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## AN EXPLICIT GENETIC MODEL FOR ECOLOGICAL CHARACTER DISPLACEMENT<sup>1</sup>

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**Abstract.** Realistic models for ecological character displacement should incorporate population genetics. In Slatkin's pioneering model (Slatkin 1980), the genetics of the quantitative character determining the competitive interactions are modeled by assuming that the character is normally distributed in each generation. Only the mean and the variance of the character distributions change over time. With symmetric ecological assumptions for the two competing species, and with normally distributed resources that are equally used by both species, this model did not yield significant displacement. This has led to the belief that ecological asymmetries or constraints on resource use, e.g., by constraining the phenotypic variances, are necessary for character displacement. I argue that the reason for the negative result in Slatkin's original model is that the genetics are modeled too rigidly. With a more flexible genetic model, obtained by explicitly modeling many loci with additive effects, character displacement occurs as a rule even for symmetric ecological assumptions and without constraints on the phenotype distributions. The model can also be used in other contexts than competition for resources. For example, character displacement in a host–parasite system can lead to parasite specialization. The results suggest that more detailed genetic models yield a finer resolution of the interaction between population genetics and ecological dynamics. Explicit genetics lead to more insights than the usual quantitative genetic assumption of normal character distribution.

**Key words:** *apparent competition; character displacement; multilocus genetics; population dynamics; quantitative genetics.*

### INTRODUCTION

If the strength of competition between individuals is determined by a quantitative character, selection could lead to a permanent difference in the distributions of the character in two competing species. Such ecological character displacement is an intuitively appealing explanation for phenotypic differences among related sympatric species. However, it is controversial how often observed patterns are due to ecological character displacement, and much theoretical work has been devoted to the study of the conditions under which it can occur (e.g., Bulmer 1974, Crozier 1974, Lawlor and Maynard Smith 1976, Roughgarden 1976, Slatkin 1980, Matessi and Jayakar 1981, Case 1982, Lundberg and Stenseth 1985, Milligan 1985, Rummel and Roughgarden 1985, Taper and Case 1985, Abrams 1986, 1987*a, b*, 1990, Brown and Vincent 1987, Gotelli and Bossert 1991, Taper and Case 1992*a*, Vincent et al. 1993). These efforts have been reviewed by Taper and Case (1992*a*). They argued that, among the models they compared, the most realistic is the quantitative genetic model of Slatkin (1980), because it incorporates population genetics and contains the fewest constraining assumptions. It can be considered as a null-model: the competing species are assumed to have the same carrying capacity curves, competition in and between the

species is symmetrical, and there are no constraints on the utilization of the resources. However, this null-model does not yield significant character displacement (Slatkin 1980, Taper and Case 1985). Substantial displacement only results when ecological asymmetries in the carrying capacity curves of the species or in the competition functions are introduced (Slatkin 1980, Milligan 1985, Taper and Case 1992*a*), or when resource use is constrained, e.g., by constraining the phenotypic variance in the species (Slatkin 1980, Taper and Case 1992*a*), or by introducing explicit resource dynamics (Taper and Case 1985). Although asymmetries and constraints are common in natural systems (Taper and Case 1992*b*), analyzing character displacement under symmetrical and unconstrained conditions is also important, for example when studying adaptive radiation from ancestral lineages into otherwise empty phenotype space (e.g., Schluter and McPhail 1993, Schluter 1994). Moreover, studying this scenario with its few assumptions can reveal mechanisms preventing or enhancing character displacement that are otherwise masked by additional assumptions, and may thus give new insights for other scenarios as well. In this paper I show that the null-result for Slatkin's null-model derives from the way the quantitative genetics are modeled, rather than from the lack of asymmetries or constraints. Thus, while character displacement is more likely under such conditions, they are not necessary for displacement to occur.

Slatkin (1980) modeled the genetics by assuming

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that random mating results in normal character distributions in each generation. This is very common in quantitative genetic theory, but it seems to be too rigid an assumption for studying ecological dynamics (Doebeli 1995*b*). It obscures important details of the dynamics of single phenotypes. I propose a more flexible quantitative genetic model that keeps track of the frequencies of single phenotypes. This explicit modeling of the genetics leads to character displacement even in the null-model, i.e., without ecological asymmetries and without constraints on the phenotype distributions. Therefore, conclusions from models for character displacement depend not only on the ecological features of the models, but also on the genetic assumptions. In particular, more detailed genetic models make displacement more likely. The reason seems to be that these models describe more subtly how competition shapes character distributions, so that inherent tendencies for divergence manifest themselves more clearly. Thus ecological character displacement might be more common than previously believed.

THE QUANTITATIVE GENETIC MODEL

Following Slatkin (1980), my starting point was the models for the dynamics of populations with discrete generations:

$$N_{t+1} = N_t f(N_t). \tag{1}$$

Here  $N_t$  is the density of the population at time  $t$ , and  $f(N)$  is the fitness function. Exploitative competition for resources is modeled implicitly by assuming that the fitness depends on the density: the higher  $N$ , the lower  $f(N)$ . Slatkin (1980) used the logistic fitness function

$$f(N) = 1 + r - \frac{r}{K}N. \tag{2}$$

Here  $1 + r$  is the intrinsic growth rate of the population, and  $K$  is its equilibrium density, i.e., its carrying capacity. Although often used, the logistic function has the drawback of negative fitness values for high densities. This is not a problem in the homogenous and deterministic setting given by Eq. 2, because such high densities are never attained if the parameter  $r$  is chosen appropriately. However, in phenotypically variable populations like those considered below, negative fitness values can occur even for biologically reasonable choices of  $r$  if the system does not have stable equilibrium dynamics. This happens because the densities of the single phenotypes in the population add up to the total density that determines the fitness, and this total density can fluctuate to values that are too high. Therefore, for some of the results I used the following alternative for the fitness function:

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

This function was considered by Bellows (1981) to be

the most generally applicable one-dimensional competition model. The parameter  $\lambda$  is the intrinsic growth rate of the population. Depending on the assumptions about the competitive process that leads to density dependence, the parameter  $b$  reflects different types of competitive interaction (e.g., Hassell 1975, Schoener 1976). The parameter  $a$  determines the carrying capacity of the population (Doebeli 1995*a*). A low  $a$  implies a high carrying capacity and vice versa. Below I will use Eq. 2 to compare my results with those of Slatkin (1980) when the ecological dynamics exhibit a stable equilibrium, and I will use Eq. 2' to show how character displacement occurs with complex dynamics. As was pointed out by Milligan (1985), exploitative competition is not the only interpretation consistent with fitness functions that have the same general properties as Eqs. 2 and 2'. Thus the results obtained here generalize to other situations in which frequency- and density-dependent selection occurs. I come back to this point in the discussion, where I indicate how the model developed here can lead to character displacement in a host-parasite system.

