Selection 2 (2001) 1–2, 41–64
Available online at http://www.akkrt.hu

Long-term Buildup of Reproductive Isolation Promoted by Disruptive Selection: How Far Does it Go?

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(Received: 6 July 2001,
Accepted: 20 September 2001)

We analyze the long-term evolution of a continuous trait subject to frequency-dependent disruptive selection, and controlled by a single diploid, additive locus. Our simple selection model is a mathematical approximation to many complex systems of ecological interactions resulting in disruptive selection, like, for example, scramble competition and habitat heterogeneity. A polymorphism of two specific alleles at equal frequencies is the unique long-term equilibrium, or ESS, of this system. We then study the evolution of direct assortative mating for the selected trait, through mutations of small effect at modifier loci controlling the degree of assortment. The mating process is described by a model that allows for possible costs of assortment. Unless the cost of assortment is too high, strength of assortment always increases in populations where mating is random or weakly assortative, and also in populations that already practice very strong assortative mating. However, even if it has no cost, assortment can increase continuously from random mating to complete isolation, resulting in sympatric speciation, only if selection is sufficiently strong. In fact, only a modest degree of assortment, corresponding to a continuously stable ESS, can be attained from random mating, when selection intensity is below a certain threshold.

Keywords: Assortative mating, sympatric speciation, long-term evolution, polymorphic ESS, competition, frequency-dependent selection, MEAST, PEAST, evolutionary singularity, evolutionary branching point

1. Introduction

It is well known that under certain conditions frequency-dependent selection can maintain genetic variation (e.g. Cockerham et al., 1972; Clarke, 1979; Udovic, 1980; Wilson and Turelli, 1986; Asmussen and Basnayake, 1990). The question of how general and common these conditions are in natural populations remains largely unsettled. Recently, a new theoretical framework – the adaptive dynamics – has been developed that suggests that under certain types of ecological interactions (e.g. competition, multiple niches) biological systems naturally evolve, through a sequence of fixations of mutations, towards the areas of parameter space where genetic variation is maintained (e.g. Christiansen and Loeschcke, 1980; Christiansen, 1991; Metz et al., 1996; Eshel et al., 1997; Geritz et al., 1998; Kisdi and Geritz, 1999). An interesting consequence of the adaptive dynamics is a possibility for the maintenance of genetic variation under apparently disruptive selection. In such a situation, intermediate genotypes (e.g. heterozygotes) are selected against by frequency-dependent selection but are recreated each generation as a result of random mating, segregation and recombination.

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Given reduced fitness of intermediate genotypes, one may expect that any tendency to assortative mating for the selected trait will be favoured by natural selection whenever its possibility arises in the population, because this will decrease the production of maladaptive genotypes. This idea forms the basis of both the reinforcement scenario (e.g. Dobzhansky, 1940; Butlin, 1987; Howard, 1993) and of many scenarios of sympatric speciation (e.g. Maynard Smith, 1966; Udovic, 1980; Felsenstein, 1981; Kondrashov, 1986; Doebeli, 1996; Dieckmann and Doebeli, 1999; Kondrashov and Kondrashov, 1999; Geritz and Kisdi, 2000).

Most of the theoretical works on reinforcement and sympatric speciation have considered not the whole process of the evolution towards complete reproductive isolation but rather single (mostly initial) steps resulting in some assortative mating. The idea was that if some level of assortative mating can get established in a population which initially is random mating, then further changes that make assortative mating stronger and stronger would proceed easily, leading eventually to complete reproductive isolation. A limited amount of simulations focusing on the whole process of speciation seem to confirm this expectation (e.g. Dieckmann and Doebeli, 1999; Kondrashov and Kondrashov, 1999). However, the complexity of the models used in these simulations and a large number of parameter combinations to be explored make any generalizations very difficult. For this reason, it would be beneficial to complement numerical studies of complex models with analytical (and numerical) studies of much simpler models with transparent interpretation.

Here, we will consider a type of model that according to Felsenstein (1981) should provide the most favorable conditions for the evolution of reproductive isolation. We will assume that there is a diploid additive locus controlling a continuous trait which simultaneously is under frequency-dependent disruptive selection and is also used in assortative mating. By the latter assumption our model is of the type called “one-allele” by Felsenstein (1981) in opposition to the “two-allele” type – considered less favorable to evolution of reproductive isolation – where selection and assortment act on distinct characters. Assortment for the character subject to selection may be achieved indirectly, through the development of linkage disequilibrium with the character subject to assortment. We assume that the mechanics of assortment is according to the model of Gavrilets and Boake (1998), which allows for a possible cost of assortment due to delays in mating caused by mate choice. First, we will show that for the selected trait there is a polymorphic long-term equilibrium or ESS, namely a stable equilibrium of two specific alleles that cannot be invaded by any other mutant allele. We then will assume that a second genetically controlled continuous trait determines the intensity of assortment, so that its range of variation spans the whole spectrum of mating behavior, from random mating to complete isolation among the two homozygotes of the primary trait occurring at the polymorphic ESS. By considering the dynamics of single locus mutations of small effect for this mating trait, we will show that, provided the cost of assortment is not too high, a small degree of assortment can always be introduced in a random mating population while, conversely, a weakening of assortment is always prevented whenever complete isolation prevails in the population. But, in spite of these results, we also will demonstrate, quite surprisingly, that unless selection on the primary trait is strong enough, evolution of assortment starting from random mating cannot progress by small changes all the way to complete isolation because it will stop at some intermediate stage that has the characteristics of a (weak) ESS level of assortment. Apparently in contrast with recent simulation results based on a model very similar to ours (e.g. Dieckmann and Doebeli, 1999), but in agreement with old and recent results based on models of the “two-allele” type (e.g. Felsenstein, 1981; Dieckmann and Doebeli, 1999; Kondrashov and Kondrashov, 1999;), we thus conclude that disruptive selection *per se* is not sufficient for sympatric speciation to occur because it is also required that the level of selection against intermediate types be strong enough.

2. Frequency-dependent disruptive selection in a random mating population

2.1. Types of evolutionary singularities

In the long-term evolution of continuous traits there are points of the phenotypic space that have

the nature of "evolutionary singularities" (Geritz et al., 1998) in the sense that a qualitative change of the evolutionary dynamics of monomorphic populations takes place in their vicinity. The two most important kinds of such singularities were discussed and contrasted for the first time by Christiansen (1991) who named them MEAST and PEAST, respectively (acronyms for monomorphic or polymorphic evolutionarily attainable stable traits). Both these singularities have the property – called continuous stability (Eshel and Motro, 1981; Eshel, 1983), or *m*-stability (Taylor, 1989), or convergence stability (Christiansen, 1991) – that mutations of small effect can invade a monomorphic population with a trait value near the singular point if and only if they change the trait in its direction. Thus, both MEAST and PEAST are evolutionary attractors in the sense that in long-term evolution a trait tends to move toward one of such points, as long as the population remains monomorphic. The difference between the two kinds of singularities is that a monomorphic population at a MEAST cannot be invaded by any mutation, while, on the opposite, any mutation can invade a PEAST. A MEAST is the same as a continuously stable ESS as defined by Eshel and Motro (1981) and Eshel (1983). Thus it identifies a monomorphic long-term equilibrium which, at least for traits with the simple clonal (asexual) genetics, is locally stable in the long-term dynamics (Eshel et al., 1997). This means that a population not too far away from a MEAST will indeed converge – through a sequence of invading mutations – to the monomorphic state identified by the singularity.

The long-term implications of a PEAST are not as firmly established. What is certain that once a population is sufficiently close to a singularity of this kind a protected polymorphism necessarily arises which is maintained by disruptive selection, the opposite of what would occur near a MEAST where, if a polymorphism arises it would be maintained by stabilizing selection (through heterozygote advantage). As a consequence of this selection regime, further evolution through invading mutations tends to be in the direction of increasing the phenotypic variance of the population (Christiansen and Loeschcke, 1980; Christiansen, 1991; Eshel et al., 1997; Geritz et al., 1998; Kisdi and Geritz, 1999). According to the theory of adaptive dynamics PEASTs might be a major cause of branching of the phylogenetic tree. This view is based mostly on results from models with asexual reproduction (Geritz et al., 1998, 1999; Kisdi, 1999; Doebeli and Dieckmann, 2000), but also in the more important case of sexual reproduction and mendelian genetics it is supported by results on secondary evolution of assortative mating and sympatric speciation (Dieckmann and Doebeli, 1999; Geritz and Kisdi, 2000). Here we will analyze the role of PEAST in sexual diploid species with respect to both maintenance of polymorphism and speciation.

Singular points of the PEAST kind have been discovered in a great variety of adaptation problems, including intraspecific exploitative competition (Christiansen and Loeschcke, 1980), sex-ratio (Uyenoyama and Bengtsson, 1982), resource-exploitation intensity, susceptibility to predation and intraspecific contests (Abrams et al., 1993), multiple niches (Geritz et al., 1998), asymmetric competition (Kisdi, 1999), seed size (Geritz et al., 1999), mutualism and prey-predator interactions (Doebeli and Dieckmann, 2000). Accordingly, the details of the fitness representations specifically appropriate to each of these situations are quite diverse and might be very complicated. We instead, rather than focussing on a particular example, prefer to adopt a fitness representation that, on the one hand, is mathematically simple and, on the other hand, can be taken as a good approximation for any adaptation problem involving a PEAST if the current values of the evolving character are close enough to the singularity.

2.2. A simple model of frequency-dependent disruptive selection

The context in which evolutionary singularities appear is that of the initial evolution of a mutation introduced at a small frequency in a monomorphic resident population in equilibrium. In this context, assuming discrete generations, let $v(y,x)$ denote the intrinsic (asymptotic) rate of increase of the subpopulation of mutants when the trait value of residents is x and that of mutants is y, and let $v(x,x)=1$. Hence, the mutation invades if $v(y,x)>v(x,x)=1$, while it cannot invade if $v(y,x)<1$. If $v(y,x)$ is such as to admit a PEAST singularity in the interior of the phenotypic domain, without loss of generality

we may assume it to be located at $x=x^\circ=0$. It then follows from the definition of PEAST that, avoiding non-generic cases, $v(y,x)$ must have the following properties (Eshel, 1983; Taylor, 1989; Christiansen, 1991; Geritz et al., 1998)

$$v_y(0,0)=0 , \quad (1.1)$$

$$v_{yy}(0,0)>0 , \quad (1.2)$$

$$v_{yy}(0,0)+v_{yx}(0,0)<0 , \quad (1.3)$$

where

$$v_y(x,x)=\left.\frac{\partial v(y,x)}{\partial y}\right|_{y=x} , \; v_{yx}(x,x)=\left.\frac{\partial^2 v(y,x)}{\partial y \partial x}\right|_{y=x} , \text{ etc.}$$

Conditions (1.1) and (1.2) ensure that $v(y,x)$ has a local minimum with respect to y at $y=x=x^\circ=0$, so that x° can be invaded by any mutant of small effect. Condition (1.3) ensures the continuous stability of $x^\circ=0$, so that this singularity is an evolutionary attractor for monomorphic populations. The simplest function with these properties is the quadratic in y and x

$$v(y,x)=1+\alpha y^2-(\alpha+\beta)xy+\beta x^2 , \text{ with } 0<\alpha<\beta . \quad (2)$$

The limitations on the parameters α and β are imposed by (1.2) and (1.3). In fact, (1.2) is violated if $\alpha\leq 0$, while (1.3) is violated if $\alpha\geq\beta$. As long as x and y are not too far from $x^\circ=0$, this choice for $v(y,x)$ is a good approximation to the rate of increase of a mutant in a monomorphic population for any adaptation problem that admits a PEAST (Metz et al., 1996).

