to density-dependence (Hassell, 1975; Schoener, 1976). The parameter a influences the carrying capacity.

The model can be extended to a phenotypically variable population in which a quantitative character z determines the competitive interactions. Without loss of generality, the character z is scaled to take on values in the interval $[0, c]$, where c is a positive integer. This is convenient for introducing genetics, as c will be the number of loci involved. At the beginning of generation t , $p_t(z)$ is the frequency of the haploid phenotype with character value z , and selection acts on the distribution $p_t(z)$ before mating takes place. Selection is determined by phenotypic fitness functions $f_z(N_t, p_t)$, $z \in [0, c]$, that depend on the total population density N_t and on the phenotype distribution p_t . To determine them we use the following Gaussian function $\alpha(z, z')$ to measure the strength of competition between individuals of phenotypes z and z' :

$$\alpha(z, z') = \exp\left(\frac{-(z - z')^2}{2\sigma_z^2}\right) \quad (3)$$

(Slatkin, 1980). It implies that competition is more intense between individuals with similar phenotypes. The parameter σ_z^2 determines the niche width: the niche is wider, and competition is less intense, for smaller σ_z^2 . Using $\alpha(z, z')$, we can calculate the "effective density" $N_{eff,t}(z)$ that an individual of phenotype z experiences in a population with total density N_t and phenotype distribution p_t :

$$N_{eff,t}(z) = N_t \int_0^c p_t(z') \alpha(z, z') dz' \quad (4)$$

$N_{eff,t}(z)$ is a weighted sum over the densities of all phenotypes, the weights reflecting the intensity of competition. The phenotypic fitness functions f_z have the same general form as the fitness function (2), but with the total size N_t replaced by $N_{eff,t}(z)$:

$$f_z(N_t, p_t) = \frac{\lambda}{1 + [a(z)N_{eff,t}(z)]^b} \quad (5)$$

It is implicitly assumed that all phenotypes have the same growth rate λ and the same competition parameter b . However, the parameter $a(z)$ depends on the phenotype, allowing different phenotypes to have different carrying capacities.

Eq. (5) means that the fitness of a phenotype is both density- and frequency-dependent. The density dependence comes from using the fitness function (2) as the basic competition model. Frequency dependence occurs because the effective density $N_{eff,t}(z)$ depends on the frequency distribution $p_t(z)$ via the function $\alpha(z, z')$, eq. (3), which measures the competitive impact that phenotypes have on each other.

Multiplying the frequency of each phenotype by its fitness, after normalizing with the mean fitness, yields the phenotype distribution after selection, denoted by $q_t(z)$:

$$q_t(z) = \frac{p_t(z) \cdot f_z(N_t, p_t)}{\bar{W}_t} \quad (6)$$

where

$$\bar{W}_t = \int_0^c p_t(z) f_z(N_t, p_t) dz \quad (7)$$

is the mean fitness at time t . Mating occurs after selection, i.e. the genetics now mould the distribution $q_t(z)$ to give the phenotype distribution at the start of the next generation, $p_{t+1}(z)$. To determine the phenotypic frequencies after mating from $q_t(z)$, I use a simple genetic model in which the character z is determined additively by c haploid loci (see Doebeli (1996a, b) for more details). Each locus has two alleles, 0 and 1, and the phenotype of an individual is given by the number of 1-alleles in its genome. Thus, phenotypes range from 0 to c . For mating, I assume that the alleles of an offspring at any locus have probability 1/2 to come from either parent. Given two parents with phenotypes i and j , let l denote their genetic overlap, i.e. the number of loci at which they both have the 1-allele. It is then easy to see that the offspring phenotypes lie in the interval $[l, i + j - l]$, and that the phenotypic distribution in this interval is binomial:

$$p_{ij}^l(z) = \begin{cases} \binom{i+j-l}{z-l} \cdot \left(\frac{1}{2}\right)^{i+j-2l} & \text{for } z \in [l, i+j-l] \\ 0 & \text{otherwise,} \end{cases} \tag{8}$$

where $p_{ij}^l(z)$ denotes the offspring phenotype distribution from parents with phenotypes i and j and genetic overlap l . The total offspring distribution p_{ij} from parents with phenotypes i and j is a weighted sum of the distributions determined by different overlaps:

$$p_{ij}(z) = \sum_{\substack{\text{all possible} \\ \text{overlaps } l}} \text{Pr}(\text{overlap} = l) \cdot p_{ij}^l(z). \tag{9}$$