Models 2 and 2' were first extended to describe a phenotypically variable population in which the phenotypes are determined by a quantitative character  $z$ . Then the extent of competition between two individuals depends on their relative character value: competition is more intense between individuals with similar phenotypes. For simplicity I assumed that the character  $z$  takes on values in the interval  $[0, c]$ , where  $c$  is a positive integer. This facilitates introducing genetics ( $c$  will be the number of loci), and it does not imply any loss of generality, since an appropriate rescaling always leads to such a situation. At the beginning of generation  $t$ ,  $p_t(z)$  is the frequency of the phenotype with character value  $z$ . Selection due to competition acts on the distribution  $p_t(z)$  before random mating in each generation. It is determined by the phenotypic fitness functions  $f_z$ ,  $z \in [0, c]$ . These functions now depend on the total density of the population,  $N_t$ , as well as on the phenotypic distribution  $p_t$ . Following Slatkin (1980), they have the form

$$f_z(N_t, p_t) = 1 + r - \frac{r}{K(z)} N_t \int_0^c p_t(z') \alpha(z, z') dz' \tag{3}$$

for the logistic model, and

$$f_z(N_t, p_t) = \frac{\lambda}{1 + \left\{ a(z) N_t \int_0^c p_t(z') \alpha(z, z') dz' \right\}^b} \tag{3'}$$

for the alternative model. The function  $\alpha(z, z')$  measures the strength of competition between individuals of phenotypes  $z$  and  $z'$ , so that the weighted sum

$$N_t \int_0^c p_t(z') \alpha(z, z') dz' \tag{4}$$

is the density experienced by an individual of phenotype  $z$  in a population with total density  $N_t$  and phenotype distribution  $p_t$ . Like Slatkin (1980), I used a symmetric form for  $\alpha(z, z')$ :

$$\alpha(z, z') = \exp\left(\frac{-(z - z')^2}{2\sigma_\alpha^2}\right). \quad (5)$$

The parameter  $\sigma_\alpha^2$  determines the niche width. Although commonly used, this function has the caveat of implying density-independent competition coefficients. It has been argued that density-dependent competition coefficients are more realistic (Abrams 1980), but I have not explored this further complication.

The functions  $K(z)$  and  $a(z)$  in Eqs. 3 and 3' determine the carrying capacities for the different phenotypes. Again following Slatkin (1980), I assumed that they are of the form

$$K(z) = a_0 \exp\left(\frac{-\left(z - \frac{c}{2}\right)^2}{2\sigma_k^2}\right) \quad (6)$$

and

$$a(z) = a_0 \exp\left(\frac{\left(z - \frac{c}{2}\right)^2}{2\sigma_k^2}\right), \quad (6')$$

respectively.

This implies that the carrying capacity of the phenotypes declines in a Gaussian form towards the edges of the character interval  $[0, c]$ . Thus extreme phenotypes have less resources. The choice of a symmetric  $\alpha(z, z')$  and of Gaussian  $K(z)$  corresponds to Slatkin's null-model that did not yield character displacement. He showed that displacement is more likely with increasingly asymmetric  $\alpha(z, z')$  and non-Gaussian  $K(z)$  (Slatkin 1980). Although such asymmetries may be common in nature, I want to show here that they are not necessary for character displacement. When the genetics are described explicitly, displacement occurs even with Slatkin's original symmetric assumptions.

Let  $q_t(z)$  denote the distribution of the phenotypes after selection. Then

$$q_t(z) = \frac{p_t(z)f_z(N_t, p_t)}{\bar{W}_t}, \quad (7)$$

where

$$\bar{W}_t = \int_0^c p_t(z)f_z(N_t, p_t) dz \quad (8)$$

is the mean fitness of the population at time  $t$ . The frequency  $q_t(z)$  is the frequency of gametes that phenotype  $z$  contributes to the gamete pool. The basic question is, how does the distribution  $q_t(z)$  determine the distribution  $p_{t+1}(z)$ ? In principle, this question can be answered if all the genotype frequencies are known. In

practice, this is not feasible if there is a large number of loci. The classical quantitative genetic method is to assume that the distribution  $p_{t+1}(z)$  is normal, with mean and variance calculated from the mean and variance of the distribution  $q_t(z)$ . In contrast, I used the following procedure.

I assumed the simplest genetic model, in which the character  $z$  is determined additively by  $c$  haploid loci (recall that I assumed the upper boundary  $c$  of the character interval to be an integer). Each locus has two alleles whose effects are 0 and 1, respectively. Thus the haploid individuals are described by a string of 0's and 1's of length  $c$ , corresponding to which allele is present at each locus, and I assumed that the phenotype of an individual is given by the number of 1's in its genetic string: if it has the 1-allele at  $z$  loci and the 0-allele at the remaining  $c-z$  loci, its phenotype is  $z$ . This means that there is no environmental variance in the phenotype. There were three reasons for this assumption.

First, my goal was to see the effect of explicitly modeling the genetics with as few confounding factors as possible. Second, it follows from the definition of the fitness function of the phenotypes that the results obtained without environmental variance will remain qualitatively the same if a small amount of environmental variance is introduced. This was confirmed with a few numerical simulations. Small environmental variances were used in the models of Slatkin (1980) and Taper and Case (1985), on which the present model is based. Last, and not least, there is no conceptual difficulty in introducing environmental variance in the model, but it slows down the computer simulations substantially.

Phenotypes thus ranged from 0, displayed by individuals having the 0-allele at each locus, to  $c$ , which corresponds to having the 1-allele everywhere. To see the effect of mating, I had to determine the phenotypic distribution of the offspring, given the phenotypes of the parents. To do this, I assumed that the allele of an offspring at a particular locus has probability 1/2 to come from either parent. This corresponds to assuming Mendelian segregation and free recombination between loci.

