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FREQUENCY-DEPENDENT SELECTION, DISRUPTIVE SELECTION, AND THE EVOLUTION OF REPRODUCTIVE ISOLATION

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Mechanisms for the evolution of reproductive isolation in natural populations have been the subject of considerable controversy. The view that allopatric speciation is essentially the only mechanism for gradual species formation (Mayr 1947, 1963) has been challenged in recent years by both theoreticians (Basykin 1965; Maynard Smith 1966; Dickinson and Antonovics 1973; Endler 1977; Caisse and Antonovics 1978; Rosenzweig 1978; Gibbons 1979 and laboratory population biologists (e.g., Thoday and Gibson 1962, 1970; Soans et al. 1974; Hurd and Eisenberg 1975), as well as by examples of probable sympatric speciation events, particularly among insects (Bush 1969, 1975; Knerer and Atwood 1973; Richardson 1974; Phillips and Barnes 1975; Khasimuddin and DeBach 1976; Tauber and Tauber 1977; Gibbons 1979).

Despite its importance, mathematical theory of the evolution of reproductive isolation is still in the early stages of development. Maynard Smith (1966) used models of populations occupying two niches to demonstrate that multiple-niche polymorphisms could result in reproductively isolated populations through disruptive selection. His results were extended by Dickinson and Antonovics (1973), who used computer simulation to show that the degree of reproductive isolation depends on the intensity of disruptive selection and the amount of gene flow between niches.

Further development and analysis of mathematical models of the evolution of reproductive isolation are difficult because of the inherent complexity of the process. Most evolutionary models start out with a set of genotypic frequencies as state variables of a dynamic system and an algorithm or a system of equations is constructed which determines genotypic frequencies at some later time. The goal of analysis of such a dynamic system is to predict the future behavior of the state variables from a knowledge of the parameters or control variables. However, because of the large number of state variables and parameters involved in speciation models, analysis is at best cumbersome and difficult and results are difficult to interpret biologically.

In this paper, I report on the construction and analysis of models designed to explore the theoretical potential for the evolution of reproductive isolation via disruptive selection. As is often useful in analysis of models of complex systems, emphasis has been placed on only a few variables and parameters which are of

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primary interest. Analysis has also been simplified by the use of indices, which are functions of the state of the system or of the parameters, but which are more easily interpreted and measured than the state variables and parameters. These indices may be divided into two classes: *state indices*, which are defined solely in terms of the state variables and which provide useful measures of certain aspects of the state of the system; and *parametric indices*, which may be defined as functions of both parameters and state variables and which provide useful measures of the effects of certain processes on the dynamic behavior of the system. By analysis of the models I then attempt to determine the effect of the parametric indices (e.g., index of disruptive selection) on the behavior of the state indices (e.g., index of reproductive isolation).

In general, the models described below will involve two interacting polymorphic loci in one large potentially panmictic population. One locus will be polymorphic with respect to some trait that is undergoing frequency-dependent selection. Under some circumstances, the heterozygote genotype will be less fit at a polymorphic equilibrium even though the equilibrium is stable. I will call this the *disruptive selection* locus or the DS locus. The second locus will be polymorphic with respect to some character that is associated with the mating process in such a way that assortative mating may occur. This locus will be referred to as the *assortative mating* locus or the AM locus. The DS locus and AM locus may or may not be linked. As with earlier models of the evolution of reproductive isolation, analysis of the models will attempt to define the nature of the conditions which will lead to an epistatic association between the DS locus and the AM locus and hence the establishment of partial premating reproductive isolating mechanisms (RIMs) between populations of opposite homozygotes at the DS locus. The multiple-niche models of Maynard Smith (1966) and Dickinson and Antonovics (1973) may be viewed as special cases of this model. However, the populations modeled in this paper are potentially panmictic; any restrictions on gene flow result entirely from the intrinsic barriers established by interaction between the DS locus and the AM locus.

I. FREQUENCY-DEPENDENT SELECTION

The first prerequisite for the evolution of reproductive isolation via disruptive selection is the establishment and maintenance of a stable disruptive selection regime (e.g., heterozygote inferiority). It is well known that such polymorphic regimes are unstable when fitness values are considered constants. However, when fitness values depend on gene frequency (e.g., when rare genotypes have a fitness advantage), a stable polymorphic equilibrium is possible (Lewontin 1958). In this section the conditions for the local stability of a polymorphism at a single locus in a randomly mating population will be determined for the frequency-dependent case. The results can also be obtained using the results of Li's (1955) general analysis of the stability of polymorphisms and are similar to results derived previously by Lewontin (1958).

The models of the evolution of reproductive isolation presented in section III are based on the general model of frequency-dependent selection described

below, and are somewhat independent of the actual mechanism causing fitness values to depend on gene frequency. A large number of potential mechanisms have been studied or proposed (Ayala and Campbell 1974), including mating behavior (e.g., Petit and Ehrman 1969; Ehrman and Spiess 1969), predator foraging strategies (Clarke 1962, 1969; Udovic et al. 1976), mimicry (Fisher 1958; O'Donald and Pilecki 1970), intergenotypic facilitation or interference (Weisbrot 1966), coevolutionary interactions between populations (Levin and Udovic 1977), and niche heterogeneity (Levene 1953; Deakin 1968; Prout 1968; Ayala and Campbell 1974, p. 121). Models of niche heterogeneity (multiple-niche polymorphisms) formed the basis for earlier models of the evolution of reproductive isolation (Maynard Smith 1966; Dickinson and Antonovics 1973). The qualitative results obtained in this paper will apply to most of the suggested mechanisms for frequency dependence, including niche heterogeneity.

Assume that at a given locus there are two alleles, A_0 and A_1, with frequencies p and $(1 - p)$, respectively. Let W_0, W_1, and W_2 be the fitnesses of the three genotypes, A_0A_0, A_0A_1, and A_1A_1, which occur with frequencies Y_0, Y_1, and Y_2, respectively. Assume that genotypic frequencies are measured after the formation of zygotes, but before selection. Hence, if mating is random they will follow Hardy-Weinberg expectations. The W_i's are assumed to be nonnegative, continuous, and differentiable functions of p. Thus the mean fitness of the population at any point in time will be

$$\bar{W} = \sum_{i=0}^{2} Y_i W_i(p). \tag{1}*$$

As in all models described in this paper, generations are discrete and the dynamics are assumed to be deterministic. Following the notation of Lewontin (1958), I define the following parametric indices: the indices of fitness differences,

$$\begin{aligned} a(p) &= W_0(p) - W_1(p) \\ b(p) &= W_2(p) - W_1(p), \end{aligned} \tag{2}*$$

and the ratio of the fitness differences

$$r(p) = b(p)/a(p). \tag{3}*$$

Note that because of relaxation of the assumption of random mating, the equations derived below will not apply in later sections. However, the notation defined above and any expressions followed by an asterisk will be preserved.