To produce a simple frequency-dependent selection model in which invasion dynamics is governed by (2), we stipulate that fitness (viability) of individuals in a population of arbitrary phenotypic composition is determined by the outcome of random pairwise interactions in which the payoff to an individual of phenotype y who has an opponent of phenotype x is given by $v(y,x)$. Hence, in any very large population where the trait has mean $\bar{x}$ and variance s^2, the average fitness of generic individuals of phenotype y is given by

$$w(y,\bar{x},s^2)=\mathbf{E}\{v(y,x)|y\}= =1+\alpha y^2-(\alpha+\beta)\bar{x}\,y+\beta(\bar{x}^2+s^2) . \quad (3)$$

Obviously, this fitness is a linear function of the phenotypic frequencies, with positive coefficients, $v(y,x)$, that depend on the particular set of trait values, x, currently available in the population. Hence, as ever new genetic variants invade and spread in the population, the intensity and characteristics of selection changes in the course of long-term evolution. The mean fitness of the population is

$$\overline{w}=\mathbf{E}\{w(y,\bar{x},s^2)\}=1+(\alpha+\beta)s^2 . \quad (4)$$

Hence, the trait values that are favored by selection in the current population are those that satisfy

$$w(y,\bar{x},s^2)>\overline{w} \text{ , or } \alpha y^2-(\alpha+\beta)\bar{x}\,y+\beta\bar{x}^2>\alpha s^2 . \quad (5)$$

The disruptive character of selection becomes conspicuous when $\bar{x}\approx x^\circ=0$. In fact for $\bar{x}=0$ condition (5) indicates that, since $\alpha>0$, a trait y is favored if and only if $y^2>s^2$.

It is interesting that, in spite of its rather abstract derivation, the fitness regime represented by (3) is fully equivalent to that of the model of intraspecific exploitative competition of Christiansen and Loeschcke (1980), later adopted also by Dieckmann and Doebeli (1999). Formally this model – based on Lotka–Volterra competition equations parameterized in terms of Gaussian resource spectrum and utilization functions – appears quite complex. But then it is reduced to (3) by the approximations permitted by the assumptions that all individual niche locations are close to the mean of the resource spectrum, equal to zero, and that the total population size is always at the equilibrium corresponding to the current population composition. With this interpretation the parameters α and β satisfy the limitations of (2) if the resource spectrum is wider than the individual niches, and both decrease as the width of individual niches increases.

2.3. Genetic assumptions

We complete the formulation of our model with the two additional assumptions that variation of the selected trait is determined by a single additive, diploid locus, and that genetic or developmental constraints limit such variation to the finite range

$[-x_L,x_L]$. Without loss of generality we may let $x_L=1$, so that the phenotypic domain in our model is given in fact by the interval [–1,1]. The assumption of one additive locus is also made by Christiansen and Loeschcke (1980), Kisdi and Geritz (1999), and Geritz and Kisdi (2000), and genetic additivity is central to the analysis of sympatric speciation of Dieckmann and Doebeli (1999).

If mating is at random with respect to the selected trait, the trait value of a random individual from the newborn population is $x=e+e'$, where e and e' are independent, identically distributed random variables representing, respectively, the effects of the maternal and the paternal allele in the genotype of the given individual. Hence, if the allelic effects in the population have mean $\bar{e}$ and variance s_a^2, we easily derive from (3) and (4) that in this population the mean fitness of an allele with effect d is

$$w_a(d,\bar{e},s_a^2)=1+\alpha d^2-2\beta\bar{e}d+ \\ +(2\beta-\alpha)\bar{e}^2+(2\beta+\alpha)s_a^2, \qquad (6.1)$$

and the overall mean fitness is

$$\bar{w}=1+2(\alpha+\beta)s_a^2. \qquad (6.2)$$

2.4. The polymorphic ESS

As demonstrated by Christiansen and Loeschcke (1980), in a population conforming to the above model no equilibrium may involve more than two distinct alleles. Additivity, together with the limitation of the phenotypic domain to [–1,1], implies that the allelic domain – the set of possible allele effects – is limited to the interval $\left[-\frac{1}{2},\frac{1}{2}\right]$. By a standard analysis it can be shown that the two extreme alleles of this set, of effects $e_-=-\frac{1}{2}$ and $e_+=\frac{1}{2}$, may coexist in a locally stable equilibrium where each has a frequency of $\frac{1}{2}$, and that this is the only equilibrium with these two alleles. This polymorphism is protected because the two alleles can invade each other, as it is easy to prove from the general condition for a mutant d to invade a population where the effects of the resident alleles have mean $\bar{e}$ and variance s_a^2, namely

$$w_a(d,\bar{e},s_a^2)>\bar{w}\Leftrightarrow\alpha d^2-2\beta\bar{e}d+(2\beta-\alpha)\bar{e}^2>\alpha s_a^2. \qquad (7)$$

In a population where the two alleles $\{e_-,e_+\}$ are in equilibrium obviously $\bar{e}=0$ and $s_a^2=\frac{1}{4}$, which is the largest possible variance for a distribution over the interval $\left[-\frac{1}{2},\frac{1}{2}\right]$. It follows that no mutant allele can invade such a population. In fact, condition (7) reduces in the present case to $d^2>\frac{1}{4}$, which can never be satisfied since any mutant different from the resident alleles is limited by $-\frac{1}{2}<d<\frac{1}{2}$. We conclude that the population state $\mathbf{E}_0$, consisting of the two extreme alleles, $\{e_-,e_+\}$, in equal frequency, is a polymorphic long-term equilibrium. Moreover $\mathbf{E}_0$ is the only long-term equilibrium possible in the present system, because for every other population state a mutant can be found that can invade it. In fact, consider the mutant e_- if in the resident population $\bar{e}\geq 0$, or the mutant e_+ if $\bar{e}<0$. In either case invasion condition (7) reduces to

$$\alpha\frac{1}{4}+\beta|\bar{e}|+(2\beta-\alpha)\bar{e}^2>\alpha s_a^2,$$

which is always true because $s_a^2<\frac{1}{4}$ for any population state different from $\mathbf{E}_0$. As a conclusion of this analysis we may therefore state the following

RESULT 1. In a system of frequency-dependent disruptive selection defined by the fitness function of Eq. (3), operating on a continuous trait controlled by a single additive, diploid locus with allelic effects limited to the finite range $\left[-\frac{1}{2},\frac{1}{2}\right]$, there is, under random mating, a unique long-term equilibrium or ESS, identified by the polymorphic population state, $\mathbf{E}_0$, where each of the two alleles with extreme effects is present with a frequency of $\frac{1}{2}$.

3. Assortative mating for a trait under disruptive selection

Once a random mating population has settled in the polymorphic long-term equilibrium $\mathbf{E}_0$, its phenotypic variance is only $s^2=\frac{1}{2}$. Hence its mean fitness, equal to $1+\frac{\alpha+\beta}{2}$, by Eq. (4), is far from the maximum value it could have, namely $1+\alpha+\beta$. This is because half of its members, being heterozygotes, have a trait value, $x=0$, that gives them the smallest fitness, $w(0,0,\frac{1}{2})=1+\frac{\beta}{2}$ (see Eq. 3), while the homozygotes, with phenotype $x=-1$ or $x=1$, have the largest fitness, namely $1+\alpha+\frac{\beta}{2}$. Since assortative mating for the trait would tend to decrease the frequency of heterozygotes, individuals adopting such mating habit should have, on average, offspring with a larger phenotypic variance and therefore a greater fitness than the resident population. Thus, it is reasonable to expect that assortative mating should evolve in this population.

3.1. The model of assortative mating

Most studies of the evolution of reproductive isolation under disruptive selection concentrate on the establishment of a correlation, by way of linkage disequilibrium, between the selected trait and another trait which, for some reason, happens to be already a target of assortative mating in the population (Maynard Smith, 1966; Felsenstein, 1980; Kondrashov and Kondrashov, 1999; Geritz and Kisdi, 2000). This is certainly reasonable. But, if the selected trait is a morphological feature that can be perceived and appreciated by conspecifics of the opposite sex it is not less plausible, from a biological point of view, that this trait itself might become the focus of mating preferences. We therefore suppose (as in Dieckmann and Doebeli, 1999) that there exist genes – to which we will refer as *mating genes* – different from the one controlling the primary trait, which with the appropriate alleles could induce in females a degree of mating preference of assortative type with respect to the selected trait.

We assume that the mating process resulting in assortment agrees with the model introduced by Gavrilets and Boake (1998). Preferences are expressed by females, who mate only once, while males may participate in multiple matings. Unmated adult females encounter males at random. An encounter of a female of trait value x with a male of trait value y results in their mating with probability $\pi(|x-y|)$, where the *mating preference* function π is decreasing and $\pi(0)=1$, since here we are concerned only with positive assortment. A female who refuses a male will try again at the next encounter, unless she had made already a total of $n=1,2,\ldots$ unsuccessful encounters, in which case she must remain unmated, an assumption that reflects the idea that the time interval available to females for mating is limited. Thus, if $p(y)$ represents the frequency of trait y in the population, the probability that for a female x an encounter results in mating is

$$\bar{\pi}(x)=\sum_y \pi(|x-y|)p(y), \qquad (8.1)$$

and the probability that she eventually mates with a male y is given by $\theta(x,y)p(y)$, where

$$\theta(x,y)=\sum_{i=0}^{n-1}[1-\bar{\pi}(x)]^i\,\pi(|x-y|). \qquad (8.2)$$

We refer to this function as *mating rate*. In case of $n=1$ the model can also be conceived as a model of fertility selection (Bodmer, 1965; Hadeler and Liberman, 1975), or as a model of parental selection (Gavrilets, 1998). Thus, in this case the model can be interpreted as describing both *postmating* and *premating* reproductive isolation, whereas the case of $n>1$ is for premating isolation only. Ecological situations where the encounter rate between females and males is very high are approximated by the limit of $n=\infty$. In this case there is no cost to females for being choosy, because every female is sure to find a mate irrespective of her phenotype. In fact

$$\theta(x,y)=\frac{\pi(|x-y|)}{\bar{\pi}(x)}, \text{ so that}$$

$$\sum_y \theta(x,y)p(y)=1 \quad \text{for all } x. \qquad (9)$$

Hence, in this form, assortative mating does not introduce an additional factor of selection among females. But even in this case a component of sexual selection for the primary trait is present among males, because in general $\sum_x \theta(x,y)p(x) \neq \sum_x \theta(x,y')p(x)$ for yy'. Published studies of sympatric speciation through evolution of assortative mating have not considered the possibility of a cost to choosiness and use models of assortment where, as in our case of $n=\infty$, all females are certain to mate (e.g. Felsenstein, 1981; Doebeli, 1996; Dieckmann and Doebeli, 1999; Kondrashov and Kondrashov, 1999; Geritz and Kisdi, 2000).