For phenotypes $i \leq j$, the possible overlaps range from l_{\min} to j , where l_{\min} is equal to the larger of the numbers $i + j - c$ and 0. For $l \in [l_{\min}, j]$, the probability for overlap l is

$$\text{Pr}(\text{overlap} = l) = \frac{\binom{i}{l} \cdot \binom{c-i}{j-l}}{\binom{c}{j}}. \tag{10}$$

The total distribution $p_{t+1}(z)$ is now given as the sum of the offspring distributions p_{ij} , weighted by the frequency of matings between phenotypes i and j . If mating is random, this frequency is $2q_t(i)q_t(j)$ for $i \neq j$, and $q_t^2(i)$ otherwise, where $q_t(z)$ is the phenotype distribution after selection, eq. (6). Thus, for random mating we have

$$p_{t+1}(z) = \sum_i \sum_j [q_t(i)q_t(j)] \cdot p_{ij}(z). \tag{11}$$

To introduce assortative mating, we use the mating function

$$m(i, j) = \exp\left(\frac{-(i-j)^2}{2\rho^2}\right), \tag{12}$$

and replace the factors $q_t(i)q_t(j)$ in eq. (11) by factors $m(i, j)q_t(i)q_t(j)$. Thus, with

assortative mating, eq. (11) is replaced by

$$p_{t+1}(z) = \sum_i d_i \sum_j [m(i,j)q_t(i)q_t(j)] \cdot p_{ij}(z), \quad (13)$$

where the d_i are normalizing constants needed to obtain a frequency distribution for the offspring of the single phenotypes. Eq. (12) implies that matings between similar phenotypes occur relatively more often. The parameter ρ in eq. (12) measures the degree of assortative mating. A small ρ implies a high degree of assortativeness, while $\rho = \infty$ corresponds to random mating.

Iterating eqs. (6) and (13), together with the recursion

$$N_{t+1} = N_t \cdot \bar{W}_t, \quad (14)$$

where \bar{W}_t is the mean fitness, eq. (7), gives the dynamics of the quantitative genetic model. In the 1-dimensional model given by eqs. (1) and (2), the parameters λ and b determine the dynamics (Bellows, 1981), which range from stable equilibria to chaos (May, 1976). In the phenotypically variable model, these dynamics are also influenced by frequency-dependent competition and by the distribution of resources, which is given by the function $a(z)$ in the phenotypic fitness functions (5). The carrying capacity in the basic model (2), i.e., the density N^* for which $f(N^*) = 1$, is given by $N^* = (\lambda - 1)^{1/b}/a$. Therefore, the phenotypic carrying capacities are given by

$$N^*(z) = \frac{(\lambda - 1)^{1/b}}{a(z)}, \quad (15)$$

so that a low $a(z)$ implies a high carrying capacity for phenotype z .

Results

We first assume that the function $a(z)$ reflects a symmetric bimodal resource distribution $N^*(z)$, eq. (15). For this we superimpose the two functions

$$a_1(z) = a_0 \cdot \exp\left(\frac{\left(z - \frac{c}{4}\right)^2}{2\mu^2}\right)$$

and

$$a_2(z) = a_0 \cdot \exp\left(\frac{\left(z - \frac{3c}{4}\right)^2}{2\mu^2}\right),$$

i.e. we set

$$a(z) = \min\{a_1(z), a_2(z)\}. \quad (16)$$

This function has two minima at $c/4$ and $3c/4$, an intermediate local maximum at $c/2$, and two local maxima at the edges of the character interval, hence the opposite is true for the phenotypic carrying capacity given by eq. (15) (Fig. 1a). The