The following expression describes the change in p from one generation to the next (values of variables in the next generations are denoted by primes):

$$\begin{aligned} p' &= \frac{p(1-p)}{\bar{W}}[p(a+b) - b] + p \\ &= \frac{p(1-p)}{\bar{W}}a[p(1+r) - r] + p. \end{aligned} \tag{4}$$

This system will be at equilibrium whenever $\Delta p = p' - p = 0$; that is, whenever $p = 0$, $p = 1$ or

$$\hat{p} = \frac{b(\hat{p})}{a(\hat{p}) + b(\hat{p})} = \frac{r(\hat{p})}{1 + r(\hat{p})}. \tag{5}$$

For any polymorphic equilibrium, $0 < \hat{p} < 1$, expression (5) indicates that $\hat{a}$ and $\hat{b}$ must be the same sign and therefore that the heterozygote cannot be intermediate in fitness at the equilibrium. This is a well-known condition which requires no further explanation.

The local stability characteristics of a polymorphic equilibrium (whether it returns to equilibrium after small perturbations) depend on the rate of change of Δp as a function of gene frequency ($d\Delta p/dp$), which is a quantitative measure of the direction and intensity of feedback loops. For local stability the following conditions must be satisfied (Goldberg 1958):

$$-2 < \frac{d\Delta p}{dp}\Big|_{p=\hat{p}} < 0. \tag{6}$$

The left condition occurs solely because changes in gene frequency occur in discrete intervals rather than continuously, therefore allowing for the possibility of overshoot. It is theoretically possible to have such strong negative feedback loops caused by frequency-dependent selection that this condition is not satisfied. However, it can be shown that the left condition will almost always be satisfied when the parameters of the fitness functions are assigned values which are biologically reasonable. Hence consideration will be restricted to the condition on the right. This condition, which must be satisfied no matter what the time scale of response of the system, is the necessary and sufficient condition for intrinsic stability (Levin and Udovic 1977). By substitution and simplification, we can obtain

$$\frac{d\Delta p}{dp}\Big|_{p=\hat{p}} = \frac{\hat{p}}{\hat{W}}\hat{a} + (1-\hat{p})\left[\hat{p}\frac{\partial a}{\partial p}(\hat{p}) - (1-\hat{p})\frac{\partial b}{\partial p}(\hat{p})\right]. \tag{7}$$

Further algebraic simplification yields the following condition for intrinsic stability,

$$S + M < 0, \tag{8}$$

where S and M are parametric indices of the feedback effects of heterozygote superiority or inferiority and frequency dependent selection, respectively, at a polymorphic equilibrium and are defined as follows

$$S = a(\hat{p}) + b(\hat{p}) = a(\hat{p})\,[1 + r(\hat{p})] \tag{9}*$$

$$M = \hat{p}\frac{\partial a}{\partial p}(\hat{p}) - (1-\hat{p})\frac{\partial b}{\partial p}(\hat{p}). \tag{10}*$$

Condition (8) depends only on the relationship between fitness values and not on the values themselves. However, in order to standardize values for S and M, the fitness values at equilibrium will always be defined relative to $W_{\hat{a}}(\hat{p}) = 1$.

Theoretically, values for S and M could be obtained for any value of p, but interpretation of the meaning of the indices would change for $p \neq \hat{p}$. Furthermore, it is the polymorphic equilibrium values that are of interest. At equilibrium, S is positive when the heterozygote is inferior, negative when the heterozygote is superior, zero when all genotypes are of equal fitness, and reaches a maximum of 2 when heterozygote fitness is zero. The M will be negative when frequency-dependent selection is stabilizing (the rare genotypes have a fitness advantage), positive when frequency-dependent selection is destabilizing (the common genotypes have a fitness advantage), and zero when fitness values are independent of gene frequency. Here S will be referred to as the index of disruptive selection, and M will be referred to as the index of frequency-dependent selection.

Condition (8) shows the potential complementarity between the two feedback processes defined by S and M. Stabilizing frequency-dependent selection can override the destabilizing effect of heterozygote inferiority (disruptive selection) and heterosis can override the effect of destabilizing frequency-dependent selection. Furthermore, the feedback effects are additive. In the case of heterozygote inferiority, $S > 0$, and the feedback effects of frequency dependence must be strong enough that $-M > S$.

II. ASSORTATIVE MATING MODELS

The second prerequisite for the evolution of reproductive isolation via disruptive selection is the development of a genetic basis for increasing the probability of intragenotypic crosses among the extreme types and reducing the probability of crosses which would result in less-fit intermediates. Maynard Smith (1966) and Dickinson and Antonovics (1973) discuss several possible genetic mechanisms for this process. In this paper only one of these is considered: the potential epistatic association of the DS locus discussed in the previous section with a second locus which provides a genetic basis for mating assortatively by phenotype.

The two simple models of assortative mating which will be used in this paper were chosen from a large number of possible alternatives (Scudo and Karlin 1969; Karlin and Scudo 1969) because they have two characteristics which help simplify analysis. First, assortative mating does not result in changes in gene frequency from generation to generation. Thus, in the absence of stochastic effects or differences in the average fitness values of genotypes as a result of differences in genetic background, gene frequency remains invariant. As Scudo and Karlin (1969) point out, this is not characteristic of most assortative mating models. Most often the mating process itself generates fitness variability because of genotypic differences in the probability of finding a mate. However, selection at the AM locus compounds difficulties in analysis and interpretation, and for the present it is ignored. Since selection resulting from assortative mating tends to operate against the formation of stable polymorphisms at the AM locus (Scudo and Karlin 1969), it will be important to consider the sensitivity of the results of this paper to the introduction of this factor into the models. Second, the extent of assortative mating at the AM locus is defined by a single invariant parameter, α, which indicates the probability that an individual will mate assortatively ($1 - \alpha$ will then

TABLE 1

FREQUENCIES OF INTRA- AND INTERGENOTYPIC CROSSES (ξ_{ij}) AND RECURSIVE EQUATIONS DESCRIBING CHANGES IN GENOTYPIC FREQUENCIES FOR ASSORTATIVE MATING MODEL 1