3.2. The invasion dynamics of mating modifiers

Our general method of analysis of the evolution of assortative mating with disruptive selection is the following. We start from a resident population where the primary locus is polymorphic for the two extreme alleles (e_-,e_+) and all mating loci are monomorphic, and assume it to be at a locally stable equilibrium under disruptive selection, as specified by (3), and under some level of assortative mating characterized by a mating preference π, as specified in (8). We then let a mutation of a mating gene occur, introducing at a small frequency a mutant allele m' besides the resident allele m. The rate of recombination between the mating locus affected by the mutation and the primary locus is r. The mutation has the effect that heterozygote females $m|m'$ have mating preference π' instead of π – and mating rate θ' instead of θ – but we assume that the difference between π' and π is small. We then analyze the dynamics of this population to determine whether the mutant allele m' is able to invade the resident population by increasing in frequency while rare.

Table 1 shows the structure of this population. Since with assortative mating genotypes are not in Hardy–Weinberg proportions, we have to use genotypic frequencies rather than gametic frequencies to represent the composition of the population. The total frequency of adult mutant heterozygotes, $u_1+u_{21}+u_{22}+u_3=u$, is small and the adult genotypic frequencies of residents are only slightly different from the equilibrium frequencies (p_1,p_2,p_3), that they had before mutation occurred, so that the perturbations v_1, v_2 and v_3 are small. Matings among heterozygote mutants are ignored because their frequency is $o(u)$. Therefore, the homozygote mu-

TABLE 1

Structure of the population after mutation of a mating gene

	genotype →			RESIDENTS		
				$\frac{e\_m}{e\_m}$	$\frac{e\_m}{e_+m}$	$\frac{e_+m}{e_+m}$
	↓	primary trait ↓	→	–1	0	1
			frequency	p_1+v_1	p_2+v_2	p_3+v_3
MUTANTS	$\frac{e\_m'}{e\_m}$	–1	u_1	$(\theta_{11}+\theta'_{11})p_1u_1$	$(\theta_{21}+\theta'_{12})p_2u_1$	$(\theta_{31}+\theta'_{13})p_3u_1$
	$\frac{e_+m'}{e\_m}$	0	u_{21}	$(\theta_{12}+\theta'_{21})p_1u_{21}$	$(\theta_{22}+\theta'_{22})p_2u_{21}$	$(\theta_{32}+\theta'_{23})p_3u_{21}$
	$\frac{e\_m'}{e_+m}$	0	u_{22}	$(\theta_{12}+\theta'_{21})p_1u_{22}$	$(\theta_{22}+\theta'_{22})p_2u_{22}$	$(\theta_{32}+\theta'_{23})p_3u_{22}$
	$\frac{e_+m'}{e_+m}$	1	u_3	$(\theta_{13}+\theta'_{31})p_1u_3$	$(\theta_{23}+\theta'_{32})p_2u_3$	$(\theta_{33}+\theta'_{33})p_3u_3$

Note: genotypic frequencies refer to the adults, after selection. $\theta_{11}=\theta(-1,-1)$, $\theta_{12}=\theta(-1,0)$, $\theta_{13}=\theta(-1,1)$, … , $\theta'_{11}=\theta'(-1,-1)$, … . Each θ_{ij} and θ'_{ij} is computed, according to (8), with respect to a population of composition (p_1,p_2,p_3).

tants, which are produced by these matings, are also ignored. It follows that $v_1+v_2+v_3 = -u+o(u)$ because the sum of all genotypic frequencies must be one. In the main body of the table we report the frequencies of matings among mutants and residents, neglecting $o(u)$ terms. The mutation affects only the mating behaviour of females. Hence, in each of these matings, the appropriate mating rate is θ when the female is a resident, and θ' when she is a mutant.

The equilibrium genotypic frequencies among the adults of the resident population (p_1,p_2,p_3), are determined as follows. Adult females mate and generate the newborn of next generation. If (q_1,q_2,q_3) denote the genotypic frequencies among these – before the occurrence of viability selection – then

$$Mq_1 = \theta_{11}p_1^2 + \frac{1}{2}(\theta_{12}+\theta_{21})p_1p_2 + \frac{1}{4}\theta_{22}p_2^2, \quad (10.1)$$

$$Mq_2 = \frac{1}{2}(\theta_{12}+\theta_{21})p_1p_2 + (\theta_{13}+\theta_{31})p_1p_3 + \\ + \frac{1}{2}\theta_{22}p_2^2 + \frac{1}{2}(\theta_{23}+\theta_{32})p_2p_3, \quad (10.2)$$

$$Mq_3 = \theta_{33}p_3^2 + \frac{1}{2}(\theta_{23}+\theta_{32})p_2p_3 + \frac{1}{4}\theta_{22}p_2^2 \quad (10.3)$$

$$M = \sum_{ij}\theta_{ij}p_ip_j\,. \quad (10.4)$$

M is the normalization factor needed to ensure that $q_1+q_2+q_3=1$. It is equal to one only if all females are certain to mate, which occurs only when $n=\infty$ (see Eqs 8, 9). The population of newborn with this composition now undergoes frequency-dependent viability selection according to (3). Hence the adult genotypic frequencies, which at equilibrium must be identical to those of the previous generation, are given by

$$\overline{W}p_i = W_iq_i,\ i=1,2,3,\ \overline{W} = \sum_i W_iq_i \quad (10.5)$$

where W_i is the fitness of the i-th genotype and $\overline{W}$ then is the mean fitness. Hence

$$W_1 = 1+\alpha(q_2+2q_3)+2\beta q_3\,, \quad (10.6)$$

$$W_2 = 1+\beta(q_1+q_3), \quad (10.7)$$

$$W_3 = 1+\alpha(q_2+2q_1)+2\beta q_1\,, \quad (10.8)$$

$$\overline{W} = 1+(\alpha+\beta)[4q_1q_3+(q_1+q_3)q_2]. \quad (10.9)$$

We know that with random mating ($\theta_{ij}=1$ for all i,j) the unique polymorphic solution of (10) is $p_1=p_3=\frac{1}{4}$, and with complete assortment ($\theta_{ij}=0$ for all ij) it is obvious that the solution is $p_1=p_3=\frac{1}{2}$. It is natural to expect that the same symmetry, $p_1=p_3$, carries over to polymorphic solutions of (10) at intermediate levels of assortment. Such expectation is grounded in the intrinsic symmetry of the viability selection structure (cf. 10.6–8) and of the mating preference function π. We therefore limit our consideration to solutions of (10) having the property that $p_1=p_3$. The actual existence – and stability – of these equilibria for intermediate assortment will be considered later. With this restriction it follows immediately that

$$\theta_{12}=\theta_{32},\ \theta_{13}=\theta_{31},\ \theta_{21}=\theta_{23},\ q_1=q_3, W_1=W_3. \quad (10.10)$$

As long as the mutant allele is rare (small u) the state of the population is adequately described by the vector ($v_1,v_2,v_3,u_1,u_{21},u_{22},u_3$) and the – short-term – evolutionary dynamics of the population is represented with sufficient accuracy by the recurrence equations for this vector, approximated by neglecting $o(u)$ terms. It is easy to verify that in the resulting linear recurrence equations the dynamics of (u_1,u_{21},u_{22},u_3) is independent of (v_1,v_2,v_3), and that the transition matrix for $(u_1,u_{21},u_{22},u_3)^T$, taking into account (10.10) is

$$\mathbf{Q}(\pi,\pi') = \frac{W_1}{M\overline{W}}\begin{pmatrix} a & rb & (1-r)b & 0 \\ 0 & h(1-r)b & hrb & hc \\ hc & hrb & h(1-r)b & 0 \\ 0 & (1-r)b & rb & a \end{pmatrix}, \quad (11.1)$$

where

$$a = \frac{1}{2}(\theta_{11}+\theta'_{11})p_1 + \frac{1}{4}(\theta_{21}+\theta'_{12})p_2\,,$$

$$b=\frac{1}{2}(\theta_{12}+\theta'_{21})p_1+\frac{1}{4}(\theta_{22}+\theta'_{22})p_2,$$

$$c=\frac{1}{2}(\theta_{13}+\theta'_{13})p_1+\frac{1}{4}(\theta_{21}+\theta'_{12})p_2,$$

$$h=\frac{W_2}{W_1}=\frac{1+2\beta q_1}{1+\alpha+2\beta q_1}. \qquad (11.2)$$

Notice that the quantity $1-h$ is a measure of the intensity of disruptive selection.

Invasion by the mutant mating allele m' is decided by the dominant eigenvalue, $\lambda(\pi,\pi')$ say, of $\mathbf{Q}(\pi,\pi')$. The elements of this matrix are non-negative and the matrix is irreducible – because the elements of $\mathbf{Q}^i$ are strictly positive for $i \geq 2$; the only exception is when $c=0$, which occurs if assortment is complete and m' has no effect on mating preferences. Therefore, by Perron–Frobenius theorem for non-negative matrices, λ is a real, simple and positive root of the characteristic equation, and its associated (right) eigenvector, $\xi(\pi,\pi')$ say, is the only (right) eigenvector of $\mathbf{Q}$ with positive elements (Gantmacher, 1960). Thus, m' invades if $\lambda>1$, it does not invade and is eliminated if $\lambda<1$. If m' is neutral (i.e. $\pi=\pi'$), not surprisingly, $\lambda(\pi,\pi)=1$ and $\xi(\pi,\pi)=\left(p_1,\frac{1}{2}p_2,\frac{1}{2}p_2,p_3\right)^{\mathrm{T}}$, as proved by the fact that the equations

$$\left(p_1,\frac{1}{2}p_2,\frac{1}{2}p_2,p_3\right)^{\mathrm{T}}=$$

$$=\mathbf{Q}(\pi,\pi)\left(p_1,\frac{1}{2}p_2,\frac{1}{2}p_2,p_3\right)^{\mathrm{T}}$$

are necessarily satisfied, being identical to the equilibrium conditions (10.1–5); since the elements of $\left(p_1,\frac{1}{2}p_2,\frac{1}{2}p_2,p_3\right)^{\mathrm{T}}$ are positive, 1 is the dominant eigenvalue of $\mathbf{Q}(\pi,\pi)$.

The eigenvalues of $\mathbf{Q}$ can be calculated explicitly for all π and π' (Appendix A). In particular, the dominant eigenvalue λ is given by

$$\lambda=\frac{W_1}{M\overline{W}}\eta, \qquad (12.1)$$

where η is the largest root of the quadratic equation

$$\eta^2-(a+hb)\eta+hb(a-c)=0. \qquad (12.2)$$

By inspecting (12), (11.2) and (10), we notice immediately that λ is independent of the rate of recombination, r, between the mating gene and the primary locus. Hence we can state the following general result.