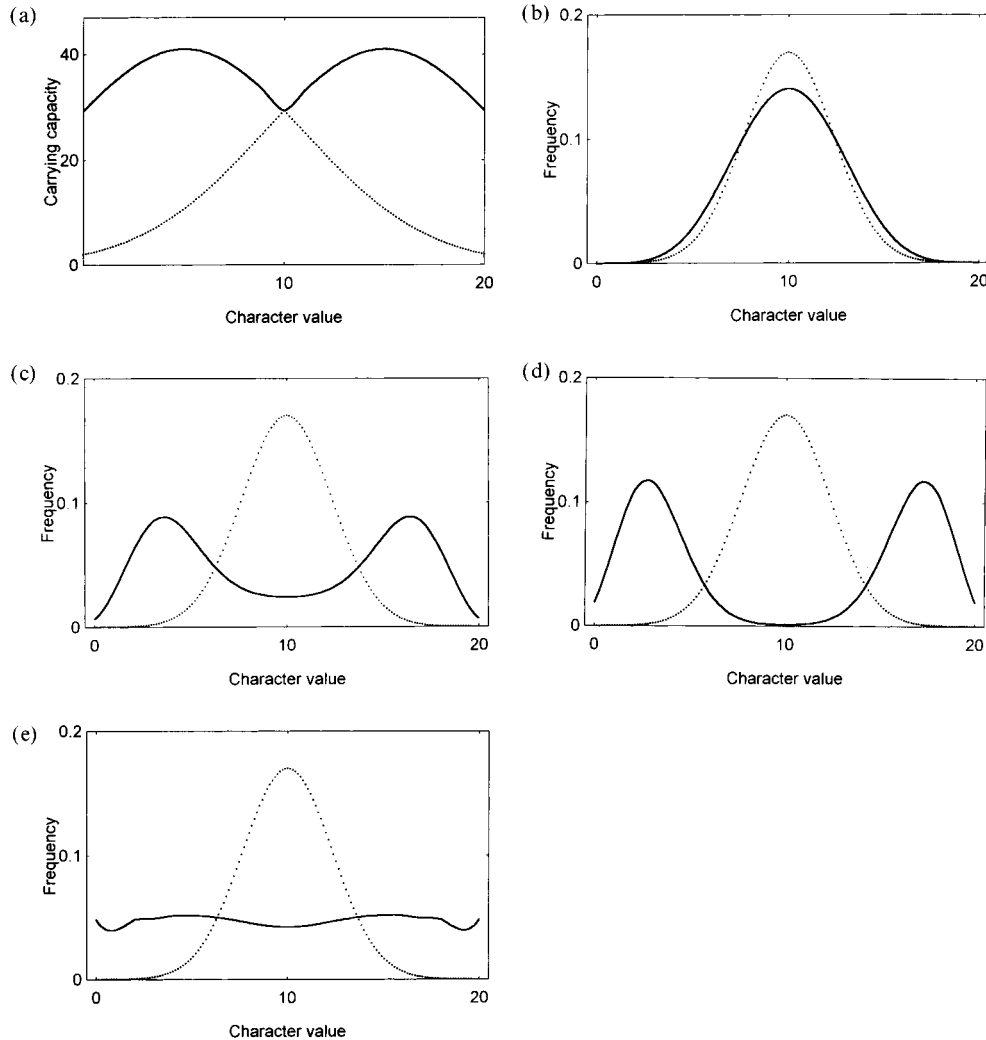


Fig. 1. Assortative mating with a bimodal resource distribution. 1a shows the phenotypic carrying capacity, eq. (15), in which $a(z)$ is given by eq. (16) as the minimum of $a_1(z)$ and $a_2(z)$ (see text), which correspond to the dotted curves shown. In 1b–1d, the dotted distribution is the initial unimodal distribution with which the runs were started. The continuous lines show the equilibrium distributions reached after ca. 100 generations. Despite bimodal resources, no split in the character distribution occurs with random mating (1b). With assortative mating, the character distribution splits into two modes (1c, 1d). The split is greater for higher degrees of assortative mating (1d), i.e. for smaller values of ρ in the mating function (12). Fig. 1e shows that without frequency-dependent competition, i.e. with a very large niche width, even assortative mating for which only matings between the same phenotypes are possible (very low ρ) does not induce a bimodal split of the character distribution. The parameter values were $\sigma_z = 7.07$ in eq. (3), $\lambda = 10$ and $b = 14/9$ in eq. (5), and $a_0 = 0.1$ and $\mu = 6.1$ in eq. (16) for 1a–1d; $\sigma_z = 0.3$ for 1e; $\rho = \infty$ (i.e. random mating, model (11)) for 1b, $\rho = 5$ for 1c, $\rho = 3.1$ for 1d, and $\rho = 0.1$ for 1e. The number of loci was $c = 20$ in all simulations.

parameter a_0 determines the maximal carrying capacity, while μ controls the width of the distribution. The bimodality of the resource adds to the disruptive effect of frequency-dependent competition. In this situation, assortative mating can split an initially unimodal character distribution into two distinct modes (Fig. 1).