	FEMALES (j)		
MALES (i)	B_0B_0 (0)	B_0B_1 (1)	B_1B_1 (2)
B_0B_0 (0)	$\alpha \frac{X_0^2}{X_0 + X_1} + (1 - \alpha)X_0^2$	$\alpha \frac{X_0X_1}{X_0 + X_1} + (1 - \alpha)X_0X_1$	$(1 - \alpha)X_0X_2$
B_0B_1 (1)	$\alpha \frac{X_0X_1}{X_0 + X_1} + (1 - \alpha)X_0X_1$	$\alpha \frac{X_1}{X_0 + X_1} + (1 - \alpha)X_1^2$	$(1 - \alpha)X_1X_2$
B_1B_1 (2)	$(1 - \alpha)X_0X_2$	$(1 - \alpha)X_1X_2$	$X_2 + (1 - \alpha)X_2^2$

$$X_0' = \frac{\alpha}{X_1 + X_1} + (1 - \alpha)q^2 \quad \text{(11a)}$$

$$X_1' = \frac{\alpha X_1}{X_0 + X_1} q + 2(1 - \alpha)q(1 - q) \quad \text{(11b)}$$

$$X_2' = \alpha\left(X_2 + \frac{X_1^2}{4(X_0 + X_1)}\right) + (1 - \alpha)(1 - q)^2 \quad \text{(11c)}$$

be the probability that an individual will choose a mate "randomly"). In more realistic models, α might have a different value for each genotype and each of those values might be frequency-dependent.

Assume that there are two alleles at an assortative mating locus (B_0 and B_1), with gene frequencies q and $(1 - q)$ respectively. Let X_0, X_1 and X_2 be the frequencies of the B_0B_0, B_0B_1, and B_1B_1 genotypes, respectively. We make the following simplifying assumptions.

(1) The AM locus is autosomal.
(2) Given identical genetic backgrounds, each AM genotype has the same viability and fertility.
(3) Individuals which mate assortatively always mate with an individual of the same AM phenotype. In model 1, B_0 is dominant and hence the dominant homozygote and the heterozygote have the same phenotype. In model 2, the heterozygote is distinguishable from both homozygotes and hence there is a one to one correspondence between genotype and phenotype.
(4) There is no loss in fertility associated with either mating assortatively or randomly.

Model 1 is a special case of model 1 of Scudo and Karlin (1969) and is the model used by Maynard Smith (1966) and Dickinson and Antonovics (1973). Model 2 has the advantage that it is "symmetrical", which simplifies analysis.

The frequencies of various intra- and intergenotypic crosses for models 1 and 2 are presented in tables 1 and 2, together with recursive equations describing changes in genotypic frequencies from one generation to the next. These tables are based on the assumption that no selection is occurring at the AM locus. If selection occurs before mating begins, the genotypic frequencies after selection

TABLE 2

FREQUENCIES OF INTRA- AND INTERGENOTYPIC CROSSES (ξ_{ij}) AND RECURSIVE EQUATIONS DESCRIBING CHANGES IN GENOTYPIC FREQUENCIES FOR ASSORTATIVE MATING MODEL 2

	FEMALES (j)		
MALES (i)	B_0B_0 (0)	B_0B_1 (1)	B_1B_1 (2)
B_0B_0 (0)	$\alpha X_0 + (1-\alpha)X_0^2$	$(1-\alpha)X_0X_1$	$(1-\alpha)X_0X_2$
B_0B_1 (1)	$(1-\alpha)X_0X_1$	$\alpha X_1 + (1-\alpha)X_1^2$	$(1-\alpha)X_1X_2$
B_1B_1 (2)	$(1-\alpha)X_0X_2$	$(1-\alpha)X_1X_2$	$\alpha X_2 + (1-q)X_2^2$

$$X_0' = \alpha(X_0 + \tfrac{1}{4}X_1) + (1-\alpha)q^2 \quad (12a)$$
$$X_1' = \tfrac{1}{2}\alpha X_1 + 2(1-\alpha)q(1-q) \quad (12b)$$
$$X_2' = \alpha(X_2 + \tfrac{1}{4}X_1) + (1-\alpha)(1-q)^2 \quad (12c)$$

could be substituted into the tables in the appropriate locations. Then the recursive equations would simply define the genotypic frequencies before selection in the next generation as a function of genotypic frequencies after selection in the current generation.

The dynamics of assortative mating systems are often expressed in terms of the heterozygote deficiency (F), a state index which measures the deviation in heterozygote frequency from the expected Hardy-Weinberg equilibrium.

$$F = 1 - \frac{X_1}{2q(1-q)}. \quad (13)^*$$

The following recursive equations for F have been derived assuming no selection pressures:

model 1
$$F' = \alpha \frac{1 - q(1-F)}{1 + (1-q)(1-F)} \quad (14)$$

model 2
$$F' = \frac{\alpha}{2}(1+F). \quad (15)$$

The equilibrium values for heterozygote deficiency can be expressed as a function of q and α. For model 1, $\hat{F}$ is the unique root between 0 and 1 of

$$\hat{F}^2 - \left(\frac{1-\alpha q}{1-q} + 1\right)\hat{F} + \alpha = 0. \quad (16)$$

For model 2:

$$\hat{F} = \frac{\alpha}{2-\alpha}. \quad (17)$$

These equations are presented graphically in figure 1. Note that in model 2, $\hat{F}$ is independent of gene frequency.

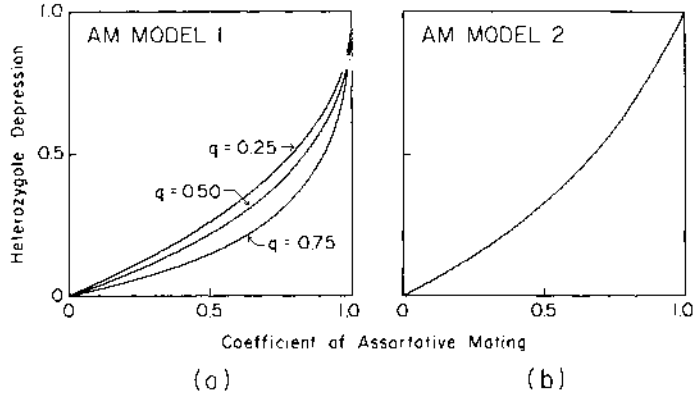

FIG. 1.—Graphical representation of the heterozygote depression at the AM locus at equilibrium, $\hat{F}$, as a function of the coefficient of assortative mating, α. *a*, AM model 1. The three curves represent different values of the gene frequency, q. *b*, AM model 2. For this model, $\hat{F}$ is independent of gene frequency.

The following three points deserve emphasis. First, for any value of α between 0 and 1, heterozygote deficiency will result. There is no threshold which must be reached before $\hat{F}$ becomes positive. Second, $\hat{F}$ is a monotonically increasing function of α. Third, except for boundary values of $\alpha(\alpha = 0$ or $\alpha = 1)$, $\hat{F}$ is always less than α.