RESULT 2. Invasion of weak modifiers of assortative mating in a resident population at a symmetric polymorphic equilibrium under frequency-dependent disruptive selection does not depend on the rate of recombination between modifier genes and the trait locus.

3.3. Inception of assortment and maintenance of complete isolation

Our first use of these mathematical results will be to investigate whether a small degree of assortment can be successfully introduced in a random mating population and, at the opposite end, whether a population with complete assortment is immune from invasion by mutations that slightly decrease the strength of assortment. In case of random mating we already know (Result 1) that the resident population has a globally stable polymorphic equilibrium, $\mathbf{E}_0$, where genotypic frequencies satisfy $p_1=p_3$ for adults and $q_1=q_3=\frac{1}{4}$ for the newborn. A symmetric equilibrium – with genotypic frequencies $q_1=q_3=p_1=p_3=\frac{1}{2}$, a state that will be indicated by $\mathbf{E}_\infty$ – obviously exists also in case of complete assortment. It can be proved that with no cost of mate choice, at $n=\infty$, this equilibrium is globally stable provided only that selection is disruptive ($\alpha,\beta>0$). On the other hand, for any finite n the stability of $\mathbf{E}_\infty$ requires sufficiently strong disruptive selection and is only local, because it can be shown that the two monomorphic equilibria, corresponding to fixation of either alleles, e_- or e_+, are always stable.

In a random mating resident population, where obviously mating preferences are

$$\pi(0)=\pi(1)=\pi(2)=1,$$

we consider a mating gene mutation which has the effect to change these to

$$\pi'(0)=1\ ,\ \pi'(1)=1-k\varepsilon\ ,\ \pi'(2)=1-K\varepsilon,$$

where k, K and ε are positive but ε is small ($\varepsilon\approx 0$). Straightforward calculations of the dominant eigenvalue $\lambda(\pi,\pi')$ from (12), using (11.2) and (8) give the following result

$$\lambda(\pi,\pi')=1-\frac{(1+h)^2k+hK}{4(1+h)^2}\varepsilon+o(\varepsilon) \qquad \text{for } n=1,$$

$$\lambda(\pi,\pi')=1+\frac{(1+h)K}{8(1+h)^2}\varepsilon+o(\varepsilon) \qquad \text{for } n=2,\ldots,\infty.$$

It is then clear that if the cost of assortment is very high – because by rejecting the first male she encounters a female forfeits mating altogether – a small amount of assortment cannot invade a random mating population, irrespective of the intensity of disruptive selection, $1-h$, because $\lambda<1$ always. In all other cases, even if mate choice entails a cost ($2\le n<\infty$), we see that $\lambda>1$, meaning that random mating can always be invaded by weak assortment, as long as there is some disruptive selection ($h<1$) in the population.

In a resident population with complete assortment, where mating preferences therefore are

$$\pi(0)=1\ ,\ \pi(1)=0\ ,\ \pi(2)=0,$$

we consider a mutation which slightly reduces assortment, changing mating preferences to

$$\pi'(0)=1\ ,\ \pi'(1)=k\varepsilon\ ,\ \pi'(2)=K\varepsilon,$$

where k, K and ε are positive and $\varepsilon\approx 0$. Notice that the resident population contains only the two extreme types of individuals, of phenotype $x=-1$ and $x=1$, respectively, which effectively constitute two fully isolated species. Computing again $\lambda(\pi,\pi')$ from (12) we find

$$\lambda(\pi,\pi')=1+\frac{hkK}{4}\varepsilon^2+o(\varepsilon^2) \qquad \text{for } n=1,$$

$$\lambda(\pi,\pi')=1-\frac{(2^n-n-1)K}{2(2^n-1)}\varepsilon+o(\varepsilon) \qquad \text{for } n=2,3,\ldots,$$

$$\lambda(\pi,\pi')=1-\frac{(2-h)K}{4-h}\varepsilon+o(\varepsilon) \qquad \text{for } n=\infty.$$

This result is entirely consistent with the previous one. If cost of mate choice is high ($n=1$) complete mating isolation cannot be maintained; in fact mutations that slightly reduce assortment can always invade because $\lambda>1$. Hence, in this case assortment is disadvantageous at both extremes of its range. In all other cases ($n>1$), on the other hand, complete assortment is protected; in fact mutations that reduce it can never invade because $\lambda<1$. Hence, for $n>1$ assortment is advantageous at both extremes of its range. As a summary, we can state the following

RESULT 3. Excepting the case of high cost of mate choice ($n=1$), an equilibrium random mating population under disruptive selection – as defined in Result 1 – is invaded by small levels of assortment, while complete reproductive isolation between the two extreme types coexisting under this selective regime is protected against small reductions of assortment.

4. Buildup of reproductive isolation

Result 3 suggests the possibility that, in the model system we are considering, disruptive selection might generally cause the following long-term process: (1) increase of phenotypic variance toward the maximum level allowed by random mating, $s^2=\frac{1}{2}$, at the symmetric polymorphism $\mathbf{E}_0$; (2) inception of assortative mating; (3) progressive reinforcement of assortment through a sequence of invasions and fixations of mating gene mutants; (4) attainment of complete assortment, resulting in full reproductive isolation between the two extreme phenotypes which alone remain at state $\mathbf{E}_\infty$ of the population, where phenotypic variance is $s^2=1$, the largest possible value for a distribution over $[-1,1]$. If phases (1), (2) and (4) of this process are essentially validated by previous results, the central and most critical phase (3) needs a number of results

yet to be ascertained, namely: (i) that a stable polymorphic equilibrium, $\mathbf{E}_S$, of the two extreme alleles, (e_-,e_+), for the primary trait persists with continuity from $\mathbf{E}_0$ to $\mathbf{E}_\infty$ as assortment increases throughout all intermediate levels, S, from random mating to complete isolation; (ii) that at each $\mathbf{E}_S$ mating gene mutants invade if and only if they increase the degree of assortment; (iii) that each $\mathbf{E}_S$ is an ESS with respect to mutations of the primary locus; (iv) that invasion generally does not result in polymorphism of mating genes but in replacement of the resident allele by the mutant. Ascertainment of these points is complicated by the difficulty of solving analytically equilibrium equations (10). Thus several of our results will be based on evidence from numerical experiments rather than on analytical proofs.

4.1. Polymorphism under disruptive selection with partial assortative mating

In order to quantify degrees of assortment, from now on we will assume (Lande, 1981) that the mating preference, $\pi(|x–y|)$, of a female of trait x for a male y is given explicitly by

$$\pi(|x-y|)=\exp\{-S(x-y)^2\}, \qquad (13.1)$$

so that $S\in[0,\infty)$ measures the strength of assortment, $S=0$ corresponding to random mating and $S=\infty$ to complete isolation. As long as the only trait values in the population are (–1, 0, 1), we need to be concerned with only three values of this function and in this respect, for convenience, we will also use the alternative notation

$$\pi(0)=1\ ,\ \pi(1)=\exp\{-S\}=1-\upsilon\ ,$$

$$\pi(2)=\exp\{-4S\}=(1-\upsilon)^4 \qquad (13.2)$$

in which the strength of assortment is measured by $\upsilon\in[0,1)$.

Although existence and local stability of polymorphic equilibria of the primary locus with partial assortment cannot be ascertained analytically, we can easily obtain some hint by looking at the stability of fixations of the two alleles e_- and e_+. By the symmetries of the model the two fixations are stable or unstable simultaneously. These symmetries also imply that if there is an odd number of polymorphic equilibria one of these must be symmetric (i.e. with $p_1=p_3$). Thus, if instability of fixations prevails, a likely possibility – but by no means a certainty – is that there is a globally stable symmetric equilibrium. Conversely, if fixations are stable a possibility is that there is an unstable symmetric equilibrium or that the symmetric polymorphism is stable, but is "flanked" by two unstable polymorphisms. In the latter case the domain of attraction to the symmetric equilibrium might be so small as to devoid in practice such equilibrium of any evolutionary significance, because small random fluctuations may easily throw a polymorphic population into the domain of attraction of either fixation.

If we denote by p_i and p_i' (i=1,2,3) the genotypic frequencies of the primary locus among the adults of two successive generations, the recurrence equations linking these variables are

$$p_i'=\frac{W_i}{\overline{W}}q_i,\ i=1,2,3, \qquad (14)$$

where the genotypic frequencies of the intervening newborn stage, q_i, are determined in terms of the p_i's by Eqs (10.1–4) and the fitnesses W_i and $\overline{W}$ are given by Eqs (10.6–9). Near fixation of e_-, where $p_2,p_3\approx0$, with incomplete assortment these equations are approximated by

$$p_2'=(1+\beta)\frac{\theta_{12}+\theta_{21}}{2}p_2+o(p_2),\quad p_3'=o(p_2).$$

Thus, fixation of e_- is stable if

$$(1+\beta)\frac{\theta_{12}+\theta_{21}}{2}<1,$$

where the mating rates, θ_{ij}, are evaluated at $p_1=1$. Computing these rates explicitly according to (8) we conclude that fixation of e_-, and simultaneously that of e_+, is stable if

$$(1+\beta)\frac{2-\upsilon}{2}<1, \qquad (15)$$

when there is no cost of mate choice ($n=\infty$). Thus, stability of fixations is facilitated by high levels of assortment (high υ) and low levels of selection (low β). In fact, without selection fixations would

be stable with any positive amount of assortment. But, if selection is sufficiently strong (β>1) fixations never become stable, irrespective of the strength of assortment. When there is some cost of mate choice, stability of fixations is more easily achieved, and it always occurs if assortment is sufficiently strong, irrespective of the strength of selection.

We have made a numerical survey of the stable equilibria of (14), in case of no cost of mate choice, by iterations of these recurrence equations for a large number of parameter values. We found that the symmetric equilibrium is always present and that it is globally stable whenever the monomorphic equilibria are unstable. Moreover the local stability of such equilibrium does not necessarily vanish when the monomorphic equilibria are stable, that is when (15) is satisfied. Recalling (13.2), this condition may be rewritten in terms of S as

$$S > S_L = \ln\left(\frac{1+\beta}{1-\beta}\right). \qquad (16)$$

The way the size of the domain of attraction to the symmetric equilibrium depends on selection (α,β) and intensity of assortment, S, when (16) is true is illustrated by Table 2. The table shows examples of results obtained from numerical iterations of (14) beginning from a population with a given frequency, $p<0.5$, of allele e_+ (e_-) and genotypic frequencies in Hardy–Weinberg proportions. All such iterations converged either to fixation of e_- (e_+) or to the symmetric equilibrium, depending on the value of p. The relative size of the domain of attraction to the symmetric equilibrium can thus be estimated by $1–2p_{max}$, where p_{max} is the largest value of p such that convergence is to fixation of e_- (e_+). The symmetric equilibrium cannot be reached from any Hardy–Weinberg population and is probably unstable when $p_{max}=0.5$. The fixations are unstable when $p_{max}=0$, which, by (16), occurs when $S\leq S_L$. Table 2 reports values of p_{max} for selected values of {α,β,S}. For any given {α,β}, p_{max} is not monotone with respect to $S\in[S_L,\infty)$. In fact, as S increases, p_{max} first increases up to a maximum and then decreases, apparently going to zero as S goes to infinity. The maximum of p_{max} decreases as α or β increase, and does not reach 0.5 unless both α and β are small. In summary, from this numerical analysis it emerges that a stable symmetric equilibrium with a substantial domain of attraction always exists, unless selection is weak and assortment is neither too weak nor too strong.