If mating is random, the character distribution remains unimodal (Fig. 1b), whereas increasing degrees of assortative mating lead to bimodality with increasing distance between the two humps of the distribution (Figs. 1c, d). Thus, assortative mating allows the tendency to avoid competition to manifest itself in a bimodal split. The higher the degree of assortativeness, the easier it is for frequency dependence to break up the character distribution. Since mating is assortative, the probability of mating between phenotypes corresponding to the means of the two modes in the distributions shown in Figs. 1c, d is very low. Thus the modes are reproductively isolated, although isolation is not complete, and there is still some gene flow between the two subpopulations. With increasing degree of assortative mating the isolation increases as well. Extensive simulations showed that results similar to those in Fig. 1 occur for a wide range of parameters. As long as the degree of assortative mating is high enough, a bimodal equilibrium distribution is typically reached after ca. 100 generation, starting from a unimodal distribution in the center of the character interval. For example, with the niche width as in Fig. 1b–d, speciation occurs for all ρ with $2\rho^2 \leq 65$. On the other hand, with degrees of assortative mating as in Fig. 1c or 1d, speciation occurs for all niche widths, with the exception of very large niches ($\sigma_x^2 \leq 2.5$), that is, if there is no frequency dependence in the competition for resources (Fig. 1e). This highlights the importance of frequency-dependent selection, which is what actually causes the bimodal split, while assortative mating is a precondition for bimodality. In particular, disruptive selection due to bimodal phenotypic carrying capacities alone is not enough to induce a split character distribution even if the degree of assortative mating is very high (Fig. 1e).

Qualitatively, these results do not depend on the implicit assumption of the model that there is no environmental variance in the phenotypic characters, except if this variance is very large. With environmental variance, the effect of assortative mating is diluted. However, a few numerical simulation showed that the dilution is typically not strong enough to prevent the bimodal split of the character distribution, although the modes become less pronounced. For example, with moderate environmental variance, c.g., if the character of a genotype is drawn from a Gaussian distribution with mean equal to the number of I-alleles and variance equal to 20% of the character interval, the equilibrium distribution corresponding to the parameters of Fig. 1d would look more like the one shown in Fig. 1c. Only a very large environmental variance would cancel the effect of assortative mating and lead to an equilibrium distribution that would still be unimodal. Similar observations concerning the effect of environmental variance have been made by Seger (1985).

We have seen that the main driving force in the system is frequency dependence. In fact, the splitting of the character distribution due to frequency dependence when mating is assortative can also be observed when the resource distribution is

unimodal, i.e., when selection due to differential phenotypic carrying capacities is stabilizing, not disruptive. Two examples are shown in Fig. 2, for which the curve $a(z)$ has the form

$$a(z) = a_0 \cdot \exp\left(-\frac{\left(z - \frac{c}{2}\right)^2}{2\mu^2}\right) \tag{17}$$

so that the phenotypic carrying capacity has a maximum at $c/2$ and is minimal at the edges of the character interval. Again, the tendency to split is greater with stronger assortativeness. If in addition the parameter σ_x^2 in the competition function $\alpha(z, z')$, eq. (3), is smaller, that is, if there is more niche space and less intense competition, a third mode appears in the character distribution (Fig. 2b). This new hump is in the center of the character interval, and there is again only little gene flow to the marginal modes because mating is assortative, suggesting that a simultaneous split into more than two descendant lines is possible during sympatric speciation.

Qualitatively similar results to those shown in Fig. 2 can be obtained for a wide range of parameters. As with bimodal resources, a certain degree of frequency dependence is necessary, and bimodal character distributions do not result if the niche width is very large. However, with unimodal resources speciation does not occur if the niche width is too small. Thus, with the degree of assortative mating fixed as in Fig. 2a, bimodality is not seen for σ_x with $2\sigma_x^2 \geq 140$. In contrast to the case of bimodal resources, where the split of the character distribution occurs for all values of σ_x (except for very small ones which eliminate the frequency dependence),