III. MODELS OF REPRODUCTIVE ISOLATION

In this section the models of the two previous sections are combined to describe the dynamics of a population in which disruptive selection is operating at one locus (DS locus) and assortative mating at a second locus (AM locus). Each generation in the models is divided into two phases: a selection phase in which differential mortality occurs based on each individual's allelic configuration at the DS locus without regard to its AM genotype; and a mating phase, in which partners are chosen according to their AM genotype without regard to their DS genotype.

As in previous models the dynamics of the system may be described by a system of recursive equations which express genotypic frequencies in one generation as a function of genotypic frequencies in the previous generation. In the general case, there are 10 distinct genotypes (table 3), including two double heterozygote genotypes: one with gametes in the coupling phase (A_0B_0/A_1B_1); the other with gametes in the repulsion phase (A_0B_1/A_1B_0). The frequency of genotype ij (the genotype with i A_1 alleles at the DS locus and j B_1 alleles at the AM locus) is denoted by g_{ij}. The frequencies of the coupling and repulsion double heterozygotes are denoted by $g_{\text{II},C}$ and $g_{\text{II},R}$, respectively. Although the number of variables in multiple-locus population genetics models which assume random mating can be reduced significantly by considering gametic frequencies rather

TABLE 3

GENOTYPIC FREQUENCIES (g_{ij}) FOR THE TEN POSSIBLE GENOTYPES IN THE MODELS OF REPRODUCTIVE ISOLATION

	AM LOCUS			
DS LOCUS	B_0B_0	B_0B_1	B_1B_2	Σ
A_0A_0	g_{00}	g_{01}	g_{02}	Y_0
A_0A_1	g_{10}	g_{11}*	g_{12}	Y_1
A_1A_1	g_{20}	g_{21}	g_{22}	Y_2
Σ	X_0	X_1	X_2	1

NOTE.—Note that, in general, there are two distinct double-heterozygote genotypes, corresponding to the coupling and repulsion phases.

* $g_{11} = g_{11,C} + g_{11,R}$.

than genotypic frequencies (Lewontin 1964), genotypic frequencies in the models considered here cannot be ascertained solely from a knowledge of gametic frequencies. In general, nine independent recursive equations are required to describe the dynamics of these two-locus models.

The basic assumptions of previous models will also hold for this section (e.g., generations are discrete and processes are deterministic). In addition, the following assumptions are made concerning the interaction of the DS locus and AM locus.

1) Both loci are autosomal. They may or may not be linked.
2) Fitness values at the DS locus are functions of allelic frequencies at that locus, and are independent of allelic or genotypic frequencies at the AM locus.
3) The selection phase and the mating phase occur sequentially (i.e., zygote formation → selection phase → mating phase → zygote formation).
4) Fitness variability at the DS locus is due solely to differential viability. Each genotype is assumed to have the same average fertility. This assumption is a direct consequence of the previous one, since differential fertility rates would imply that the selection and mating phase would be in reverse order, if not simultaneous.

Since the fitness of each genotype depends only on its DS alleles, the following set of equations expresses genotypic frequencies after the selection phase as a function of preselection genotypic frequencies:

$$g_{ij}^{S} = g_{ij} W_i / \overline{W}, \tag{18}*$$

where the mean fitness of the population is

$$\overline{W} = \sum_{j=0}^{2} \sum_{i=0}^{2} g_{ij} W_i. \tag{19}*$$

The W_i's are functions of p, the frequency of the A_0 allele at the DS locus, but can be expressed as functions of genotypic frequencies by determining the relationship between the genotypic frequencies and p.

Let

$$\phi_i = g_{0i} + \tfrac{1}{2}g_{1i}. \tag{20}*$$

Then

$$p = \sum_{i=0}^{2} \phi_i = \sum_{i=0}^{2} (g_{0i} + \tfrac{1}{2}g_{1i}). \tag{21}*$$

The next objective is to obtain a set of equations for the mating phase which express the genotypic frequencies after mating and zygote formation in terms of their postselection, premating values. As shown in tables 1 and 2, there are nine possible crosses at the AM locus. Given a particular cross (e.g., $B_0B_0 \times B_0B_1$), since mating choice is not affected by the DS locus, zygote formation can be viewed as the union of gametes chosen randomly from two gamete pools corresponding to the two parental genotypes at the AM locus. Let $(\gamma_{ij})_\mu$ represent the frequency of gamete A_iB_j among individuals of genotype μ at the AM locus ($\mu = 0$ for B_0B_0; $\mu = 1$ for B_0B_1; $\mu = 2$ for B_1B_1). Formulas for $(\gamma_{ij}^S)_\mu$ are easily derived and are presented in table 4. Note that for genotype B_0B_1 the gametic frequencies depend on the frequency of recombination between the AM locus and the DS locus, which is called R ($0 \leq R \leq \frac{1}{2}$), the recombination fraction. Let $\tau_{\mu\nu}(\gamma_{kl}^S, \gamma_{mn}^S)$ represent the frequency of the union of gametes A_kB_l and A_mB_n among matings between males of genotype μ at the AM locus and females of genotype ν at the AM locus. Then if gametes are selected at random,

$$\tau_{\mu\nu}(\gamma_{kl}^S, \gamma_{mn}^S) = (\gamma_{kl}^S)_\mu (\gamma_{mn}^S)_\nu + (\gamma_{mn}^S)_\mu (\gamma_{kl}^S)_\nu \tag{22}*$$
$$\text{for } k, l, m, n = 0, 1;\ \mu, \nu = 0, 1, 2.$$

From the values for $\tau_{\mu\nu}$, the frequencies of each of the 10 genotypes among offspring of each of the nine crosses can be easily determined. Let $T_{\mu\nu}(i,j)$ represent the proportion of offspring from matings between males of genotype μ at the AM locus and females of genotype ν at the AM locus which are of genotype ij. Then

$$T_{\mu\nu}(i,j) = \sum_{k,l,m,n=0}^{1} \tau_{\mu\nu}(\gamma_{kl}^S, \gamma_{mn}^S) \qquad \text{for } i,j = 0,1,2 \tag{23a}*$$
$$\mu, \nu = 0,1,2$$

$$T_{\mu\nu}^R(1,1) = \sum_{k,l,m,n=0}^{1} \tau_{\mu\nu}(\gamma_{kl}^S, \gamma_{mn}^S) \quad \text{for } \mu, \nu = 0,1,2 \tag{23b}*$$
$$m = l \neq k = n$$

$$T_{\mu\nu}^C(1,1) = \sum_{k,l,m,n=0}^{1} \tau_{\mu\nu}(\gamma_{kl}^S, \gamma_{mn}^S) \qquad \text{for } \mu, \nu = 0,1,2 \tag{23c}*$$
$$k = l \neq m = n$$

where $T_{\mu\nu}^R(1,1)$ and $T_{\mu\nu}^C(1,1)$ represents the frequencies of the repulsion and coupling-phase double heterozygotes, respectively, among offspring of a $\mu \times \nu$