TABLE 2

Domain of attraction to fixation of e_- or e_+

β=0.1			β=0.3		
	p_{max}			p_{max}	
S	α/β=0.1	α/β=0.9	S	α/β=0.1	α/β=0.9
0.201	0.0000	0.0000	0.619	0.0000	0.0000
0.202	0.0069	0.0061	0.620	0.0002	0.0002
0.21	0.04	0.03	0.65	0.008	0.006
0.22	0.09	0.07	0.66	0.010	0.007
0.25	0.24	0.16	0.68	0.014	0.010
0.28	0.50	0.25	0.70	0.017	0.013
0.30	0.50	0.28	0.80	0.028	0.019
0.40	0.50	0.41	0.90	0.031	0.020
0.50	0.50	0.35	1.00	0.030	0.018
0.80	0.37	0.17	1.50	0.016	0.007
1.00	0.17	0.10	2.00	0.012	0.002

Note: for the indicated values of the selection parameters, β and α/β, and of the strength of assortment, S, the table shows the largest value, p_{max}, of the frequency of allele e_+ (e_-) such that a population, starting from an Hardy–Weinberg composition with this allelic frequency, converges to fixation of e_- (e_+). Whenever $p_{max}<0.5$, a population with initial frequency, p, such that $p_{max}<p\leq 0.5$ converges to the symmetric polymorphic equilibrium.

4.2. Progress of assortment

We now analyze the conditions under which a resident population at a stable symmetric equilibrium $\mathbf{E}_S$ due to disruptive selection and assortative mating of strength S can be in-

vaded by a mutant mating gene changing the strength of assortment to S'. As demonstrated in § 3, the basic tool for this analysis is the eigenvalue, λ, defined by Eqs 11–12. Given the assumption of (13), this eigenvalue is a function of both S and S' or, equivalently, of $\upsilon = 1-\exp\{-S\}$ and $\upsilon' = 1-\exp\{-S'\}$, so that we may write $\lambda=\lambda(\upsilon,\upsilon')$. Since we are concerned only with mutations of weak effect, such that $\upsilon'\approx\upsilon$, and recalling that $\lambda(\upsilon,\upsilon)=1$, we may approximate λ by

$$\lambda(\upsilon,\upsilon') = 1 + \left.\frac{\partial\lambda(\upsilon,\upsilon')}{\partial\upsilon'}\right|_{\upsilon'=\upsilon}(\upsilon'-\upsilon) + o(\upsilon'-\upsilon).$$

This means that the criterion for invasion by (weak) mutations that increase the strength of assortment is

$$\left.\frac{\partial\lambda(\upsilon,\upsilon')}{\partial\upsilon'}\right|_{\upsilon'=\upsilon} > 0, \qquad (17.1)$$

while the reversed inequality is the criterion for invasion by mutations that reduce the strength of assortment. Considering that, by (12.1), the relation between λ and the largest root, η, of the quadratic equation (12.2) is of the form $\lambda=C\eta$, where the factor C is independent of υ' and is positive, we see that (17.1) reduces to the equivalent condition

$$\left.\frac{\partial\eta(\upsilon,\upsilon')}{\partial\upsilon'}\right|_{\upsilon'=\upsilon} > 0. \qquad (17.2)$$

We shall limit our analysis to the type of assortative mating that has no cost of mate choice ($n=\infty$; Eq. 9) which is mathematically simpler, and is the most favorable to the evolution of assortment as indicated by previous results. In Appendix B we demonstrate that in this case condition (17.2) reduces to the following equivalent criterion

$$\Delta(\upsilon) > 1, \qquad (18.1)$$

where

$$\Delta(\upsilon) = \frac{1+\alpha+\beta(1-q_2)}{1+(\alpha+\beta)(1-q_2)}\left[1+\frac{1}{2}p_2(\theta_{21}-\theta_{12})\right], \qquad (18.2)$$

$$\theta_{21}-\theta_{12} = \frac{1-\upsilon}{p_2+(1-\upsilon)(1-p_2)} - \frac{2(1-\upsilon)}{2(1-\upsilon)p_2+[1+(1-\upsilon)^4](1-p_2)}, \qquad (18.3)$$

and the equilibrium frequencies, q_2 and p_2, of newborn and adult heterozygotes $e_-|\,e_+$, respectively, in the resident population are, by (10), solutions of the following pair of equations

$$q_2 = \frac{1}{2}p_2 + \frac{(1-\upsilon)(1-p_2)p_2+(1-\upsilon)^4(1-p_2)^2}{2(1-\upsilon)p_2+[1+(1-\upsilon)^4](1-p_2)}, \qquad (18.4)$$

$$p_2 = \frac{1+\beta(1-q_2)}{1+(\alpha+\beta)(1-q_2)}q_2. \qquad (18.5)$$

A complete analysis of this invasion criterion would need the explicit general solution of equilibrium equations (18.4–5), which is not feasible because it amounts to finding the roots of a fourth degree algebraic equation. However, by using standard perturbation techniques (e.g. Nayfeh, 1981), we can determine accurate conditions of invasion in the limiting cases of (i) strong assortment ($\upsilon\approx1$), (ii) weak assortment ($\upsilon\approx0$) and (iii) weak assortment vis-à-vis weak selection ($\upsilon,\alpha,\beta\approx0$).

Consider first the case of strong assortment, where $1-\upsilon\approx0$. Our approach will be to determine $\Delta(\upsilon)$ as an expansion in powers of $(1-\upsilon)$,

$$\Delta(\upsilon) = c_0 + c_1(1-\upsilon) + c_2(1-\upsilon)^2 + \ldots,$$

of which it is sufficient to retain only terms of lower power, as long as $1-\upsilon$ is small. To find the sequence of coefficients, $(c_0, c_1, \ldots)$, of this expansion we first have to solve (18.4–5) for (p_2,q_2), again in the form of power expansions in $(1-\upsilon)$. Let

$$p_2 = p^*(\upsilon) = a_0 + a_1(1-\upsilon) + a_2(1-\upsilon)^2 + \ldots,$$

$$q_2 = q^*(\upsilon) = b_0 + b_1(1-\upsilon) + b_2(1-\upsilon)^2 + \ldots,$$

be the solution. Then, if we substitute (p^*,q^*) for (p_2,q_2) in (18.4–5) we obtain a pair of identities, each of which equates two power series in $(1-\upsilon)$, which must be satisfied for all υ. This means that the coefficients of equal powers of $(1-\upsilon)$ on the two sides of each identity must be equal. This condition then provides a pair of equations for each power of $(1-\upsilon)$ that can be solved to obtain the

pairs of coefficients (a_0,b_0), (a_1,b_1), etc. Following this procedure we find that $a_0=b_0=\ldots=a_3=b_3=0$ and

$$p^*(\upsilon)=\frac{2(1+\beta)}{1+2\alpha+\beta}(1-\upsilon)^4+\ldots,$$

$$q^*(\upsilon)=\frac{2(1+\alpha+\beta)}{1+2\alpha+\beta}(1-\upsilon)^4+\ldots.$$

Substituting this solution in (18.2) and expressing the right hand side of this equation as a power series in (1–υ) we finally find

$$\Delta(\upsilon)=1+\frac{\alpha}{1+\alpha+\beta}(1-\upsilon)^4+\ldots\ . \qquad (19)$$

Comparing this result to the criterion (18.1), we immediately conclude that, irrespective of the strength of selection on the primary trait, provided that it is disruptive (α,β>0), only mutants that increase the strength of assortment can invade a resident population in which assortment is already sufficiently strong. In case of weak assortment (υ≈0), proceeding in a similar way to determine Δ(υ) as a power series in υ, we find

$$\Delta(\upsilon)=1+\frac{\alpha}{2+\alpha+\beta}-\left[\frac{(2+\beta)(2+2\alpha+\beta)}{4(2+\alpha+\beta)^2}+\frac{\alpha(1+\alpha+\beta)(2+2\alpha+\beta)^2}{(2+\alpha+\beta)^4}\right]\upsilon+\ldots\ . \qquad (20)$$

Hence, for any fixed (α,β) provided that α,β>0, Δ(υ)>1 if assortment is weak enough, so that only mutants that increase the amount of assortment can invade.

These two results extend to the respective neighborhoods the invasion properties that in § 3 were demonstrated to hold at the two extremes of assortment level, υ=1 and υ=0 (Result 3), suggesting that under disruptive selection assortment always tends to increase in strength. This is not true, however, because in (20) we can observe that if α is let tend to zero for any fixed υ, then Δ(υ) becomes less than one. Indeed, if we expand Δ(υ), as given by (20), in power series also with respect to α and β, retaining only terms linear in α, β and υ, it results that for weak selection and weak assortment

$$\Delta(\upsilon,\alpha,\beta)=1+\frac{1}{2}\alpha-\frac{1}{4}\upsilon+\ldots, \qquad (21)$$

showing that, for small α, β and υ, Δ is greater than one only if υ<2α, approximately. This equation, together with (19) proves the following

RESULT 4. At least for weak selection there are two thresholds (singularities) in the evolution of assortment, $\upsilon_0(\alpha,\beta)$ and $\upsilon_1(\alpha,\beta)$, where $\Delta(\upsilon_0)=\Delta(\upsilon_1)=1$, and such that all levels of assortment, υ, in the ranges $0\le\upsilon<\upsilon_0$ and $\upsilon_1<\upsilon<1$ are invaded only by mutants that increase assortment. Of the levels of assortment in the remaining middle range, $\upsilon_0<\upsilon<\upsilon_1$, at least those that are close to υ_0 are invaded only by mutants that decrease assortment. For small α and β, $\upsilon_0(\alpha,\beta)\approx 2\alpha$.

This result is obviously quite important because it implies that, at least when disruptive selection is weak, evolution of assortative mating through mutations of small effect cannot carry a population all the way from random mating to sympatric speciation. Some degree of assortment can indeed accumulate in all cases, but for weak selection it cannot progress beyond the rather low level υ_0. It is therefore essential to ascertain whether a sufficiently high level of selection removes such limitation, allowing sympatric speciation to evolve. There is also some interest in refining the above result with respect to the behavior of Δ(υ) in the middle range (υ_0,υ_1), to verify in particular whether assortment tends to diminish [i.e. Δ(υ)<1] in the whole interval, or whether more complex behaviors [e.g. other roots of Δ(υ)=1] occur. We have obtained partial answers to these questions by solving numerically for many values of (α,β) the equation Δ(υ)=1 in the interval 0<υ<1, which of course requires simultaneously solving equations (18.4–5). Some examples of the results are reported in Table 3 which, for selected values of (α,β), shows the values of $S=-\ln(1-\upsilon)$ that solve $\Delta(\upsilon)=\Delta\left(1-e^{-S}\right)=1$.