Suppose that the phenotypes of the parents are  $i$  and  $j$ , so that one parent has  $i$  1's and  $c-i$  0's in the genome, while the other has  $j$  1's and  $c-j$  0's. The number of loci at which both parents have the 1-allele is defined as the overlap  $l$ . From the  $l$  overlap loci, the offspring inherits the 1-allele. Also, there are  $i+j-2l$  loci at which one parent has the 1-allele and the other one has the 0-allele. By assumption, the offspring has either one of the alleles with probability 1/2 at these loci. At the remaining loci both parents have the 0-allele, hence so does the offspring. It follows that the phenotype of the offspring lies in the interval

$$[l, i+j-l], \quad (9)$$

and that the phenotype distribution in this interval is binomial. Therefore, if  $p_{ij}^l$  denotes the phenotype frequency distribution of offspring having parents with phenotypes  $i$  and  $j$  and with overlap  $l$ , we have

$$p_{ij}^l(z) = \begin{cases} \binom{i+j-l}{z-l} \left(\frac{1}{2}\right)^{i+j-l} & \text{for } z \in [l, i+j-l] \\ 0 & \text{otherwise.} \end{cases} \quad (10)$$

To determine the frequency distribution  $p_{ij}$  for the offspring phenotype of parents  $i$  and  $j$ , we have to take the sum of the distributions determined by the overlaps, weighted by the probability that a particular overlap occurs:

$$p_{ij} = \sum_{\text{all overlaps}} \text{Pr}(\text{overlap} = l) \cdot p_{ij}^l \quad (11)$$

Eq. 11 means that the phenotype distribution  $p_{ij}$  of the offspring is a weighted sum of nested binomial distributions. The weights are easily calculated: for phenotypes  $i \geq j$ , the probability to have overlap  $l$  is

$$\text{Pr}(\text{overlap} = l) = \frac{\binom{i}{l} \cdot \binom{c-i}{j-l}}{\binom{c}{j}} \quad (12)$$

Here  $l$  lies in the interval of all possible overlaps  $[l_{\min}, j]$ , where  $l_{\min}$  is the minimal possible overlap (since  $i \geq j$ ,  $j$  is the maximal possible overlap). It is easy to see that  $l_{\min}$  is equal to the larger of the numbers  $i + j - c$  and 0. To save computer time, one can approximate the correct distribution Eq. 11 by using only the outermost of the nested distributions in Eq. 11. That is, one can assume that the overlap is always minimal and use the approximation

$$p_{ij} \approx p_{ij}^{l_{\min}} \quad (13)$$

This approximation assigns slightly higher probabilities to extreme offspring phenotypes, and slightly lower probabilities to common phenotypes. The approximation makes the numerical simulations faster by a factor of more than four. Thus, the dynamic behavior of the models can be explored much more efficiently when using Eq. 13 instead of Eq. 11, especially when the number of loci is large. A few simulations with the exact formula Eq. 11 showed that approximation 13 yields qualitatively correct results. An indication of how good the approximation is, is given below in Fig. 1.

Given the frequency of gametes  $q_i(z)$  coming from phenotypes with character  $z$ , we can now determine the distribution  $p_{t+1}(z)$ . It is given as the sum of the offspring distributions  $p_{ij}$ , weighted by the frequency of matings between parents of phenotypes  $i$  and  $j$ , i.e., by the factor  $q_t(i)q_t(j)$ . Thus

$$p_{t+1}(z) = \sum_{i,j} q_t(i)q_t(j)p_{ij}(z). \quad (14)$$

Together with the recursion equation

$$N_{t+1} = N_t \bar{W}_t \quad (15)$$

where  $\bar{W}_t$  is the mean fitness Eq. 8, Eqs. 7, 11, and 14 determine the dynamics of this quantitative genetic model. I emphasize that this model is ecologically the exact analogue of the basic model in Slatkin (1980). What is different here is the genetics, i.e., the way the phenotype distribution  $p_{t+1}$  is determined from the gamete distribution  $q_t(z)$ . To reiterate, Slatkin assumed that  $p_{t+1}(z)$  is normal, with the mean and variance determined from  $q_t(z)$ , while I used Eqs. 11 and 14 to describe the transition from  $q_t(z)$  to  $p_{t+1}(z)$ . Eq. 11 was derived under the assumption of free recombination between haploid loci. In principle, analogous equations could be derived for more complicated genetic assumptions such as diploidy and linkage, but I have not explored these possibilities. In the next section I report the results obtained for the competition between two species whose phenotypic fitness functions depend on the joint distributions of the phenotypes in both species, and whose genetics are described by the model presented here.

RESULTS

To model competition between two species I assumed that both species have the same range of possible phenotypes given by the quantitative character  $z \in [0, c]$ , and that both have the same ecological parameters. Thus each species is limited by the same resources, and individuals of each species utilize these resources in the same way, which means that the functions  $K(z)$  and respectively,  $a(z)$ , Eqs. 6 and 6', which determine the carrying capacity of phenotype  $z$ , are the same in both species. Moreover, the competitive impact of the individuals on each other does not depend on belonging to the same or different species. This implies that the effective density that an individual of phenotype  $z$  experiences under competition at a given time  $t$  is

$$N_{\text{eff},t}(z) = N_{1,t} \int_0^c p_{1,t}(z') \alpha(z, z') dz' + N_{2,t} \int_0^c p_{2,t}(z') \alpha(z, z') dz' \quad (16)$$

Here  $N_{i,t}$  are the total densities of the two species at time  $t$ , and  $p_{i,t}$  are the phenotype distributions in the two species, where  $i = 1, 2$ . Formula 16 holds for individuals of phenotype  $z$  regardless of which species they belong to. It follows that the phenotypic fitness functions in both species are

$$f_{i,z}(N_{1,t}, N_{2,t}, p_{1,t}, p_{2,t}) = 1 + r - \frac{r}{K(z)} N_{\text{eff},t}(z) \quad (i = 1, 2) \quad (17)$$

for the logistic model, and

$$f_{i,z}(N_{1,t}, N_{2,t}, p_{1,t}, p_{2,t}) = \frac{\lambda}{1 + [a(z)N_{\text{eff},i}(z)]^b} \quad (i = 1, 2) \quad (17')$$

for the alternative model. After selection given by these functions, random mating in each species determines the phenotypic distributions  $p_{i,t+1}$ ,  $i = 1, 2$ , according to the model described in the previous section. The total densities  $N_{i,t+1}$  are given by

$$N_{i,t+1} = N_{i,t} \bar{W}_{i,t} \quad (18)$$

where  $\bar{W}_{i,t}$  is the mean fitness of species  $i$ , which is given by an equation analogous to Eq. 8.