TABLE 4

GAMETIC FREQUENCIES AFTER SELECTION $(\gamma_{ij})_\mu$ FOR EACH GENOTYPE AT THE AM-LOCUS

AM GENOTYPE	GAMETE FREQUENCIES			
	$\gamma^S_{00} = (A_0B_0)$	$\gamma^S_{01} = (A_0B_1)$	$\gamma^S_{10} = (A_1B_0)$	$\gamma^S_{11} = (A_1B_1)$
B_0B_0 ($\mu = 0$)	ϕ^S_0/X^S_0	0	$1 - \frac{\phi^S_0}{X^S_0}$	0
B_0B_1 ($\mu = 1$)*	λ_C/X_1	λ_R/X_1	$\frac{1}{2} - \frac{\lambda_R}{X_1}$	$\frac{1}{2} - \frac{\lambda_C}{X_1}$
B_1B_1 ($\mu = 2$)	0	ϕ^S_2/X^S_2	0	$1 - \frac{\phi^S_2}{X^S_2}$

* where

$$\lambda_C = \tfrac{1}{2}(g^S_{01} + \rho g^S_{11})$$

$$\lambda_R = \tfrac{1}{2}(g^S_{01} + (1 - \rho)g^S_{11})$$

$$\rho = \frac{(1 - R)g^S_{11,C} + Rg^S_{11,R}}{g^S_{11,C} + g^S_{11,R}},$$

and R ($0 \leq R \leq 0.5$) is the fraction of recombinant gametes.

cross. Finally, the frequency of each genotype in the next generation (g'_{ij}) can be obtained by multiplying the proportion of offspring of each cross which result in that genotype [$T_{\mu\nu}(i,j)$] by the frequency of that cross ($\xi_{\mu\nu}$) and summing over all nine crosses:

$$g'_{ij} = \sum_{\mu=0}^{2}\sum_{\nu=0}^{2} \xi_{\mu\nu}T_{\mu\nu}(i,j) \qquad \text{for } i,j = 0,1,2 \tag{24a}*$$

$$g'_{11,R} = \sum_{\mu=0}^{2}\sum_{\nu=0}^{2} \xi_{\mu\nu}T^R_{\mu\nu}(1,1) \tag{24b}*$$

$$g'_{11,C} = \sum_{\mu=0}^{2}\sum_{\nu=0}^{2} \xi_{\mu\nu}T^C_{\mu\nu}(1,1). \tag{24c}*$$

Note that only the frequencies of each cross ($\xi_{\mu\nu}$) depend on the particular assortative mating scheme. For AM models 1 and 2, formulas for ξ have already been derived (tables 1 and 2). However, the AM genotypic frequencies (X_i) in the tables must be replaced by the AM genotypic frequencies after selection (X^S_i) for use in expression (24).

IV. ANALYSIS

The goal of analysis of these models is to determine when epistatic interactions between the AM locus and the DS locus will result in a stable association between the two, and hence in partial reproductive isolation. There are several possible state indices which could be used to measure the degree of association between the AM locus and the DS locus or the resulting degree of isolation. The gametic-phase disequilibrium coefficient (D) measures the degree to which alleles at the

two loci covary in their distribution among gametes,

$$D = \gamma_{00}\gamma_{11} - \gamma_{01}\gamma_{10}. \quad (25)^*$$

Values of D vary from -0.25 to $+0.25$, with a value of zero indicating no association. The degree of reproductive isolation at the DS locus can be measured either by the degree of heterozygote deficiency at the DS locus or by measuring the degree of nonrandomness in matings or inseminations among the DS genotypes. Measures based on the latter are commonly used in laboratory experiments on reproductive isolation. For example, Stalker's (1942) *isolation index*, developed by Donald Charles, has been used by a number of investigators (e.g., Dobzhansky and Mayr 1944; Hurd and Eisenberg 1975):

$$\frac{\text{No. of homogamic matings} - \text{No. of heterogamic matings}}{\text{Total no. of matings}}.$$

Levene's (1949) *coefficient of isolation*, a refinement of Stalker's index, is also used in laboratory studies (e.g., Khasimuddin and DeBach 1976).

Here, the degree of heterozygote deficiency at the DS locus (I) will be used as a state index of reproductive isolation:

$$I = 1 - \frac{Y_1}{2p(1-p)}. \quad (26)^*$$

Values of I vary from -1 to 1. If the AM locus and DS locus are not associated, mating will be random at the DS locus and, if fertility rates are equal, I will be zero. An excess of heterozygotes yields a negative value for I, while a deficit yields a positive value. Reproductive isolation is complete if $I = 1$.

Indices such as Stalker's or Levene's which are based on the frequencies of various matings are direct measures of the degree of premating isolation. Heterozygote deficiency, on the other hand, may result from differential fertility of the various genotypic crosses as well as from nonrandom mating, and hence may include postmating as well as premating isolation. However for the models analyzed in this paper, heterozygote deficiency measures only the degree of premating isolation because fertility rates are assumed to be equal for all crosses. Furthermore, the specific measures introduced by Stalker and Levene are designed for controlled laboratory experiments and lack the generality necessary to be useful in the context of these models. More general indices based on mating frequencies have yet to be developed.

The basic analytical problems are: (1) to determine what conditions on the parameters and parametric indices (α, S, M, and R) must be satisfied for a polymorphic equilibrium with $\hat{I} > 0$, $\hat{D} \neq 0$ to be stable, and (2) to express the equilibrium values of the state indices ($\hat{I}$ and $\hat{D}$) as functions of the parameters and parametric indices. For analysis of the models of reproductive isolation described in this paper, a combination of numerical and algebraic approaches has been used. Although a general algebraic solution for $\hat{I}$ or $\hat{D}$ has not been found, solutions have been obtained for special cases where one or two ancillary parameters or parametric indices (R or $\hat{r}$) are restricted to specific values (see Appendix). These results have led to inferences about the relationships between parameters and parametric

indices and the equilibrium values of the state indices for the general case. Finally, the inferences have been tested by obtaining numerical solutions using the computer. Except in very limited circumstances, algebraic approaches to stability analysis (e.g., the Routh-Hurwitz criteria; Arnold 1973) are too unwieldy. Fortunately, straightforward inferences about the stability conditions for these models can be made from numerical solutions.