As illustrated in Table 3, if selection is sufficiently weak (β or α/β small) there are exactly two thresholds, so that assortment tends to decrease anywhere in the middle range (υ_0,υ_1). As selection is made stronger this interval becomes narrower

because υ_0 increases and υ_1 decreases. When β or α/β are large enough the two thresholds merge and disappear. Hence, for sufficiently strong selection, increasing assortment is always favored irrespective of the current degree of assortative mating in the population.

Comparing these numerical results about the roots of $\Delta(\upsilon)=1$ with those about the stability of the symmetric equilibrium discussed above (cf. Table 2) it can be seen that the symmetric equilibrium is always stable at the parameter points, $\{\alpha,\beta,\upsilon\}$, where invasion is by mutants that increase assortment [i.e. $\Delta(\upsilon)>1$]. For example, for $\alpha/\beta=\beta=0.1$ we see from Table 3 that invading mutants increase assortment whenever $0<S<0.02$ or $S>0.855$, and from Table 2 we see that in the first of these ranges the symmetric equilibrium is globally stable (both fixations are unstable), while in the second it is locally stable with a reasonably large domain of attraction ($p_{max}>0.37$).

We made some numerical test to verify whether the symmetric equilibrium, besides being stable, is also an ESS with respect to primary trait mutants at points, $\{\alpha,\beta,\upsilon\}$, of the parameter space where $\Delta(\upsilon)>1$. The tests were made by iterating the recurrence equations for the genotypic frequencies of a population at a symmetric equilibrium which had been contaminated by a small frequency of a mutant allele of the primary locus with effect chosen at random in $\left(-\frac{1}{2},\frac{1}{2}\right)$. For each given point of the parameter space a thousand random mutants were checked. The results suggest that the symmetric equilibrium is indeed a polymorphic ESS of the primary trait wherever assortment tends to increase. In fact in all cases where $\Delta(\upsilon)>1$ the mutant allele disappeared from the population and the original symmetric equilibrium was restored. Interestingly, we also found cases where the symmetric equilibrium did not have the ESS property, and this occurred when $\Delta(\upsilon)<1$, that is when a decrease of assortment was favored.

TABLE 3

Thresholds of assortment where evolution changes direction

	α/β				
β	0.1	0.3	0.5	0.7	0.9
0.1	0.020	0.060	0.105	0.155	0.220
	0.855	0.805	0.750	0.690	0.620
0.3	0.055	0.190	–	–	–
	0.815	0.660			
0.5	0.090	–	–	–	–
	0.780				
1.0	0.150	–	–	–	–
	0.720				
2.0	0.250	–	–	–	–
	0.635				
4.0	0.410	–	–	–	–
	0.495				
4.2	–	–	–	–	–

Note: for given pairs of β and α/β the table shows the roots of $\Delta\left(1-e^{-S}\right)=1$ for $S>0$. Dashes indicate that for the given (α,β), $\Delta>1$ for all $S>0$, so that only mutants that increase assortment invade.

4.3. Fixation of invading mutants of assortment

Let υ, υ' and υ'' be the levels of assortment associated respectively to genotypes $m|m$, $m|m'$ and $m'|m'$ of a mating gene of which m is the resident allele and m' is a mutant that has invaded, and suppose that $\Delta(\upsilon)>1$. Under the assumption that mutations have small effect both υ' and υ'' are very close to υ, and $\upsilon'>\upsilon$ because m' was able to invade. If partial dominance prevails in the expression of mating genes, the heterozygote phenotype, υ', is between the phenotypes of the two homozygotes, so that $\upsilon<\upsilon'<\upsilon''$. Hence, if m' were the resident allele m would not be able to invade it if $\Delta(\upsilon'')>1$, while such invasion would occur if $\Delta(\upsilon'')<1$. In other words, in the short-term dynamics of the two alleles m and m', fixation of m is unstable and fixation of m' is stable in the first case, while both fixations are unstable in the second. It is then certain that for $\Delta(\upsilon'')<1$ a polymorphism of resident and mutant allele is maintained. On the other hand, it is plausible, although by no means certain in such a complex selection

system, that for $\Delta(\upsilon'')>1$ there is no polymorphism of m and m', and that invasion by m' is followed by its fixation.

Based on these considerations we could expect that, with partial dominance of mating genes, every invasion is followed by fixation of the mutant allele at all assortment levels $\upsilon\in[0,1)$ when the disruptive selection parameters, $\{\alpha,\beta\}$, are so large that $\Delta(\upsilon)>1$ anywhere in [0,1) (see Table 3). When the strength of selection is small enough that the threshold $\upsilon_0(\alpha,\beta)$ exists, fixation of invading alleles can be expected to occur for assortment levels $\upsilon\in[0,\upsilon_0)$ only if υ is far enough from υ_0 that $\upsilon''<\upsilon_0$. But when υ is in the vicinity of υ_0 and a mutation appears such that $\upsilon<\upsilon_0<\upsilon''$, a polymorphism of resident and mutant allele is established.

These conjectures have been completely confirmed in a large number of numerical tests that we performed by iterations of the exact recurrence equations of the genotypic frequencies for the two-locus system with the two alleles (e_-,e_+) at the primary locus and a pair of alleles, (m,m'), at a mating locus. The mutant allele m' was introduced at the frequency of 10^{-5} in a population that had reached the one-locus symmetric equilibrium under assortative mating of level $S=-\ln(1-\upsilon)$ due to the resident homozygotes, $m|m$. The levels of assortment due to mutant heterozygotes, $m'|m$, and homozygotes, $m'|m'$, were measured by $S'=-\ln(1-\upsilon')$ and $S''=-\ln(1-\upsilon'')$ respectively, and S' was chosen in agreement with the assumption of partial dominance and such as to ensure that m' would invade m. After introduction of the mutation, iterations were ended when either the distance between genotypic frequencies in two consecutive generations had become less than 10^{-14} – indicating attainment of an equilibrium – or generation number 300,000 had been reached. If at the end of iterations the frequency of the mutant allele m' was larger than 0.99 we assumed that the mutation would go to fixation. In some cases evolution could have been so slow that at the end of iterations the frequency of m' was still below the threshold of 0.99, although its fixation would occur eventually. To control for this possibility, so as to distinguish it from genuine polymorphism, we made a second series of iterations starting at the same one-locus equilibrium as in the first series but with the initial frequency of m' set equal to 0.99. When these iterations ended by the same criterion as before, it was assumed that a stable polymorphism of resident and mutant allele existed if the frequency of m' was now less than its initial frequency of 0.99. Otherwise it was assumed that the mutation would go to fixation.

The numerical tests were made for each of the 48 combinations of four values of α/β (0.1, 0.2, 0.5, 1), six values of β (0.1, 0.5, 1, 2, 4, 5) and two values (0.05, 0.5) of the rate of recombination, r, between the primary locus and the mating locus. For each pair $\{\alpha,\beta\}$ such that $\Delta(\upsilon)>1$ everywhere in [0,1), five sets of mating phenotypes, $\{S,S',S''\}$, were tested with the two recombination values. Each set was chosen at random subject to the following constraints:

$$0<S<1\ ,\ S+0.01<S''<S+0.1,$$

$$S+0.01<S'<S''.$$

These were the tests of type I. For each pair $\{\alpha,\beta\}$ where the threshold $S_0=-\ln[1-\upsilon_0(\alpha,\beta)]$ exists, five sets of mating phenotypes for tests of type II and five for tests of type III were used with the two values of r. These ten sets were generated at random subject to the following constraints:

$$S_0-0.02<S<S_0-0.01,\ S+0.001<S'<S'',$$

for both types of tests and

$$S+0.001<S''<S_0-0.001,\ \text{for type II,}$$

$$S_0+0.001<S''<S_0+0.02,\ \text{for type III.}$$

In total 150 tests of type I and 90 tests each of types II and III were made. In agreement with our conjecture all tests of types I and II ended with fixation of the mutant allele, while all tests of type III ended with polymorphism of the resident and mutant alleles.

4.4. Long-term evolution of assortative mating

We have thus provided analytical proofs or numerical evidence in favor of all the elements required to validate the third phase of the long-term evolutionary process outlined at the beginning of this section, uncovering meanwhile the important

qualification that unlimited progress of assortative mating to sympatric speciation can only occur if disruptive selection is strong enough. To summarize the findings of this section we can therefore state the following:

RESULT 5 (based in part on numerical evidence). If the strength of disruptive selection – as measured by $\{\alpha,\beta\}$ – is sufficiently high, and if mate choice can occur without cost, assortative mating for the selected trait emerges in a random mating population and becomes progressively stronger, through a sequence of invasions and fixations of mutations of small effect, until complete reproductive isolation between the two extreme morphs in the population is achieved, effectively concluding a process of sympatric speciation. In the course of this process, with respect to the primary trait the population remains in a state of symmetric polymorphic equilibrium, $\mathbf{E}_S$, which changes with continuity, from the initial state $\mathbf{E}_0$ to the final state $\mathbf{E}_\infty$, as the strength of assortment, S, increases from zero to infinity. In particular the phenotypic variance increases monotonically from $\frac{1}{2}$ to 1. Every equilibrium $\mathbf{E}_S$ is stable in the short-term dynamics and cannot be invaded by any allele of the primary locus different from the resident alleles (e_-,e_+). On the other hand, if the strength of disruptive selection is too small, the process just described does not proceed to complete isolation because there exists a threshold $S_0(\alpha,\beta)$ such that, when the increasing strength of assortment S reaches its vicinity, a stable polymorphism of assortment levels, both smaller and larger than S_0, arises in the population.

How evolution will further proceed in the long term, once the vicinity of S_0 has been reached, depends on the nature of this singular point, which is determined by the second derivative (with respect to υ') of the dominant eigenvalue, $\lambda(\upsilon_0,\upsilon')$, of the invasion dynamics for a resident population with assortment level $S_0 = -\ln(1-\upsilon_0)$. This is difficult to analyze because υ_0 cannot be computed exactly. However, numerical calculations of $\lambda(\upsilon,\upsilon')$, for υ very close to υ_0 and for a wide range of values of υ', indicate that $\lambda(\upsilon_0,\upsilon')=1$ for all υ'. In this case then no mutant can actually invade S_0, but some might persist in the population as rare neutral variants. This means that S_0 is a "weak" ESS, an evolutionary singularity of a kind that is well known in several adaptation problems. For example, the 1:1 sex allocation ratio of classical sex-ratio theory is an ESS of this type (e.g. Charnov, 1982; Karlin and Lessard, 1986). We may then conclude that if disruptive selection is not strong enough, evolution of assortment through mutations of weak effect, starting from random mating cannot progress to complete isolation and stops at the moderate ESS level S_0.