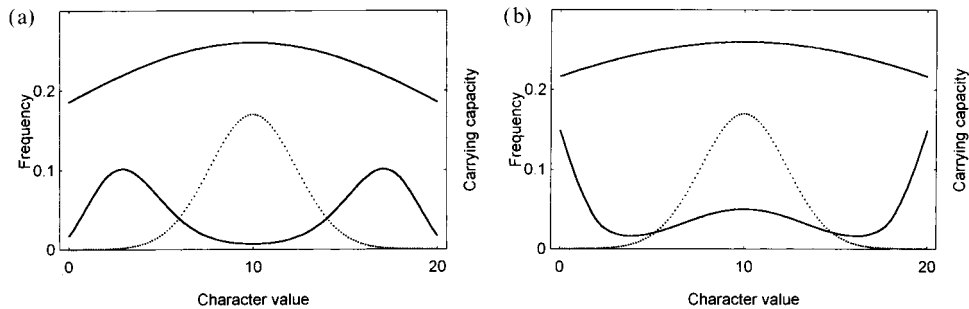


Fig. 2. Assortative mating with a unimodal resource distribution. In equation (15) for the phenotypic carrying capacity the function $a(z)$ is given by eq. (17). The phenotypic carrying capacities are shown (displaced vertically) above the character distributions. The dotted curves show the initial character distributions at the start of the runs, while the (lower) continuous curves show the equilibrium distributions reached after ca. 100 generations. Even with unimodal resources, assortative mating leads to a bimodal equilibrium distribution (2a). If competition is less intense (σ_x^2) and the degree of assortative mating is high (2b), the two principal modes of the distribution move towards the edges of the character interval and a third mode appears in the center. Parameter values were $\sigma_x = 7.07$ in eq. (3), $\rho = 3.25$ in eq. (12) and $\mu = 12.2$ in eq. (17) for 2a; $\sigma_x = 4$ in eq. (3), $\rho = 1.87$ in eq. (12) and $\mu = 16.6$ in eq. (17) for 2b. The other parameters were the same as in Fig. 1.

the stabilizing selection induced by unimodal resources prevents the split if there is not enough niche space. Thus, unimodal resources limit the range of niche widths for which speciation is possible. Nevertheless, the results show that, in contrast to what is commonly believed, bimodal resource distributions are not a *sine qua non* for sympatric speciation when competition is strong and frequency-dependent.

In all the examples shown in Figs. 1 and 2 the system exhibited stable equilibrium dynamics. By changing the parameters λ and b , more complicated dynamics can be obtained, although the system may behave very differently from the basic one-dimensional model given by (2) (Doebeli, 1996a). With more complex dynamics similar phenomena to those shown in Figs. 1 and 2 can be seen. Initially unimodal character distributions again split into different modes with little mating between them, although now the resulting distributions fluctuate over time. However, the reproductive separation typically remains intact, because the fluctuations do not lead to a significant overlap of different modes. A corresponding result in the related context of ecological character displacement was reported by Doebeli (1996b).

Evolution of assortative mating

Up to now I assumed a fixed degree of assortative mating and showed that it can initiate sympatric speciation. However, the evolution of assortative mating, i.e. the evolution of the parameter ρ in eq. (12), can also be studied in the present model. For this one assumes that, given a resident population with an equilibrium distribution corresponding to a certain ρ , mutants with a different parameter value ρ_m occur in the population prior to mating. To determine whether these mutants can increase in number, one has to first determine their offspring distribution and then the mean fitness of these offspring. If this mean fitness is larger than 1, the mutants can invade. This is the usual criterion for invasion into a population with a stable equilibrium: since the equilibrium is not affected by the initially rare mutants, the conditions for the mutants do not change, and the first generation fitness is a valid approximation for determining the success of invasion (Metz et al., 1992).

The mutant offspring distribution will depend on ρ_m and on the initial distribution of the mutants in which they occur. I assumed that the mutants occur in the same phenotypic distribution as the resident. Their offspring distribution is then obtained by the genetic model described previously, using ρ_m in eq. (12). Again because mutants are initially rare, the mean fitness of the mutant offspring is determined solely by the phenotype distribution of the resident and by its total density. If the mean fitness is larger than 1, the new value ρ_m is advantageous, and I made the simplifying assumption that in this case the mutants take over the population, so that the whole population now has a mating function determined by ρ_m .