V. RESULTS

For every polymorphic equilibrium which exists at the DS locus when mating is random ($\hat{I} = 0$), there may exist two other equilibria which are due to epistatic association with the AM locus. Interpretation of the results of algebraic analysis and numerical solutions for both AM models (Appendix, figs. 2, 3, and 4) leads to the following conclusions about the existence, magnitude, and stability characteristics of these alternative equilibria.

1. In general, the dynamic behavior of I is coupled to the dynamic behavior of D. Figure 2 shows the qualitative relationship between $\hat{I}$ and $\hat{D}$ for AM model 2 with $\hat{r} = 1$. In this case, for every nonzero equilibrium value for I, two nonzero equilibria exist for D, one positive and one negative. Depending on the initial conditions, reproductive isolation can arise either through the association of allele A_0 with allele B_0 and allele A_1 with allele B_1 ($\hat{D} > 0$), or through the reciprocal association of A_0 with B_1 and B_0 with A_1 ($\hat{D} < 0$). In general, the values of $\hat{I}$ associated with the positive equilibrium for $D(\hat{I}_+)$ and negative equilibrium for $D(\hat{I}_-)$ are not equal, although $\hat{I}_+ = \hat{I}_-$ for all parameter sets for AM model 2 and for $\hat{r} = 1$ for AM model 1. Furthermore, for any given set of parameters, $\hat{I}_+$ and $\hat{I}_-$ have the same stability characteristics.

2. For any nonzero values of the recombination fraction, R, and the assortative mating coefficient, α, there are two threshold values of the index of disruptive selection, $0 < S_b < S_c < 2$. If $S \leq S_b$, there are no nonzero equilibria for I associated with the polymorphic equilibrium in question (fig. 2, region A). If $S_b < S < S_c$, nonzero equilibrium values for I exist which are negative (fig. 2, region B). The S_b is called a bifurcation threshold or bifurcation point (Keller and Antman 1969; Stakgold 1971) because as the parametric index, S, increases in value past S_b each solution of the models of repoductive isolation defined by recursive equations (24) splits into three solutions. Positive equilibrium values for I will exist if and only if $S > S_c$ (fig. 2, regions C and D). Hence, $S > S_c$ is a necessary (but not sufficient) condition for the evolution of reproductive isolation.

3. Both S_b and S_c are functions of the assortative mating coefficient, α, and the recombination fraction, R, but are independent of the index of frequency dependence, M. As illustrated in figures 3 and 4, the smaller the AM coefficient and the more recombination between the AM locus and the DS locus, the greater the value of the S required both for bifurcation of the solutions and for partial reproductive isolation. This result has been derived analytically for AM model 2 where $\hat{r} = 1$ ($\hat{p} = 0.5$), and numerically for other cases.

4. As illustrated in figures 2, 3, and 4, the values for the nonzero equilibria of the index of reproductive isolation, I, are: (1) increasing functions of the index of

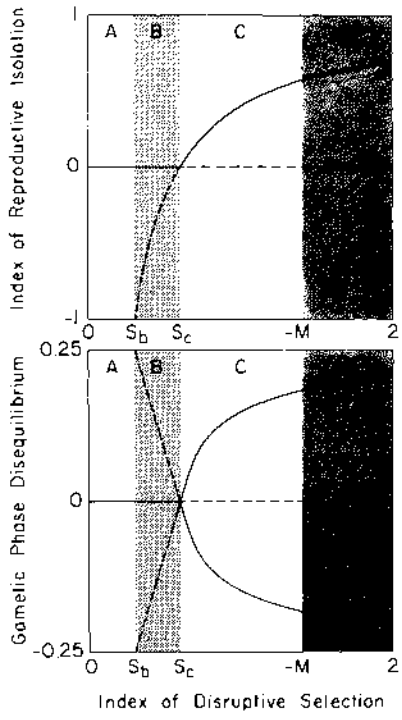

FIG. 2.—Stability characteristics of polymorphic equilibria as a function of the index of disruptive selection, S. Curves are drawn for AM model 2, using representative values of the assortative mating coefficient ($\alpha = 0.7$) and the recombination fraction ($R = 0.5$). Solid lines represent stable equilibria, dashed lines represent unstable equilibria, and dotted lines represent equilibria whose stability characteristics are undetermined. Sufficient conditions for the evolution of partial premating reproductive isolation will be satisfied if S falls within region C. Note that if $-M < S_c$, region C will not exist.

disruptive selection, S, and the assortative mating coefficient, α; (2) decreasing functions of the recombination fraction, R; and (3) independent of the index of frequency dependence, M. For any value of R and α, the maximum value for $\hat{I}$ ($\hat{I} = \alpha$) is attained when $S = 2$ (heterozygote viability = 0).

5. For any polymorphic equilibrium which exists at the DS locus when mating is random and $S > 0$, condition (8) (the stability condition derived for the models of frequency-dependent selection in section I) is a sufficient, but not necessary, condition to insure the local asymptotic stability of either the above equilibrium (with $\hat{I} = 0$) or of the alternative epistatic equilibria ($\hat{I}_+$ and $\hat{I}_-$). Assuming that condition (8) is satisfied, if $S < S_c$, the epistatic equilibria will be unstable, and the DS locus and AM locus will not become associated (fig. 2, region B). If $S > S_c$, the epistatic equilibria will be stable, the random mating equilibrium will be unstable, and partial reproductive isolation will result (fig. 2, region C). Hence the following

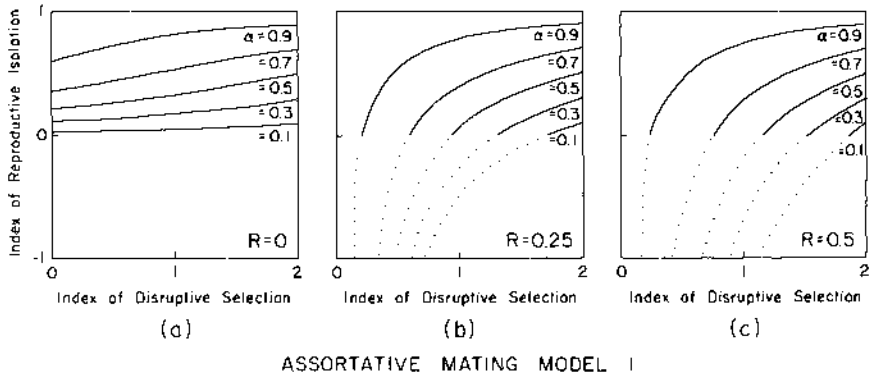

FIG. 3.—The index of reproductive isolation at equilibrium, $\hat{I}$, as a function of the index of disruptive selection, S, the coefficient of assortative mating, α, and the recombination fraction, R, for AM model 1. Each curve shows the relationship between $\hat{I}$ and S for fixed values of α and R. The effects of α on $\hat{I}$ can be assessed by comparing curves within each box. The effect of R on $\hat{I}$ can be assessed by comparing the families of curves in each of three boxes. Solid lines represent stable equilibria. Dotted lines represent unstable equilibria whose existence is inferred from comparison with AM model 2 (see fig. 4).