5. Discussion

Our primary intent in this article was to examine the long-term consequences, in a sexually reproducing species, of the regime of disruptive selection on a continuous trait characterized by that particular type of evolutionary singularity – named PEAST (Christiansen, 1991) or "evolutionary branching point" (Geritz et al., 1998) – which, being continuously stable (convergence stable), is an evolutionary attractor for monomorphic populations but at the same time can be invaded by any mutation. In this inquiry we addressed the case where the genetic control of the selected trait is additive. The non-additive case, which behaves very differently, will be examined in another paper (Matessi and Gimelfarb, 2002, in preparation). To permit a mathematical analysis, we simplified the problem by the assumption that variation of the trait is determined by a single locus and is confined to the finite interval [–1,1]. Moreover, we assumed a simple frequency-dependent selection model that approximates, in the vicinity of the singularity at $x^\circ=0$, the many diverse and complex systems of ecological interactions that give rise to a PEAST singularity, including scramble competition (Christiansen and Loeschcke, 1980; Christiansen, 1991; Doebeli, 1996; Dieckmann and Doebeli, 1999) and habitat heterogeneity (Geritz et al., 1998; Kisdi and Geritz, 1999).

The first and most characteristic consequence of this selection regime is not only the emergence of genetic polymorphism, described in Christiansen and Loeschcke (1980) and in Christiansen (1991), but also, and more specifically, the likely attainment of a polymorphic long-term equilibrium (polymorphic ESS) as demonstrated in Kisdi and

Geritz (1999) and in this article. Thus, we could say that MEAST – or continuously stable ESS – and PEAST type singularities are pointers, respectively, to *monomorphic* and *polymorphic* long-term equilibria. There is, however, a major difference between the two cases. While in case of a MEAST the equilibrium and its stability are direct consequences of the local properties of selection at the singularity, in case of PEAST the very existence of a polymorphic long-term equilibrium depends critically on the global properties of the fitness landscape. For example, in our model the equilibrium is unique while in the model of Kisdi and Geritz (1999) there may be up to three equilibria although both models entail a single PEAST. The examples of extinction of evolutionary branches found by Geritz et al. (1999) in a model with clonal reproduction indicate that with sexual reproduction there could be cases where the buildup of polymorphism around a PEAST is only transient and is followed, for example, by attainment of a monomorphic equilibrium at a MEAST.

The presence of polymorphism renders the mathematical analysis of long-term evolution near a PEAST rather difficult, even with single locus genetics as assumed in this article and in Kisdi and Geritz (1999). The difficulties would be forbidding with multilocus genetics. From the point of view of the method of analysis of polymorphic ESS there is an important difference between our approach and that of Kisdi and Geritz (1999). We assume that, irrespective of the state of the population, a mutation of the primary locus can produce any one of the alleles that are possible for this locus. Hence, at any time the effect of a mutant allele can be any value in the interval $\left[-\frac{1}{2},\frac{1}{2}\right]$ because this is the set of all possible allelic effects in our model. It follows that for us a long-term equilibrium (ESS) is a population state that cannot be invaded by any of the alleles with effect in $\left[-\frac{1}{2},\frac{1}{2}\right]$. Kisdi and Geritz (1999) make a very different assumption with respect to which mutations are possible at any given moment. In fact they assume that the effect of a mutant allele is always close to the effect of the resident allele from which the mutation originally arose. Thus if **E**, say, is the set of effects of all possible alleles of the primary locus and if, for example, e_1 and e_2 are the effects of the resident alleles at a certain moment, then the effect of any mutant that could appear in the population at this moment cannot be an arbitrary element of **E** but only a value sufficiently close to either e_1 or e_2. It follows that, according to Kisdi and Geritz, a polymorphism with the pair of alleles (e_1,e_2) is an ESS if and only if it cannot be invaded by any allele e that is close either to e_1 or to e_2. This difference means that what for Kisdi and Geritz is a necessary and sufficient condition of ESS, from our point of view is just a necessary condition. From a biological point of view, and if we were to measure distances between alleles, for example, by the number of different nucleotides between the corresponding DNA sequences, the assumption of Kisdi and Geritz would be entirely justified, because generally more than one mutation event separates two homologous sequences that differ at more than one nucleotide site. Unfortunately, it is not generally true that distances in this molecular metric correlate with the distances at the level of expressed phenotype.

At the unique polymorphic ESS that exists in our model a half of the population consists of heterozygotes which, because of their intermediate phenotype, x=0, have the lowest fitness. The homozygotes that make up for the rest are evenly distributed among the two extreme and most fit phenotypic values, $x = -1$ and $x = 1$. It is then natural to expect that in such a situation assortative mating for the selected trait will be advantageous and that progressive reinforcement of this mating pattern by natural selection will result in complete reproductive isolation between the two extreme types with disappearance of the intermediate heterozygotes. This, of course, is the classical scenario of sympatric speciation about which a rich theoretical literature has accumulated, probably beginning with the paper by Maynard Smith (1966). A large majority of these studies belong to the class of the "two-allele" models as defined by Felsenstein (1981). Their common feature is that evolution of assortment for the selected trait is conceived as resulting from the establishment of a strong correlation – due to linkage disequilibrium – between the selected trait and an initially independent trait for which a given level of assortative mating already existed in the population for some unspecified reason. The general result of this approach is that assortment for the selected trait is established only if the strength of disruptive selec-

tion is above a certain threshold, that decreases as the preexisting level of assortment for the other trait increases (e.g. Maynard Smith, 1966; Udovic, 1980; Felsenstein, 1981; Dieckmann and Doebeli, 1999; Kondrashov and Kondrashov, 1999; Geritz and Kisdi, 2000). According to Felsenstein (1981), in this class of models, speciation through evolution of assortment is more difficult to achieve than in the other class identified by him, that of the "one-allele" models, to which our model also belongs. Evolution of assortment in this case is conceived as resulting from the accumulation of modifiers of mating behavior that induce and reinforce direct assortment for the selected trait (e.g. Maynard Smith, 1966; Doebeli, 1996; Dieckmann and Doebeli, 1999). Probably the reason why models of the first type have been preferred that they were considered biologically more plausible (Maynard Smith, 1966). This opinion may be correct in cases where the primary trait subject to disruptive selection measures biochemical or physiological activities which cannot be perceived by potential mates. But there is no compelling reason to question the possibility of direct assortative mating for the primary trait if it reflects aspects of external morphology which at the same time are directly involved in ecological interactions, as for example body size, or bill size in birds, which often are strongly related to the trophic niche (Hutchinson, 1959; MacArthur, 1972; Cody, 1974).

In theoretical studies of sympatric speciation through assortative mating it is generally assumed that mate choice occurs without cost. However, mating preferences could significantly prolong the time required to find a mate, particularly if the preferred type is rare. Thus there may be cases in which, due to, for example, low population density or short-mating season, the probability of mating and reproducing is substantially reduced by a strong preference or by the rarity of the preferred mate. The assortative mating model of Gavrilets and Boake (1998) that we adopted accounts for the possibility of such effects through the parameter n, representing the maximum number of males that a female is allowed to encounter while searching for a mate. Although we analyzed progress of assortment only in case of no cost ($n=\infty$), other results that we obtained indicate that evolution of assortment is more difficult if mate choice has a cost. Thus, when $n=1$ assortment cannot invade random mating, and complete assortment is invaded by a reduction of its intensity. Hence, we do not expect any evolution of assortative mating in cases where females can mate only by accepting the first male they encounter. Moreover, our stability analysis of the equilibria of the primary trait under assortment indicates that stable polymorphism – obviously a prerequisite for any progress of assortment – is less likely to occur if $n<\infty$.

When there is no cost of mate choice ($n=\infty$), or even if such cost is moderate ($1<n<\infty$), our model agrees in some respects with the expectations implied by Felsenstein's classification. First of all, the rate of recombination between the primary locus and the mating modifiers has no influence on the evolution of assortment. Second, there is no threshold amount of selection necessary for inception of assortative mating. Provided that selection is disruptive, modifiers inducing some assortment for the selected trait can always invade a random mating population. If $n=\infty$, the same is true when assortment is already strong because, irrespective of the strength of disruptive selection, mutants that further increase the amount of assortment can always invade, and complete isolation cannot be destroyed by invasion of modifiers that reduce the amount of assortment. Thus, if mate choice is not costly, complete isolation is a continuously stable ESS of mating behavior at any level of disruptive selection. If cost of mate choice is moderate, complete isolation is still an ESS whenever coexistence of the two extreme types is stable, but such stability occurs only if selection is sufficiently strong.

The concordance with the expected properties of Felsenstein's "one-allele" models however vanishes, even in case $n=\infty$, with regard to the crucial question of whether sympatric speciation (complete assortment) can evolve starting from a random mating population. In this respect, our result rather resembles that of Felsenstein's "two-allele" models because we find that an increase of the strength of assortment is favored at all assortment levels, from random mating to complete isolation, only if the disruptive selection parameters, α and β, are large enough. If α and β are small, evolution of assortment changes direction at two thresholds, S_0 and S_1, of the strength of assortment $S\in[0,\infty)$: assortment increases when it is either very weak, $0\le S<S_0$, or very strong, $S>S_1$, but it decreases when its strength is intermediate, $S_0<S<S_1$. Thus, starting

from random mating (S=0), assortment does indeed increase but it cannot progress, through mutations of small effect, beyond the rather modest ESS level S_0=$S_0(\alpha,\beta)$ (see Table 3), where its evolution comes to an end.

An apparently similar behavior is briefly reported by Doebeli (1996), among the results of a numerical study of sympatric speciation due to direct assortative mating for a continuous trait subject to disruptive selection, caused by scramble competition. The genetics of the trait is simulated by an hypergeometric model of segregation for twenty additive, haploid and unlinked loci. The rather complex selection model has many parameters, none of which is indicated as related to strength of selection. However, the parameter whose qualitative effects on the evolution of assortative mating are described is the variance of the competition function, which is Gaussian as in the disruptive selection model used by Christiansen and Loeschcke (1980). Hence, from the equivalence of the latter with our model, we might infer that such parameter is inversely related to our selection parameters, $\{\alpha,\beta\}$. Doebeli finds that if the variance of the competition function is high (weak selection), starting from random mating the degree of assortment evolves to a relatively moderate equilibrium value, but it evolves to a much higher level if initially assortment was already intense. On the other hand, if competition is sufficiently loose (low variance, strong selection) the lower equilibrium level disappears so that very strong assortment can evolve starting from random mating. It seems clear that these numerical results of Doebeli reflect the two thresholds, S_0 and S_1, of which we proved here the existence with weak selection and the disappearance with strong selection.