The basic density-dependent models given by Eqs. 1 and 2 or 2' can exhibit very complicated dynamics (May and Oster 1976). Although phenotypic variability given by the quantitative character has a stabilizing effect (Doebeli 1995b), the system can still have complicated dynamics. But first I considered the case where the two populations exhibit stable equilibrium dynamics. This was always the case in Slatkin (1980), and to make the comparison with his results I used the logistic Eqs. 2 and 17, respectively, for this scenario. Results similar to the ones reported here can be obtained for the alternative model (Eq. 17'). At the equilibrium, the character distributions in the two species have means  $\bar{z}_1$  and  $\bar{z}_2$  and variances  $\sigma_1^2$  and  $\sigma_2^2$ . By definition, significant character displacement is said to occur if the difference between the means is greater than the averaged standard deviation (Taper and Case 1985), i.e., if

$$\frac{|\bar{z}_1 - \bar{z}_2|}{(\sigma_1 + \sigma_2)/2} > 1. \quad (19)$$

This inequality was never satisfied in Slatkin's model. In the present model, however, it holds as a rule. Two typical situations are shown in Fig. 1, where the means and variances of the character distributions in the two species are plotted as a function of time. Taper and Case (1985) reanalyzed Slatkin's model and noted that some displacement occurs for certain parameter values, although never enough for Inequality 19 to hold. A critical parameter is the ratio  $\sigma_k^2/\sigma_\alpha^2$ , where  $\sigma_k$  is the variance of the resource utilization curve Eq. 6, and  $\sigma_\alpha$  determines the niche width in Eq. 5. Intuitively, one would expect that a larger niche, i.e., a smaller  $\sigma_\alpha$ , and a higher  $\sigma_k$ , hence a higher  $\sigma_k^2/\sigma_\alpha^2$ , would facilitate displacement, because there is room for more niches along the resource axis. Indeed, Taper and Case (1985) showed that some displacement occurs as  $\sigma_k^2/\sigma_\alpha^2$  increases above 2. This trend was confirmed in the present model, but significant displacement as defined by Inequality 19 occurred even for  $\sigma_k^2/\sigma_\alpha^2$  that are much smaller than 2 (Fig. 1a). The displacement was larger for larger values of  $\sigma_k^2/\sigma_\alpha^2$  (Fig. 1b).

At the equilibrium, the character distributions were approximately normal (Figs. 1c and d). This shows that

it is not normality per se that prevented displacement in Slatkin's model, but how normal phenotype distributions were achieved. In Slatkin's model, normality was an assumption. Here the corresponding assumption was a binomial offspring distribution, Eq. 10. Normality of the phenotype distributions emerged as a consequence both of this assumption and of the ecological dynamics of the single phenotypes. In contrast, Slatkin's model is more rigid in allowing only the total density of the population and the mean and the variance of the phenotype distributions to change over time. In terms of genetics, what prevents character displacement in his model are the variances: they evolve to values that are too high to allow divergence of the phenotypic means. However, displacement can occur if the variances are constrained by assuming them fixed at a value that is much lower than the one they would attain if they were free to evolve (Slatkin 1980, Taper and Case 1992a). Small variances imply a small denominator in Inequality 19, but, more importantly, they allow the phenotypic means to diverge further than with unconstrained variances, because constraints lead to underutilized resources and consequently to more niche space (Milligan 1985). That constraints on the variance of the phenotype distributions can be important for ecological character displacement has been pointed out repeatedly (Slatkin 1980, Matessi and Jayakar 1981, Milligan 1985).

In the explicit genetic model presented here, however, no additional assumptions constraining the phenotypic variance were needed for displacement. The variances typically evolved to values that were low enough for significant displacement to occur. Like normality of the phenotype distributions, the evolution of the variances to relatively low values was an emergent property of the system, determined by the explicit genetics and the ecological dynamics resulting from selection through competition for resources. The evolution of the variance can be seen in Figs. 1b and 2. (In Fig. 1a, plotting started after 50 generations, after which the variance was already very close to the equilibrium value.)

The explicit genetic model and Slatkin's model with fixed variances can be compared as follows. For a given set of parameters  $\sigma_\alpha$ ,  $\sigma_k$ , etc., one can determine the equilibrium value to which the variances evolve in the explicit model. One can then build the version of Slatkin's original model corresponding to the given parameters, and fix the variance at the value obtained from the explicit model. From this it can be seen that if displacement occurred in the explicit model, it also occurs in Slatkin's model (where it would not occur if the variance were free to evolve). Moreover, the variance obtained from the explicit model typically lies well below the upper limit of values that still yield displacement in Slatkin's model (recall that only fixed variances below a certain limit lead to displacement in this model). Thus the two approaches can be reconciled