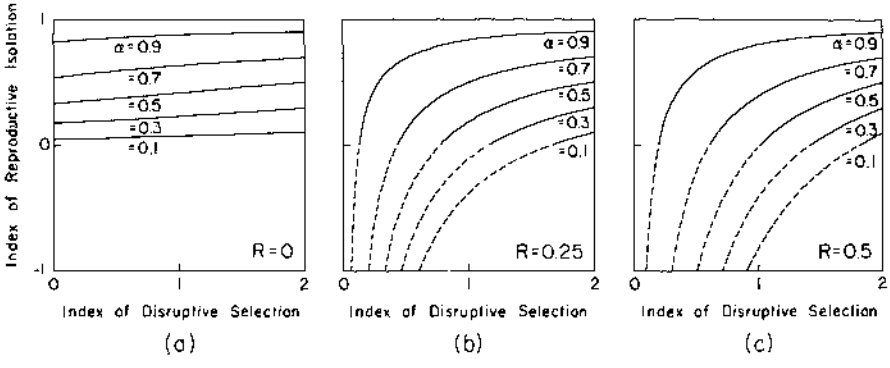

FIG. 4.—The index of reproductive isolation at equilibrium, $\hat{I}$, as a function of the index of disruptive selection, S, the coefficient of assortative mating, α, and the recombination fraction, R, for AM model 2. Each curve shows the relationship between $\hat{I}$ and S for fixed values of α and R. The effects of α on $\hat{I}$ can be assessed by comparing curves within each box. The effect of R on $\hat{I}$ can be assessed by comparing the families of curves in each of three boxes. Solid lines represent stable equilibria and dashed lines represent unstable equilibria.

is a sufficient condition for the evolution of partial reproductive isolation via disruptive selection,

$$0 \leqslant S_c < S < -M. \tag{27}$$

This result has not been derived analytically, but it is a straightforward inference from a large number of numerical solutions.

6. Given a stable disruptive selection regime, the mean population fitness ($\overline{W}$) is an increasing function of I, and is maximized when $I = 1$. Hence for these models of reproductive isolation $\overline{W}$ is in general not maximized at stable equilibria. However, stable polymorphic equilibria always have higher values for $\overline{W}$ than associated unstable equilibria. There are two constraints preventing fitness maximization. First, unless $\alpha = 1$, even complete association of the DS locus with the AM locus cannot lead to complete reproductive isolation because a proportion $(1 - \alpha)$ of the individuals at the AM locus are mating randomly. Second, inertia due to recombination (Fisher 1958; Lewontin 1971) acts as a force opposing the association of the AM locus and the DS locus. If $S < S_c$, small changes in α and R will not result in any change in the stable values for $\hat{I}$ ($\hat{I} = 0$). However, once partial reproductive isolation has evolved ($\hat{S} > S_c$), small increases in α or decreases in R will cause an increase in $\hat{I}$ and $\overline{W}$. Hence genetically based mechanisms for increasing α and restricting recombination should be selected for, leading ultimately to complete reproductive isolation. For example, modifier genes which tend to cause a higher proportion of individuals to mate assortatively on the basis of their AM phenotype should be favored by natural selection. Likewise chromosomal rearrangements which result in tighter linkage or which mechanically restrict recombination between the AM locus and the DS locus should be favored by selection. To the extent that the parameters α and R are under genetic control, they should evolve in the direction of increased reproductive isolation.

VI. DISCUSSION

Although models of the evolution of premating reproductive isolating mechanisms (RIMs) via disruptive selection are usually viewed as models of sympatric speciation, their application is actually more general (Maynard Smith 1966). Essentially, postmating RIMs (e.g., hybrid inviability) may be viewed as a form of disruptive selection, and hence disruptive selection is an equally crucial element in the origin of premating RIMs in zones of secondary contact after allopatric speciation has occurred. Furthermore, frequency-dependent processes such as differential resource utilization may be required for coexistence of the new species in zones of contact. The difference between allopatric and sympatric modes of speciation lies primarily in the mechanism for origin of postmating RIMs. In the former, postmating RIMs are the fortuitous result of genetic changes occurring during geographic isolation, while in the latter, postmating RIMs (selection against intermediates) are the result of selection pressures arising from a population's ecological milieu (Bush 1975). While the results presented in this paper corroborate the results of earlier theoretical work (Mather 1955; Maynard Smith 1966;

Dickinson and Antonovics 1973; Rosenzweig 1978) that sympatric origin of postmating RIMs is feasible, the models are also applicable to the formation of premating RIMs after allopatric formation of postmating RIMs (the Wallace effect; Mayr 1963; Grant 1971).

The generality of the results presented here is limited by the simplifying assumptions of the models. Hence determining the robustness of the models is of considerable importance. For example, gene flow has not been considered as a parameter in the above models. Work is now in progress, based on extensions of these models, which explores the significance of extrinsic barriers to gene flow. Analyses of extensions of the models can also be performed to determine whether the results can be generalized to other mechanisms of assortative mating or nonrandom mating, including selfing and inbreeding, and to stochastic models. Sensitivity to other critical assumptions, such as the assumptions that disruptive selection and assortative mating are each occurring at single loci, and that dynamics at these loci are independent of the rest of the genome, is more difficult to determine by mathematical models. However, it is at least theoretically feasible to test sensitivity to these assumptions by experiments on laboratory population systems. Duplication of the qualitative results using laboratory populations would strongly suggest that the models are robust.

The results presented here demonstrate that frequency-dependent selection may contribute to the maintenance of disruptive selection regimes, which may in turn result in the evolution of reproductive isolation, even in the total absence of physical barriers to gene flow. Hence, in theory, frequency-dependent processes may play an important role in the evolution of new species. How commonly are fitness values frequency dependent in natural situations? Although more convincing field data would certainly be useful in answering this question, ubiquity of the process would not be a surprising result, given the wide variety of proposed mechanisms (Petit and Ehrman 1969; Ayala and Campbell 1974; Levin and Udovic 1977). More importantly, how strong are the feedback loops generated by frequency-dependent selection? One overall implication of the results is that selection against intermediate types must be relatively intense if reproductive isolation is to be expected. However, the stability conditions imply that such disruptive selection regimes will not be maintained without equally intense stabilizing feedback due to frequency-dependent fitnesses. Finding answers to the above questions is imperative if a thorough understanding of the role of disruptive selection in the evolution of reproductive isolation is to be obtained.