In a recent individual-based simulation study of sympatric speciation due to disruptive selection, Dieckmann and Doebeli (1999) do not report any evidence of the threshold effect of selection intensity on the evolution of assortative mating, in case of direct assortment for the selected trait. This is surprising because their model of mating is the same as that used by us (with n=∞) and by Doebeli (1996), and their representation of disruptive selection due to scramble competition is even closer to ours than that of Doebeli (1996), because it consists of a Lotka–Volterra competition model with Gaussian resource spectrum and Gaussian competition function, exactly as in Christiansen and Loeschcke (1980). In fact it appears that in these simulations almost complete isolation between two extreme types is achieved provided only that the variance of the resource spectrum is greater than the variance of the competition function. From the equivalence of the model of Christiansen and Loeschcke with ours we can see that this is simply the condition that α>0, or that selection be disruptive. On the other hand, in accordance with the general behavior of the "two-allele" models of Felsenstein's classification, in the simulations dealing with the case of assortment directed to a neutral marker trait, reproductive isolation is attained only if the variance of the competition function is less than some low threshold, which, in the terms of our model, is equivalent to the requirement that both α and β be sufficiently large. It is likely that the apparent discrepancy between the simulations of Dieckmann and Doebeli (1999) and our result on the existence of the two thresholds, S_0 and S_1, under weak selection can be explained by the fact that our analysis, as well as the numerical study of Doebeli (1996), assumes mutations of mating loci with a very small effect and considers only invasions by one mutation at the time. But these assumptions do not really apply to the simulations of Dieckmann and Doebeli (1999) because there the genetic assumptions were such that, starting from random mating, only five mutation events on different loci were sufficient to produce an haplotype that, in homozygote condition, would code for essentially complete assortment, while mutation rate, population size and number of loci were such that about 10 mutations occurred each generation, on average. It seems quite possible that under these conditions, simply by the pressure of mutation and genetic drift, a population could in a few generations, so to speak, jump over the region $[S_0,S_1]$, where an increase of assortment is not permitted, thereby landing in the basin of attraction toward complete assortment.

Acknowledgements

This work was partially financed by "Progetto Strategico: Metodi e Modelli Matematici per lo Studio dei Fenomeni Biologici" of the CNR. SG was also supported by the National Institute of Health grant GM56693.

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APPENDIX

A)

In this appendix we compute the eigenvalues of the transition matrix **Q** of the invasion dynamics of mating modifiers, defined by (11).

Let **P** be the 4×4 matrix defined as $\mathbf{P}=\frac{M\overline{W}}{W_1}\mathbf{Q}$. Thus, if ω is an eigenvalue of **P**, $\frac{W_1}{M\overline{W}}\omega$ is an eigenvalue of **Q**. Let $\mathbf{z}=(z_1,z_2,z_3,z_4)^{\mathrm{T}}$ be the (right) eigenvector of **P** associated to ω. By definition, **z** and ω must satisfy the system of linear equations $\omega\mathbf{z}=\mathbf{P}\mathbf{z}$, which, by (11), corresponds explicitly to

$$az_1+rbz_2+(1-r)bz_3=\omega z_1, \tag{A.1}$$

$$h(1-r)bz_2+hrbz_3+hcz_4=\omega z_2, \tag{A.2}$$

$$hcz_1+hrbz_2+h(1-r)bz_3=\omega z_3, \tag{A.3}$$

$$(1-r)bz_2+rbz_3+az_4=\omega z_4. \tag{A.4}$$

Summing (A.1) to (A.4) and (A.2) to (A.3) we get the two equations

$$z_2+z_3=\frac{\omega-a}{b}\,(z_1+z_4)\,,\ z_2+z_3=\frac{hc}{\omega-hb}\,(z_1+z_4),$$

which are compatible if and only if either

$$\frac{\omega-a}{b}=\frac{hc}{\omega-hb}, \tag{A.5}$$

or

$$z_2+z_3=z_1+z_4=0. \tag{A.6}$$

Condition (A.6) cannot apply to the eigenvector associated to the dominant eigenvalue of **P** since such eigenvector must be strictly positive because **P** is irreducible (for $c>0$). Hence the dominant eigenvalue of **P** must satisfy (A.5). This condition is equivalent to the following quadratic equation in ω

$$\omega^2-(a+hb)\omega+hb(a-c)=0, \tag{A.7}$$

which is the same as (12.2). The two distinct real roots of such equation provide two of the eigenvalues of **P**. The largest of these in absolute value, which is positive, is the dominant eigenvalue of **P**.

If **z** satisfies (A.6), then (A.1) and (A.3) reduce, respectively, to

$$z_1=\frac{(1-2r)b}{a-\omega}z_2,\ \ z_1=\frac{(1-2r)hb-\omega}{hc}z_2\,,$$

which are compatible if and only if $\frac{(1-2r)b}{a-\omega}=\frac{(1-2r)hb-\omega}{hc}$. This condition is equivalent to the following quadratic equation in ω

$$\omega^2-[a+hb(1-2r)]\omega+hb(1-2r)(a-c)=0,$$

which provides the two remaining eigenvalues of **P**.

B)

Let λ be the dominant eigenvalue of the transition matrix **Q**, defined in (11) and discussed in Appendix A. Then, as we know, $\lambda=\frac{W_1}{M\overline{W}}\eta$, where η is the largest in absolute value of the two real roots of the quadratic equation given in (12.2) and in (A.7), namely

$$\omega^2-(a+hb)\omega+hb(a-c)=0\,, \tag{B.1}$$

where the parameters a, b, c and h are defined in (11.2) and (10). Under the assumptions of (13), η is a function, $\eta=\eta(\upsilon,\upsilon')$, of the strength of assortment in the resident population, $\upsilon\in[0,1]$, and of the strength of assortment, $\upsilon'\in[0,1]$, due to a mutation of a mating gene. For $\upsilon'-\upsilon=\varepsilon\approx 0$, we are interested in the expansion

$$\eta=\eta_0+\eta_1\varepsilon+o(\varepsilon). \tag{B.2}$$

In particular, for the case of assortment with no cost of mate choice ($n=\infty$), we want to compute $\eta_1=\left.\frac{\partial\eta(\upsilon,\upsilon')}{\partial\upsilon'}\right|_{\upsilon'=\upsilon}$ and to prove that $\eta_1>0$ is equivalent to (18).

We know that $\lambda=1$ if $\upsilon'=\upsilon$. Thus, recalling that $M=1$ for $n=\infty$, we immediately have that

$$\eta_0=\eta(\upsilon,\upsilon)=\frac{\overline{W}}{W_1}. \qquad \text{(B.3)}$$

To determine η_1 from (B.1) we will use simple perturbation techniques (e.g. Nayfeh, 1981). Thus, the first step is to represent the coefficients of (B.1) as power series expansions in $\varepsilon=\upsilon'-\upsilon$. It is clear from (11.2) that the parameter h is independent of υ'. With no cost of mate choice, a case in which (9) is true, b is also independent of υ'. In fact, by (11.2), recalling that $p_1=p_3$ because the resident population is at a symmetric equilibrium and that $\theta'_{21}=\theta'_{23}$, by (10.10), we have

$$b=\frac{1}{4}\left[\left(2\theta_{12}p_1+\theta_{22}p_2\right)+\left(2\theta'_{21}p_1+\theta'_{22}p_2\right)\right]=$$

$$=\frac{1}{4}\left[\left(2\theta_{12}p_1+\theta_{22}p_2\right)+\left(\theta'_{21}p_1+\theta'_{22}p_2+\theta'_{23}p_3\right)\right]=$$

$$=\frac{1}{4}\left(2\theta_{12}p_1+\theta_{22}p_2+1\right).$$

For analogous reasons we also have that

$$a+c=\frac{1}{2}\left(\theta_{11}p_1+\theta_{21}p_2+\theta_{13}p_3+1\right)=$$

$$=1+\frac{1}{2}\left(\theta_{21}-\theta_{12}\right)p_2, \qquad \text{(B.4)}$$

so that $\frac{\partial(a+c)}{\partial\upsilon'}=0$ and therefore $\frac{\partial a}{\partial\upsilon'}=-\frac{\partial c}{\partial\upsilon'}$. From these observations it follows that (B.1) can be written as

$$\omega^2-(a_0+hb+a_1\varepsilon)\omega+hb(a_0-c_0+2a_1\varepsilon)+o(\varepsilon)=0, \qquad \text{(B.5)}$$

where

$$a_0=a(\upsilon,\upsilon),\ c_0=c(\upsilon,\upsilon),\ a_1=\left.\frac{\partial a(\upsilon,\upsilon')}{\partial\upsilon'}\right|_{\upsilon'=\upsilon}. \qquad \text{(B.6)}$$

Since $\omega=\eta$ is a root of (B.5), substituting (B.2) for ω in this equation produces the following equations for η_0 and η_1

$$\eta_0^2-\left(a_0+hb\right)\eta_0+hb\left(a_0-c_0\right)=0, \qquad \text{(B.7)}$$

$$\eta_1=a_1\frac{\eta_0-2hb}{2\eta_0-a_0-hb}. \qquad \text{(B.8)}$$

Of the two roots of (B.7) we have to take the largest in absolute value, which is given by (B.3). With this choice for η_0, from (B.8) we obtain for η_1

$$\eta_1=a_1\frac{\overline{W}-2W_2b}{2\overline{W}-W_1a_0-W_2b}. \qquad \text{(B.8)}$$

It is clear that $a_1>0$. In fact, by (11.2), we have

$$\frac{\partial a}{\partial\upsilon'}=\frac{1}{4}\frac{\partial}{\partial\upsilon'}\left(\theta'_{11}p_1+\theta'_{12}p_2+\theta'_{11}p_3\right)=$$

$$=\frac{1}{4}p_3\frac{\partial}{\partial\upsilon'}\left(\theta'_{11}-\theta'_{13}\right)$$

which is positive because, obviously, $\theta_{11}-\theta_{13}$ increases with the strength of assortment, υ.

By taking into account equations (10.1), (10.2), (10.5), (10.10), (11.2) and (B.6), and by recalling that $M=1$ and $p_1=p_3$, we can easily deduce the following relations

$$2a_0p_1+bp_2=2\frac{\overline{W}}{W_1}p_1\ ,\ 2c_0p_1+bp_2=\frac{\overline{W}}{W_2}p_2,$$

which, with simple rearrangements, become, respectively

$$2p_1\left(\overline{W}-W_1a_0\right)=W_1bp_2, \qquad \text{(B.9)}$$

$$p_2\left(\overline{W}-W_2b\right)=2W_2c_0p_1. \qquad \text{(B.10)}$$

These two relations show that the left-hand side of each of them is a positive quantity, so that the denominator in the expression for η_1 given in (B.8) is also positive. Hence, η_1 can be written as

$$\eta_1=C'\left(\overline{W}-2W_2b\right), \qquad \text{(B.11)}$$

where C' is a positive quantity. Moreover, if we multiply (B.9) by (B.10) we obtain

$$\left(\overline{W}-W_1a_0\right)\left(\overline{W}-W_2b\right)=W_1W_2bc_0,$$

which can be solved for b, giving

$$b = \frac{\overline{W}}{W_2} \frac{\overline{W} - W_1 a_0}{\overline{W} - W_1 a_0 + W_1 c_0}.$$

Note that, again by (B.9), the denominator on the right-hand side of this equation is positive. Thus, when we substitute this expression for b in (B.11) we obtain that

$$\eta_1 = C\left[\frac{W_1}{\overline{W}}(a_0 + c_0) - 1\right],$$

where C is a positive quantity. Hence, using (B.4) and (B.6), we conclude, in agreement with (18), that

$\eta_1 > 0$ if and only if

$$\Delta(\upsilon) = \frac{W_1}{\overline{W}}\left[1 + \frac{1}{2}(\theta_{21} - \theta_{12})p_2\right] > 1.$$