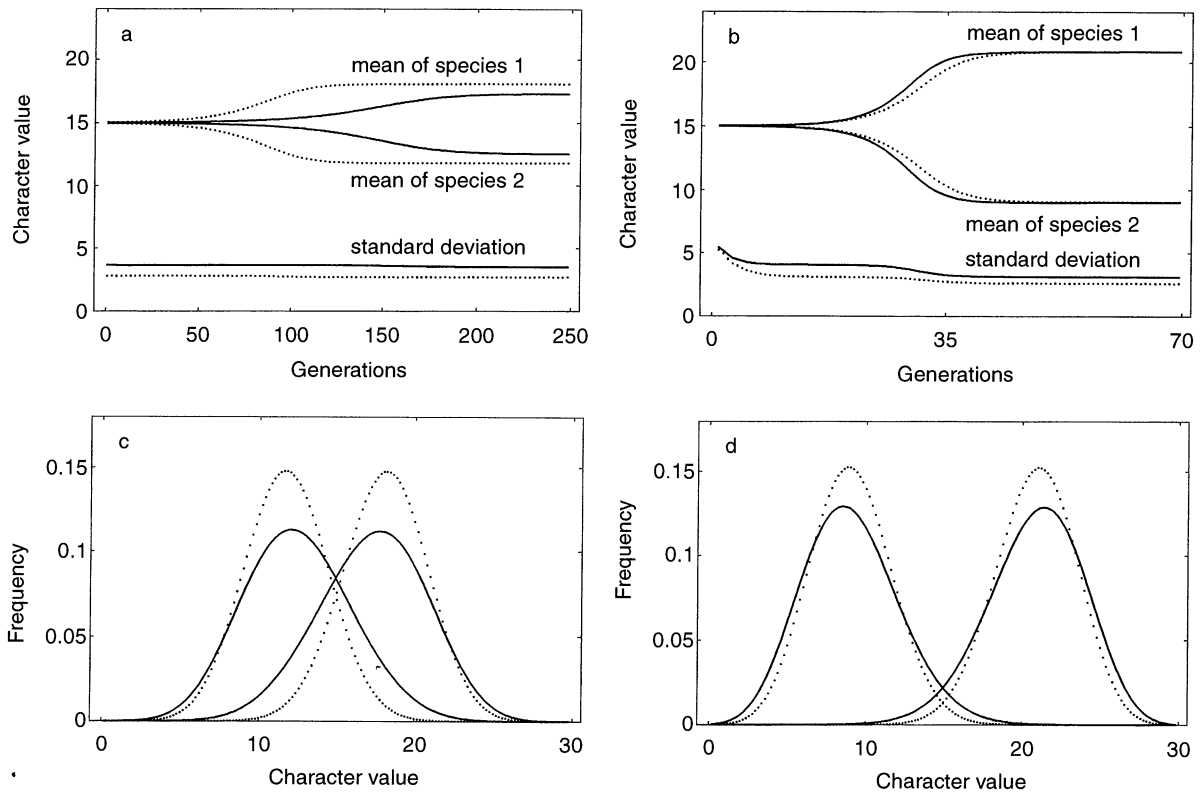


FIG. 1. In parts a and b, the means and standard deviations of the character distributions in two competing species are shown as a function of time for two different values of  $\sigma_k^2/\sigma_\alpha^2$ . The systems have globally stable equilibria, in which the species have evolved to different niches in phenotype space. The niche separation is symmetric because of the symmetric ecological assumptions. Because at the equilibrium both species have the same standard deviation, it is shown for only one species. The logistic fitness functions (Eq. 17) were used. In all panels, the dotted lines represent results obtained with the exact formula (Eq. 11) in the genetic model, while the continuous lines show results obtained with Approximation 13. In part a, the value of the critical parameter  $\sigma_k^2/\sigma_\alpha^2$  (see Results) is well below 2, yet the condition (Inequality 19) for significant displacement is satisfied at the equilibrium. Displacement increases and occurs more rapidly for larger  $\sigma_k^2/\sigma_\alpha^2$  (part b). For the lower value of  $\sigma_k^2/\sigma_\alpha^2$ , displacement is larger and occurs faster with the exact genetic model than with the approximation. For the higher value of  $\sigma_k^2/\sigma_\alpha^2$  the difference between the two models is very small. In parts c and d, the character distributions in the two populations are shown at the equilibria corresponding to parts a and b. The distributions are approximately normal, and for both values of  $\sigma_k^2/\sigma_\alpha^2$  the exact model yields a slightly lower variance. The parameter values for the figures were: number of loci  $c = 30$ ;  $r = 1.6$ ,  $K_0 = 1000$  in the basic fitness function (Eq. 2) ( $K_0$  determines the maximal carrying capacity in Eq. 6, see *The quantitative genetic model*);  $\sigma_k^2 = 59.3$  in Eq. 6, and  $\sigma_\alpha^2 = 40$  in Eq. 5 for parts a and c, hence  $\sigma_k^2/\sigma_\alpha^2 = 1.48$ ;  $\sigma_k^2 = 122.7$  and  $\sigma_\alpha^2 = 30$  for parts b and d, hence  $\sigma_k^2/\sigma_\alpha^2 = 4.09$ . The initial character distributions for the simulation runs were obtained by applying one bout of mating according to the genetic model to a uniform distribution. In part a, plotting started after 50 generations.

with additional assumptions in the classical quantitative genetic model. The basic difference remains that displacement occurs in the explicit genetic model without constraints on the phenotypic variances.

In this model, phenotypic variation is constantly produced by mating, for even if two parents have the same phenotype, the offspring can have a range of phenotypes, because the parent phenotypes are determined by 1-alleles at different loci. The non-zero equilibrium value of the variance is determined by frequency-dependent selection, which is operating because the fitness of a phenotype decreases if more individuals of the same phenotype are present. The effect of frequency dependence can be illustrated by plotting the fitness of the different phenotypes in the equilibrium state (Fig.

2). In this state, the fitness is not constant as a function of the character value. Rather, it has two minima, corresponding to the two peaks of the equilibrium character distributions in the two species: fitness is  $< 1$  for the most common phenotypes due to frequency dependence. Fitness has two maxima at the edges of the character interval: even though there are fewer available resources, fitness is  $> 1$  for extreme phenotypes because they are rare. There is also an intermediate local fitness maxima, which lies exactly in the middle of the character interval because of the symmetry in the model. Mean fitness is 1, but in each generation frequency-dependent selection tends to flatten the character distributions and hence to increase the phenotypic variance. Random mating then restores the original dis-