For the results shown in Fig. 3, I started with a very high initial value of ρ corresponding to random mating, and then chose mutant value ρ_m from a Gaussian distribution whose mean was the resident value. In case of invasion I let the

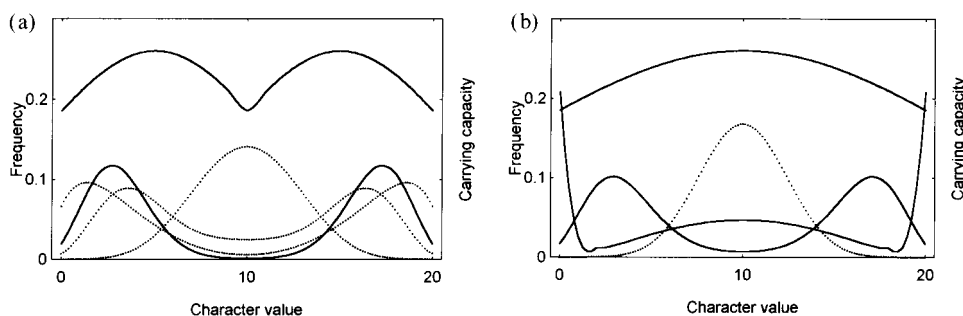


Fig. 3. Evolution of assortative mating. In 3a, the equilibrium distributions (lower curves) from Figs. 1b–1d are shown with reference to the carrying capacity curve of Fig. 1a (upper curve, displaced vertically). In addition, an equilibrium distribution for a very high degree of assortative mating is shown ($\rho = 1.2$ in eq. (12), other parameters as in Fig. 1). This distribution has the largest separation between the two modes, i.e. the modes are closest to the edge of the character interval. Of the four distributions, the one shown with a continuous curve (same as Fig. 1d) permits the highest equilibrium population size. It corresponds to the value of ρ in the mating function (12) that evolves in the selection process described in the text. Fig. 3b shows the situation for a unimodal carrying capacity curve (upper continuous curve, same as in Fig. 2a). The unimodal dotted character distribution results from random mating. It matches the resource better than the continuous distributions. However, less intense competition due to the bimodality compensates for the worse fit, and the continuous curves result from the selection process described in the text, applied to different niche widths σ_x^2 . The bimodal curve shown is the same as in Fig. 2a and results for the parameters used there when the evolutionary walk starts out from random mating. In this case, the walk ends at $\rho = 3.25$. If the walk is started at a high degree of assortativeness (e.g. at $\rho = 2$), then it ends up at the other evolutionarily stable value of $\rho = 1.18$ (distribution not shown). With a larger niche width (smaller σ_x , $\sigma_x = 5$, other parameters as before), there is only one evolutionary stable degree of assortativeness. Starting out from random mating, the evolutionary process ends at $\rho = 0.97$, resulting in the distribution with the two very peaked marginal modes.

successful mutants reach a stable equilibrium distribution before simulating the next mutation. At the beginning of this process the variance of the Gaussian distribution from which the mutant values ρ_m were drawn was relatively high, say 5% of the resident value. Initially, only values of ρ_m that were lower than the resident value were advantageous, so that evolution was away from random mating. However, eventually the successful ρ -values started to wander up and down, confined to a small interval of intermediate values of ρ , which indicated that the process had reached the vicinity of an evolutionary stable value. After reaching this parameter region, I decreased the variance of the Gaussian distribution from which the mutants were drawn in order to increase the accuracy of the selective process. Thus, the “true” values that are favoured by selection lie within 1% of the values given in Fig. 3. The simulations showed that for a wide range of niche width parameters and resource distributions, the evolutionary walk leads away from random mating towards locally evolutionarily stable degrees of assortativeness allowing frequency-dependent competition to induce sympatric speciation.

The assumption that advantageous mutants sequentially take over the whole population corresponds to a “playing the field” selection process (Maynard Smith,

1982) in which a stable polymorphism of strains with different values of ρ is not possible. A justification comes from the observation that in stable populations a polymorphism of phenotypes with different carrying capacities is usually not possible, and that different values of ρ lead to different equilibrium sizes, i.e., carrying capacities, of the population. Indeed, considering equilibrium densities provides an intuitive explanation for the evolution of assortative mating: populations with random mating have lower carrying capacities than populations with assortative mating. In the case of a bimodal resource distribution (Fig. 3a), this is intuitively clear, since a bimodal character distribution leads to a better match and consequently a better exploitation of the resources. If assortative mating is too weak, frequency dependence is not able to separate the phenotype distributions sufficiently far apart for effective use of resources, and if it is too strong, then the modes get pushed to the edge of the character interval where carrying capacities are low.