VII. Summary

Mathematical models have been developed and analyzed to determine the conditions required for the evolution of premating reproductive isolation via disruptive selection in a potentially panmictic population. The models describe changes in genotypic frequencies through time for a population which is polymorphic at two loci. Disruptive selection due to reduced viability of the heterozygote at a polymorphic equilibrium is assumed to operate at the first locus. Mating is assumed to be occurring assortatively within the population, based on phenotypic

characters determined by the second locus. Reproductive isolation results from epistatic interaction of these two loci which reduces the number of intergenotypic crosses at the disruptive selection locus. Analysis of the models involves searching for conditions which yield stable polymorphic equilibria with the two loci epistatically associated, and determining the relationship between the degree of association at equilibrium (and hence the degree of reproductive isolation) and the conditions of the models.

Results indicate that the equilibrium degree of reproductive isolation depends on the following factors: (1) the intensity of disruptive selection; (2) the proportion of individuals in the population which mate assortatively (i.e., the penetrance of the genes for assortative mating behavior); (3) the amount of recombination between the disruptive selection locus and the assortative mating locus; and (4) the extent to which fitness values of genotypes at the disruptive selection locus depend on gene frequency. Premating reproductive isolation will not evolve at all unless the following two criteria are satisfied: (1) As a result of recombinational inertia, the intensity of disruptive selection must be greater than a threshold value which is an increasing function of the amount of recombination between the two loci and a decreasing function of the degree of assortative mating, and (2) the destabilizing effect of disruptive selection on polymorphic equilibria must be compensated for by the stabilizing effect of frequency-dependent selection. Assuming these criteria are satisfied, the degree of reproductive isolation at equilibrium is an increasing function of the intensity of disruptive selection, an increasing function of the degree of assortative mating, a decreasing function of the amount of recombination, and independent of the degree of frequency-dependent selection. Once partial premating isolation has been achieved, it may be enhanced by evolution of some of the parameters of the model. For example, natural selection should favor genetically based mechanisms for increasing the penetrance of the assortative mating genes and for decreasing the amount of recombination between the disruptive selection locus and the assortative mating locus.

To the extent that these results can be generalized, they suggest that frequency-dependent processes (resulting from niche heterogeneity or from a number of other potential mechanisms) can play an important role in the evolution of reproductive isolation. Hence obtaining an understanding of the causes and relative significance of frequency-dependent processes in natural systems is an important task for the development of evolutionary theory.

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APPENDIX

The following algebraic expressions have been obtained for nonzero equilibrium values of the index of reproductive isolation when there is no recombination between the AM

locus and the DS locus. For AM model 1 (with dominance):

$$\hat{r}[2S - (1 + \hat{r})]\hat{I}^2 + [S\{\alpha(1 - \hat{r}) - 2\} + (1 + \hat{r})\{(1 + \hat{r}) + (1 - \alpha)\}]\hat{I} + \alpha[S(1 - \hat{r}) - (1 + \hat{r})] = 0 \qquad \text{for } R = 0. \tag{A1}$$

For AM model 2:

$$(2\hat{r}S)\hat{I}^2 + [(2 - \alpha)\{(1 + \hat{r})^2 - S\} - \alpha\hat{r}S]\hat{I} + \alpha[S(1 - \hat{r}) - (1 + \hat{r})^2] = 0 \qquad \text{for } R = 0. \tag{A2}$$

When $R = 0$ the models essentially reduce to one-locus models where disruptive selection and assortative mating occur at the same locus. Hence, $\hat{I} = \hat{F}$, and equations (16) and (17) can be viewed as special cases of (A1) and (A2), respectively, where $S = 0$.

An expression for $\hat{I}$ for AM model 2 has also been obtained for the special case, $\hat{r} = 1$ ($\hat{p} = 0.5$), where the recombination fraction, R, is allowed to vary:

$$(\tfrac{1}{2}S)\hat{I}^2 + [2 - \tfrac{1}{2}S - \alpha + 2R(1 - \tfrac{1}{2}S)]\hat{I} - \left[\alpha - 2R\left(\frac{2 - S}{S}\right)(2 - \tfrac{1}{2}S - 2\alpha)\right] = 0 \tag{A3a}$$

$$0 \leqslant R < \tfrac{1}{2}$$

$$\hat{I} = \frac{S - 2(1 - \alpha)}{S} \qquad R = \tfrac{1}{2}. \tag{A3b}$$

Over the range for which parameters and parametric indices are defined, these expressions yield at most one value for $\hat{I}$ between -1 and 1. For all parameter values, except when $R = 0$, another equilibrium exists at $\hat{I} = 0$. These expressions are depicted graphically in figures 3*a*, 4*a*, 4*b*, and 4*c*, for representative parameter values. The stability characteristics of these equilibria, as elicited by numerical solutions, are shown in figure 2. Solid lines indicate regions of stability while dashed lines indicate regions of instability.

Algebraic expressions equivalent to expressions (A3a) and (A3b) have not been derived for AM model 1 because of complications in the equations introduced by dominance at the AM locus. However, numerical solutions have been obtained for a large number of parameter values for the case, $\hat{r} = 1, \hat{p} = 0.5$. Figures 2*B* and 2*C* show how the equilibrium values of $\hat{I}$ obtained by numerical solution vary as a function of the parameters and parametric indices.

The special case, $\hat{r} = 1, \hat{p} = 0.5$, was chosen because it is the only case in which the parametric indices, S and M, which depend on $\hat{p}$, have been parameterized (i.e., determined a priori solely from a knowledge of the fitness parameters). In the general case, $\hat{p}$ depends on $\hat{I}$,

$$\hat{p} = \frac{\hat{r} - \hat{I}}{(1 + \hat{r})(1 - \hat{I})}, \tag{A4}$$

and hence a priori knowledge of $\hat{p}$ implies a priori knowledge of $\hat{I}$. However, if parameters of the fitness functions are chosen which yield $\hat{p} = 0.5$ as an equilibrium value when mating is random at the DS locus (i.e., $r(0.5) = b(0.5)/a(0.5) = 1$), then the equilibrium value for p will not be altered by changes in I. Because of difficulties in parameterizing S and M, parameter space has not been explored systematically when $\hat{r} \neq 1$. However, numerical solutions for a variety of sets of parameters indicate that the qualitative relationships between $\hat{I}$ and S, M, α and R remain the same for other values of $\hat{r}$.

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