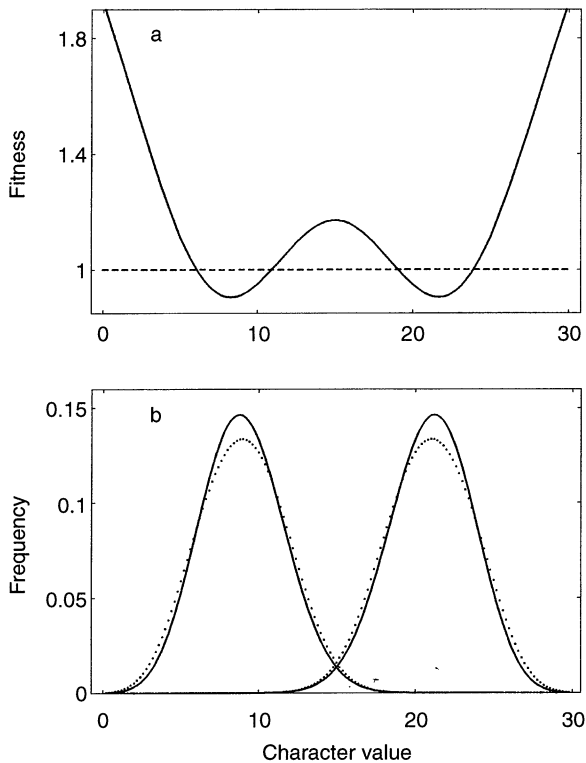


FIG. 2. Phenotypic fitnesses, and character distributions before and after selection, in the equilibrium state. (A) Due to frequency dependence, the fitness has two minima at the peaks of the character distributions in the two species. The fitness has an intermediate local maximum and two maxima at the edges of the character interval. Mean fitness is 1. The dotted line is drawn for reference. (B) The continuous curves show the phenotypic distributions in the two species at the beginning of each generation in the equilibrium state. The dotted lines show the distributions after selection, but before mating. Selection flattens the distributions and hence increases their variance. When mating is applied to the dotted distributions, the continuous distributions are restored. The exact genetic model (Eq. 11) was used for the figure. The parameter values were: number of loci  $c = 30$ ;  $r = 1.6$ ,  $K_0 = 1000$ ,  $\sigma_k^2 = 150$ , and  $\sigma_\alpha^2 = 20$ .

tributions that were present at the beginning of the generation. Thus, at the equilibrium, phenotype distributions are constant when censused at the same stage in different generations, but within generations the distribution after selection, given by  $q_i(z)$  in Eq. 7, is different from the distribution before selection,  $p_i(z)$ . Random mating exactly compensates the selective forces, so that  $p_{i+1}(z) = p_i(z)$ . An example of this is shown in Fig. 2. In Fig. 2a, the fitness is plotted as a function of the character value in the equilibrium state, and in Fig. 2b the equilibrium character distributions are shown before (continuous line) and after (dotted line) selection in a single generation. The difference between the two distributions illustrates the effect of frequency-dependent selection, which decreases the frequency of common phenotypes and increases the frequency of rare ones.

Frequency dependence is one of several mechanisms thought to play a role in the maintenance of phenotypic and genetic variability (Barton and Turelli 1989). Other such mechanisms are mutation–selection balance, heterosis, and pleiotropy (Barton and Turelli 1989, Bulmer 1989), which do not occur in the model studied here. In general, the evolution of genetic variances and covariances is a complicated problem (Turelli 1988, Turelli and Barton 1990). In many quantitative genetic models, this problem is decoupled from the evolution of phenotypic means, because genetic covariances are parameters in these models that do not themselves evolve (Turelli 1988). One approach to couple these problems is to study explicit genetic models, in which the evolution of both the means and the variances emerges from the dynamics of the interactions between single genotypes or phenotypes. The present model constitutes a very simple example of this approach. It shows how frequency-dependent selection on single phenotypes, together with a simple algorithm for the effect of mating on phenotypic distributions, determines non-zero equilibrium variances. How competition for a resource between individuals of a single species can maintain phenotypic variance has been discussed by Slatkin (1979). If the present model is applied to a single species, the variance that is maintained at equilibrium is larger than when two species are competing with each other, which again illustrates the effect of frequency dependence. Competition between two species reduces the equilibrium variance because the niches get smaller. However, it can reduce the variances to zero only if it is intense enough for the species' phenotypic means to evolve to the extreme values 0 and  $c$ , which does not happen for reasonable choices of the parameters, because the extreme phenotypes are selected against due to their low carrying capacities.

The amount of variation that was maintained at equilibrium depended on the parameters  $\sigma_k$  and  $\sigma_\alpha$ , and on the ratio  $\sigma_k^2/\sigma_\alpha^2$ . On the one hand, a larger range of available resources, expressed by a higher  $\sigma_k$ , and a smaller  $\sigma_\alpha$  (i.e., less intense competition) both tended to increase the variance of the character distributions at equilibrium. On the other hand, larger values of  $\sigma_k^2/\sigma_\alpha^2$  tended to decrease the variance, because character displacement was enhanced, which moved the species' phenotypic means to more extreme values. Now  $\sigma_k^2/\sigma_\alpha^2$  is increased by both larger  $\sigma_k$  and smaller  $\sigma_\alpha$ , and these opposing trends determined the equilibrium variance. Its value did not depend on the initial densities and character distributions assumed at the beginning of the simulations. This reflects another difference to Slatkin's model. While there was neutral stability with many different stable equilibria in his model, the numerical simulations indicated that there were only two globally stable equilibria in the systems studied here, corresponding to the interchangeability of the two species.

The results also did not depend qualitatively on using



Approximation 13 instead of the exact formula (Eq. 11) for the genetic model. Other numerical simulations confirmed what is shown in Fig. 1: if anything, displacement was larger in the exact model. This justifies the use of the approximation, at least in the present context. That the variances of the phenotype distributions were slightly smaller when using the exact model was expected, since the approximation implies a spreading of the offspring distribution.

Character displacement also occurred as a rule when the system exhibited more complicated dynamics. To study these cases, I used Eq. 2' instead of the logistic function (Eq. 2) as the basic model. With fluctuating population sizes, the logistic fitness (Eq. 17) can attain negative values, which happens when the densities of the single phenotypes add up to effective population sizes (Eq. 16) that are too high. This does not occur for expression 17', which makes this model better suited for studying character displacement when the ecological dynamics are complicated. Such complex dynamics lead to non-equilibrium dynamics also for the character distributions in the two species. They were not constant over time, and their means and variances fluctuated. However, the size of the fluctuations was usually much smaller than the average difference between the means, and this average difference was again larger than the average variance in either species. Even though the variances were not constant, they were on average low enough to allow divergence of the phenotypic means: the characters were displaced. This is shown in Fig. 3, for a system with chaotic dynamics. Significant displacement occurred for a wide range of parameters. Only two factors tended to prevent it. First, it did not occur for small values of  $\sigma_k^2/\sigma_a^2$ . Second, if the ecological dynamics exhibited very large fluctuations, then the fluctuations in the means of the character distributions also became large and of similar size to the average difference between the means, so that no permanent displacement was discernible.