However, the resource distribution is not the only determinant of the equilibrium population size. At the equilibrium, the mean fitness in the population is 1, i.e., the effective densities $N_{eff,t}(z)$ experienced by phenotype z are such that the corresponding fitness values f_z have mean 1. The effective densities are determined by the total population density and by the phenotype distribution as described by eq. (4). Thus, given a fixed total density, different phenotype distributions lead to different effective densities $N_{eff,t}(z)$, and hence to different phenotypic fitness values f_z : if a phenotype distribution implies a low average strength of competition in the population, the effective densities are low, so that the average phenotypic fitness is high. In a population with such a phenotype distribution, the average fitness will reach 1 at a high total density, so that such a population will have a high equilibrium density, i.e., a high total carrying capacity. Conversely, a population with a phenotype distribution implying a high average strength of competition will have a low equilibrium density.

It is intuitively clear that the average competitive impact of the individuals on each other is greater in a population with a unimodal phenotype distribution than in a bimodal population, in which the individuals of one mode only weakly compete with those of the other mode. Thus, bimodality per se is selectively advantageous when competition is frequency-dependent. However, with a unimodal resource distribution, a bimodal phenotype distribution implies a disadvantage, because phenotypes with the highest phenotypic carrying capacity are rare, which tends to decrease the equilibrium density. In this situation, two opposite selective forces impinge on assortative mating. Since it leads to bimodal phenotype distributions, it is favoured because it relaxes the average strength of competition in the population, but it is disadvantageous because it induces a bad fit of the resource distribution. In the evolutionary “playing the field process” described above, it turns out that avoiding competition is more important: if the unimodality of the resource is not too pronounced, assortative mating is favoured over random mating (Fig. 3b). In other words, with assortative mating, the advantage of less intense competition due to the bimodality of the character distribution more than compensates the disadvantage of a fit of the resource that is worse than that of the unimodal character

distribution resulting from random mating, and the evolutionary walk again leads away from random mating to an intermediate degree of assortativeness. Interestingly, this intermediate degree is not globally stable if the niche width is not too large. If the evolutionary walk starts out from a degree of assortativeness that is substantially higher (say $2\rho^2 \leq 16$ in Fig. 3b) than the locally stable intermediate value, then the walk does not lead back to the intermediate value and instead ends up at a very high degree of assortativeness ($2\rho^2 = 2.8$). Thus there can be more than one evolutionarily locally stable values of ρ . However, with very large niche widths, the intermediate value loses its stability. There is then only one globally stable value of ρ , for which the equilibrium distribution is similar to the one shown in Fig. 2b: there is a wide and flat central mode together with two very peaked modes at the edges of the character interval (Fig. 3b). In effect, these two modes constitute different species.

Discussion

In this article I argue that, on theoretical grounds, sympatric speciation could be quite a common phenomenon. I used a competition model based on a quantitative character to show that assortative mating with respect to this character can lead to a bimodal split of the character distribution in the population. Because phenotypes with very different characters have a very low probability of mating, the two modes of the distribution are reproductively separated. The rupture in the character distribution is more pronounced if resource availability, i.e., the carrying capacity curve of the phenotypes, is described by a bimodal function, but even with a unimodal resource distribution, frequency- and density-dependent competition can induce sympatric speciation. This conforms with and extends the theory of competitive speciation (Rosenzweig, 1978, 1995).

Of the existing models for sympatric speciation, the present model is closest to those of Seger (1985) and Kondrashov (1986). It combines the ecological approach of the former with the multilocus genetics of the latter, although both competition for resources and genetics are handled differently. The explicit quantitative genetics I use make it particularly easy to model assortative mating, and if mating is assortative, then sympatric speciation occurs for a wide range of parameters.

With a bimodal resource distribution, assortative mating leads to a better resource use than random mating, and hence to a higher equilibrium density. Therefore, assortativeness is favoured by selection. However, even with unimodal resources assortative mating implies higher equilibrium densities and is therefore advantageous, because it dampens the effect of frequency-dependent competition. This more than outweighs the worse fit of the resource. Thus, the traditional view that sympatric speciation only occurs if it leads to a better exploitation of resources (Ridley, 1993) must be modified. With frequency-dependent competition, sympatric speciation can occur even if it implies underused resources, because it reduces the competitive impact.

One assumption of the model is that assortative mating is based on the character that determines the ecological interactions. It is questionable how general this is

(Maynard Smith, 1996), and alternative models have been proposed in which the character determining mate choice is different from the one on which selection acts (Udovic, 1980; Seger, 1985). However, especially when the quantitative character that regulates competition is morphological and polygenic, e.g., beak size in birds, it is likely that there is genetic variation for mate choice based on this character (Kondrashov and Mina, 1986). Recently, Schluter and Nagel (1995) have examined the role of natural selection for speciation by looking at four groups of closely related species. In almost all of their examples they noted that reproductive isolation between sister species mainly depends on assortative mating with respect to body size or shape. Indeed they argue that this may be a general phenomenon explaining the tendency for closely related species to diverge in body size. All of the examples of Schluter and Nagel (1995) involve characters that may be important for competitive interactions. For instance, beak size and shape in Darwin's Finches influence both mate choice and the dietary intake of the birds. Thus, either because resources are underutilized or competition too intense with random mating, evolution of assortative mating may have driven the speciation in this group of birds. The theory presented here provides an explanation of how this might have happened: it fits the bill.

An alternative interpretation of the mating function (12) and how it determines the phenotypic distribution (13) is that mating between distant phenotypes do not occur less often, but are instead less successful, i.e., that isolation is post- instead of prezygotic. The parameter ρ in eq. (12) would then be a measure of postzygotic fertility selection: the smaller ρ , the less fertile are matings between distant phenotypes, and the higher is the proportion of offspring from matings between similar phenotypes. With this interpretation, the results indicate that density- and frequency-dependent selection can induce sympatric speciation if the fertility of matings between genetically very different individuals is lower than that of matings between genetically similar organisms.

The model presented here actually describes only one critical phase of speciation. Given the ecological boundary conditions and an initially unimodal character distribution, it describes the relatively fast (ca. 100 generations) transition to reproductively distant subpopulations, which results in a dumb-bell structure of the population (Kondrashov and Mina, 1986). However, the subpopulations are not completely isolated, and there is still some gene flow. Thus, two questions arise: which processes generate the ecological conditions that favour bimodality, and which processes complete reproductive isolation? In general, the first question can only be answered by studying community ecology and coevolution of species. However, the fact that even unimodal resource distributions can lead to speciation suggests that sympatric speciation can occur in quite general ecological scenarios. In combination with the analysis of genetic phenomena such as linkage disequilibrium (Udovic, 1980; Felsenstein, 1981), the second question should probably also be answered in the context of community ecology. After the split into bimodality, the two subpopulations occupy different niches, and divergent evolution due to different selective forces, e.g. adaption to different predators, or further evolution of mate choice based on niche characteristics, may lead to complete reproductive isolation and to the formation of new species.

Whatever the answer to these questions are, there is now much evidence for sympatric speciation in nature. Examples include fishes (Schliewen et al., 1994; Schluter, 1994; Schluter and McPhail, 1992; Meyer et al., 1990), frogs (Barendse, 1984), many insects (Feder, 1995; Hirai et al., 1994; Tauber and Tauber, 1989; Feder et al., 1988; Gibbons, 1979; Tauber and Tauber, 1977; Knerer and Atwood, 1973; Bush, 1969), and plants (Manning and Linder, 1992). Lazarus et al. (1995) report a case of sympatric speciation inferred from marine fossils. Some of these are probably examples of competitive speciation (Rosenzweig, 1978) as envisaged in the present paper, while others may be cases of speciation due to predator-prey or host-parasite interactions (e.g., Feder, 1995). This suggests that frequency- and density-dependent selection can induce speciation under quite general conditions.

Abrams et al. (1993) have given examples of predator-prey models in which frequency-dependence leads to evolutionarily stable fitness minima. Living on a fitness minimum means experiencing disruptive selection, and if mating is not random as in Abrams et al. (1993), but assortative, this can lead to speciation. In fact, I have applied my haploid genetic model with assortative mating to the predator-prey model in Abrams et al. (1993, example 2, p. 476 and Appendix), and the result is speciation in the prey. Similarly, one can introduce assortative mating in the quantitative genetic host-parasite model of Doebeli (1996a, b), and the result is often speciation in both host and parasite. To better understand the general conditions that favour sympatric speciation, one could take a true community ecology approach by modeling not just one population, but the dynamics of both the consumer and the resource, the prey and the predator, or the host and its parasite. This could be done using genetics of the type described in this article, preferably extended to diploid organisms.

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