With the present model one can also study the effect of the number of loci on character displacement. To do this, I assumed a given maximal carrying capacity  $a_0$  in Eq. 6, and a given fraction of  $a_0$  as the carrying capacity for the extreme phenotypes 0 and  $c$ . This means that  $\sigma_k^2$  was larger when there were more loci. To make the comparison with different numbers of loci for fixed values of the critical parameter  $\sigma_k^2/\sigma_a^2$ , I also assumed that the niche width  $\sigma_a$  was larger when there were more loci. This compensated for the larger range of available resources. The general effect of increasing the number of loci was then to decrease the value of  $\sigma_k^2/\sigma_a^2$  for which character displacement first occurred. In particular, when the number of loci was large enough, displacement occurred even if  $\sigma_k^2/\sigma_a^2 < 1$ , i.e., even if the niche width was larger than the range of available resources. In general, the higher flexibility gained from a larger number of loci made displacement more likely. This conforms with the general theme of

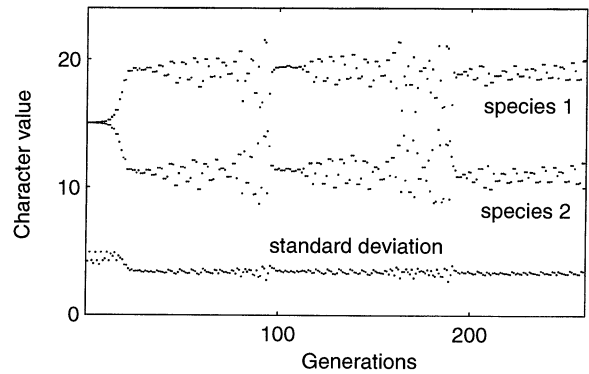


FIG. 3. Mean and standard deviations of the character distributions for two populations that do not exhibit equilibrium dynamics. Here the fitness functions had the alternative form (Eq. 17'), and Approximation 13 was used in the genetic model. Again, the standard deviation  $\sigma$  is shown only for one species, since it has similar dynamics in both species. The ecological dynamics are chaotic, and the means and variances of the character distributions fluctuate. The size of the fluctuations is smaller than the average difference between the means. This average difference is much larger than the average standard deviation, which shows that character displacement has occurred. Note the intermittent outbursts of erratic fluctuations of the means that interrupt longer periods of more regular dynamics. The parameter values for the figure were: number of loci  $c = 30$ ;  $\lambda = 15$ ,  $b = 4.18$ ,  $a_0 = 0.1$  in the basic fitness function (Eq. 2');  $\sigma_k^2 = 42.6$  in Eq. 6', and  $\sigma_a^2 = 16$  in Eq. 5.

this paper: less rigid and more detailed genetic models facilitate character displacement.

## DISCUSSION

### *Character displacement under exploitative competition*

The ecological assumptions in the model presented here are the same as in Slatkin's (1980) basic model: two species are limited by a common resource; resource utilization depends on a quantitative character and is the same in both species; the extent of competition between two individuals either of the same or of different species depends on their relative character value, and competition is symmetric, given by the function  $\alpha(z, z')$ , Eq. 5; finally, the resource utilization curve (Eq. 6) is assumed to be Gaussian. The models only differ in the way the genetics of the quantitative character are modeled, i.e., in the way mating determines the phenotypic distribution in the next generation, given the phenotypic distribution after selection due to competition has occurred. Slatkin assumed that the distribution after mating is normal with the mean and variance determined from the distribution after selection, but before mating. In contrast, I modeled the genetics of many loci with additive effects explicitly. Significant character displacement does not occur in Slatkin's model, while it is the rule rather than the exception in the model presented here. This suggests that modeling the

genetics in more detail can lead to very different predictions under the same ecological assumptions.

The central step in the genetic model is to determine the phenotypic distribution of the offspring given the phenotypes of the parents. This was done here for a very simple genetic scenario, letting the character value be determined by the number of 1-alleles, regardless of their position in the genome. In principle, the transition from the parent phenotypes to the offspring distribution can also be done under more complicated genetic assumptions, e.g., with diploidy or linkage, but the description of the model would be more complicated. I do not know to what extent the results would be different, but I suspect that displacement would still be very likely. The basic feature of this model is the greater flexibility obtained from explicitly describing the frequencies of single phenotypes. This allows more subtlety in the way phenotype distributions are molded by competition. That greater flexibility facilitates character displacement is confirmed by the observation that increasing the number of loci that determine the quantitative character makes displacement more likely.

A list of examples of character divergence in nature is given in Taper and Case (1985: Table 1). Slatkin's basic model can only explain these patterns if additional genetic or ecological assumptions are made. For example, displacement occurs in his model if resource use is constrained, either by explicitly modeling the resource dynamics (Taper and Case 1985), or by constraining the phenotypic variance in the competing species (Slatkin 1980). Thus, variances that are fixed at a value that is sufficiently lower than the one to which they would evolve without constraints can lead to divergence of the phenotypic means and to character displacement. Such constraints are often thought to be necessary for ecological character displacement. (Slatkin 1980, Matessi and Jayakar 1981, Milligan 1985), but they are not needed in the explicit genetic model presented here, in which the variances typically evolve to values that are consistent with displacement.

Another possibility to explain displacement with Slatkin's model is to assume asymmetries in the effects of competition, or in the way resources are used by the competing species (Slatkin 1980, Milligan 1985, Taper and Case 1992a). Such ecological conditions are likely to be satisfied often in natural systems, and indeed could probably be argued in most examples given in Taper and Case (1985). Nevertheless, studying the symmetric case with unconstrained resource use may also be important. For example, for adaptive radiation from a common ancestor into otherwise empty phenotype space, symmetric and unconstrained conditions are at least initially realistic. Therefore, it may be necessary to assume these conditions in order to explain adaptive radiation with ecological character displacement (Schluter and McPhail 1993, Schluter 1994). Moreover, finding the mechanisms that enhance or prevent displacement may be easier in this case, because there are

fewer confounding factors. I do not claim that ecological asymmetries and constraints are rare in nature, but that they are not a necessary condition for ecological character displacement. Theoretically, none of the extensions mentioned above is needed. Instead, modeling the genetics of the quantitative character explicitly is enough.

Two of the weaknesses of the type of models studied here are the assumptions of a fixed-carrying-capacity curve and of density-independent competition coefficients (Abrams 1980, Taper and Case 1992b). It would be interesting to see how explicit genetics affect more general models in which these assumptions are relaxed. Extrapolating from the present results, one would expect that explicit genetic models facilitate character displacement in these situations as well. Thus, to the extent that such models are more realistic than those assuming normal character distributions, one could expect that ecological character displacement plays a more important role in structuring communities than previously believed. This is supported by recent experimental evidence showing that competition promotes adaptive radiation (Schluter 1994).

#### *An example of character displacement under apparent competition*

The use of the genetic model described here is not restricted to competition for resources. In principle, it can be applied whenever interactions in a population with discrete generations are determined by a quantitative character. I briefly outline its use in a host-parasite system that was introduced by May and Anderson (1983). Their model describes a host population whose density is regulated by parasites that spread in each generation of the host before reproductive age is reached, thereby killing a fraction  $I(N)$  of the host population  $N$ . Therefore, if  $\lambda > 1$  is the intrinsic growth rate of the host in the absence of parasites, the fitness function in a phenotypically homogeneous host population is

$$f(N) = \lambda[1 - I(N)]. \quad (20)$$

May and Anderson (1983) derived the density-dependent fraction  $I(N)$  from epidemiological considerations as the solution of the equation

$$1 - I = \exp\left(-\frac{IN}{N_T}\right). \quad (21)$$

$N_T$  is the threshold density of the host: if  $N < N_T$ , the only solution to Eq. 21 is 0, i.e., the parasite cannot spread. May (1985) showed that a host population with fitness given by Eq. 20 exhibits chaotic dynamics for all growth rates  $\lambda > 1$ , regardless of the size of the threshold density  $N_T$ .

Guided by the gene-for-gene principle for host-parasite interactions (Hamilton 1980), one can extend this model to a host population in which the interaction with the parasites is determined by a character that is

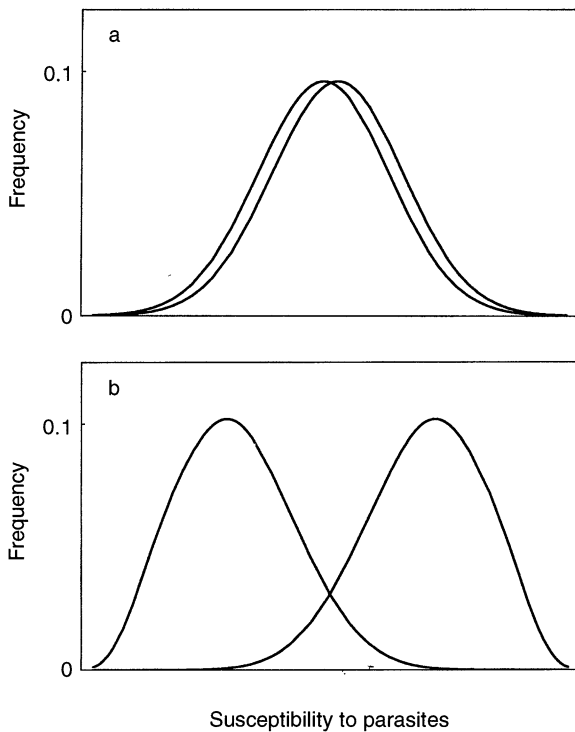


FIG. 4. The distributions of a host character that determine the interaction with parasites are shown at the beginning (part a) and at the end (part b) of character displacement due to competition for phenotype space between two host populations. The intrinsic growth rate  $\lambda$  is 1.7 in both populations, and the threshold density is 1 (see *Discussion*). The number of loci is 30, and Approximation 13 was used in the genetic model. The system exhibits stable equilibrium dynamics. That the populations evolve to different niches in phenotype space means that they evolve susceptibilities to different types of parasites, and could be a mechanism for parasite specialization. Note that the homogeneous host population given by Eq. 20 has chaotic dynamics for the same growth rate  $\lambda$ . This indicates that quantitative genetics can have a stabilizing effect on population dynamics.

quantitative in the sense that it is additively determined by many haploid loci according to the model described in *The quantitative genetic model*. The character takes on discrete integer values in the interval  $[0, c]$ , and the corresponding phenotypes are susceptible to a unique parasite. There are as many parasite types as there are host phenotypes, and a particular parasite can attack a phenotype if the latter has the right number of 1-alleles, regardless of where these alleles occur in the genome. Thus the host-parasite interaction is based on additive matching. The fitness function of each host phenotype is of the form of Eq. 20. It depends only on that phenotype's density, because the parasite attack on a particular phenotype does not depend on the densities of other phenotypes. After selection due to the parasites, random mating leads to a reshuffling of phenotypes according to the quantitative genetic model.

If two host populations coexist, and if the parasites attack them equally, then the fitness of a phenotype in

one population decreases if the same phenotype is also present in the other population. This leads to apparent competition for phenotype space, which could lead to character displacement: the two populations could evolve to separate niches in phenotype space. Indeed, this is what happened (Fig. 4). The phenotype distributions in the two populations diverged. Since susceptibility to parasites is determined by the phenotype, this means that the populations evolve susceptibilities to different types of parasites. Thus character displacement due to apparent competition for phenotype space could explain the tendency for each species to have its own parasite (S. Stearns, *personal communication*). That apparent competition for parasite free space could be an important factor for the structure of host species communities has been suggested by Holt and Lawton (1993).

This example extends Milligan's (1985) remark that character displacement could result whenever negative fitness interactions occur, not just when competition is exploitative. It also shows that the genetic model presented in this paper can be used in many different contexts. Moreover, one can use the model to study other problems such as the influence of phenotypic variability and quantitative genetics on population dynamics (Doebeli 1995b). The flexibility gained from explicit genetics allows a finer resolution of the interaction between population genetics and ecological dynamics. This can lead to different results than the usual quantitative genetic assumption of normal character distributions.

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